



PhD-FLSHASE-2016-23
The Faculty of Language and Literature,
Humanities, Arts and Education

Faculty of Psychology and Educational
Sciences

DISSERTATION

Defence held on 13/10/2016 in Luxembourg

to obtain the degree of

DOCTEUR DE L'UNIVERSITÉ DU LUXEMBOURG

EN PSYCHOLOGIE

AND

DOCTOR IN DE PSYCHOLOGIE

by

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THE ROLE OF SELF-REGULATORY CAPACITY IN THE
ADAPTATION TO PAIN

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Science is organized knowledge.

Wisdom is organized life.

Immanuel Kant (1724 – 1804)

Acknowledgements

Many great people accompanied me through my PhD and I would like to thank all of them. But I want to start with the one person who left me much too early and whom I could not share this important part of my life with, my Dad. You raised me to be curious and explore the world and you taught me to believe in myself. You are the primary reason that I actually got here. It would make you incredibly proud to see where I stand right now and if there is one thing I regret about getting to the point of finishing my PhD is the fact that you simply cannot be part of it. Thank you for having been the greatest Dad and for having guided me to be who I am.

Then of course I am in deep gratitude to my supervisors Claus Vögele and Geert Crombez. Administration not always made it easy for us, but I would like to thank you for having gone this whole way of a joint doctorate which I benefitted so much of. Thank you Claus that you put so much faith in me and that you trusted me. Thank you for having offered me your precious help and advice whenever I needed it and thank you for always having had my back. Thank you Geert that you believed in me and my ideas. I still remember my first presentation in Ghent, oh God... Thank you that you always found time for me when I visited. I highly appreciate your invaluable support and help and want to thank you that, despite the distance, you've always been "there".

I've also been guided by great people from whom I've learnt enormously and who have gained a special place, not only as mentors but also as friends. Dimitri, I don't even know where to start. Thank you for your scientific guidance and your patience, for your countless hours invested in my PhD, for inspiring discussions, for always being reachable and always offering help, for believing in my work as a researcher and for having supported me in every moment of the last four years! But also thank you for the fun we had and for sharing personal commonalities. Stefan, thank you for having created the opportunity for "this" in the first place. You have been so essential for me in the last years and I want to thank you for your scientific and moral support. Thank you for never doubting me and for always having turned my perspective from the problems to the achievements. And a special thanks for having pulled me out of my deep hole 10 days ago and motivated me to finish my PhD. André, thank you for your support, for your interest and your belief in me. Thank you also to the members of my dissertation defence committee for having agreed to be part of this. Thank you to Dieter Ferring for having accompanied me too.

THANK YOU also to all my colleagues in Luxembourg and in Ghent. Thank you to the Self-Regulation and Health team and the Health Psychology Lab. It was such a great pleasure working with all of you. A special thanks to Yacine, Zoé and Gunthora for having been there to support me even in the hardest times, for never giving up the optimism and for having become great friends. Thank you Nele for the great ASEF-I-time. Thank you Wouter, Annick, Charlotte, Sophie and Lien for the exciting conferences we spent together. Thank you also to Raymonde who has been supporting me throughout these last four years. I would also like to thank all the great participants who have supported my research!

And a very warm THANKS goes of course to my family and friends who have backed me where they could, who have not stopped believing in me, who have helped me to see the bright side of life, who have been patient with me and spent comfort during hard times. Thank you Mum, my siblings Kai and Frauke, thank you my parents-in-law for your interest in my work, for your esteem and for your unconditional support. Thank you Romy, Martine, Patty, Yasmine, Anne and all my other great friends, I couldn't wish for better friends. Thank you to all my wonderful friends in Berlin who have, despite the distance, always been very close and whom I'd never want to miss again. Thank you also to my friends from the "Leidelenger Musek", you have helped me to unwind and find new energy in music. And finally I want to thank the two most important persons in my life. Sebastian, for being the greatest partner I could imagine. Thank you for having come with me to Luxembourg, for having supported my stay in Ghent, for having supported me with much care in the most difficult moments and for always having been on my side. You made me smile in the moments I was about to forget what smiling is and you always made me feel valued as your partner, as a psychologist, as a scientist and as a mum. Loki, today you may not really understand what I say, but I would like to thank you too. I cannot hide that you made me go through very hard times with your very special sleep-pattern for weeks and months. But the truth is that you have opened a door to another life for me and I would never want to go back. You are the best thing that happened to me and I will always be grateful that you unremittingly kept spoiling me with the most amazing moments of my life, no matter how hard I was fighting to reach the end of my PhD.

With the last words of my PhD, I feel completely exhausted and amazingly light and happy to reach the end. Thank you to every other amazing person who has shared my life and this special part of it, who has been indulgent, motivating and caring!

THANK YOU.

Abstract

Pain is an everyday experience and serves an important evolutionary function. It attracts attention and interrupts ongoing activity to assure survival. Chronic pain, however, loses its warning signal function and constitutes a common problem in healthcare. It is associated with important personal and social impairments as well as considerable costs which are mostly due to work absenteeism. The gate control theory has opened a new window and allowed the field of pain research to move from a biomedical to a biopsychosocial perspective which considers medical, social, and psychological factors as potentially influencing the experience of pain. Popular contemporary explanations of chronic pain conditions are based on the idea of dysfunctional attentional processes. One can distinguish between two modes of attentional selection. The bottom-up, involuntary, capture of attention by pain is a critical feature of its alarm function. It interrupts ongoing behaviour and thereby allows an individual to prevent further injury. Top-down factors can modulate this unintentional capture of attention. Clinical observations have argued for chronic pain patients, particularly fibromyalgia (FM) patients, to be hypervigilant, i.e. excessively attend to threat-related information, and thereby report amplified pain experience. Distraction, on the other hand, can be defined as an attentional strategy to cope with pain by directing one's attention away from pain, thereby decreasing pain experience. Moreover, pain, by definition, is a sensory and emotional experience and the disentanglement of both pain and emotion is a challenge for research. To gain a better understanding of the dynamic interplay between emotions and pain, it may be necessary to switch from a categorical to a more flexible and dynamic perspective. Emotions are shaped not only by intensity, but also by time features. Temporal patterns rely on regulation processes and provide unique information that is relevant for psychological well-being. Emotion regulation can be integrated in the more general notion of self-regulation which refers to the capacity to control one's thoughts, emotions, and behaviour in order to pursue a goal. Thereby, self-regulation refers to a higher-order process which controls lower-order processes to guide behaviour, primarily operating by inhibitory control. According to the model of neurovisceral integration, self-regulatory capacity reflects the adaptability of an organism to respond to changing environmental demands and can be operationalized by heart rate variability (HRV). Self-regulatory processes involve attention which is either directed towards environmental or towards internal sources. One of the most important internal targets of regulation certainly refers to an emotion. Numerous studies have revealed a close association between dysregulated affect,

such as affective instability which is defined as large and frequent mood shifts over time, and psychopathology. Besides its importance for mental well-being, emotion regulation was also suggested as a key factor in the adaptation to chronic pain. Evidence is scarce, but still emphasizes a dynamic relationship between self-regulatory demands, self-regulatory capacity, and persistent pain. The purpose of the current project was to investigate the role of self-regulatory capacity in the adaptability to pain by focussing on emotional and attentional processes in pain. In study 1, we investigated the role of affective instability in daily functioning of chronic pain patients. We applied an innovative measure of emotional fluctuations which takes into account the variability and temporal dependency of emotions. Results showed an association between negative affect (NA) instability and daily disability and cognitive complaints. The results further indicated a moderating effect of NA instability between daily pain severity and daily pain outcomes. These findings mean that patients with lower emotion regulation capacity (as indexed by NA instability) have poorer functioning in general, and show a greater decrease in functioning on days when they experience higher than usual levels of pain. Study 2 expanded on these findings by adding a physiological index of emotion regulation capacity, HRV, and a healthy control group. Results indicated that NA instability is associated with HRV and that FM patients showed higher levels of NA instability. It may be reasonable to assume that regulatory demands in patients rather relate to the down-regulation of NA. Findings from studies 1 and 2 suggest that affective instability, particularly NA instability, plays an important role in the adjustment to daily pain and may yield clinical implications such as targeting NA instability in therapeutic interventions to offer patients tools that help them to better adapt to their conditions. Study 3 focussed on self-regulatory capacity in regard to attentional processes involved in symptom perception. The generalized hypervigilance hypothesis states that FM patients are hypervigilant not only to pain-related information, but to all kinds of sensations. Therefore, we assessed the perception of internal bodily signals using the heartbeat tracking task. Results indicated no differences in interoceptive accuracy between patients and healthy controls, but showed lower levels of HRV in patients. We argue for a dynamic view of hypervigilance as it seems context dependent and modality-specific rather than a general characteristic. Study 4 investigated self-regulatory capacity as one possible underlying mechanism of distraction efficacy. Evidence regarding distraction efficacy is inconclusive and research suggests that it depends on specific factors such as inhibitory control. Students performed a distraction task in which they were cued to either localize a visual or a somatosensory (painful or non-painful) stimulus while both stimuli were

present in each trial. Results showed that pain intensity was reduced when attention was directed towards the visual stimulus compared with the somatosensory stimulus. Also, reaction times were longer in the presence of a painful stimulus. We found, however, no evidence for an association between HRV and distraction efficacy and attentional interference. HRV may not be sensitive enough in this homogenous sample. Overall, this project yields several clinical implications. Firstly, it may be an important strategy for clinicians to target the regulation of affect, particularly NA. This may be possible through a regulations skills or mindfulness-based training. Secondly, decreased levels of HRV in patients may be targeted by HRV biofeedback training. Finally, the entity of findings from the current project justifies the suggestion to measure HRV in clinical practice to tailor therapeutic interventions. Some limitations should be acknowledged. It may be useful to include additional measures, such as putative mediating variables (e.g. rumination, fear of pain), external events in regard of emotional experiences and the perception of signals originating from other sensory systems. Furthermore, we assessed self-regulatory capacity using a single measurement of heart rate. It may be preferable to use multiple assessments to minimize occasion-specific variance and ensure the measurement of HRV as a consistent trait. Future research should also investigate other chronic pain conditions to allow for a generalization of findings. To conclude, this project has yielded important insights into the role of regulatory processes in the adjustment to pain. Thereby, it raises directions and challenges for future research. A primary question relates to the conceptualization of self-regulatory capacity. This term is broad and relates to multiple processes. There is a need to further elaborate its exact function and to develop measures allowing for the assessment of regulatory processes at different levels (e.g. behavioural, cognitive). A practical challenge refers to the identification of patients who show regulatory deficits which may only be relevant in a subset of patients. Moreover, it is important to note the possible bias by medication on HRV. Future research may aim at investigating the effect of medication on HRV. Finally, it would be desirable to explore self-regulatory capacity more systematically in experimental settings.

Nederlandstalige samenvatting

Pijn is een alledaagse ervaring en dient een belangrijke evolutionaire functie door het trekken van de aandacht en het onderbreken van lopende activiteiten om de overleving te verzekeren. Chronische pijn verliest echter deze functie als waarschuwingssignaal en is een veelvoorkomend probleem in de gezondheidszorg. Het gaat gepaard met grote persoonlijke en sociale beperkingen en aanzienlijke kosten die meestal het gevolg zijn van ziekteverzuim. De ‘gate control theory’ heeft de deur geopend om het pijnonderzoek van een biomedisch naar een biopsychosociaal perspectief te bewegen, zodat medische, sociale en psychologische factoren nu beschouwd worden als mogelijke invloeden op de pijnbeleving. Populaire hedendaagse verklaringen van chronische pijnandoeningen zijn gebaseerd op het idee van dysfunctionele aandachtsprocessen. De aandacht kan op twee wijzen gericht worden. Het bottom-up trekken van de aandacht door pijn is een essentieel kenmerk van de alarmfunctie. Het onderbreekt het lopende gedrag waardoor verdere schade voorkomen kan worden voor een individu. Top-down factoren kunnen dit onopzettelijk vatten van de aandacht moduleren. Klinische observaties hebben geleid tot de aanname dat chronische pijnpatiënten, in het bijzonder fibromyalgie (FM) patiënten, hypervigilant zijn voor lichamelijke sensaties. Dat wil zeggen dat zij overmatig veel aandacht schenken aan pijn-gerelateerde informatie, waardoor de pijnbeleving en andere pijnuitkomsten versterkt kunnen worden. Afleiding, anderzijds, kan worden gedefinieerd als een strategie om met pijn om te gaan door het richten van de aandacht weg van pijn, waardoor de pijnbeleving afneemt. Bovendien is pijn, per definitie, een sensorische en emotionele ervaring en de ontwarring van pijn en emotie is een uitdaging voor onderzoek. Om een beter inzicht te krijgen in de dynamische wisselwerking tussen emoties en pijn, kan het nodig zijn over te schakelen van een categorisch naar een meer flexibel en dynamisch perspectief. Emoties worden niet alleen gevormd door de intensiteit, maar ook door temporele eigenschappen. Temporele patronen worden bepaald door regulatieprocessen en bieden unieke informatie relevant voor het psychisch welbevinden. Emotieregulatie kan worden geïntegreerd in het meer algemene begrip van zelfregulatie, wat verwijst naar de capaciteit om gedachten, emoties en gedrag te reguleren om een doel na te streven. Zelfregulatie verwijst dus naar een hoger orde proces dat lagere orde processen reguleert om gedrag te sturen, en werkt voornamelijk door inhibitie. Volgens het ‘model van neurovisceral integration’, weerspiegelt zelfregulatievermogen het aanpassingsvermogen van een organisme om te reageren op veranderende omgevingseisen en kan dit worden geoperationaliseerd door hartslagvariabiliteit (HRV).

Zelfregulatieprocessen hebben betrekking op aandacht die wordt gericht op omgevings- of interne bronnen. Emoties zijn ongetwijfeld één van de belangrijkste interne doelwitten van zelfregulatie. Talrijke studies hebben een verband onthuld tussen psychopathologie en onregelde emoties, zoals affectieve instabiliteit, gedefinieerd als grote en frequente stemmingswisselingen. Naast het belang voor het psychisch welbevinden heeft emotieregulatie ook een belangrijke functie in de aanpassing aan chronische pijn. De evidentie is schaars, maar benadrukt desondanks een dynamische relatie tussen zelfregulatie-eisen, zelfregulatievermogen en aanhoudende pijn. Het doel van het huidige project was om de rol van het zelfregulatievermogen in het aanpassen aan pijn te onderzoeken door de nadruk te leggen op emotionele en aandachtsprocessen bij pijn. In studie 1 hebben we de rol van affectieve instabiliteit in het dagelijks functioneren van patiënten met chronische pijn onderzocht. We gebruikten een innovatieve maat voor emotionele fluctuaties die rekening houdt met de variabiliteit en temporele afhankelijkheid van emoties. De resultaten toonden een verband tussen negatief affect (NA) instabiliteit en dagelijkse beperkingen en cognitieve klachten. De resultaten gaven verder een modererende invloed aan van NA instabiliteit op de positieve relatie tussen dagelijkse ernst van de pijn en dagelijkse gevolgen van pijn. Deze bevindingen betekenen dat patiënten met kleinere emotieregulatie capaciteit (gemeten door NA instabiliteit) een slechter algemeen functioneren hebben, en een grotere afname in functioneren vertonen op dagen wanneer zij een hoge mate van pijn ervaren. Studie 2 breidde deze resultaten uit door het toevoegen van een fysiologische index voor emotieregulatie capaciteit, HRV, en een gezonde controlegroep. Resultaten gaven aan dat NA instabiliteit gerelateerd is aan HRV en dat FM patiënten grotere NA instabiliteit vertoonden. Het is redelijk om te veronderstellen dat de regulatie-eisen bij patiënten eerder betrekking hebben op de down-regulatie van NA. Bevindingen uit studies 1 en 2 suggereren dat affectieve instabiliteit, met name NA instabiliteit, een belangrijke rol speelt in de aanpassing aan dagelijkse pijn en hebben mogelijk klinische implicaties, zoals het zich richten op NA instabiliteit in therapeutische interventies, om patiënten instrumenten aan te bieden die hen helpen om zich beter aan te passen aan hun aandoeningen. Studie 3 richtte zich op zelfregulatievermogen ten aanzien van aandachtsprocessen betrokken bij symptoomperceptie. De 'generalized hypervigilance' hypothese houdt in dat FM patiënten niet alleen hypervigilant zijn voor pijn-gerelateerde informatie, maar voor allerlei sensaties. Daarom onderzochten wij de waarneming van interne lichamelijke signalen met behulp van de 'heartbeat tracking' taak. Resultaten toonden geen verschillen in interoceptieve accuraatheid tussen FM patiënten en gezonde proefpersonen, maar toonden lagere HRV

bij patiënten. Wij stellen dat hypervigilantie misschien geen algemeen kenmerk is, maar eerder een dynamisch gevolg van de context of de modaliteit. Studie 4 onderzocht zelfregulatievermogen als een mogelijk onderliggend mechanisme voor de effectiviteit van aandachtsafleiding. Beschikbare evidentie over de effectiviteit van aandachtsafleiding in alle contexten is niet overtuigend en onderzoek suggereert dat het afhangt van specifieke factoren zoals inhibitie. Studenten voerden een afleidingstaak uit waarin zij ofwel een visuele of een somatosensorische (pijnlijke of niet-pijnlijke) stimulus moesten lokaliseren, terwijl beide stimuli in elke trial aanwezig waren. De resultaten toonden aan dat de pijnintensiteit lager was wanneer de aandacht werd gericht op de visuele stimulus dan op de somatosensorische stimulus. Verder werd ook gevonden dat pijn zorgt voor interferentie van de uitgevoerde taak. De reactietijden waren langer in de aanwezigheid van een pijnlijke stimulus. We vonden echter geen bewijs voor een associatie tussen HRV en de effectiviteit van aandachtsafleiding en aandachtsinterferentie. HRV is wellicht niet sensitief genoeg in deze homogene (i.e., studenten) steekproef. Alles samen wijzen de resultaten van deze studies op een aantal klinische implicaties. Ten eerste zou het zich richten op de regulatie van emoties, in het bijzonder NA, een belangrijke strategie kunnen zijn voor klinici bij de behandeling van chronische pijnpatiënten. Dit zou mogelijk kunnen zijn door training van regulatievaardigheden of op mindfulness gebaseerde trainingen. Ten tweede, verlaagde HRV bij patiënten zou aangepakt kunnen worden door HRV biofeedback training. Tot slot rechtvaardigt het geheel van bevindingen van het huidige project de suggestie om HRV in de klinische praktijk te meten om therapeutische interventies op maat te ontwikkelen. Enkele beperkingen dienen te worden erkend. Het zou nuttig kunnen zijn aanvullende maten toe te voegen, zoals vermeende mediërende variabelen (b.v. ruminatie, angst voor pijn), externe gebeurtenissen ten aanzien van emotionele ervaringen en de waarneming van signalen afkomstig van andere sensorische systemen. Verder hebben we zelfregulatievermogen onderzocht aan de hand van één enkele meting van de hartslag. Het is wellicht beter om meerdere metingen te doen om gelegenheid-specifieke variatie te minimaliseren en om de meting van HRV als een consistente persoonlijkheidstrek te verzekeren. Toekomstig onderzoek moet ook andere chronische pijn aandoeningen bestuderen om de bevindingen te kunnen generaliseren. In conclusie heeft dit project belangrijke inzichten opgeleverd over de rol van zelfregulatieprocessen in de aanpassing aan pijn. Hierbij brengt het richtingen en uitdagingen voor toekomstig onderzoek naar voren. Een belangrijke vraag betreft de conceptualisering van zelfregulatievermogen. Deze term is breed en betreft meerdere processen. Er is behoefte aan de verdere uitwerking van de exacte functie en aan maten voor de beoordeling van de regulatieprocessen op

verschillende niveaus (bijvoorbeeld gedrag, cognitief). Een praktische uitdaging heeft betrekking op de identificatie van patiënten die regulatietekorten hebben omdat deze mogelijk alleen relevant zijn in een subgroep van patiënten. Bovendien is het belangrijk om de eventuele beïnvloeding van HRV door medicatie op te merken. Toekomstig onderzoek zou zich kunnen richten op het effect van medicatie op HRV. Tot slot zou het wenselijk zijn om zelfregulatievermogen systematischer in experimentele labo-situaties te onderzoeken.

Abbreviations

ACR	American College of Rheumatology
ADHD	Attention Deficit Hyperactivity Disorder
ASEF-I	Ghent Attention and Self-Regulation in Fibromyalgia-I-study
ASI	Anxiety Sensitivity Index
BSCS	Brief Self-Control Scale
BVS	Body Vigilance Scale
CAN	Central Autonomic Network
DASS	Depression Anxiety Stress Scales
DRM	Day Reconstruction Method
EMA	Ecological Momentary Assessment
FM	Fibromyalgia
GHH	Generalized Hypervigilance Hypothesis
GPD-I	Ghent Pain Disability-I-study
HADS	Hamilton Depression Scale
HF	High Frequency band (of Heart Rate Variability)
HFabs	absolute power in the High Frequency band
HRV	Heart Rate Variability
IA	Interoceptive Accuracy
IASP	International Association for the Study of Pain
lnHFabs	log transformed absolute power of the High Frequency band
lnSSD	log transformed Squared Successive Differences
MBSR	Mindfulness-Based Stress Reduction
MPI	Multidimensional Pain Inventory
MSSD	Mean Square of Successive Differences
NA	Negative Affect
PA	Positive Affect
PANAS	Positive And Negative Affect Schedule
PCS	Pain Catastrophizing Scale
PDI	Pain Disability Index
pNN50	percent of difference between adjacent RR intervals that are greater than 50 ms
RMSSD	Root Mean Square of Successive Differences
RSAnorm	normalized Respiratory Sinus Arrhythmia

SSD

Squared Successive Differences

STAI

State-Trait Anxiety Inventory

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GENERAL INTRODUCTION

Pain

The word “pain” is derived from the Latin *poena*, meaning penalty or punishment. Pain is often seen as bad or maladaptive, but it actually serves an important evolutionary function. By attracting the organism’s attention and interrupting ongoing activity, pain promotes withdrawal or active defence and is, thereby, considered essential for survival. Although pain appears in everyday life and is a common experience (e.g. having a headache, bruising oneself, burning one’s tongue with hot tea), it remains hard to clearly describe one’s pain experience. The International Association for the Study of Pain (IASP) defines pain as “*an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage*” which is “*always subjective*” (IASP, 1979, p. 250). This definition underlines three central facets of pain. Firstly, pain experience is subjective and not exclusively determined by the measurable tissue damage. Secondly, pain is not only an aversive sensory experience, but is related to (negative) emotions. Third, pain can occur in the absence of tissue damage (e.g. phantom pain), whereas actual tissue damage must not imperatively lead to pain experience (e.g. athletes’ injuries during important competition). The most widely used differentiation in terms of pain experiences refers to its duration, i.e. *acute* versus *chronic* pain (King, 2000; Turk & Melzack, 2001). Acute pain appears in the short-term, and can usually be treated (e.g. with medication). Chronic pain, however, is generally not well localized, and may persist in the absence of an identifiable physical pathology and despite efforts to relieve it. Thus, chronic pain loses its warning signal function. Chronic pain is commonly identified as pain lasting for longer than 3 months (e.g. Reid, Harker, Bala, & Truysers, 2011) or 6 months (e.g. Breivik, Collett, Ventafridda, Cohen, & Gallacher, 2006) after the initiation of pain.

Persistent pain is a common problem in healthcare. In Europe, the prevalence of chronic pain is estimated at 19% in the general population (Breivik et al., 2006). It is more prevalent in women than in men and it occurs more often in older age (Bouhassira, Lanteri-Minet, Attal, Laurent, & Touboul, 2008; Català et al., 2002; Chung & Wong, 2007). Chronic pain is usually associated with major personal and social impairments as well as considerable financial costs, especially through work absenteeism (Dagenais, Caro, & Haldeman, 2008).

Biopsychosocial perspective

The current consensus that psychological factors contribute to the experience of pain was not always shared. For a long time, a dualistic, biomedical vision dominated the field of pain. This vision was conform to the Cartesian view that mind and body function independently. A direct link between tissue damage and pain experience was assumed (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). Several observations could, however, not be explained with this model. For example, pain may be experienced in the absence of tissue damage (e.g. Nikolajsen & Jensen, 2001) or placebos may alter the experience of pain (Wager et al., 2004). The main model that allowed for the evolution from a biomedical toward a biopsychosocial perspective on pain is the Gate Control Theory (Melzack & Wall, 1965). This model refutes a simple one-to-one relationship between nociceptive input and pain experience. Instead, nociceptive input is stipulated to be modulated at many physiological levels, mainly the spinal cord. Specifically, the authors suggest that the dorsal horn acts like a gate, either inhibiting or facilitating afferent transmission. Pain sensation is thought to arise only if the output exceeds a certain level and passes through the gate. The model was extended by differentiating sensory-discriminative, motivational-affective and cognitive-evaluative aspects of pain (Melzack & Casey, 1968). These aspects are all thought to contribute equally to the experience of pain. Today, the importance of the interaction between biological, social and psychological factors for pain experiences is widely recognized. In the past decades, a range of psychological factors has been investigated to better understand pain perception. One such factor is attention. Popular explanations for chronic pain conditions are based on the idea of dysfunctional attentional processes (Crombez, Van Damme, & Eccleston, 2005).

The role of attentional selection in pain

Attention is an intuitive construct and not a scientific term: *“Everyone knows what attention is. It is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalization, concentration, of consciousness are its essence. It implies withdrawal from some things in order to deal effectively with other.”* (James, 1890, pp. 403-404). Attention is a complex process, and its requirements may seem to be contradictory: *“the need for continuity of attentional engagement, against the need for its interruptibility”* (Allport, 1989, p. 652). Firstly, attention should ensure that behaviour is automatically triggered in the pursuit of

specific goals and is not distracted by conflicting but less important demands. Secondly, attention must allow for the interruption of ongoing behaviour if a new, more important, goal emerges. The protection of an organism from danger must be guaranteed by a flexible attentional system, which can switch to a new subordinate goal if required (Shallice & Burgess, 1993). Pain is a perfect example to illustrate this mechanism. Its evolutionary warning function may activate the goal of self-protection and, thereby, interrupt ongoing behaviour in order to enable an individual to escape from danger (Chapman, Tuckett, & Song, 2008).

Contemporary models of attention distinguish between bottom-up and top-down modes of attentional selection (Corbetta & Shulman, 2002; Sarter, Givens, & Bruno, 2001; Yantis, 2000). Suppose one twists one's ankle while dancing. It is highly probable that the sensation caused by this event will capture attention and prevent from continuing to dance and further injuring oneself. This is one example of the involuntary, stimulus-driven or *bottom-up* capture of attention by pain, which is a critical feature of its alarm function. This mechanism is important for survival as it informs us of potential bodily harm and urges an adequate response to prevent further injury (Eccleston & Crombez, 1999). The involuntary capture by pain has repeatedly been investigated in experimental settings by using the primary task paradigm (Crombez, Baeyens, & Eelen, 1994; Eccleston, 1994). This paradigm consists of one task which participants execute (e.g. detection of specific stimuli) while painful stimuli are occasionally administered but cued to be ignored. Attentional interruption by pain is operationalized as impaired task performance during pain stimulation, e.g. longer reaction times. Numerous studies have demonstrated the attentional interference by pain using this paradigm (Buhle & Wager, 2010; Crombez, Eccleston, Baeyens, & Eelen, 1997; Van Damme, Crombez, & Eccleston, 2004; Vancleef & Peters, 2006). These findings are even more pronounced when pain is intense (Crombez, Eccleston, Baeyens, & Eelen, 1998), novel (Crombez, Eccleston, Baeyens, & Eelen, 1996) or salient (Crombez, Baeyens, & Eelen, 1994). *Top-down* factors can modulate the unintentional capture of attention by pain by facilitating (directing attention toward) or inhibiting (directing attention away from) pain. Legrain and colleagues (2009) postulate that this top-down modulation of attention to pain depends on working memory, and acts through (1) attentional load and (2) attentional set. Attentional load refers to the amount of attention deployed to achieve a task. As such, pain ratings can be reduced by increasing the load of attentional resources of a non-pain-related task (e.g. Romero, Straube, Nitsch, Miltner, & Weiss, 2013). Attentional set refers to the mental set of stimulus features that is used to identify task-relevant stimuli. A stimulus is more likely to be processed if it shares

features of the task-relevant targets (e.g. Van Ryckeghem, Crombez, Eccleston, Legrain, & Van Damme, 2013). Figure 1 depicts the dynamic interplay between bottom-up and top-down processes of attention to pain.

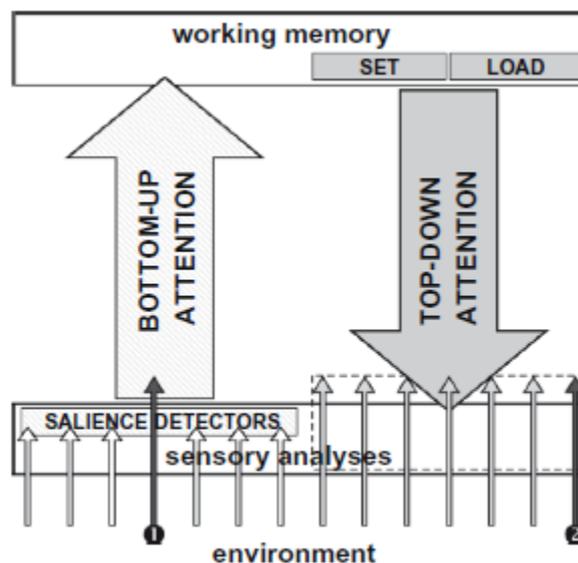


Figure 1. The neurocognitive model of attention to pain (Legrain et al., 2009). The orientation of attention to pain depends on both bottom-up and top-down influences. Bottom-up selection of attention is an involuntary capture of attention by the stimuli which can be modulated by top-down variables such as cognitive goals which are activated in working memory.

Top-down mechanisms can either inhibit or facilitate pain perception. One well-studied example of top-down attentional *inhibition* mechanism is distraction, which is defined as directing attention away from pain. While most of these studies have reported pain-reducing effects of distraction (Tracey & Mantyh, 2007; Van Ryckeghem, Crombez, Van Hulle, & Van Damme, 2012), some have failed to find this effect (Roelofs, Peters, van der Zijden, & Vlaeyen, 2004) or even found opposite results (Goubert, Crombez, Eccleston, & Devulder, 2004; Keogh, Hatton, & Ellery, 2000). Recent research suggests that distraction efficacy depends on psychological factors. For example, attentional bias towards pain-related information (Van Ryckeghem et al., 2012) and catastrophic thinking (Verhoeven et al., 2010) reduces distraction efficacy. In contrast, the capacity of working memory (Legrain, Crombez, & Mouraux, 2011) and the spatial location of the distracting information (Van Ryckeghem et al., 2011) increases distraction efficacy. Van Damme and

colleagues (2010) have emphasized the role of motivation in the attentional processing of pain, which has been shown to increase pain reduction effects (Verhoeven et al., 2010).

A highly prevalent clinical observation relates to the fact that chronic pain patients show a tendency to increase attentional allocation to pain-related information, i.e. hypervigilance. This construct was introduced to describe a tendency to attend to somatic distress signals and to continuously scan for somatic sensations (Chapman, 1978). Hypervigilance to pain-related information has extensively been studied in patients with chronic pain conditions, identifying it as a potential top-down attentional *facilitation* mechanism. Numerous studies report higher scores on self-report measures of vigilance toward pain and pain-related information in chronic pain patients compared with healthy controls (Peters, Vlaeyen, & van Drunen, 2000; Roelofs, Peters, McCracken, & Vlaeyen, 2003; Tiemann et al., 2012). Experimental research has further demonstrated reduced thresholds and tolerance levels for pain in these patients (Kosek, Ekholm, & Hansson, 1996; McDermid, Rollman, & McCain, 1996) and the presence of an 'attentional bias to pain' (Crombez, Van Ryckeghem, Eccleston, & Van Damme, 2013; Pincus & Morley, 2001; Van Damme, Legrain, Vogt, & Crombez, 2010).

Fibromyalgia

Hypervigilance has intensively been investigated as one potentially aetiological factor for a specific chronic pain condition, i.e. fibromyalgia (FM). This condition is characterized by widespread musculoskeletal pain (Wolfe et al., 1990). The term was first suggested by Kahler Hench in 1976, Muhammed Yunus published first observations in the 1980s (Yunus, Masi, Calabro, Miller, & Feigenbaum, 1981). In 1990, a committee of the American College of Rheumatology (ACR) formulated criteria for the FM syndrome (Wolfe et al., 1990):

- History of widespread pain, which includes all four quadrants of the body and axial skeletal pain
- Pain in 11 out of 18 tender points: occipital, low cervical, trapezius, supraspinatus, second rib, lateral epicondyle, gluteal, greater trochanter, and anserine knee bursal regions
- Both these criteria must be present for at least 3 months

It is important to note that the ACR criteria only focus on pain, whereas other important FM symptoms such as fatigue, cognitive disturbances, and psychological distress have also been acknowledged as further key features (Choy et al., 2009; Mease et al., 2007, 2009). In 2010, new ACR preliminary diagnostic criteria for FM were presented, which include these key symptoms and add a quantitative measure of widespread pain and symptom severity (Wolfe et al., 2010). The prevalence of FM in Europe has been estimated at 4.7%, with approximately twice the prevalence rates in women than men (Branco et al., 2010). The aetiology of FM is still not fully understood, but is considered to be multifactorial, including genetic, stress-related, and psychological factors (Bellato et al., 2012).

The association between emotions and pain

Although theories based on dysfunctional attentional processes are widely accepted in pain research, it is important to also take into account the affective aspect of pain experiences, to fully capture pain perception. It is hard to imagine pain without emotion. Their disentanglement remains a challenge for research, and may not even be possible based on the IASP definition explicitly pointing out the emotional component of pain. The question of how pain and emotions, specifically negative emotions, relate to each other is an intriguing scientific problem, which has generated abundant research. It seems obvious that pain can lead to feelings of frustration, worry or anxiety. But, we can also think of examples which highlight the power emotions can have over pain: A thrilling moment letting us forget a toothache or a fight with a colleague aggravating a headache. Indeed, experimentally induced negative emotions or mood changes have been shown to lead to pain or the aggravation thereof (Wiech & Tracey, 2009). Meagher, Arnau and Rudy (2001) showed that viewing pictures representing fear and disgust decreased pain thresholds, and fear even decreased pain tolerance, whereas viewing erotic pictures increased pain thresholds in men. Another study demonstrated increased pain reactivity after the induction of anxiety compared to the induction of fear, which resulted in decreased pain reactivity (Rhudy & Meagher, 2000), although their operationalization of fear (exposure to brief electrical shocks) versus anxiety (anticipation of shocks) may be disputed. The amplifying effect of anger and sadness on pain has also been reported in patients with FM (van Middendorp, Lumley, Jacobs, Bijlsma, & Geenen, 2010).

Individuals experiencing persistent pain usually report low levels of positive affect (Zautra et al., 2005) and higher levels of negative emotions such as anger (Trost,

Vangronsveld, Linton, Quartana, & Sullivan, 2012), anxiety (Asmundson & Katz, 2009), depression (Linton & Bergbom, 2011; Robinson et al., 2009), fear (Vlaeyen & Linton, 2012) or worry (Eccleston & Crombez, 2007). The co-occurrence of pain and negative emotions can have detrimental consequences, e.g. treatment failure, relapse or absenteeism due to sickness (Lumley, 2010; Nicholas, Linton, Watson, & Main, 2011; Roberts, Adebajo, & Long, 2002). Conclusive evidence concerning the direction of association between negative emotions and pain, is, however, lacking (Asmundson & Katz, 2009; Gatchel et al., 2007). Several studies have suggested that mood and anxiety disorders can increase the risk of developing chronic pain syndromes (Larson, Clark, & Eaton, 2004; Roy-Byrne et al., 2008). Nevertheless, chronic pain can also lead to long lasting emotional disturbances such as worrying (Price, 2000). The currently available evidence points to a reciprocal relationship between negative emotions and pain.

Psychophysiological perspective on emotional intensity

One important determinant of emotions is the intensity in which they are experienced. The emotional intensity differs between individuals and these differences have been described in a psychophysiological framework, i.e. the somatic marker hypothesis (Damasio, Everitt, & Bishop, 1996). The model posits that somatic states mark response options, which guide behaviour, particularly decision-making. More precisely, internal and external stimuli elicit somatic states, which involve physiological modifications and are processed in specific brain structures (i.e. amygdala, ventromedial prefrontal cortex). These neural representations of physiological conditions elicit emotions, which provide the individual with options to respond to a stimulus, and guide behaviour. Such models of emotion allow for the investigation of individual differences in the ability to perceive signals originating from the body. The perhaps most commonly investigated aspect refers to the ability to accurately discern internal bodily states, i.e. interoceptive accuracy. This is generally quantified by measuring an individual's ability to accurately report one's heartbeats at rest (Schandry, 1981) and has been conceptualized as a trait (Mussgay, Klinkenberg, & Rüdell, 1999; Tsakiris, Tajadura-Jiménez, & Costantini, 2011). Findings from neuro-imaging studies have linked interoceptive accuracy to the activation of the insula, which, in turn, relates to emotional intensity (Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Zaki, Davis, & Ochsner, 2012). Numerous studies have confirmed that interoceptive accuracy facilitates bottom-up processing of emotions (Dunn et al., 2010; Herbert, Pollatos, & Schandry, 2007; Pollatos, Kirsch, & Schandry, 2005;

Wiens, 2005). Experimental studies on pain have demonstrated the importance of autonomic-nervous-system activity for the experience of pain [as indicated by cardiovascular parameters, e.g. heart rate and heart rate variability (HRV; Koenig, Jarczok, Ellis, Hillecke, & Thayer, 2013; Loggia, Juneau, & Bushnell, 2011), blood pressure (Bruehl, Carlson, & McCubbin, 1992; Fillingim & Maixner, 1996) and spontaneous baroreflex sensitivity (Chung et al., 2008)]. Research on the role of interoceptive accuracy in pain, however, is still in its infancy. Pollatos and colleagues (2012) demonstrated that individuals who are more accurate in perceiving their heartbeats show decreased pain threshold and tolerance levels. It remains, to date, poorly understood whether somatic symptom reporting in chronic pain relates to changes in interoceptive accuracy.

To gain a better understanding of the dynamic interplay between emotions and pain, it may be necessary to switch from a categorical perspective to a more flexible and dynamic perspective. The experience of emotions as well as of pain seems not to be static, but rather fluctuating (Strand et al., 2006; Zautra et al., 2005; Zautra, Smith, Affleck, & Tennen, 2001). Emotional experiences are not only shaped by their intensity, but also by other features, i.e. dynamic time patterns such as variability and temporal dependency. These patterns provide unique information that is relevant for psychological well-being (Houben, Noortgate, & Kuppens, 2015). In line with this research, adaptive regulation of emotions has been suggested as a key factor in the adaptation to chronic pain (Connelly et al., 2007; Hamilton, Zautra, & Reich, 2005; Sturgeon, Zautra, & Arewasikporn, 2014). On the one hand, the capacity to regulate emotions depends on bottom-up emotional processing leading to more or less pronounced challenges for individual regulation abilities. On the other hand, affect regulation also depends on top-down processes involving inhibition and can be integrated in the more general notion of self-regulation, which will be introduced in the following paragraph.

Self-regulation in pain

Self-regulation is an essential homeostatic process referring to the capacity to exert control over one's thoughts, emotions and behaviour in order to achieve a desired state or pursue a goal (Baumeister, 1998; Carver & Scheier, 1998). As such, self-regulation can be classified as a higher-order process, which controls lower-order processes in order to guide behaviour. One of the key features of self-regulation is the capacity for inhibition (Muraven & Baumeister, 2000). Diamond (2013) regards the concepts of inhibitory control and self-regulation as synonymous, in the model of executive functions. The importance of self-regulation for everyday life is easily comprehensible when looking at the plethora of requirements of our modern life, e.g. to delay gratification, and resist and succeed in various contexts: resisting a big variety of unhealthy food or persisting in school to ensure academic achievements.

The neural underpinnings of self-regulatory processes are found in the prefrontal cortex (Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012), a structure which is primarily associated with inhibition. Consequently, frontal hypo-activity is related to psychological disorders characterized by behavioural dysregulation such as attention deficit hyperactivity disorder (Fassbender, 2006), obsessive-compulsive disorder (Stein, 2000), substance dependence (Kaufman, Ross, Stein, & Garavan, 2003) or emotional dysregulation such as depression (Koenigs & Grafman, 2009).

Heart rate variability as an index for self-regulatory capacity

Thayer and Lane have introduced the model of neurovisceral integration (Thayer & Lane, 2000, 2009) to elaborate the intimate connection between the brain and the heart, already postulated by Claude Bernard in the 19th century. The central autonomic network (CAN) has been identified as functional unit to support goal-directed behaviour and adaptability (Benarroch, 1993). The CAN receives and integrates visceral, humoral and environmental information in order to form responses to changing environments. It is under tonic inhibitory control (via GABAergic neurons) and plays a key role in the reciprocal cortico-cardiac interactions with a primary output through sympathetic and parasympathetic neurons. The CAN innervates the heart via stellate ganglia and the vagus nerve at the sino-atrial node. The output of the CAN, thus, directly links to the beat-to-beat intervals of the heart. Figure 2 illustrates the pathways by which the prefrontal cortex exerts inhibitory control over the heart rate.

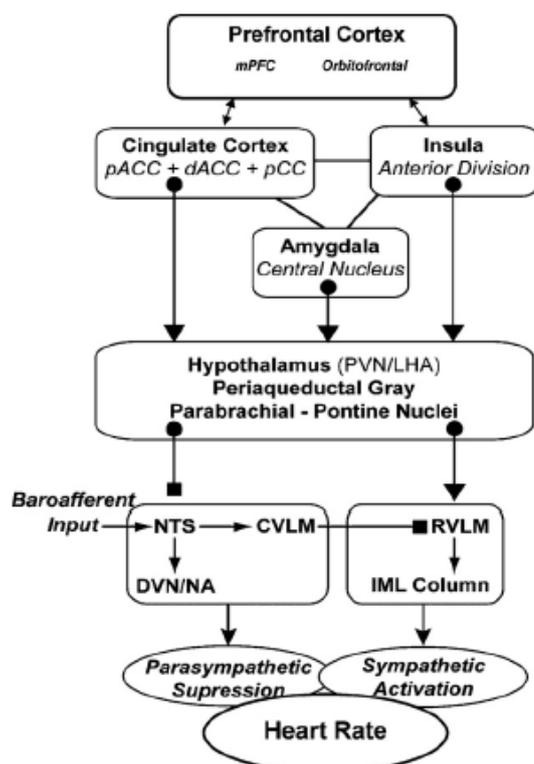


Figure 2. Illustration of the pathways by which the prefrontal cortex influences heart rate based on the neurovisceral model of integration (Thayer & Lane, 2009).

Heart rate variability (HRV), therefore, indexes the activity of a flexible network of neural structures that is dynamically organized in response to environmental challenges, i.e. self-regulatory capacity. Accordingly, prefrontal inhibitory control plays a major role when it comes to the ability to – consciously or unconsciously – decide which information to attend to (attention regulation; Thayer & Johnson, 2004), and which emotional states to modify (emotion regulation; Appelhans & Luecken, 2006; Thayer & Brosschot, 2005) in order to allow for the promotion of a specific behaviour in a specific situation.

Attentional regulation

Attention is a crucial aspect of self-regulation, as all self-regulatory processes involve attention (Shapiro & Schwartz, 2000). Exerting control over the focus of attention and inhibiting pre-potent but inappropriate responses are important for health in a complex environment. Attentional selection can either facilitate the processing of goal-supporting information or inhibit the processing of information relating to competing goals (Luszczynska, Diehl, Gutiérrez-Doña, Kuusinen, & Schwarzer, 2004). Attentional

regulation is considered a relatively stable characteristic of an individual (Kandel, Schwartz, & Jessell, 2000) and refers to the ability to organize incoming stimuli in order to create an appropriate response to the selected stimuli (Luszczyńska et al., 2004). One established example of deficits of attentional regulation is attention deficit hyperactivity disorders (ADHD). In ADHD, deficits have been specifically reported for sustained attention, i.e. the ability to focus on one activity for an extended period of time (Hooks, Milich, & Lorch, 1994). Porges (1992) suggests that the capacity to attend is limited by the neurophysiological organization of an individual and proposes HRV as a mediator of attention. As in the model of neurovisceral integration (Thayer & Lane, 2000, 2009), HRV is considered a marker of efficiency of neural feedback mechanisms and, therefore, indexes an individual's capacity to organize physiological resources to respond appropriately (Porges, 1992). Several studies have demonstrated that HRV relates to the ability to allocate psychophysiological resources to meet environmental demands. For example, individuals with high HRV show better performance in a task requiring sustained attention (Hansen, Johnsen, Sollers, Stenvik, & Thayer, 2004), and odontophobic patients with low HRV show poorer attentional performance in a modified Stroop paradigm reflected by difficulties in inhibiting pre-potent but inappropriate responses (Johnsen et al., 2003).

This selective aspect of attention is crucial for the regulation of goal-directed activities across changing circumstances (Luszczyńska et al., 2004). The regulation of attention is, however, not limited to the area of environmental sources, but can also aim at internal sources (Porges, 1992). Emotions certainly constitute one of the most important internal targets of regulation when it comes to understanding pain perception in its complexity, and improve treatments for chronic pain conditions.

Emotion regulation

Emotions arise in situations which one appraises as being relevant to one's current goals (Gross & Jazaieri, 2014). They are central to people's lives and can have a powerful impact on their functioning, both positive and negative. Emotion regulation refers to a set of processes – automatic or controlled, conscious or unconscious – to increase, maintain or decrease one's emotional state (Gross & Thompson, 2007). The capacity to regulate emotions has been described as vital to social functioning (Eisenberg, 2001) and for maintaining mental health (Gross & Muñoz, 1995). Many mental disorders are characterized by a lack in emotion regulation. Emotion dysregulation refers to the inability

to flexibly respond to and manage emotions (Carpenter & Trull, 2013). Gross and colleagues (2014) state that emotion dysregulation can result from problematic patterns of emotion intensity, duration, frequency or type. Affective instability reflects one aspect of dysregulated emotion, which is generally conceptualized as a pattern of frequent and large mood shifts over time (Jahng, Wood, & Trull, 2008). Affective instability has been found to be associated with various forms of psychopathology and lower psychological well-being (Ebner-Priemer, Eid, Kleindienst, Stabenow, & Trull, 2009; Houben et al., 2015; Trull et al., 2008). Recent research has suggested vagally mediated HRV as an objective measure to index emotion regulation (Appelhans & Luecken, 2006). In line with this assumption, Koval and colleagues (2013) reported a negative relationship between HRV and affective instability in healthy individuals.

Self-regulatory capacity in chronic pain

Dysregulated affect, however, is only one precise example of dysfunctional regulation. Our society presents a lot of examples of failed self-regulation (e.g. obesity, substance dependence), which can be considered as lapses rather than intended acts. Experimental evidence suggest that the capacity to self-regulate does not only differ inter-individually, but is a resource that can be depleted (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Muraven, Tice, & Baumeister, 1998). Solberg and colleagues (2010) suggest that chronic pain patients experience self-regulatory fatigue as they constantly aim to adapt to their condition. Chronic pain conditions pose complex challenges through interactions of cognitive, emotional and physiological disturbances (Solberg Nes, Roach, & Segerstrom, 2009). Domains of self-regulation and symptoms/deficits in chronic pain patients are closely linked (Solberg Nes et al., 2009). To list only a few examples: Control thoughts, problem solving, behavioural disengagement, emotional instability, worry and rumination or staying involved in treatment. Further evidence for decreased self-regulatory capacity is provided by studies reporting lower HRV in patients with chronic pain conditions (Cohen et al., 2000; Koenig, Loerbroks, Jarczok, Fischer, & Thayer, 2016; Terkelsen et al., 2012). The little research that exists in this domain emphasizes that it is essential to take the aspect of self-regulatory capacity into account when aiming to better understand and treat these complex conditions. Solberg and colleagues (2009) propose an interactive view

on how self-regulatory demands can lead to self-regulatory fatigue when faced with pain, thereby preventing successful adjustment to pain conditions (Figure 3).

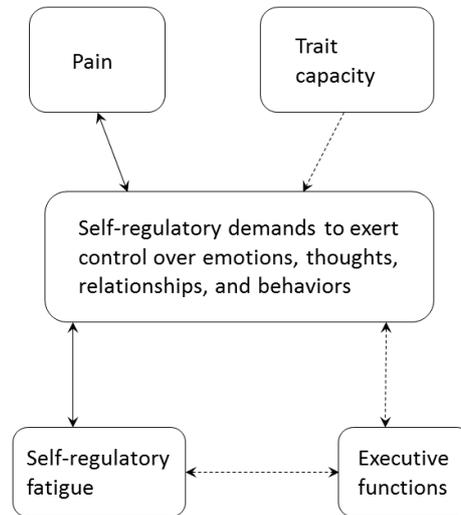


Figure 3. Proposed relationships between pain, self-regulatory demands, self-regulatory fatigue and executive functions according to Solberg and colleagues (2009). Solid arrows indicate positive relationships and dashed arrows indicate inverse relationships.

Firstly, self-regulatory capacity can be fatigued by excessive demands. This results in reduced capacity to execute additional self-regulatory demands. Secondly, self-regulatory demands and executive functions co-vary inversely. That is, self-regulatory demands reduce executive functions, leading to weakened capacity to meet further demands. Thirdly, with a high trait capacity (e.g. HRV) one can better respond to self-regulatory demands and thereby reduce their impact. On the contrary, pain increases the magnitude of self-regulatory demands as it is accompanied by cognitive, emotional and physical demands. It is important to note that the connections among these constructs show a potential for a downward spiral in which self-regulatory demands cause self-regulatory fatigue, reduce executive resources for further self-regulation and thereby increase difficulties in meeting further demands. The authors support the notion that self-regulatory deficits are fundamental contributing factors to the phenomenology of chronic pain conditions and motivate future research addressing the impact of self-regulatory deficits on illness.

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STUDY AIMS

Study aims and dissertation outline

In this PhD thesis, we aimed to address the shortcomings of research on the role of self-regulatory capacity in the adaptation to pain. To do so, we used a range of different methodologies (e.g. diary methodology, psychophysiological measures, experimental distraction paradigm) and examined non-clinical as well as clinical populations in experimental and natural settings. This strategy allowed for a broad investigation of the impact of self-regulatory capacity on processes of pain experience specifically with regard to attentional and emotional processes. The present project aimed at expanding insight into mechanisms relevant for the adaptation to pain and, thereby, to reveal implications for the improvement of treatment strategies for chronic pain patients in a self-regulatory perspective. We investigated the role of self-regulatory capacity

- In the daily functioning of chronic pain patients (Chapter 1)
- In emotion regulation of chronic pain patients compared with healthy individuals (Chapter 2)
- In the accuracy of perceiving internal bodily signals under the assumption of chronic pain patients being hypervigilant (Chapter 3)
- In distraction efficacy and attentional interference by pain in healthy individuals (Chapter 4)

Study 1 establishes the relationship between affective instability, indicating dysregulated emotion, and daily functioning of chronic pain patients. More specifically, we investigated the predictive power of affective instability for daily disability and cognitive complaints in a natural context over a period of 14 consecutive days. The conceptualization of pain as a sensory and emotional experience allows for assuming a link between dysregulated affect (affective instability) and dysfunctional regulation of pain (interference with daily activities and cognitive processes). We used an innovative approach of measuring affective instability in daily life with an index incorporating both variability (i.e., average magnitude of affective changes) and temporal dependency (i.e., average frequency of affective changes).

Study 2 builds on these findings and adds a psychophysiological proxy of self-regulation to (1) compare affective instability between fibromyalgia patients and healthy controls, (2) investigate the role of emotional top-down regulatory capacity in daily pain, and (3) replicate findings with regard to the role of affective instability in daily pain

outcomes. In this study, we investigated the link between two supposedly complementary (physiological: HRV and experiential: affective instability) indices of emotion regulation capacity in fibromyalgia patients and healthy controls with a diary method. Assuming that dysfunctional affect regulation may not only be linked to mental but also to physical problems, we investigated group differences in affective instability. We further expected to replicate previous findings indicating that patients with higher levels of affective instability report poorer functioning in general, and show greater reductions in functioning on days when they experience higher than usual levels of pain.

Study 3 investigates hypervigilance as one aspect of top-down facilitation in fibromyalgia patients. Based on the Generalized Hypervigilance Hypothesis (Hollins et al., 2009; McDermid et al., 1996), we expected for fibromyalgia patients to be hypervigilant to bodily sensations and show increased interoceptive accuracy as operationalised with a heartbeat tracking task (Schandry, 1981). In this task, patients and healthy controls were asked to silently count all the heartbeats they perceived without taking their pulse. Heart rate was monitored during rest as we further expected trait-HRV to be decreased in fibromyalgia patients. Lower HRV indicates insufficient inhibitory control, which, in turn, would relate to hypervigilance.

Study 4 completes the picture by attempting to validate the self-regulatory biomarker HRV in the context of top-down inhibition and bottom-up capture of attention. We investigated the possible association between HRV and distraction efficacy as well as the inverse association between HRV and attentional interference by pain. In this study, healthy participants performed a distraction task. During this paradigm, they were instructed to localize either a somatosensory (electrocutaneous or vibrotactile) stimulus (focus trial) or a visual stimulus (distraction trial). Both stimuli were simultaneously presented. We expected that self-reported pain intensity and unpleasantness of the electrocutaneous stimuli would be lower in the distraction than in focus trials. We also expected that reaction times in the distraction trials would be longer when electrocutaneous stimuli are presented. Our last hypothesis concerned the predictive power of HRV, indexing inhibitory capacity, for distraction efficacy and attentional interference by pain.

Finally, the **general discussion** highlights the main findings of the studies and offers an interpretation and integration of these findings. Furthermore, limitations of the studies and clinical implications as well as perspectives for future research are discussed.

EMPIRICAL STUDIES

1

CHAPTER 1

**Affective Instability in Patients with Chronic Pain:
A Diary Approach¹****Abstract**

Affective instability, conceptualized as fluctuations in mood over time, has been related to ill-health and psychopathology. In this study, we examined the role of affective instability upon daily pain outcomes in 70 chronic pain patients ($M_{\text{age}} = 49.7$ years; 46 females) using an end-of-day diary. During a baseline phase, patients completed self-reported questionnaires of pain severity, pain duration, disability, depression, and anxiety. During a subsequent diary phase, patients filled out an electronic end-of-day diary over 14 consecutive days assessing daily levels of pain severity, disability, cognitive complaints, negative affect (NA) and positive affect (PA). Affective instability was operationalized as the mean square of successive differences (MSSD) in daily mood (separately for NA and PA), which takes into account the size of affective changes over consecutive days. Results indicated that NA instability was positively associated with daily disability, beyond the effects of daily pain severity. Furthermore, NA instability moderated the relationship between daily pain severity and daily disability and the relationship between daily pain severity and daily cognitive complaints. PA instability, however showed to be unrelated to all outcomes. Current findings extend previous results and reveal the putative role of affective instability upon pain-related outcomes and may yield important clinical implications. Indeed, they suggest that targeting NA instability by improving emotion regulation skills may be a strategy to diminish disability and cognitive complaints in patients with chronic pain.

¹ Rost, S., Van Rykceghem, D., Koval, P., Sütterlin, S., Vögele, C., & Crombez, G. (2016) Affective instability in patients with chronic pain: A diary approach. *Pain*, 157(7), 1783-90.

Introduction

Emotions change following the ebb-and-flow of daily life. However, experiencing unusually large and/or frequent changes in emotion, labeled affective instability, can be considered dysfunctional (Thompson et al., 2012; Trull et al., 2008). Affective instability may reflect problems in regulating affect (Carpenter & Trull, 2013; Selby et al., 2015), which is a key feature of mental disorders (Gross & Jazaieri, 2014). In line with this view, affective instability has been found to be associated with lower well-being and various forms of psychopathology (Ebner-Priemer, Eid, Kleindienst, Stabenow, & Trull, 2009; Houben, Noortgate, & Kuppens, 2015; Thompson et al., 2012; Trull et al., 2008). To our knowledge, there are no studies that have investigated the role of affective instability in patients with chronic pain.

There is abundant research demonstrating that patients with chronic pain report high levels of negative affect (NA) and low levels of positive affect (PA) on self-report questionnaires (Affleck, Tennen, Urrows, & Higgins, 1994; Potter, Zautra, & Reich, 2000; Zautra et al., 1995). Questionnaire research, however, does not capture well the temporal dynamics of affect. Indeed, chronic pain patients experience fluctuations not only in pain (Solberg Nes, Roach, & Segerstrom, 2009), but also in NA and PA (Strand et al., 2006; Zautra, Johnson, & Davis, 2005; Zautra, Smith, Affleck, & Tennen, 2001). Investigating moment-to-moment variations in affect may further our understanding of chronic pain and associated problems. As an example, Sturgeon and colleagues (2014) showed that the relationships between daily PA and pain as well as daily NA and pain were mediated by positive interpersonal events and pain catastrophizing. Similarly, Connelly and colleagues (2007) found that changes in PA and NA from the prior day to the current day predicts significantly greater decreases in pain that day. In the current study, we examined the potential role of affective instability, an index that captures both the frequency and size of affective fluctuations (Jahng, Wood, & Trull, 2008), upon pain and its outcomes. This is in line with previous research which suggests that affect regulation may be a key factor for the adjustment to pain (Hamilton, Zautra, & Reich, 2005). Affective instability may be considered an indicator of dysregulated affect (Carpenter & Trull, 2013; Selby et al., 2015). As pain is conceptualized as a sensory and emotional experience, affective instability is likely to be closely related to the dysfunctional regulation of pain. With increasing affective instability, we may expect pain to interfere more with daily activities and cognitive processes (Attridge, Crombez, Van Ryckeghem, Keogh, & Eccleston, 2015; Eccleston & Crombez, 1999; Van Damme, Legrain, Vogt, & Crombez, 2010). Based on the

results of a recent meta-analysis on the role of affective instability in psychological well-being (Houben et al., 2015), we expected NA instability to have a stronger influence on daily pain outcomes.

To investigate the role of affective instability, the current study assessed day-to-day fluctuations in levels of pain and mood using end-of-day diaries over 14 consecutive days. This method allowed us to investigate the impact of the dynamics of PA and NA on daily pain outcomes, i.e. disability and cognitive complaints. In particular, we were interested (1) to what extent NA and PA instability were related to standard measures of emotional functioning, such as anxiety and depression, (2) whether NA and PA instability were related to daily pain outcomes (disability and cognitive complaints), and (3) whether NA and PA instability moderated the relationship between daily pain severity on the one hand and daily disability and cognitive complaints on the other hand.

Methods

Participants

This study reports secondary analyses of data from the Ghent Pain Disability – I (GPD-I) Study. Previous research has been reported using a part of these data, but had other research objectives (see Van Ryckeghem et al., 2013). Detailed information on recruitment, primary research objectives and variables assessed can be found on the website (<http://hdl.handle.net/1854/LU-3050986>). We briefly report the most relevant information. 518 members of the Flemish Pain League responded to an invitation letter of which 315 agreed to be contacted by phone. 276 persons were actually contacted by phone. Patients were recruited in February and March 2011. Inclusion criteria were: 1) age between 18 and 65 years; 2) sufficient knowledge of the Dutch language; and 3) pain that lasted for at least six months. Individuals were excluded if headache pain was the most important pain ($n = 1$; cf. Morley, Eccleston, & Williams, 1999), when they were unable to use both index fingers ($n = 1$) or when their eyesight was not normal or corrected-to-normal ($n = 2$; e.g. by glasses). The latter two exclusion criteria were used as participants had to be able to perform a computer task. However, this task was not part of this study. 81 patients fulfilled the inclusion criteria and agreed to participate. The need to travel to the university campus to participate in this study was mentioned as the most frequent reason for nonparticipation. Seven more patients did not participate because of health problems. The final sample of patients consisted of 74 individuals with chronic pain.

Patients filled out some standard questionnaires (STAI-T, HADS-D, PDI, MPI) via an online assessment system at home. When arriving at the research lab, patients filled out the STAI-S. All these measures are labeled as “baseline” measures. Next, they participated in an experiment (see also Van Ryckeghem et al., 2013) and received instructions for the diary study. The diary study started one day after the visit to the research lab. Patients filled out an online end-of-day diary for 14 consecutive days. Measures from the diary are labeled “daily” measures. The study design was approved by the Ethics Committee of the Faculty of Psychology and Educational Sciences of Ghent University. Patients provided written informed consent and received a monetary compensation for their participation.

Questionnaires

State and trait anxiety were measured with the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983; Van der Ploeg, Defares, & Spielberger, 1980). This questionnaire consists of 40 items asking people to report how they feel in general (e.g. “I feel nervous”) and at present (e.g. “I feel at ease”) using a scale from 1 (*almost never/not at all*) to 4 (*almost always/very much so*). Scores for the trait and state version may vary between 20 and 80. This questionnaire shows good reliability and validity (Barnes, Harp, & Jung, 2002; Spielberger et al., 1983). In the present study Cronbach’s alpha was .93 for the trait version and .93 for the state version.

Depressive mood was assessed by means of the depression subscale of Hospital Anxiety and Depression Scale (HADS-D; Zigmond & Snaith, 1983). The HADS-D is designed to screen for depression during the last week and has been developed for patients with “medical conditions”. It consists of seven items rated on a 4-point Likert scale. Scores may range from 0 to 21. In the present study, Cronbach’s alpha of the HADS-D was .81.

Disability because of pain was measured with the Pain Disability Index (PDI; Pollard, 1984). Patients indicate the degree of disability experienced in seven life domains (e.g. family and occupation) using a scale from 0 (*no disability*) to 10 (*total disability*). They are asked to respond to each category by indicating the overall impact of pain in their life, not just when pain is at its worst. Scores may vary between 0 and 70. In the present study, Cronbach’s alpha of the PDI was .81.

Pain severity was assessed with the pain severity subscale of the Multidimensional Pain Inventory (MPI; Kerns, Turk, & Rudy, 1985; Lousberg et al., 1999). Part I of the MPI consists of 5 subscales assessing the impact of pain (i.e. pain severity, pain interference, social support, perceived life control and affective distress) on a 7-point Likert scale. Pain severity was assessed by means of two items (i.e., “Rate the level of your pain at the present moment” and “On average, how severe has your pain been during the last week?”). The third item (“How much suffering do you experience because of your pain?”) was not taken into account as its content relates to suffering rather than pain severity (see Parenteau & Haythornthwaite, 2011). The MPI showed a good reliability and validity (Rudy, 1989). In the present study, Cronbach’s alpha of the MPI severity subscale was .82.

End-of-day diary assessment

Patients were asked to fill out an online diary at the end of each day for 2 consecutive weeks. They were reminded to do so each evening via a text message. The diary took approximately 5 minutes to complete. In this study, only the items which are of relevance for the current research aim are described. Items were developed iteratively by a group of pain researchers and piloted for feasibility in patients with chronic pain.

Daily affect: Items assessing daily PA and NA were rated on a scale from 0 (*not at all agree*) to 6 (*totally agree*). We used six adjectives for PA and NA respectively: glad [blij], enthusiastic [enthousiast], happy [gelukkig], relaxed [ontspannen], strong [sterk] and proud [trots] for PA and afraid [bang], irritated [geirriteerd], angry [kwaad], powerless [machteloos], sad [triest] and nervous [zenuwachtig] for NA. Items were derived from a validation study investigating the representation of emotion terms in a general population (Veirman & Fontaine, 2015). PA and NA scales were calculated by averaging PA and NA items respectively. We calculated within-person reliability of the PA and NA scales using the Generalizability theory approach described by Bolger and Laurenceau (2013). Estimates of within-person reliability were .84 for PA and .77 for NA, indicating that both scales assessed within-person changes reliably.

Daily pain severity was assessed using two items: “On average, how severe has your pain been today?” and “Which number would you ascribe to the pain you experienced the most today?” both rated on a scale from 0 (*no pain*) to 10 (*worst imaginable pain*). As the two pain items were highly correlated (within-person correlation was .78, $p < .001$), we calculated an average score of both items.

Daily pain disability was assessed by the item “To what extent did pain interfere with your planned activities?” rated on a scale from 0 (*not at all*) to 10 (*very much*). This item corresponds to the PDI questioning, but asks more generally about the degree to which pain prevents patients from their planned activities.

Daily cognitive complaints were assessed by three items: distractibility (“To what extent were you distracted today?”), forgetfulness (“To what extent were you forgetful today?”) and clear thinking (“To what extent were you able to think clearly today?”) all rated on a scale from 0 (*not at all*) to 10 (*very much*). The clear thinking item was reverse-coded (subtracting scores from 10) and scores on these three items were averaged to form a score for daily cognitive complaints with a range from 0 to 10. The within-person reliability of the cognitive complaints scale was .80. These items were selected based on previous research in which authors reported frequent cognitive problems of chronic pain patients (McCracken & Iverson, 2001; Roth, Geisser, Theisen-Goodvich, & Dixon, 2005).

Affective instability

Affective instability, conceptualized as frequent mood shifts over time (Jahng, Wood, & Trull, 2008), takes into account the size of affective changes as well as their temporal order. It is related to the magnitude of the difference from one time-point to another. The mean square of successive differences (MSSD) has been proposed as an index of affective instability (Jahng et al., 2008). It reflects the extent to which consecutively assessed mood states differ from each other and, therefore, provides a measure of both variability (i.e., average magnitude of affective changes) and temporal dependency (i.e., average frequency of affective changes). In our study, this measure reflects both the frequency and size of fluctuations in daily mood over 14 days. The squared difference between successive observations at occasions $i + 1$ and i for a time series of n measurement occasions is given by:

$$MSSD = \frac{1}{N - 1} \sum_{i=1}^{N-1} (x_{i+1} - x_i)^2$$

This index was calculated separately for PA and NA (see Houben et al., 2015; Koval et al., 2013; Thompson et al., 2012).

Procedure and data handling

Diary reports were included in the analyses only if they filled out the diary for at least 10 out of the 14 days. The data of four patients were excluded for this reason, providing a final sample of 70 patients.

Statistical model

Descriptive statistics and correlation analyses were performed using SPSS statistical software, version 19.0 for Windows (SPSS Inc., Chicago/IL). The nested structure of the data (multiple observations nested within individuals) requires a multilevel regression approach. The HLM software package (Version 7.01; Scientific Software International, Skokie/IL) was used (Raudenbush & Bryk, 2002). Level 1 variables consisted of the daily diary measures of affect (PA and NA), daily pain intensity, pain disability, and cognitive complaints. All Level 1 variables were continuous and entered group mean centered (Enders & Tofghi, 2007). Level 2 variables consisted of baseline questionnaire measures of gender, age, pain duration, and baseline disability. Gender was dummy coded (0 = female; 1 = male) and entered un-centered, while all continuous Level 2 variables were standardized to facilitate interpretation (Nezlek, 2012). Full maximum likelihood estimation was used for all models. We followed a model building procedure in our analyses (Raudenbush & Bryk, 2002). To maximize stability and reliability of the findings, we excluded control variables from further steps in model building if their effects proved to be non-significant (Kreft & de Leeuw, 1998). As suggested by some authors, we controlled for the mean level of daily affect in order to get the effect specific to within-individual variability (Ebner-Priemer & Trull, 2009; Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007). Mean levels of daily PA and NA were therefore included in the final analyses at Level 2. The moderating role of affective instability was investigated in the last step of model building. Models included random intercepts and random slopes and included the subscripts i representing days and j representing persons:

$$\text{Level 1:} \quad \text{daily pain outcome} = \beta_{0j} + \beta_{1j} * \text{daily pain severity}_{ij} + r_{ij}$$

$$\text{Level 2:} \quad \beta_{0j} = \gamma_{00} + \gamma_{01} * \text{mean daily affect}_j + \gamma_{02} * \text{affect instability}_j + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11} * \text{mean daily affect}_j + \gamma_{12} * \text{affect instability}_j + \mu_{1j}$$

Results

Participant characteristics

The average age of the patients was 49.7 years ($SD = 9.76$; range 22-64 years), and 46 were female (65.7%). Most were married (63%) or living together (10%). A total of 42.6% reported a higher education level (college or university degree), 55.9% of the patients had a secondary school degree and the remaining 1.5% had a primary school degree. The sample consisted of a mixed group of chronic pain patients. All of them were seeking for help, taking medication, receiving some kind of pain treatment (e.g. occupational therapy, chiropractic, manual therapy) and/or seeing a specialist (e.g. orthopedist, rheumatologist, oncologist, neurosurgeon, physiotherapist, psychologist). The mean pain duration was 168.99 months ($SD = 111.74$) and almost every patient indicated having more than one pain location ($M = 3.81$, $SD = 1.89$; range 1-9). Most frequently reported pain locations were the back (92.9%), neck (67.1%) and leg (66.7%). Mean pain severity was 3.86 as measured by the MPI ($SD = 0.97$) and mean disability was 39.17 on the PDI ($SD = 11.34$); these mean levels compare well with previously reported data from chronic pain patients ($M_{MPI} = 4.2$, $SD_{MPI} = 1.1$; $M_{PDI} = 44.6$, $SD_{PDI} = 13.4$; Chibnall & Tait, 1994; Nicholas, Asghari, & Blyth, 2008). On average, the level of daily PA was 2.46 ($SD = 1.24$) and the level of daily NA was 1.75 ($SD = 1.12$). The mean level of instability for PA was 1.08 ($SD = 0.98$) and for NA 1.06 ($SD = 1.01$), indicating that, on average, patients' PA and NA levels varied by approximately 1 point (on a scale from 0 to 6) from one day to the next. Those with higher (+1 SD) levels of instability showed differences of approximately 2 scale points in their affect levels on successive days, whereas patients with lower (-1 SD) levels of instability had near to zero differences in their affect levels from day-to-day. Mean scores for state ($M = 38$, $SD = 9.49$) and trait anxiety ($M = 47.17$, $SD = 11.35$) were comparable with those in other chronic pain studies (Asmundson, Carleton, & Ekong, 2005; Crombez, Hermans, & Adriaensen, 2000). Depression scores ($M = 8.39$, $SD = 4.03$) were mildly elevated compared to available norms (Snaith & Zigmond, 1994).

Correlation analyses

Pearson correlations were calculated between NA and PA instability, assessed via end-of-day diary, and measures assessed via questionnaires (i.e. depression, state anxiety, trait anxiety, baseline pain severity, baseline disability). Further Pearson correlations were calculated between NA and PA instability and the mean levels of daily PA and NA, measures based on the daily reported emotions. The two measures of affective instability were not related to the mean levels of daily affect. Further, NA instability was related to more trait anxiety (STAI), more pain (MPI) and more disability (PDI). For PA instability there were no significant relationships, except with NA instability. An overview of the means and correlations is presented in Table 1.

Table 1
Means (M), Standard Deviations (SD) and Pearson Correlation Coefficients for All Measures

	M	SD	2	3	4	5	6	7	8	9
1. Instability of PA (MSSD)	1.08	0.98	.683***	-.053	.008	.069	.194	.151	.028	-.043
2. Instability of NA (MSSD)	1.06	1.01		.069	.058	.284*	.313**	.304*	-.120	.088
3. Depression (HADS-D)	8.4	4.03			.529***	.749***	.463***	.331**	-.679***	.690***
4. State anxiety (STAI-S)	38	9.49				.609***	.184	-.024	-.408**	.618***
5. Trait anxiety (STAI-T)	47.17	11.35					.402**	.286*	-.599***	.777***
6. Pain severity (MPI)	3.86	0.97						.554***	-.350**	.376**
7. Disability (PDI)	39.17	11.34							-.252*	.260*
8. Mean daily PA	2.46	1.24								-.585***
9. Mean daily NA	1.75	1.12								

Note. PA = positive affect, NA = negative affect, MSSD = Mean Square Successive Difference, HADS-D = Depression scale of the Hospital Anxiety and Depression Scale, STAI-S = State subscale of the State-Trait Anxiety Inventory, STAI-T = Trait subscale of the State-Trait Anxiety Inventory, MPI = Multidimensional Pain Inventory, PDI = Pain Disability Index, $n = 67$ for HADS-D, * $p < .05$, ** $p < .01$, *** $p < .001$.

Multilevel analyses

Separate multilevel models were run with daily disability and daily cognitive complaints as outcome measures. We predicted that NA instability would be positively related to daily disability and daily cognitive complaints and moderate the relationship between daily pain severity and its outcomes. We derive similar hypotheses to investigate the relationship with PA instability.

Negative affect instability.

Daily disability.

Initial analyses indicated that there was substantial variance in reported daily disability between (43%) and within (57%) patients. Firstly, we included daily pain severity as a Level 1 predictor. The model proved to be better [$X^2(3) = 367.50, p < .001$] than the model without any predictor. Secondly, we added NA instability as a Level 2 predictor, and additional Level 2 measures, i.e. age, gender, pain duration, baseline pain severity, pain disability and the mean daily level of NA, as control variables. The proposed model proved to be better than the model with only daily pain severity as a Level 1 predictor [$X^2(7) = 44.18, p < .001$]. Only the mean level of daily NA was a significant predictor for the intercept of daily disability [coefficient = 0.895, $t(62) = 5.5, p < .001$], indicating that patients reporting more daily NA also reported more daily disability. We, therefore, excluded age, gender, pain duration, baseline pain severity and pain disability. Third, we included the interaction between NA instability (Level 2) and daily pain severity (Level 1) as a cross-level moderator of the Level 1 intercept and slope. The resulting model was better than without the cross-level interaction term [$X^2(3) = 0.847, p = .037$]. NA instability was a significant predictor for the Level 1 intercept, meaning the mean level of daily disability [coefficient = 0.445, $t(67) = 2.27, p = .026$], and the Level 1 slope [coefficient = 0.094, $t(67) = 2.09, p = .041$], indicating a stronger within-person association between daily pain severity and disability for patients who were more unstable in their NA. The final model is shown in Table 2. It shows that (1) the mean level of daily NA and NA instability are related to daily disability and (2) NA instability moderates the association between daily pain severity and daily disability. Some further analyses were performed to test the robustness of our findings. For example, when including trait anxiety as a Level 2 variable, there were no significant changes for the predictive values of the Level 2 variables.

Daily cognitive complaints.

Initial analyses indicated the presence of substantial variance in reported daily cognitive complaints between (60%) and within (40%) patients. Firstly, we investigated the predicting effect of daily pain severity at Level 1. The resulting model proved to be better [$X^2(3) = 70.13, p < .001$] than the model without any predictor. Secondly, we added NA instability as a Level 2 predictor, as were control variables, i.e. age, gender, pain duration, baseline pain severity and the mean level of daily NA. This model was better than the model with daily pain severity as single Level 1 predictor [$X^2(6) = 28.06, p < .001$]. Only the mean level of daily NA was a significant predictor for the intercept [coefficient = 0.77, $t(63) = 3.94, p < .001$], indicating that patients reporting more daily NA also reported a higher level of daily cognitive complaints. Age, gender, pain duration and baseline pain severity were excluded from the model as they did not show any significant effects. Third, we added the interaction between NA instability (Level 2) and daily pain severity (Level 1) as a cross-level moderator of the Level 1 intercept and slope to the model. The model proved to be better than without cross-level interaction [$X^2(2) = 8.73, p = .013$]. NA instability was a significant predictor only for the Level 1 slope [coefficient = 0.13, $t(67) = 2.39, p = .020$], indicating a stronger within-person association between daily pain severity and cognitive complaints for patients who were more unstable in their NA. The final model (Table 2) suggests that (1) the mean level of daily NA is related to daily cognitive complaints and (2) NA instability moderates the association between daily pain severity and daily cognitive complaints. The robustness of these findings was tested by some further analyses. For example, when including trait anxiety as a Level 2 variable, trait anxiety showed to be a significant predictor [coefficient = 0.63, $t(66) = 2.12, p = .038$], but not the mean level of daily NA [coefficient = 0.39, $t(66) = 1.48, p = .145$]. There was no change in the moderating role of NA instability.

In a series of posthoc-analyses we explored whether the effect of affective instability was moderated by age. Age only moderated the effect of NA in the model predicting cognitive complaints. Specifically, there was a significant interaction between age and NA instability on the within-person association between daily pain and cognitive complaints [coefficient = -0.185, $t(65) = -2.34, p = .023$], meaning that the effect of NA instability was larger in younger patients. Age did not moderate the effects of affective instability in any of the other three models ($ps > .33$).

Table 2
Final Hierarchical Linear Models in Regard of Negative Affect Instability

Dependent variable (diary items)	coefficient	SE	t
Daily disability			
Intercept (γ_{00})	4.484	0.16	27.549***
NA_MSSD (γ_{01})	0.445	0.20	2.272*
Mean NA (γ_{02})	1.020	0.17	5.856***
Daily pain severity (γ_{10})	0.916	0.05	18.457***
Daily pain severity x NA_MSSD (γ_{11})	0.094	0.04	2.085*
Daily pain severity x Mean NA (γ_{12})	0.022	0.05	0.411
Daily cognitive complaints			
Intercept (γ_{00})	3.233	0.17	18.635***
NA_MSSD (γ_{01})	0.238	0.16	1.521
Mean NA (γ_{02})	0.872	0.16	5.427***
Daily pain severity (γ_{10})	0.277	0.05	5.395***
Daily pain severity x NA_MSSD (γ_{11})	0.128	0.05	2.386*
Daily pain severity x Mean NA (γ_{12})	0.070	0.04	1.630

Note. NA = negative affect, MSSD = mean square successive difference, * $p < .05$, ** $p < .01$, *** $p < .001$.

Positive affect instability.

Daily disability.

Initial analyses indicated that there was substantial variance in reported daily disability between and within patients, and daily pain severity to be a significant Level 1 predictor (see above). In a next step, we added PA instability as a Level 2 predictor and further Level 2 control variables, i.e. age, gender, pain duration, baseline pain severity, baseline pain disability and the mean level of daily PA, to our model. The proposed model proved to be better than the model with only daily pain severity as Level 1 predictor [$X^2(7) = 32.02, p < .001$]. Only the mean level of daily PA was a significant predictor for the Level 1 intercept [coefficient = -0.69, $t(62) = -4.15, p < .001$], reflecting the fact that patients reporting more daily PA showed less daily disability. Age, gender and pain duration,

baseline pain severity and disability were dropped from the model, because they were not significant. In a last step, we included the interaction between PA instability (Level 2) and daily pain severity (Level 1) as a cross-level moderator of the Level 1 intercept and slope. Although pointing in the same direction, the model failed to reach significance [$X^2(2) = 3.43, p = .178$]. The final model (Table 3) shows that the mean level of daily PA is related to daily disability. Including trait anxiety at Level 2, showed trait anxiety to be a significant predictor [coefficient = 0.46, $t(66) = 2.28, p = .026$], similar to the mean level of daily PA [coefficient = -0.63, $t(66) = -2.88, p = .005$].

Daily cognitive complaints.

Initial analyses indicated that there was substantial variance in reported daily cognitive complaints between and within patients, and daily pain severity to be a significant Level 1 predictor (see above). In a next step, we included PA instability as a Level 2 predictor, as were control variables i.e. age, gender, pain duration, pain severity at baseline and the mean level of daily PA. The proposed model proved to be better than the model with only daily pain severity as Level 1 predictor [$X^2(6) = 18.38, p = .006$]. The mean level of daily PA [coefficient = -.48, $t(63) = -2.43, p = .018$] and baseline pain severity [coefficient = 0.435, $t(63) = 2.04, p = .046$] were significant predictors for the intercept, indicating that patients reporting higher levels of daily PA or lower levels of baseline pain severity reported less cognitive complaints in daily life. Due to the non-significant effects of age, gender and pain duration, they were dropped from the model. In a last step, we entered the interaction between PA instability (Level 2) and daily pain severity (Level 1) as a cross-level moderator of the Level 1 intercept and slope. The resulting model was not better than without the cross-level interaction term [$X^2(2) = 0.860, p > .500$]. Results of the final model are summarized in Table 3. They reveal that the mean level of daily PA is related to daily cognitive complaints. When including trait anxiety as a Level 2 variable, it proved to be a significant predictor [coefficient = 0.78, $t(65) = 3.15, p = .002$], whereas the significant predictive effect of the mean level of daily PA of $p = .028$ changed to $p = .857$.

Additional analyses including depression scores (HADS) showed that despite a positive relation to mean levels of daily pain severity, disability and cognitive complaints (correlations ranging from .35 to .43), depression did not reliably moderate the within-person associations between daily pain and either disability or cognitive complaints ($ps > .18$). PA instability remained a significant moderator of the associations between daily pain and both disability and cognitive complaints ($ps < .03$) even after controlling for depressive

symptoms. PA instability became a statistically significant moderator of the association between daily pain and disability ($p = .024$).

Table 3
Final Hierarchical Linear Models in Regard of Positive Affect Instability

Dependent variable (diary items)	Coefficient	SE	t
Daily disability			
Intercept (γ_{00})	4.485	0.18	24.963 ^{***}
PA_MSSD (γ_{01})	0.307	0.16	1.865
Mean PA (γ_{02})	-0.909	0.19	-4.884 ^{***}
Daily pain severity (γ_{10})	0.926	0.05	19.056 ^{***}
Daily pain severity x PA_MSSD (γ_{11})	0.073	0.05	1.589
Daily pain severity x Mean PA (γ_{12})	-0.044	0.05	-0.894
Daily cognitive complaints			
Intercept (γ_{00})	3.233	0.18	17.919 ^{***}
Baseline pain severity (γ_{01})	0.531	0.19	2.856 ^{**}
PA_MSSD (γ_{02})	-0.094	0.18	-0.525
Mean PA (γ_{03})	-0.456	0.20	-2.241 [*]
Daily pain severity (γ_{10})	0.286	0.06	5.092 ^{***}
Daily pain severity x PA_MSSD (γ_{11})	0.045	0.04	1.063
Daily pain severity x Mean PA (γ_{12})	-0.019	0.04	-0.430

Note. PA = positive affect, MSSD = mean square successive difference, * $p < .05$, ** $p < .01$, *** $p < .001$.

Discussion

This study investigated the role of affective instability in the daily experience of pain, disability and cognitive complaints using an end-of-day diary over 14 days. The results can be readily summarized. Firstly, the results show a significant relationship between mean levels of daily affect and pain severity and disability, confirming previously reported results about the relationship between NA, PA and pain (Affleck et al., 1994; Potter et al., 2000; Zautra et al., 1995). Secondly, NA, but not PA, instability predicted daily disability over and above the mean level of daily NA, and even after controlling for age, gender, baseline pain severity, pain chronicity and trait anxiety. Thirdly, NA instability moderated the relationship between daily pain severity and pain-related outcomes: Daily pain severity was more strongly associated with daily disability and cognitive complaints in patients with higher levels of NA instability.

This study is the first to reveal the role of NA instability in the adjustment to chronic pain. Our findings demonstrate that not only the intensity but also the time course and variability of NA is related to daily pain outcomes. As this study is the first of its kind, research will need to further corroborate our findings, and also investigate underlying processes. One explanation for current findings may be found in the type and quality of emotion regulation skills. Indeed, affective instability has previously been interpreted as reflecting a dysfunctional regulation of emotions (Carpenter & Trull, 2013; Selby et al., 2015). Although this view mostly stems from the literature on psychopathology, similar processes may play a role when chronic pain patients have to cope with the daily demands of pain (Eccleston & Crombez, 1999). It may well be that individuals who have problems with regulating NA also have difficulties to cope with pain and, thus, experience more interference by pain in daily life. This interference by pain may result in disability and cognitive complaints. If this is true, there may be a strong overlap in regulating pain and regulating NA. This may be a reasonable assumption as pain involves a sensory and an emotional experience. Also, Linton (2013) argued for commonalities in problems of regulation in chronic pain and emotional disorders. An important strategy for clinicians may then be to target NA instability. As yet we do not know how this is best accomplished. If there are deficits in particular regulation skills, interventions may include skills training, such as the Affect Regulation Training (Berking & Whitley, 2014), which is a module-based intervention to improve emotion regulation skills. If, on the other hand, there is evidence for rigid overregulation, mindfulness training (Grossman, Tiefenthaler-Gilmer, Raysz, & Kesper, 2007; Rosenzweig et al., 2010), or Acceptance and Commitment Therapy (Hayes,

Strosahl, & Wilson, 1999; McCracken, Vowles, & Eccleston, 2005; Vowles, McCracken, & O'Brien, 2011) may be promising approaches.

A remarkable finding of our study is that NA instability but not PA instability predicted pain-related outcomes, although the association between NA and PA instability was high. This pattern of results is not unusual. Previous research has shown a similar relationship between instability measures of PA and NA (Koval et al., 2013; Maciejewski, Lier, Branje, & Koot, 2015). These findings may be explained by shared method variance between instability measures of NA and PA: They are based on similar mathematical algorithms. The finding, however, that NA instability, but not PA instability, predicts pain-related outcomes is in line with previous research in psychopathology investigating affective instability in natural contexts (Houben et al., 2015; Thompson et al., 2012; Trull et al., 2008). For example, depressed patients show higher NA instability, but not PA instability, compared to healthy volunteers (Thompson et al., 2012).

At first sight, our findings are not in line with the predictions of the dynamic model of affect (Davis, Zautra, & Smith, 2004). This model states that NA and PA are relatively independent during non-stressful periods, whereas they merge to one bipolar dimension under stressful or painful conditions. From this perspective, one may have expected a similar pattern of results for NA and PA instability. Nevertheless, although a relatively strong relationship was found between the mean levels of NA and PA instability, only NA instability was found to moderate the relationship between pain severity and disability or cognitive complaints. One explanation for not finding such a pattern for PA may relate to the fact that there are fewer fluctuations in PA. This is, however, not the case in our study: The level of instability was similar for NA and PA. Another explanation may be that our measure of affective instability captures a unique parameter that is not addressed by the dynamic model of affect. In line with this argument are the findings that NA instability and PA instability are not related to the mean levels of daily NA and PA.

Our findings corroborate the notion that affective instability does not measure the intensity of emotional experiences, but rather their temporal variability. Recently, the examination of the dynamics of affect has increased in order to improve our understanding of psychological maladjustment and psychopathology (Ebner-Priemer et al., 2009; Houben et al., 2015; Koval et al., 2013; Thompson et al., 2012; Trull et al., 2008). Within this emerging field, emotional responding reflects a dynamic process that takes place in response to changing contextual demands. Thus, emotional responding that is adaptive, is believed to be flexible, rather than rigid or stereotypical (Thompson, 1994). In case of

affective instability, an individual tends to show frequent mood shifts with a transient, fluctuating course (Trull et al., 2008). The distinction between intensity and temporal variability completes the picture of dynamically fluctuating emotions in daily life and demonstrates the importance of broadening the perspective in order to better understand daily emotional experiences. In addition, we found that NA instability, but not PA instability, is related to baseline pain severity and baseline disability. This finding expands upon previous research relating affective instability to mental disorders (Ebner-Priemer & Trull, 2009; Thompson et al., 2012; Trull et al., 2008) and furthers our understanding of daily functioning, i.e. disability and cognitive complaints, of chronic pain patients.

In consideration of affective instability as a reflection of dysfunctional emotion regulation (Carpenter & Trull, 2013; Selby et al., 2015), we also investigated the relationship between affective instability and measures of emotional functioning, i.e. depressive mood and trait anxiety. We only found a negative association between NA instability and trait anxiety, which is in line with the model of Hofmann and colleagues (2012), which links dysregulation of NA to anxiety disorders. Nevertheless, it is important to note that including trait anxiety in our models did not affect the effects of NA instability. Although not the focus of the current study, one may further have expected a relationship between depressive mood and affective instability. Hamilton and colleagues (2005) suggest that affect regulation may influence psychological functioning in patients with chronic pain. They further assume that the reason for the relationship between affect regulation and adjustment to pain may be due to the fact that these patients are more vulnerable to depression. In the current study, however, we neither found a relationship between affect dysregulation (i.e. affective instability) and depression, nor did the inclusion of depressive symptoms in our models change the predictive and moderating role of NA instability for daily disability and cognitive complaints. More research is needed in this regard. Nevertheless, in line with numerous studies reporting high levels of emotional distress in chronic pain patients (Bair, Robinson, Katon, & Kroenke, 2003; McCracken, Faber, & Janeck, 1998; McCracken & Iverson, 2001), we found a strong association between baseline depressive mood, anxiety and pain severity.

This study has some limitations. Firstly, we did not include a control group and therefore we cannot compare our findings with a non-clinical sample. Secondly, we investigated a heterogeneous group of chronic pain conditions so that findings cannot be ascribed to specific conditions. Thirdly, the analyses are based on end-of-day diaries, assessing fluctuations in affect over days. Our results may be further corroborated by also

studying within-day emotional fluctuations. Fourthly, neither did we have data on putative mediating variables such as emotion regulation strategies used, or indicators of failed regulation such as perseverative cognitions, i.e. worry and rumination (Eccleston & Crombez, 2007), nor did we include other variables known to influence levels of pain-related disability (e.g. pain-related fear) in the analysis. Fifthly, the effect of affective instability may be moderated by other variables. We found such an effect for age, albeit in posthoc-analyses. This finding suggests that emotional experiences may grow more stable with age (Carstensen et al., 2011). Sixthly, our measure of affective instability captures the temporal fluctuations of emotions over 14 days. Nevertheless, it remains unclear which individual or contextual factors may underlie, or be related to, this process. Seventhly, there is a strong tradition to psychometrically validate questionnaires. The validation of diary items is still in its infancy. Although items in the current study were carefully selected and piloted for understanding and feasibility in patients with chronic pain, validation of the diary items should be further developed (e.g. by using cognitive interviewing (Collins, 2003)). Eighthly, our analyses are best considered as cross-sectional. We did not perform time lag analyses. Therefore, one should be careful in inferring causality between variables. Finally, we only focus upon affective instability as a marker of dysfunctional regulation. Other measures are possible, such as psychophysiological measures of emotion regulation capacity (i.e. heart rate variability), and their use may further our understanding of their role in the context of chronic pain.

Conflict of interest statement

There are no conflicts of interest that may arise as a result of this research.

Acknowledgements

The authors want to thank Emelien Lauwerier and Lies Notebaert for their help in the data collection. Preparation of this paper was partly supported by the University of Luxembourg, Grant BOF/GOA2006/001 of Ghent University and FWO project G.017807.

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2

CHAPTER 2

**Altered Regulation of Negative Affect in
Fibromyalgia: A Diary Study¹****Abstract**

Fibromyalgia (FM) is characterized by widespread musculoskeletal pain accompanied by other physiological, cognitive and emotional challenges. Successful adaptation to FM may rely on a person's emotion regulation capacity. In this study, we examined two indices of emotion regulation capacity – affective instability, involving frequent and large fluctuations in self-reported affect, and resting heart rate variability (HRV) – among 46 FM patients ($M_{\text{age}} = 45.4$ years; 39 females) and 46 healthy controls ($M_{\text{age}} = 44.9$ years; 37 females). During a baseline phase, patients completed standard questionnaires and their heart rate was monitored under resting conditions to derive HRV. Finally, participants filled out an electronic end-of-day diary over 14 consecutive days assessing daily levels of pain severity, disability, distractibility, and negative affect (NA) and positive affect (PA). Affective instability was operationalized as the mean square of successive differences (MSSD) in daily mood (separately for NA and PA). Consistent with previous research, HRV and NA instability were inversely related. Furthermore, relative to controls, FM patients displayed increased NA instability and showed stronger associations between mean daily NA and NA instability. These findings confirm that HRV predicts dysfunctional regulation of NA and suggest that NA instability plays an important role in the adaptation to FM. These results may have clinical implications for therapeutic programs for FM.

¹ Rost, S., Van Ryckeghem, D., Koval, P., Sütterlin, S., Vögele, C., & Crombez, G. (in preparation). Altered regulation of negative affect in fibromyalgia: A diary study.

Introduction

Fibromyalgia (FM) is characterized by widespread musculoskeletal pain of uncertain aetiology and is accompanied by symptoms such as fatigue, sleep disorders and mood disturbances (Wolfe et al., 1990). FM patients are confronted with pronounced challenges at physiological, cognitive and emotional levels. Successful adaptation to this demanding condition requires strong capacity to self-regulate or exert control over one's thoughts, emotions and behaviour (Baumeister, 1998; Carver & Scheier, 1998). In particular, the regulation of emotions has been suggested as a key factor in the adjustment to chronic pain (Hamilton, Zautra, & Reich, 2005). Experiencing unusually large and/or frequent changes in affect, labelled affective instability, may reflect problems in regulating affect (Carpenter & Trull, 2013; Selby et al., 2015). Higher instability, particularly of negative affect (NA), has been found to be associated with lower psychological well-being and various forms of psychopathology, such as borderline personality disorder, depression and eating disorders (Ebner-Priemer, Eid, Kleindienst, Stabenow, & Trull, 2009; Houben, Noortgate, & Kuppens, 2015; Thompson et al., 2012; Trull et al., 2008).

Based on the conceptualization of pain as a sensory and emotional experience, one may expect affective instability to be related to dysfunctional regulation of pain, i.e. interference with daily activities and cognitive processes (Attridge, Crombez, Van Ryckeghem, Keogh, & Eccleston, 2015; Eccleston & Crombez, 1999; Selby et al., 2015). We investigated the role of affective instability in chronic pain patients and found that NA instability predicted greater overall daily disability as well as stronger associations between daily pain level and daily disability and cognitive complaints (Rost et al., 2016). These findings suggest that chronic pain sufferers with reduced emotion regulation capacity (indexed by higher NA instability) have poorer functioning in general, and show greater declines in functioning on days when they experience higher than usual levels of pain.

However, because our previous study included only individuals with chronic pain, it remains unclear whether FM patients show higher levels of affective instability than healthy controls. Furthermore, other indices of emotion regulation capacity have not been examined in chronic pain patients. A widely studied index of adaptive emotion regulation is resting heart rate variability (HRV; Appelhans & Luecken, 2006). According to the model of neurovisceral integration (Thayer & Lane, 2009), HRV is considered a proxy for prefrontal cortical inhibitory capacity and has been used as a general index for an individual's capacity to self-regulate (Segerstrom & Nes, 2007). It reflects the degree to

which cardiac activity can be modulated to meet changing situational demands and thus supports behavioural flexibility and adaptive emotion regulation (Appelhans & Luecken, 2006). Previous research has linked higher HRV with lower NA instability in daily life in a non-clinical student sample (Koval et al., 2013), suggesting that HRV and affective instability may be complementary (physiological and experiential) indices of emotion regulation capacity.

The current study used an end-of-day diary methodology to assess fluctuations in pain and affect among FM patients and healthy controls over 14 consecutive days. This combination of a dynamic measure of daily experience and the self-regulatory biomarker of HRV aims to expand knowledge about the role of emotion regulation in FM, and may guide novel therapeutic interventions for FM and other chronic pain conditions.

This study aimed to (1) investigate the link between HRV and affective instability in FM patients and healthy controls and (2) replicate previous findings concerning the role of affective instability in daily pain outcomes in FM patients, i.e. the predictive power of NA instability for daily disability and moderating effect of NA instability for the association between daily pain severity and daily pain outcomes. In particular, we predicted that (1a) NA and positive affect (PA) instability would be higher in FM patients than in healthy controls, (1b) NA and PA instability would be negatively associated with HRV in both groups, (2a) NA and PA instability would be related pain outcomes (disability and distractibility), and (2b) NA and PA instability would moderate the relationship between daily pain severity on the one hand and daily disability and distractibility on the other hand.

Methods

Participants

Participants between the ages of 18 and 65 years were recruited for participation in a large project (see protocol ASEF-I; <http://hdl.handle.net/1854/LU-5686902>) between January 2014 and March 2014. Individuals were eligible to participate if they were either diagnosed with FM and fulfilled the ACR-90 criteria for FM (FM group), or did not report a current pain problem (control group). Additional inclusion criteria were: (1) sufficient knowledge of the Dutch language; (2) absence of neurological conditions. Individuals were excluded from participation if they were unable to use both index fingers, reported abnormal sensations on one of the stimulated locations or if their eyesight was not normal

or corrected-to-normal (e.g., by glasses). The latter three criteria were exclusion criteria related to a task that was analysed for the current report (see ASEF-I). FM patients were recruited via the Multidisciplinary Pain Clinic of Ghent University Hospital (Belgium). They were informed about the study with posters in the waiting room of the hospital. Patients who were interested in taking part left their contact details and were screened for eligibility. Healthy controls were recruited using advertisements in a local newspaper, flyers and the university website. Individuals who volunteered were contacted and screened for eligibility. Both groups were matched for age, sex and educational level. A total of 98 individuals took part in the study: 49 persons with FM and 49 healthy controls.

As the study was part of an extended protocol, we only report the procedure relevant for the current research question. Before coming to the lab, participants completed several standard questionnaires, including the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995), the Pain Disability Index (PDI; Pollard, 1984) and the Multidimensional Pain Inventory (MPI; Kerns, Turk, & Rudy, 1985; Lousberg et al., 1999) online (participants who were unable to complete the questionnaires online received a paper version). Upon arriving at the lab, participants indicated whether they had consumed coffee within the last two hours (exclusion criterion). The researcher then checked the ACR-criteria (Wolfe et al., 1990) with the patients to confirm the FM diagnosis. Next, heart rate was measured for 5 minutes at rest to derive HRV. Finally, participants received instructions for the 14 days' diary protocol. The study design was approved by the Ethics Review Panel of the University of Luxembourg and the Medical Ethics Committee of the University Hospital Ghent. Participants gave written informed consent and were reimbursed 35€ for participating.

Questionnaires

Depressive mood, anxiety and stress was assessed using the DASS (Lovibond & Lovibond, 1995). Each sub-scale contains 14 items on which participants rate how much they have experienced each state (e.g. "I found it hard to wind down", "I felt I was pretty worthless") over the past week using a scale from 0 (*did not apply to me at all*) to 3 (*applied to me very much, or most of the time*). In the present study, internal consistencies of the sub-scales were excellent, (depression: $\alpha = .97$; anxiety: $\alpha = .91$; stress: $\alpha = .95$).

Pain-related disability was measured with the PDI (Pollard, 1984). Participants indicate the degree of disability experienced in seven life domains (e.g. family and occupation) using a scale from 0 (*no disability*) to 10 (*total disability*). They are asked to respond to each category by indicating the overall impact of pain in their life, not just when pain is at its worst. Scores range between 0 and 70. Cronbach's alpha was .82.

Pain severity was assessed with the pain severity subscale of the MPI (Kerns et al., 1985; Lousberg et al., 1999). Part I of the MPI consists of 5 subscales assessing the impact of pain (i.e. pain severity, pain interference, social support, perceived life control and affective distress) on a 7-point Likert scale. Pain severity was assessed with two items (i.e., "Rate the level of your pain at the present moment" and "On average, how severe has your pain been during the last week?"). The third item ("How much suffering do you experience because of your pain?") was not taken into account as its content relates to suffering rather than pain severity (see Parenteau & Haythornthwaite, 2011). The MPI has shown good reliability and validity (Rudy, 1989). In the present study, Cronbach's alpha of the MPI severity subscale was .84.

Heart rate variability

During heart rate monitoring under resting conditions, participants sat in individual cubicles and were instructed to sit quietly and relax. Interbeat intervals were assessed based on electrocardiographic recordings for 5 minutes at a sampling rate of 1000 Hz using a Polar watch RS800CX (Polar Electro Oy, Kempele, Finland).

End-of-day diary assessment

Participants were asked to complete an online diary at the end of each day for 2 consecutive weeks. They were reminded to do so each evening via a text message. The diary took approximately 5 minutes to complete and included 30 items in total. Here, we describe only the items which are of relevance to the current report. Items were developed iteratively by a group of pain researchers and piloted for feasibility in patients with chronic pain.

Daily affect: Participants rated how much they had experienced several affective states "that day" on a scale from 0 (*do not agree at all*) to 6 (*totally agree*). We used six adjectives to measure PA: glad [blij], enthusiastic [enthousiast], happy [gelukkig], relaxed

[ontspannen], strong [sterk] and proud [trots]; and 10 adjectives to measure NA: afraid [bang], irritated [geirriteerd], angry [kwaad], powerless [machteloos], sad [triest], frustrated [gefrustreerd], dejected [neerslachtig], infuriated [woedend], hopeless [hopeloos] and nervous [zenuwachtig]. Items were derived from a validation study investigating the representation of emotion terms in a general population and a previous study investigating affective instability in chronic pain patients (Rost et al., 2016; Veirman & Fontaine, 2015). PA and NA scales were calculated by averaging PA and NA items, respectively. We calculated within-person reliability of the PA and NA scales using the Generalizability theory approach described by Bolger and Laurenceau (2013). Estimates of within-person reliability were .87 for PA and .86 for NA, indicating that both scales assessed within-person changes reliably.

Daily pain severity was assessed using the item: “On average, how severe has your pain been today?” rated on a scale from 0 (*no pain*) to 10 (*worst imaginable pain*).

Daily pain disability was assessed by the item “To what extent did pain interfere with your planned activities today?” rated on a scale from 0 (*not at all*) to 10 (*very much*). This item is similar to the items in the PDI (Pollard, 1984), but asks more generally about the degree to which pain prevents patients from their planned daily activities.

Daily distractibility was assessed by the item “To what extent were you distracted today?”, rated on a scale from 0 (*not at all*) to 10 (*very much*).

Affective instability

Affective instability refers to the experience of frequent and large successive changes in feelings over time (Jahng, Wood, & Trull, 2008), and is typically measured as the mean square of successive differences (Jahng et al., 2008). This index of instability reflects the extent to which consecutively assessed affective states differ from each other and therefore incorporates both variability (i.e., average magnitude of affective changes) and temporal dependency (i.e., average frequency of affective changes). In our study, this measure reflects the average frequency and size of day-to-day fluctuations in affect over 14 days. The squared difference between successive observations at occasions $i + 1$ and i for a time series of n measurement occasions is given by:

$$MSSD = \frac{1}{N - 1} \sum_{i=1}^{N-1} (x_{i+1} - x_i)^2$$

This index was calculated separately for PA and NA (see Houben et al., 2015).

Data handling and reduction

To calculate HRV-indices, sequential interbeat intervals were downloaded using the software Polar Pro Trainer 5. All signals were visually inspected for artefacts. HRV analysis was performed using ARTiiFACT (Kaufmann, Sütterlin, Schulz, & Vögele, 2011). Firstly, measurement artefacts were identified by applying a distribution-related threshold criterion. Erroneous beats were deleted and substituted by cubic spline interpolation of neighbouring intervals. Time domain measures were directly calculated from RR-interval series. Spectral analysis of the RR-interval series was carried out using Fast Fourier Transformation. Following the recommendations of the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996), we defined the high frequency band (HF) as 0.14 to 0.4 Hz and used the following time and frequency HRV parameters for statistical analyses: root mean square of successive differences (RMSSD) and the absolute power in the HF band (HFabs). We focused on those parameters because they reflect vagal control over heart rate (Task Force, 1996), which is considered directly relevant to an individual's emotion regulation capacity (Appelhans & Luecken, 2006). The criterion for outliers in HRV measures was defined as deviations of more than 3 *SD* from the sample mean (cf. Koval et al., 2013). After correcting for outliers, HFabs was log transformed to adjust for skewness of the distribution (lnHFabs).

As recommended by (Jahng et al., 2008), we focused on the time series of squared successive differences (SSD) to model affective instability. The SSDs were calculated separately for PA and NA and log transformed to adjust for skewness of the distribution. Skewness values for SSDs before log-transformation were 4.6 (PA) and 4.1 (NA), which decreased to -0.78 (PA) and -0.46 (NA) after log transformation.

One participant (1 control) was excluded from the final analyses due to equipment failure. Further, two participants (1 FM, 1 control) were excluded because of outliers in HRV. To ensure that affective instability could be modelled reliably, participants who completed fewer than 7 out of 14 days of diary entries were excluded from analyses. Three participants (2 FM, 1 controls) were excluded because of fewer than seven diary entries. The final sample consisted of 46 FM patients and 46 healthy controls.

Statistical models

Descriptive statistics were performed using SPSS statistical software, version 24.0 for Windows (SPSS Inc., Chicago/IL). To account for the nested structure of the data (multiple observations nested within individuals) we conducted multilevel regression using HLM (Version 7.01; Scientific Software International, Skokie/IL. Full maximum likelihood estimation was used for all models. Models included random intercepts and random slopes.

Affective instability as a function of heart rate variability in fibromyalgia patients and healthy controls.

We modelled the (log transformed) within-person SSD ($\ln\text{SSD}_{\text{affect}_{ij}}$) using a multilevel random intercept model in which the Level 1 random intercept (β_{0j}) was predicted by vagally mediated HRV and group (dummy coded 0 = control; 1 = FM) at Level 2. We also controlled for the mean of daily of daily PA or NA ratings (at Level 2) to ensure that our findings were not driven by differences in prevailing affect levels (Ebner-Priemer et al., 2009; Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007). Vagally mediated HRV and mean affect were standardized to facilitate interpretation (Nezlek, 2012) and group was entered un-centred. PA and NA instability were modelled in separate analyses and the equations, including the subscripts i representing days and j representing persons, were as follows:

$$\text{Level 1: } \ln(\text{SSD}_{\text{affect}_{ij}}) = \beta_{0j} + r_{ij}$$

$$\text{Level 2: } \beta_{0j} = \gamma_{00} + \gamma_{01} * \text{group}_j + \gamma_{02} * \text{HRV}_j + \gamma_{03} * \text{mean affect}_j + \mu_{0j}$$

Association between daily pain severity and daily pain outcomes in fibromyalgia patients.

We modelled daily pain outcomes (disability and distractibility) using a multilevel random intercept and slope model. We ran analyses only in FM patients as the variance of the daily data of healthy controls was too restricted, i.e. ratings were ≤ 2 on scales from 0 to 10 in 85% for daily pain severity, 90% for daily disability and 64% for daily distractibility.

Level 1 variables consisted of the daily diary measures of pain intensity, pain disability, and distractibility. All Level 1 variables were continuous and entered person-mean centred (Enders & Tofighi, 2007). Level 2 variables consisted of age, gender and

baseline questionnaire measures of pain duration, and baseline disability. Gender was dummy coded (0 = female; 1 = male) and entered un-centred, while all continuous Level 2 variables were standardized to facilitate interpretation (Nezlek, 2012). We followed a model building procedure in our analyses (Raudenbush & Bryk, 2002). To maximize stability and reliability of the findings, we excluded control variables from further steps in model building if their effects proved to be non-significant (Kreft & de Leeuw, 1998). We included affective instability as a Level 2 variable and, again, controlled for the mean Level of daily affect. The moderating role of affective instability was investigated in the last step of model building:

$$\begin{aligned} \text{Level 1:} \quad & \text{daily pain outcome} = \beta_{0j} + \beta_{1j} * \text{daily pain severity}_{ij} + r_{ij} \\ \text{Level 2:} \quad & \beta_{0j} = \gamma_{00} + \gamma_{01} * \text{mean daily affect}_j + \gamma_{02} * \text{affect instability}_j + \mu_{0j} \\ & \beta_{1j} = \gamma_{10} + \gamma_{11} * \text{mean daily affect}_j + \gamma_{12} * \text{affect instability}_j + \mu_{1j} \end{aligned}$$

Results

Participant characteristics

Table 4 displays sample demographics and descriptive statistics for all measures. There were no significant group differences in age, gender and educational level (all $ps > .441$). For FM patients, the mean pain duration was 189.92 months ($SD = 117.85$). Mean pain severity was 3.62 as measured by the MPI ($SD = 1.10$) and mean disability was 41.98 on the PDI ($SD = 10.55$); these mean levels are comparable with previous studies of chronic pain patients ($M_{MPI} = 4.2$, $SD_{MPI} = 1.1$, $M_{PDI} = 44.6$, $SD_{PDI} = 13.4$; Chibnall & Tait, 1994; Nicholas, Asghari, & Blyth, 2008). FM patients had significantly higher scores (all $ps < .001$) on the DASS (Lovibond & Lovibond, 1995) compared with controls.

Further, relative to controls, FM patients showed lower mean levels of daily PA ($p < .001$), but did not differ significantly in their mean levels of NA ($p = .204$). Using Pillai's trace, there was a significant effect of HRV for Group [$V = 0.09$, $F(2,89) = 4.23$, $p = .018$], indicating significantly lower HRV in FM compared with controls. Table 4 gives an overview of all measures, including the follow-up ANOVAS for HRV-indices.

Table 4*Descriptive Statistics by Participant Group*

Variable	Group		Difference test
	FM (<i>n</i> = 46)	control (<i>n</i> = 46)	
Age (<i>M</i> , <i>SD</i>)	45.4 (9.2)	44.9 (12.2)	$t(84)^a = .14, p = .885, d = 0.05$
Gender (<i>n</i> women)	39	37	$\chi^2(1) = .303, p = .582, w = 0.06$
Educational level			$\chi^2(2) = 1.64, p = .441, w = 0.13$
College/University	41.3%	54.3%	
Secondary school	54.3%	41.3%	
Primary school	4.3%	4.3%	
DASS – D (<i>M</i> , <i>SD</i>)	13.02 (10.9)	5.59 (6.4)	$t(72)^a = 3.98, p < .001, d = 0.83$
DASS – A (<i>M</i> , <i>SD</i>)	11.09 (7.5)	2.8 (3.6)	$t(64)^a = 6.77, p < .001, d = 1.39$
DASS – S (<i>M</i> , <i>SD</i>)	15.04 (7.6)	7.91 (7.4)	$t(90) = 4.53, p < .001, d = 0.95$
Mean daily PA (<i>M</i> , <i>SD</i>)	2.59 (1.0)	3.4 (1.0)	$t(90) = -3.75, p < .001, d = 0.81$
Mean daily NA (<i>M</i> , <i>SD</i>)	1.42 (0.7)	1.19 (0.9)	$t(90) = 1.28, p = .204, d = 0.29$
HRV-indices			
RMSSD (<i>M</i> , <i>SD</i>)	19.12 (10.9)	26.36 (14.0)	$F(1,90) = 7.67, p = .007, \eta^2 = .08$
lnHFabs (<i>M</i> , <i>SD</i>)	4.74 (1.3)	5.24 (1.2)	$F(1,90) = 3.69, p = .058, \eta^2 = .04$

Note. FM = fibromyalgia, DASS – D = depression scale from the Depression, Anxiety and Stress Scales, DASS – A = anxiety scale from the Depression, Anxiety and Stress Scales, DASS – S = stress scale from the Depression, Anxiety and Stress Scales, PA = positive affect, NA = negative affect, HRV = heart rate variability, RMSSD = root mean square of successive differences, lnHFabs = log transformed absolute power in the high frequency band, ^a equal variances not assumed.

Multilevel analyses

Affect instability in fibromyalgia patients and healthy controls.

Firstly, we ran null models without predictors to obtain estimates of average levels of affective instability across the sample. The average levels of instability were [coefficient = -2.02, *SE* = 0.14, $p < .001$] for NA and [coefficient = -1.51, *SE* = 0.12, $p < .001$.] for PA. The random effects estimates from these models indicated significant between-person variability in instability for NA ($SD = 1.18, p < .001$) and PA ($SD = 0.91, p < .001$).

We then examined how group and HRV were related to NA instability while controlling for the mean level of NA (Models 1 and 2). As predicted, group was positively associated with NA instability, meaning that FM patients showed significantly higher NA instability. There was also evidence for a negative association between NA instability and RMSSD, linking lower HRV to higher NA instability, replicating previous findings (Koval et al., 2013). The results of Models 1 and 2 are shown in Table 5. We also investigated the relationship between group, HRV and PA instability, while controlling for the mean level of PA (Models 3 and 4). As shown in Table 5, neither group nor HRV-indices were related to PA instability.

Table 5

Final Hierarchical Linear Models Explaining Affective Instability

		Coefficient	SE	<i>p</i>
NA instability				
Model 1	Intercept (γ_{00})	-2.32	.20	< .001
	Group (γ_{01})	0.62	.26	.021
	Mean daily NA (γ_{02})	0.65	.14	< .001
	RMSSD (γ_{03})	-0.26	.12	.034
Model 2	Intercept (γ_{00})	-2.37	.20	< .001
	Group (γ_{01})	0.71	.25	.006
	Mean daily NA (γ_{02})	0.65	.15	< .001
	lnHFabs (γ_{03})	-0.15	.12	.214
PA instability				
Model 3	Intercept (γ_{00})	-1.52	.16	< .001
	Group (γ_{01})	0.02	.25	.946
	Mean daily PA (γ_{02})	-0.33	.11	.005
	RMSSD (γ_{03})	-0.16	.11	.160
Model 4	Intercept (γ_{00})	-1.54	.16	< .001
	Group (γ_{01})	0.05	.24	.826
	Mean daily PA (γ_{02})	-0.32	.11	.006
	lnHFabs (γ_{03})	-0.16	.11	.167

Note. HRV = heart rate variability, NA = negative affect, PA = positive affect, RMSSD = root mean square of successive differences, lnHFabs = log transformed absolute power in the high frequency band.

Daily disability and affective instability in fibromyalgia patients.

Initial analyses indicated that there was substantial variance in reported daily disability between (38%) and within (62%) patients. Firstly, we included daily pain severity as a Level 1 predictor. The model proved to be better [$X^2(3) = 261.21, p < .001$] than the model without any predictor.

In a second step, we added NA instability and additional Level 2 measures, i.e. age, gender, pain duration, baseline pain severity, baseline pain disability and the mean level of daily NA, as control variables. The proposed model proved to be better than the model with only daily pain severity as a Level 1 predictor [$X^2(7) = 25.35, p < .001$]. Only age was a significant predictor for the intercept of daily disability [coefficient = 0.48, $t(38) = 2.57, p = .014$], indicating that patients of older age reported more daily disability. We, therefore, excluded gender, pain duration, baseline pain severity and pain disability from this model. Third, we included the interaction between NA instability (Level 2) and daily pain severity (Level 1) as a cross-level moderator of the Level 1 intercept and slope. The resulting model was not better than without the cross-level interaction term [$X^2(2) = 0.97, p > .500$]. The Level 2 variables were, however, significant predictors for the Level 1 intercept: age [coefficient = 0.49, $t(42) = 2.20, p = .033$], mean level of daily NA [coefficient = 0.53, $t(42) = 2.09, p = .043$] and NA instability [coefficient = 0.41, $t(42) = 2.03, p = .048$], indicating that daily disability increased with higher age and higher mean levels of daily NA and NA instability.

Some further analyses were run to test the robustness of our findings. When including depression as Level 2 predictor, age showed to be only marginally significant [coefficient = 0.50, $t(41) = 1.88, p = .067$]. When including anxiety as Level 2 predictor, the same was the case for age [coefficient = 0.41, $t(41) = 1.89, p = .066$], and the mean level of daily NA was no significant predictor anymore [coefficient = 0.39, $t(41) = 1.37, p = .179$].

Similarly, in a second step, we added PA instability as a Level 2 predictor, as we did with control variables i.e. age, gender, pain duration, pain severity at baseline and the mean level of daily PA. The proposed model proved to be better than the model with only daily pain severity as Level 1 predictor [$X^2(7) = 22.97, p = .002$]. Only age [coefficient = 0.42, $t(38) = 2.15, p = .038$] was a significant predictor, and pain severity at baseline [coefficient = 0.58, $t(38) = 2.01, p = .052$] was a marginally significant predictor for the intercept, indicating that older patients reported more disability in daily life. As gender, pain duration and baseline disability were not significantly related with daily disability, they were dropped from the model. In a last step, we entered the cross-level interaction

between PA instability (Level 2) and daily pain severity (Level 1). The resulting model was not better than without the cross-level interaction term [$X^2(2) = 1.19, p > .500$], but showed that age [coefficient = 0.44, $t(41) = 2.23, p = .031$] and baseline pain severity [coefficient = 0.68, $t(41) = 3.27, p = .002$] remained significant predictors for daily disability. Results of the final models in regard to daily disability are summarized in Table 6.

When including depression at Level 2, there were no significant changes for the predictive values of the Level 2 predictors. When we included anxiety at Level 2, the predictive power of age changed from significant to marginally significant [coefficient = 0.37, $t(40) = 1.76, p = .086$].

Table 6

Final Hierarchical Linear Models Explaining Daily Disability in Fibromyalgia Patients

Daily disability	Coefficient	SE	p
Final model NA			
Intercept (γ_{00})	4.75	.23	< .001
Mean daily NA (γ_{01})	0.53	.25	.043
NA instability (γ_{02})	0.41	.20	.048
Age (γ_{03})	0.49	.22	.033
Daily pain severity (γ_{10})	0.91	.06	< .001
Daily pain severity x Mean daily NA (γ_{11})	-0.01	.06	.888
Daily pain severity x NA instability (γ_{12})	0.06	.04	.211
Final model PA			
Intercept (γ_{00})	4.78	.17	< .001
Mean daily PA (γ_{01})	-0.25	.25	.338
PA instability (γ_{02})	0.01	.15	.941
Age (γ_{03})	0.44	.20	.031
Baseline pain severity (γ_{04})	0.68	.21	.002
Daily pain severity (γ_{10})	0.88	.07	< .001
Daily pain severity x Mean daily PA (γ_{11})	-0.06	.06	.325
Daily pain severity x PA instability (γ_{12})	0.03	.05	.533

Note. NA = negative affect, PA = positive affect.

Daily distractibility and affective instability in fibromyalgia patients.

Initial analyses indicated that there was substantial variance in reported daily distractibility both between (32%) and within patients (68%). In a first step, we included daily pain severity as Level 1 predictor. This model proved to be better than without Level 1 predictor [$X^2(3) = 74.95, p < .001$]. In a second step, we included NA instability as a Level 2 predictor, as well as age, gender, pain duration, pain severity at baseline and the mean level of daily NA as control variables. The proposed model only showed a trend towards significance [$X^2(6) = 10.74, p = .096$]. Yet, gender [coefficient = -1.02, $t(39) = -2.25, p = .030$] and the main level of daily NA [coefficient = 0.58, $t(39) = 2.32, p = .026$] were significant predictors, indicating that male patients and patients reporting less daily NA experienced less distractibility in daily life. Due to insignificant effects of age, pain duration and pain severity at baseline, they were dropped from the model. In a last step, we entered the interaction between NA instability (Level 2) and daily pain severity (Level 1) as cross-level moderator into the model. The resulting model was not better than without the cross-level interaction [$X^2(2) = 1.07, p > .500$], but revealed mean daily NA as a significant predictor [coefficient = 0.68, $t(42) = 2.54, p = .015$], as well as gender as a marginally significant predictor [coefficient = -0.76, $t(42) = -1.80, p = .079$] of daily distractibility in FM patients. The results of this model are illustrated in Table 7.

Similarly, in a second step, we included PA instability as a Level 2 predictor, as we did with control variables i.e. age, gender, pain duration, pain severity at baseline and the mean level of daily PA. The proposed model proved to be better than the model with only daily pain severity as Level 1 predictor [$X^2(6) = 19.40, p = .004$]. Gender [coefficient = -1.30, $t(39) = -2.59, p = .014$] and the mean level of daily PA [coefficient = -0.88, $t(39) = -4.23, p < .001$] were significant predictors for the intercept, indicating that male patients and patients reporting higher levels of daily PA experienced less distractibility in daily life. As age, pain duration and baseline pain severity did not significantly contribute to the model, they were dropped. In a last step, we entered the cross-level interaction between PA instability (Level 2) and daily pain severity (Level 1) as a predictor. The resulting model was not better than without the cross-level interaction term [$X^2(2) = 0.76, p > .500$], but revealed that gender and the mean level of daily PA are related to daily distractibility. Results of this final model are summarized in Table 7.

Including depression and anxiety at Level 2 did not change the respective predictive power of the Level 2 variables in any model.

Table 7*Final Hierarchical Linear Models Explaining Daily Distractibility in Fibromyalgia Patients*

Daily distractibility	Coefficient	SE	p
Final model NA			
Intercept (γ_{00})	3.92	.26	< .001
Mean daily NA (γ_{01})	0.67	.27	.015
NA instability (γ_{02})	-0.13	.25	.608
Gender (γ_{03})	-0.76	.42	.079
Daily pain severity (γ_{10})	0.45	.10	< .001
Daily pain severity x Mean daily NA (γ_{11})	0.01	.14	.909
Daily pain severity x NA instability (γ_{12})	0.10	.08	.221
Final model PA			
Intercept (γ_{00})	5.21	.67	< .001
Gender (γ_{01})	-1.17	.57	.046
Mean daily PA (γ_{02})	-0.79	.20	< .001
PA instability (γ_{03})	-0.00	.16	.991
Daily pain severity (γ_{10})	.047	.10	< .001
Daily pain severity x Mean daily PA (γ_{11})	-0.03	.10	.760
Daily pain severity x PA instability (γ_{12})	0.07	.05	.197

Note. NA = negative affect, PA = positive affect.

Discussion

The aims of the current study were threefold. One part of the analyses (1) investigated group differences in affective instability between FM patients and healthy controls and (2) examined whether there was a link between HRV and affective instability. The other part of analyses aimed to (3) replicate previous findings indicating the predictive power of NA instability for daily disability and its moderating effect on the association between daily pain severity and daily pain outcomes in FM patients using an end-of-day diary over 14 days. Firstly, as predicted, FM patients showed significantly higher the NA instability than healthy controls. Secondly, also in line with predictions, HRV (RMSSD) was negatively associated with NA instability, replicating previous research (Koval et al., 2013). Thirdly, only NA instability predicted daily disability. Fourthly and contrary to predictions, affective instability did not moderate the association between daily pain severity and pain-related outcomes, i.e. disability and distractibility.

The first part of the study was dedicated to expanding research on the role of affective instability in clinical populations, specifically in the domain of chronic pain. The current findings show that FM patients report higher levels of NA instability than healthy controls. This is in line with previous studies demonstrating the relation between NA instability and psychopathology (Thompson et al., 2012; Trull et al., 2008), psychological well-being (Houben et al., 2015) and the adjustment to chronic pain (Rost et al., 2016). It is not surprising that FM patients report higher levels of NA instability, as affective instability refers to emotions and their (unsuccessful) regulation (Hollenstein, 2015; Kappas, 2011), and has been interpreted as reflecting dysregulated emotion (Carpenter & Trull, 2013; Selby et al., 2015). Hamilton and colleagues (2005) suggested that individual differences related to emotional processing and specifically emotion regulation may be a key factor in determining how people manage their pain. It may well be that persistent pain and emotion dysregulation share similar underlying mechanisms (Linton, 2013). For example, pain catastrophizing, as a form of negative repetitive thinking, may operate as a transdiagnostic process, i.e. serve as a driver for emotional and pain problems (Flink, Boersma, & Linton, 2013; Linton, 2013). The function of pain catastrophizing is to downregulate NA in stressful situations such as persistent pain or emotional distress (Flink et al., 2013). When pain catastrophizing occurs in other contexts and spins out of control, however, it becomes a form of ineffective problem solving that drives the development of emotional and physical problems (Linton, 2013). Indeed, FM patients showed higher levels of pain catastrophizing than healthy controls.

The assumption that affective instability reflects dysregulated emotion is further corroborated by the association between HRV (RMSSD) and NA instability. Vagally mediated HRV has previously been shown to predict lower levels of NA instability in a healthy sample (Koval et al., 2013). HRV reflects the activity of a network of neural structures, which is dynamically organized and allows for behavioural adaptability, thus, indexing regulated emotional responding (Appelhans & Luecken, 2006). The capacity to self-regulate emotions, as indexed by HRV, plays an important role in mental health (Gross & Muñoz, 1995) and can be seen as an indicator of how well people are actually capable of regulating their emotions. The lack of an association between HRV and PA instability is, however, not completely unexpected and may be due to a lower PA/NA balance in FM patients. It may be reasonable to assume that for FM patients, HRV is rather associated with the down-regulation of NA. Higher levels of depression, anxiety and stress in FM patients support this assumption. Likewise, most research supporting the notion that affective instability is maladaptive is specific to NA (for a review and meta-analysis, see

Houben et al., 2015). The lack of differences in PA instability between groups is, therefore, in line with numerous studies investigating affective instability in natural contexts, further demonstrating the role of NA instability rather than PA instability for psychopathology and well-being (e.g. Thompson et al., 2012; Trull et al., 2008).

Interestingly, FM patients differed in the mean level of daily PA but not in PA instability compared to healthy controls, whereas the opposite is found for NA. This finding demonstrates the distinction between the intensity and the temporal variability of emotions. It further stresses the importance of broadening the perspective to better understand dynamically fluctuating emotions in daily life and their putative role for pain conditions, and points to the need for future research to take into account affective instability when investigating adaptation to chronic pain conditions.

The second part of the current study focused on replicating previous findings on the impact of NA instability on daily pain outcomes in patients with chronic pain. Contrary to our expectation, we did not find a cross-level interaction between NA instability and daily pain severity. We have previously found that the association between daily pain severity and daily disability as well as cognitive complaints was stronger in patients who reported higher NA instability (Rost et al., 2016). One explanation for this discrepancy of findings may relate to insufficient power of the multilevel moderation analysis in the current study. The average Level 1 and Level 2 sample sizes are of major importance in determining the detection of cross-level interactions (Mathieu, Aguinis, Culpepper, & Chen, 2012). Our sample size may have been too small to allow for the detection of a cross-level interaction. Also, the number of Level 1 units was smaller because we used end-of-day diaries rather than ecological momentary assessment methods. We replicated, however, the finding that mean daily NA and NA instability significantly predicted daily disability, indicating that patients reporting higher daily NA and NA instability experienced more disability in daily life. These findings support previous findings indicating that not only the intensity but also the time course and variability of NA are related to daily disability (Rost et al., 2016). In addition, the current findings confirm that PA instability does play a role in the adaptation to daily pain (Rost et al., 2016).

This study has some limitations. Firstly, we assessed fluctuations in affect over days by end-of-day diaries; the results can, therefore, not be generalized to within-day emotional fluctuations. Secondly, we did not perform time lag analyses as these would have been beyond the scope of the current study. Thus, the current results do not allow for conclusions on causality between variables. Thirdly, affective instability is a global

index that captures temporal variability of emotions (in this case over 14 days). However, we cannot draw conclusions regarding the individual or contextual factors underlying individual differences in affective instability in our sample.

In conclusion, targeting NA instability in therapeutic settings in order to provide chronic pain patients with tools to better adapt to their conditions may be an important therapeutic strategy. For example, the inclusion of specific emotion regulation skills training (e.g. Berking & Whitley, 2014), mindfulness-based approaches (Grossman, Tiefenthaler-Gilmer, Raysz, & Kesper, 2007; Rosenzweig et al., 2010) or Acceptance and Commitment Therapy (Hayes, Strosahl, & Wilson, 1999; McCracken, Vowles, & Eccleston, 2005; Vowles, McCracken, & O'Brien, 2011) may be promising approaches for treating FM. Future research should investigate affective instability on different time scales (e.g. within-day fluctuations) and include other psychological variables, such as worry or rumination, in order to get closer to identifying possible shared underlying mechanism of both emotion and pain problems.

Conflict of interest statement

There are no conflicts of interest that may arise as a result of this research.

Acknowledgements

The authors want to thank Nele Decoene for their help in the data collection. This research was supported in part by the grant "Self-Regulation and Pain" (SEREPA; University of Luxembourg) to Claus Vögele.

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3

CHAPTER 3

**Generalized Hypervigilance in Fibromyalgia:
Normal Interoceptive Accuracy, but Reduced Self-
Regulatory Capacity¹****Abstract**

The factors underlying the aetiology of fibromyalgia (FM) are largely unknown. According to the generalized hypervigilance hypothesis (GHH), FM patients show excessive attention towards pain stimuli and other sensory events, thereby increasing pain perception and dysfunctional behaviour. We tested this notion by assessing interoceptive accuracy (IA) in FM patients and matched healthy controls. We also tested the hypothesis that FM is characterized by reduced self-regulatory capacity as indexed by heart rate variability (HRV). 47 FM patients ($M_{\text{age}} = 45.5$, 39 females) and 45 healthy controls ($M_{\text{age}} = 44.9$, 37 females) completed several self-report scales (Body Vigilance Scale, Depression Anxiety Stress Scales, Pain Catastrophizing Scale). To derive HRV, heart rate was monitored under resting conditions; for the assessment of IA participants performed a heartbeat tracking task in which they were asked to silently count their heartbeats. FM patients reported higher body vigilance than healthy controls, but there were no group differences in IA. FM patients had lower HRV compared with healthy controls. HRV did not predictor IA. In conclusion, our findings do not support the GHH. Patients reported a heightened focus on bodily sensations, which was not reflected in IA. It may be that hypervigilance is not a general and stable characteristic but is rather context dependent and modality-specific.

¹ Rost, S., Van Ryckeghem, D., Schulz, A., Crombez, G., & Vögele, C. (in revision: Journal of Psychosomatic Research). Generalized hypervigilance in fibromyalgia: Normal interoceptive accuracy, but reduced self-regulatory capacity.

Introduction

Fibromyalgia (FM) is characterized by widespread musculoskeletal pain, and is accompanied by fatigue, sleep disorders, memory problems and mood disturbances (Wolfe et al., 1990). Despite its prevalence and increasing research, the factors underlying the aetiology of FM remain elusive. One potentially important aetiological factor is generalized hypervigilance, i.e. the excessive attention towards potential threat. The generalized hypervigilance hypothesis (GHH) posits that patients with medically unexplained symptoms, such as FM, focus their attention on potential threat signals, resulting in increased pain sensitivity, and the amplified perception of non-painful sensations in other sensory modalities (Hollins et al., 2009; McDermid, Rollman, & McCain, 1996).

Despite its importance for a better understanding of hypervigilance in FM, research on the perception of internal signals in FM patients, i.e. interoception, is scarce. So far, generalized hypervigilance has been supported by (a) self-report measures, on which FM patients typically show elevated scores for vigilance to pain (Crombez, Eccleston, Van den Broeck, Goubert, & Van Houdenhove, 2004; Peters, Vlaeyen, & van Drunen, 2000; Tiemann et al., 2012) and (b) experimental measures showing decreased pain thresholds and tolerance levels for experimentally induced pain (Kosek, Ekholm, & Hansson, 1996; Lautenbacher, Rollman, & McCain, 1994; McDermid et al., 1996) and innocuous (e.g. auditory) stimuli (Geisser et al., 2003; Hollins et al., 2009) in FM patients. According to the GHH, FM patients should be hypervigilant towards internal signals and, thus, more accurate in perceiving internal bodily signals.

The accuracy of perceiving internal bodily changes has been conceptualised as a trait (Mussgay, Klinkenberg, & Rüdell, 1999; Tsakiris, Tajadura-Jiménez, & Costantini, 2011), with the process of accurately detecting and tracking bodily signals relying on actual bodily changes. The assessment of interoceptive accuracy (IA), therefore, requires the monitoring of physical changes, which can be readily measured. The heartbeat tracking task is a useful assessment paradigm, as heartbeats are easily quantifiable as discrete and determinable stimuli (Cameron, 2001; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Pollatos & Schandry, 2004). Numerous studies have used this paradigm to assess IA in panic patients in order to investigate hypervigilance towards bodily sensations (Antony et al., 1995; Ehlers & Breuer, 1992; Eley, Stirling, Ehlers, Gregory, & Clark, 2004; Van der Does, Antony, Ehlers, & Barsky, 2000; Willem Van der Does, Van Dyck, & Spinhoven, 1997).

In addition, FM may also be characterized by a deficiency in inhibiting irrelevant information or prioritizing attention towards relevant stimuli or sensations (Carrillo-de-la-Peña, Vallet, Pérez, & Gómez-Perretta, 2006). According to the Neurovisceral Integration Model (Thayer & Lane, 2000, 2009), insufficient inhibitory control can be physiologically indexed by vagally mediated heart rate variability (HRV). Heart rate reflects the combined result of sympathetic and parasympathetic activity at the sino-atrial node (Levy, 1990). Beat-to-beat variability indexes activity in this reciprocal inhibitory cortico-subcortical neural circuit, and serves as the structural link between psychological processes and health-related physiological processes. As HRV reflects activity that is dynamically organized in response to environmental challenges, it allows for the quantification of behavioural flexibility and adaptability in a changing environment, i.e. self-regulatory capacity. In line with this reasoning, previous research has found lower HRV in FM patients compared to healthy controls (Cohen et al., 2000; Raj, Brouillard, Simpson, Hopman, & Abdollah, 2000). Porges (1992) defines the ability to rapidly shift and effectively sustain attention in accord with situational demands as one critical component of self-regulation. Accordingly, lower HRV has been found to predict hypervigilance and inefficiency of attentional regulation (Johnsen et al., 2003).

The aims of the current study were threefold: (1) to investigate IA in FM patients, (2) to replicate previous findings on lower HRV in FM patients compared to healthy controls, and (3) to examine the predictive value of self-regulatory capacity for IA. We, therefore, assessed HRV and performance in a heartbeat tracking task (Schandry, 1981) in a group of FM patients, compared to age- and sex-matched healthy controls. We hypothesized that (1) FM patients are more accurate in counting their heartbeats, (2) HRV, as an index of self-regulatory capacity, is reduced in FM patients, and (3) HRV is negatively associated with IA.

Methods

Participants and procedure

Participants between the age of 18 and 65 years were recruited in the context of a larger project (see protocol ASEF-I; <http://hdl.handle.net/1854/LU-5686902>) between January 2014 and March 2014. Individuals were eligible to participate if they either were diagnosed with FM and fulfilled the ACR-90 criteria for FM (FM group) or did not report

a current pain problem (healthy control group). Additional inclusion criteria were: (1) sufficient knowledge of the Dutch language; (2) absence of neurological conditions. Furthermore, individuals were excluded when they were unable to use both index fingers, reported abnormal sensations in the arms or if their eyesight was not normal or not corrected-to-normal (e.g., by glasses). The latter three criteria were exclusion criteria in regard of a task, which was not part of this study (see ASEF-1). FM patients were recruited via the Multidisciplinary Pain Clinic of Ghent University Hospital. They were informed about the study with a poster in the waiting room of the hospital. Patients who were interested in taking part left their contact details and were screened for eligibility. Individuals of the control group were recruited using advertisements in a local newspaper, flyers and the university website. Individuals who volunteered were contacted and screened for eligibility. Both groups were matched at group level for age, sex and educational level. A total of 98 individuals took part in the study: 49 FM patients and 49 healthy controls.

As the study was part of an extended protocol, we only report the procedure relevant for the current research question. In a first step, participants filled out a set of standardised questionnaires, including the Body Vigilance Scale (BVS; Schmidt, Lerew, & Trakowski, 1997), the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995) and the Pain Catastrophizing Scale (PCS; Sullivan, Bishop, & Pivik, 1995) via an online assessment system (LimeSurvey) at home (if this was not possible, participants received a paper version to fill in). In a second step, i.e., when arriving at the research lab, participants rated their current pain and indicated whether they had consumed coffee within the last two hours (exclusion criterion). The experimenter then checked the ACR-criteria (Wolfe et al., 1990) with the patient to confirm the FM diagnosis. Next, heart rate was measured over a period of five minutes. Finally, the participant performed a heartbeat tracking task (Schandry, 1981). The study design was approved by the Ethics Review Panel of the University of Luxembourg and the Medical Ethics Committee, University Hospital Ghent. Participants gave written informed consent and received a token of gratitude of 35€ for their participation.

Self-report data

Pain intensity at the moment of testing was assessed using the item “How intense is your pain now?”. Participants answered by using a visual analogue scale from 0 (“no pain”) to 100 (“worst imaginable pain”).

Depressive mood, anxiety and stress were assessed with the DASS (Lovibond & Lovibond, 1995). Each of the subscales contain 14 items in which participants are asked to rate the extent to which they have experienced each state (e.g. “I found it difficult to relax”, “I felt sad and depressed”) over the past week using a 4-point Likert scale (0 = *did not apply to me at all* to 3 = *applied to me very much, or most of the time*). Scores may range from 0 to 42. The scales have excellent internal consistencies ($\alpha = .97$ for depression, $\alpha = .92$ for anxiety and $\alpha = .95$ for stress).

Catastrophic thinking about pain was assessed with the PCS (Sullivan et al., 1995). The scale consists of 13 items in which participants indicate the degree to which they experienced catastrophic thoughts or feelings during pain episodes (e.g. “I keep thinking about how much it hurts”, “I can’t seem to keep it out of my mind”) on a 5-point Likert scale (0 = *not at all* to 4 = *all the time*). Scores may range from 0 to 52. This scale showed a good reliability and validity in healthy populations and chronic pain patients (Van Damme, Crombez, Bijttebier, Goubert, & Van Houdenhove, 2002). Cronbach’s alpha for the current study was .95.

Vigilance for bodily sensations was assessed using the BVS (Schmidt et al., 1997). The BVS assesses attentional focus for bodily sensations and consists of four items in which participants indicate on an 11-point Likert scale (0 = *none* to 10 = *extreme*) the degree to which they agree with a particular statement regarding selective attention to bodily sensations. Scores on item 3 (“On average, how much time do you spend each day ‘scanning’ your body for sensations [e.g. sweating, heart palpitation, dizziness]”) are divided by 10. The last item involves having participants rate their attention to 15 bodily sensations (e.g. heart palpitations). Responses to the fourth item are averaged to yield a single score. Summing the four items derives a total score of the BVS with a range from 0 to 40. The questionnaire has adequate internal consistency in clinical and nonclinical populations (Schmidt et al., 1997; Zvolensky & Forsyth, 2002). Cronbach’s alpha for the current study was acceptable ($\alpha = .70$).

Heart rate variability

During the baseline period, participants sat in individual cubicles and were instructed to sit quietly and relax. Inter-beat intervals were assessed based on electrocardiographic recordings for 5 minutes at a sampling rate of 1000 Hz using a Polar watch RS800CX (Polar Electro Oy, Kempele, Finland).

Perception of bodily sensations

IA was assessed with a heartbeat tracking task based on the paradigm first introduced by Schandry (Schandry, 1981). The actual number of heartbeats was recorded with the same Polar watch used for baseline recordings and analysed via Polar ProTrainer software. Participants were asked to silently count all the heartbeats they perceived in their body without taking their pulse or attempting any other manipulation to facilitate the discrimination of their heartbeats. Instructions were given via an E-Prime-based script on a written screen to minimize bias introduced by the experimenter. The task consisted of four intervals of 25, 35, 45 and 55 seconds in randomized order and the duration of these intervals was unknown to participants. The intervals were separated by standard resting periods of 30 seconds. A visual countdown of 3-2-1 followed by a cross on the screen indicated the beginning of the counting period. The period ended with the disappearance of the cross. After the counting period, participants were asked to indicate the number of counted heartbeats. The number of counted heartbeats was compared to the recorded number of heartbeats. Participants started with one training interval of 25 seconds. IA was calculated using the formula:

$$IA_{Schandry} = 1 - \frac{|\sum HB_{actual} - \sum HB_{perceived}|}{\sum HB_{actual}}$$

Data handling and reduction

To calculate HRV-indices, sequential interbeat intervals were downloaded using the software Polar Pro Trainer 5. All signals were visually inspected for artefacts. HRV analysis was performed using the software ARTiiFACT (Kaufmann, Sütterlin, Schulz, & Vögele, 2011). Firstly, measurement artefacts were identified by applying a distribution-related threshold criterion. Erroneous beats were deleted and substituted by cubic spline interpolation of neighbouring intervals. Time domain measures were directly calculated

from RR-interval series. Spectral analysis of the RR-interval series was carried out using Fast Fourier Transformation. Following the recommendations of the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996) we defined the high frequency band (HF) as 0.14 to 0.4 Hz and used the following time and frequency HRV parameters for statistical analyses: root mean square of successive differences (RMSSD), percent of difference between adjacent RR intervals that are greater than 50 ms (pNN50) and the absolute power in the HF band (HFabs). We focused on those parameters because they reflect parasympathetic control over heart rate (Task Force, 1996). The criterion for outliers in HRV measures was defined as values more than 3 *SD* above the sample mean (cf. Koval et al., 2013). After correcting for outliers, HFabs was log transformed to adjust for skewness of the distribution (lnHFabs). Regarding the heartbeat tracking task, for 9 out of 89 participants (5 FM, 4 controls) only three valid intervals were included due to recording problems.

Four participants (1 FM, 3 controls) were excluded from the final analyses due to equipment failure. Furthermore, two participants (1 FM, 1 control) were excluded because of outliers in HRV. The final sample, therefore, consisted of 47 FM patients and 45 healthy controls.

Statistical analyses

Differences in characteristics between the FM and healthy control groups were examined using independent samples t-tests. Pearson correlations were performed between IA, HRV-indices and self-report measures. According to hypothesis one, we expected that FM patients are more accurate in counting their heartbeats than healthy controls. This was tested using an independent samples t-test (2-tailed). Effect size indices for independent samples (Cohen's *d*) and the 95% confidence interval (95% CI) were calculated (Borenstein, Hedges, Higgins, & Rothstein, 2009; Cohen, 1988). Hypothesis two states that HRV is reduced in FM patients. To test this hypothesis, a multivariate analysis of variance (MANOVA) was conducted in regard of the three related HRV-indices. Finally, we tested the hypothesis of a negative relationship between HRV and IA using a hierarchical linear regression analysis. In a first step, we entered HRV as predictor. In a next step, we aimed at controlling whether the relationship remains present when controlling for group. For this analysis, we choose HRV-RMSSD because of its robust statistical properties (cf. Task Force, 1996). Critical alpha level for all analyses was set to .05.

Results

Descriptive statistics

Table 8 summarizes sample characteristics and self-report data. There were no significant differences in age, gender or educational level (all p 's > .244). All of the FM patients, and 26.7% of the healthy controls reported pain at the moment of testing. Pain intensity at the moment of testing was significantly higher in the FM than in the healthy control group. Pain intensity scores in the FM group ranged between 8 and 84, compared to 0 and 17 in the control group. FM patients had significantly higher scores on all self-report measures as compared to healthy controls (see Table 8).

Table 8

Descriptive Statistics by Participant Group

Variable	Group		Difference test
	FM ($n = 47$)	Control ($n = 45$)	
Age (M, SD)	45.5 (9.2)	44.9 (12.2)	$t(82)^a = .26, p = .792, d = 0.06$
Gender (n women)	39	37	$\chi^2(1) = .009, p = .924, w = 0.00$
Educational level			$\chi^2(2) = 2.82, p = .244, w = 0.29$
College/University	38.3%	55.6%	
Secondary school	57.4%	42.2%	
Primary school	4.3%	2.2%	
Pain intensity at moment of testing (M, SD)	43.87 (21.5)	1.60 (3.8)	$t(49)^a = 13.28, p < .001, d = 2.73$
DASS – D (M, SD)	13.2 (10.6)	5.84 (6.4)	$t(75)^a = 3.95, p < .001, d = 0.84$
DASS – A (M, SD)	11.34 (7.6)	2.89 (3.6)	$t(66)^a = 6.91, p < .001, d = 1.42$
DASS – S (M, SD)	15.26 (7.8)	8.11 (7.4)	$t(90) = 4.51, p < .001, d = 0.94$
PCS (M, SD)	21.62 (10.8)	9.64 (9.6)	$t(90) = 5.61, p < .001, d = 1.17$
BVS (M, SD)	18.41 (6.6)	14.05 (6.4)	$t(90) = 3.21, p = .002, d = 0.67$

Note. FM = fibromyalgia, DASS – D = depression scale from the Depression Anxiety Stress Scales, DASS – A = anxiety scale from the Depression Anxiety Stress scales, DASS – S =

stress scale from the Depression Anxiety Stress scales, PCS = Pain Catastrophizing Scale, BVS = Body Vigilance Scale, ^a = equal variances not assumed.

There were no significant associations between IA and any other measure ($-.081 < r > .142$, ns). Body vigilance was related to higher pain intensity at the moment of testing as well as to higher scores in depression, anxiety, stress and pain catastrophizing. Further, we found a significant negative correlation ($r = -.223, p < .05$) between pain catastrophizing and pNN50. HRV-indices were highly interrelated. Table 9 gives an overview of means and correlations.

Table 9*Means (M), Standard Deviations (SD) and Pearson Correlation Coefficients for All Measures*

	M	SD	2	3	4	5	6	7	8	9	10
1. Interoceptive accuracy	.56	.24	.117	-.005	.024	.110	.051	-.081	.142	.112	.102
2. Pain intensity at moment of testing	23.20	26.30	.424***	.590***	.401***	.554***	.299**	-.232*	-.193†	-.147	
3. Depression (DASS)	9.59	9.67	.685***	.676***	.639***	.373***	-.114	-.112	-.125		
4. Anxiety (DASS)	7.21	7.28	.715***	.642***	.527***	-.099	-.069	-.064			
5. Stress (DASS)	11.76	8.36	.575***	.436***	-.071	-.075	-.085				
6. Pain Catastrophizing (PCS)	15.76	11.82	.450***	-.182†	-.223*	-.098					
7. Body Vigilance (BVS)	16.28	6.83	-.071	-.049	-.021						
8. RMSSD	22.60	12.89	.935***	.868***							
9. pNN50	6.20	9.37	.876***								
10. lnHFabs	4.98	1.24									

Note. DASS = Depression Anxiety Stress Scales; PCS = Pain Catastrophizing Scale; BVS = Body Vigilance Scale; RMSSD = root mean square of successive differences; pNN50 = percent of difference between adjacent RR intervals that are greater than 50 ms; lnHFabs = log transformed absolute power in the high frequency band. † $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$.

Interoceptive accuracy

IA did not differ between the FM group ($M = 0.59$; $SD = 0.25$) and the healthy control group ($M = 0.52$; $SD = 0.24$), $t(90) = 1.28$, $p = .205$, $d = 0.29$, 95% CI [-0.04 to 0.17].

Heart rate variability

Using Pillai's trace, we found a significant effect for group in all HRV-indices, $V = 0.09$, $F(3,88) = 2.94$, $p = .037$. Follow-up tests revealed lower RMSSD and pNN50 in FM patients compared with healthy controls. Results of separate univariate ANOVAs are summarized in Table 10.

Table 10

Heart Rate Variability Measures by Participant Group

HRV-indices	Group		Difference test (ANOVAs)
	FM ($n = 47$) (M , SD)	Control ($n = 45$) (M , SD)	
RMSSD	19.36 (10.6)	25.98 (14.3)	$F(1,90) = 6.41$, $p = .013$, $\eta^2 = .07$
pNN50	4.13 (6.9)	8.36 (11.1)	$F(1,90) = 4.88$, $p = .030$, $\eta^2 = .05$
lnHFabs	4.77 (1.2)	5.19 (1.2)	$F(1,90) = 2.58$, $p = .112$, $\eta^2 = .03$

Note. HRV = heart rate variability, FM = fibromyalgia, RMSSD = root mean square of successive differences, pNN50 = percent of difference between adjacent RR intervals that are greater than 50 ms, lnHFabs = log transformed absolute power in the high frequency band.

Association between interoceptive accuracy and heart rate variability

We investigated the predictive value of HRV for IA using a hierarchical regression analysis. The regression analysis indicated that HRV did not explain a significant amount of the variance in IA when entered as a single predictor, $F(1,90) = 1.86$, $p = .176$, $R^2 = .02$, with $\beta = .142$. When controlling for group as a predictor, the regression model was not significant, $F(2,89) = 2.41$, $p = .096$, $\Delta R^2 = .03$. This model is presented in Table 11.

Table 11

Hierarchical Regression Model Explaining Interoceptive Accuracy, with Standard Errors and 95% Confidence Intervals Reported in Parentheses

	<i>b</i>	<i>SE B</i>	β	<i>p</i>
Step 1				
HRV (RMSSD)	0.003 (-.001, .007)	0.002	.142	.176
Step 2				
HRV (RMSSD)	0.004 (0.000, 0.008)	0.002	.189	.080
Group	-0.088 (-0.191, 0.015)	0.052	-.182	.092

Note. $R^2 = .020$ for step 1; $\Delta R^2 = .031$ for step 2, RMSSD = root mean of square of successive differences, HRV = heart rate variability.

Discussion

The aims of the current study were (1) to assess the accuracy of perceiving interoceptive signals in FM patients using a heartbeat tracking task, (2) to compare HRV between groups, and (3) to investigate the predictive value of HRV for IA. Firstly, FM patients did not differ from healthy controls in IA. Secondly, FM patients showed decreased HRV compared to healthy controls. Thirdly, HRV did not predict IA.

Contrary to our expectations, we did not find altered perception of interoceptive signals in FM patients, as assessed with the heartbeat tracking task. Although the small CI indicates robustness of this finding, it is opposed to previous results showing increased pain sensitivity and amplified perception of painful and non-painful stimuli in FM patients (Geisser et al., 2003; Hollins et al., 2009; Kosek et al., 1996; Lautenbacher et al., 1994; McDermid et al., 1996). The present finding, however, is in line with a number of studies, which failed to demonstrate prioritization of external innocuous stimuli in FM patients (Peters et al., 2000; Van Damme et al., 2015). The current findings, therefore, do not support the assumption of generalized hypervigilance in FM patients and are in contrast to findings confirming hypervigilance, assessed using a heartbeat tracking task, in panic patients (Ehlers & Breuer, 1992, 1996; Eley et al., 2004).

Interoception entails a complex process with different aspects (Ceunen, Van Diest, & Vlaeyen, 2013; Farb et al., 2015; Garfinkel & Critchley, 2013; Garfinkel, Seth, Barrett, Suzuki, & Critchley, 2015). For example, IA can be conceptualized as a function of

sensitivity and specificity (Farb et al., 2015). It could be argued that FM patients are indeed more sensitive to interoceptive signal change, similarly as to exteroceptive stimuli (Hollins et al., 2009; Kosek et al., 1996; Lautenbacher et al., 1994; McDermid et al., 1996), but are not able to reject competing signals as proposed by Pennebaker's competition-of-cues model (Pennebaker, 1982). This model posits that only a limited amount of information can be processed at a given moment in time. In case of FM, persistent pain would then interfere with the processing of other bodily sensations, result in diminished IA and indicate the absence of generalized hypervigilance. In line with this reasoning are findings showing that individuals who report somatosensory amplification are less accurate in counting their heartbeats (Mailloux & Brener, 2002). Likewise, one recent study reported lower IA in FM patients than in healthy controls, using the same behavioural paradigm for the assessment of IA (Duschek, Montoro, & Reyes Del Paso, 2015). Several methodological differences between Duschek and colleagues' and the current study may explain these diverging results. For example, the instructions for the heartbeat tracking task in the current study were standardized and presented on a screen, whereas in Duschek and colleagues' (2015) they were signalled by the experimenter, with the latter representing a potential source of bias. In addition, our findings are based on four compared to only three counting periods used in the study by Duschek and colleagues (2015). The number of counting periods may affect the reliability of the task, but further research is necessary to provide specific evidence on this topic. Furthermore, and in contrast to Duschek and colleagues' (2015), the current study included balanced sample sizes and groups matched for age, sex and educational level.

The current results do not entirely rule out hypervigilance in FM patients. Instead, they cast doubt on the view of hypervigilance as a general characteristic, which applies to all kinds of signals equally. Rather, hypervigilance may be a dynamic process, which is associated with specific conditions or modalities, that occurs when the fear system is activated and an individual is concerned about pain (Crombez, Van Damme, & Eccleston, 2005; Eccleston & Crombez, 1999; Eccleston & Crombez, 2007; Van Damme, Legrain, Vogt, & Crombez, 2010). Hypervigilance would then be expected to only appear in the context of pain or threat, and this has even been shown for healthy individuals who respond with a stronger focus on body parts where pain or bodily threat was anticipated (Van Damme, Crombez, & Lorenz, 2007; Van Damme, Gallace, Spence, Crombez, & Moseley, 2009; Van Damme & Legrain, 2012; Vanden Bulcke, Van Damme, Durnez, & Crombez, 2013). Heartbeats are not threatening or aversive for FM patients per se, and the experimental setting did not suggest bodily threat, explaining the current lack of group differences in

IA. This may further explain why higher IA has been found in panic pain patients compared to healthy controls (Ehlers & Breuer, 1992, 1996; Eley et al., 2004), as heartbeats definitely constitute threatening bodily sensations for these patients. Further, the fact that IA and scores on the BVS (Schmidt et al., 1997) were not associated, may point to modality-specific hypervigilance. Future studies should investigate hypervigilance in different pain-related and threatening contexts as well as in regard to different modalities in FM patients to better understand the role of interoception on the aetiology of FM.

Interestingly, results of the heartbeat tracking task contrasted with self-reported hypervigilance, i.e. the tendency to focus on bodily sensations. Scores on the BVS (Schmidt et al., 1997) were significantly higher in FM patients, a result which is in line with previous findings (Peters et al., 2000; Roelofs, Peters, McCracken, & Vlaeyen, 2003; Tiemann et al., 2012). It is, however, important to note that self-reported body vigilance may be partly affected by the experience of persistent pain. Self-report measures may be biased by non-attentional factors and higher scores may reflect somatic complaints rather than excessive attention (Van Damme, Crombez, et al., 2009).

Groups differed in HRV in that FM patients showed lower HRV, specifically in the more robust time domain measures of HRV. These findings are in line with previous research comparing HRV between FM patients and healthy controls (Cohen et al., 2000; Raj et al., 2000). Chronic pain conditions are accompanied by cognitive, emotional and physiological disturbances (Wolfe et al., 1990). The adaptation to these conditions requires the capacity to control one's cognition, emotion and behaviour, i.e. self-regulatory capacity (Baumeister, 1998; Carver & Scheier, 1998). The persistent challenge posed by chronic pain may exhaust patients' self-regulatory resources (Solberg Nes, Carlson, Crofford, de Leeuw, & Segerstrom, 2010), which may be reflected in lower HRV. We further expected reduced HRV to be related to increased hypervigilance (Carrillo-de-la-Peña et al., 2006), as a link between resting cardiac vagal tone and attentional control has been previously reported (Porges, 1992). Reduced HRV, associated with increased hypervigilance, would then predict more accurate perception of interoceptive signals. We could not confirm this hypothesis. We do, however, find a non-significant trend towards a positive association between HRV and IA with a small effect size. A positive relationship between the perception of internal bodily states and the strength of controlling one's behaviour may indeed be expected based on Damasio's somatic marker hypothesis (Bechara, Damasio, & Damasio, 2000; Bechara, 2004; Damasio, Tranel, & Damasio, 1991). This theory proposes that somatic states mark response options which guide our

behaviour. More precisely, internal and external stimuli elicit somatic states which involve physiological modifications and are processed in specific brain structures (e.g. amygdala, ventromedial prefrontal cortex). These patterns of body-related responses to stimuli, i.e. emotions, provide an individual with options to respond to a stimulus and guide his behaviour. Some studies have emphasized the role of feedback of bodily signals in behavioural processes by linking higher IA to increased self-reported self-regulatory capacity or self-regulation of physical load (Herbert, Ulbrich, & Schandry, 2007; Weiss, Sack, Henningsen, & Pollatos, 2014).

Obviously, further research is important to elaborate body perception in FM patients and investigate hypervigilance with different experimental paradigms and in different contexts. Also, the direction of any association between the perception of interoceptive signals and self-regulatory processes remains unclear. Some limitations must be pointed out. Firstly, one may argue that IA is not a suitable concept, and, therefore, the heartbeat tracking task not be an appropriate operationalization for investigating generalized hypervigilance. While generalized hypervigilance is supposed to lead to amplified perception of all sensations, it is debatable whether it would imperatively lead to more accurate perception. Secondly, we did not assess body mass index which has been related to reduced IA (Herbert, Blechert, Hautzinger, Matthias, & Herbert, 2013), so that we cannot rule out for IA results to have been systematically affected by differences in body mass index. Thirdly, the present study used a cross-sectional design, which does not allow for conclusions on cause-effect relationships. Fourthly, pain medication may have affected the results.

In conclusion, the results suggest that hypervigilance is not a general characteristic of FM patients, but one that is rather context dependent or modality-specific.

Competing interest statement

The authors have no competing interests to report.

Acknowledgment

The authors would like to thank Nele Decoene for her help in the recruitment and data collection. This research was supported in part by the grant “Self-Regulation and Pain” (SEREPA; University of Luxembourg) to the senior author.

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4

CHAPTER 4

An Experimental Investigation of the Role of Self-Regulatory Capacity in the Distraction from Pain¹**Abstract**

Pain demands attention and interferes with cognitive processes and, therefore, serves as an alarm signal for potential bodily harm. This involuntary bottom-up selection of pain over other targets, i.e. attentional interference by pain, can be modulated to some extent, i.e. top down modulation of attention. Top-down modulation has often been investigated using distraction paradigms, in which individuals are requested to direct attention away from pain. Yet, the extent to which individuals are able to modulate attention away from pain has been hypothesized to depend on individual and contextual factors. In the current study, we investigated self-regulatory capacity as an underlying factor of distraction efficacy. Heart rate from 39 participants (31 women; age $M = 22.5$, $SD = 3.7$) was monitored under resting conditions to derive heart rate variability (HRV) as an index of self-regulatory capacity. Participants filled out several self-report measures (e.g. self-control) and subsequently performed a distraction task. They were instructed to either attend to a visual task (distraction trial) or to focus on the somatosensory sensation (focus trial) while painful and vibrotactile stimuli were administered. During distraction trials pain intensity ratings were lower than during focus trials. Furthermore, reaction times were delayed when painful stimuli were presented. This interference effect of pain increased with increasing intensity. There was no association between HRV and distraction efficacy or attentional interference. No relationship was found between distraction efficacy and the capacity to self-regulate as indexed by HRV and self-report. Future research should investigate other factors, such as motivation.

¹ Rost, S., Van Ryckeghem, D., Crombez, G., & Vögele, C. (in preparation). An experimental investigation of the role of self-regulatory capacity in the distraction from pain.

Introduction

Pain is an evolutionary acquired alarm signal of bodily harm. It demands attention, interrupts current activity and interferes with cognitive processes (Eccleston & Crombez, 1999; Legrain et al., 2009). This involuntary, bottom-up, capture of attention by pain is a critical feature of its alarm function. Several studies have documented attentional interference by pain as indexed by impaired task performance, e.g. delayed reaction times, during pain (Crombez, Eccleston, Baeyens, & Eelen, 1997; Crombez, Eccleston, Van Den Broeck, Van Houdenhove, & Goubert, 2002; Vancleef & Peters, 2006). Yet, this automatic capture of attention can be modulated to a certain extent by top-down selection of attention, which represents an intentional and goal-directed process (Legrain et al., 2009). Directing attention away from pain, i.e. distraction, is one of the most frequently used coping strategies (Johnson, 2005) and commonly part of pain treatment programs (Elomaa, Williams, & Kalso, 2009; Morley, Shapiro, & Biggs, 2004). Findings regarding the efficacy of distraction are, however, inconsistent (Goubert, Crombez, Eccleston, & Devulder, 2004; Hodes, Rowland, Lightfoot, & Cleeland, 1990; Petrovic, Petersson, Ghatan, Stone-Elander, & Ingvar, 2000; Valet et al., 2004), indicating that distraction may not be effective for everyone in every situation (Eccleston & Crombez, 1999; Van Ryckeghem et al., 2013).

Theoretically driven research is needed to gain a better understanding of the factors affecting distraction. Pain catastrophizing, for example, has been found to enhance attentional interference by pain and to make disengaging from pain more difficult (Van Damme, Crombez, & Eccleston, 2004; Vancleef & Peters, 2006). Another important factor relates to inhibitory capacity. Effective distraction relies on the ability to inhibit the predominant response of attending to pain and to resist being interrupted by pain (Friedman & Miyake, 2004; Nigg, 2000). Cognitive functions such as attentional selection or inhibition of pre-potent responses have been shown to depend on working memory (Garavan, Kelley, Rosen, Rao, & Words, 2000). Working memory, in turn, critically depends on inhibitory neural processes specifically related to prefrontal cortical activity (Arnsten & Goldman-Rakic, 1998; Braver et al., 1997; Garavan, 2002), which can be indexed by vagally mediated heart rate variability (HRV; Thayer & Lane, 2000, 2009). Beat-to-beat variability indexes activity in a neural network that permits the prefrontal cortex to inhibit subcortical structures associated with defensive behaviours (Lane, Reiman, Ahern, & Thayer, 2001; Thayer & Brosschot, 2005). Accordingly, HRV indexes self-regulatory

capacity and has been found to predict attention regulation (Johnsen et al., 2003; Kaufmann, Vögele, Sütterlin, & Lukito, 2012; Thayer & Lane, 2000, 2009).

The main aims of the present study were to assess distraction efficacy, to examine attentional interference by pain, and to investigate the predictive role of HRV for distraction efficacy. We hypothesized that 1) self-reported pain would be lower when attention was directed away from pain than when it was directed towards pain, 2) reaction times for localizing a visual signal would be slower in presence of a pain stimulus of high intensity than in presence of a pain stimulus of low pain intensity or a vibrotactile stimulus, and 3) self-regulatory capacity would be positively associated with distraction efficacy and negatively associated with attentional interference.

Methods

Participants

A sample of 45 volunteers was recruited from students at the University of Luxembourg (36 women; age $M = 22.6$, $SD = 3.5$). Participants were recruited via flyers, posts on the University platform, and standardized mails. They were required to be aged at least 18 years and to have normal or corrected-to-normal vision. They should further show normal sensitivity of the arms and have sufficient knowledge of German as all instructions, questionnaires and materials were in German. Exclusion criteria included (1) current or past neurological conditions (e.g. epilepsy); (2) cardiovascular diseases; (3) self-reported mental disorders; (4) chronic pain problems (e.g. back pain, migraine); (5) current use of medication that may affect relevant underlying psychobiological processes (e.g. analgesics); (6) electronic implants (e.g. pacemaker) and; (7) pregnancy.

The study design was approved by the Ethics Review Panel of the University of Luxembourg. Participants gave written informed consent and received course credits for participation and/or a token of gratitude of 20€ for their participation.

Self-report measures

The **Anxiety Sensitivity Index** (ASI-3; Taylor, Zvolensky, Cox, & Deacon, 2007) was used to assess fear of anxiety-related sensations (e.g. “When my stomach is upset, I worry that I may be seriously ill”, “It scares me when I blush in front of people”). The scale consists of 18 items in which participants indicate their degree of agreement on a 5-point

Likert scale (0 = *not at all* agree to 4 = *totally agree*). Scores my range from 0 to 72. Cronbach's alpha for the current study was .80.

Self-control was measures using the Brief Self-Control Scale (BSCS; Tangney, Baumeister, & Boone, 2004). It is a 13-item measure relating to a variety of behaviours involving self-control (e.g. breaking a habit, working toward a long-term goal). Participants are asked to indicate how well the statements describe them (e.g. "I am good at resisting temptations", "Sometimes I can't stop myself from doing something, even if I know it is wrong") on a 5-point Likert scale (1 = *not at all* to 5 = *very much*). Scores range from 13 to 65. Cronbach's alpha for the current study was .77. We used the BSCS (Tangney et al., 2004) as a self-report measure of self-regulatory capacity to complement the physiological index.

Catastrophic thinking about pain was assessed with the Pain Catastrophizing Scale (PCS; Sullivan, Bishop, & Pivik, 1995). The scale consists of 13 items in which participants indicate the degree to which they experienced catastrophic thoughts or feelings during pain episodes (e.g. "I keep thinking about how much it hurts", "I can't seem to keep it out of my mind") on a 5-point Likert scale (0 = *not at all* to 4 = *all the time*). Scores range from 0 to 52. This scale has shown good reliability and validity in healthy populations and chronic pain patients (Van Damme, Crombez, Bijttebier, Goubert, & Van Houdenhove, 2002). Cronbach's alpha for the current study was .89.

State positive and negative affect were assessed with the Positive And Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). Participants were asked to indicate to which degree they experienced positive and negative affect (e.g. "proud", "strong", "angry", nervous") at the moment on a 5-point Likert scale (1 = *not at all* to 5 = *extremely*). Ratings of both positive and negative affect can range from 10 to 50. Cronbach's alpha was .79 for PA and .82 for NA.

Heart rate variability

Heart rate was monitored under resting conditions, with participants seated in a comfortable chair and instructed to sit quietly and relax. Inter-beat intervals were assessed based on electrocardiographic recordings of 5 minutes at a sampling rate of 1000 Hz using a BIOPACTM MP150 (Biopac Systems Inc., USA). The ECG raw signal was processed using the software Acqknowledge 4.2.

Distraction task

The distraction task involved a within-subject paradigm which was developed on the basis of previous distraction and task interference research (Van Ryckeghem, Crombez, Eccleston, Legrain, & Van Damme, 2013; Van Ryckeghem, Crombez, Van Hulle, & Van Damme, 2012).

During this paradigm, participants were asked to localize either a somatosensory stimulus (electrocutaneous or vibrotactile) or a visual stimulus. Both stimuli were simultaneously present in each trial. Each trial began with a visual cue consisting of a full colored circle (either blue or yellow; 1000 ms duration) in the center of the screen that indicated the relevant modality (color of the cue and the associated target modality were counterbalanced). In 50% of the trials, participants were cued to identify whether the visual stimulus was presented on the left or right side of the screen (distraction trials). In the remaining trials, participants were instructed to identify whether the somatosensory stimulus was presented on the left or right location (focus trials). Somatosensory and visual stimuli were presented equally often at the same location and at the opposite location. Participants responded to the left targets by pressing the '4' key with the index finger and to the right targets by pushing the '6' with the ring finger of the right hand on a computer keyboard. A trial ended when a participant responded or 2000 ms had elapsed.

The task started with a practice phase during which no pain stimuli were administered. Then, a test phase followed, which consisted of 256 trials with a break after 128 trials. The trials were presented randomly. In 192 trials, the somatosensory stimulus consisted of a non-painful vibrotactile stimulus and in 64 trials, it consisted of a painful stimulus. Participants were required to rate the intensity and unpleasantness of the somatosensory stimuli after 25% of the trials with non-painful stimuli and 75% of the trials with painful stimuli. Ratings were electronically collected using two numeric rating scales ranging from 0 to 100 presented on the screen. Firstly, pain intensity was assessed (0 = not at all intense, 100 = very intense). Secondly, pain unpleasantness was assessed (0 = very pleasant, 100 = very unpleasant). Subsequently, an overall pain experience rating was computed for each condition by averaging pain intensity and pain unpleasantness ratings. A distraction index was generated by subtracting the pain ratings of the focus trials from those obtained during the distraction trials. An attentional interference index was calculated by subtracting reaction times during no pain trials (i.e. vibrotactile trials) from reaction times during pain (average of low and moderate intensity) in the distraction trials only.

The task was programmed and presented by the INQUISIT Millisecond software package (Inquisit 2.06, 2008) on a personal computer (Pentium 4, 2.8. GHz, 512 MB) with a 60 Hz, 17-inch color CRT monitor. The viewing distance was approximately 60 cm.

Stimuli.

Two full colored circles were used as spatial cues for the location of the targets. Painful stimuli were electrocutaneous stimuli (300 ms, instantaneous rise and fall time) and were delivered by a constant current stimulator (DS5, Digi Timer Ltd., Hertfordshire, UK). Stimuli were administered by 2 lubricated Medcat surface electrodes (1 cm diameter) at the proximal (left location) and distal (right location) radius-ulnar articulations of the left arm. Pain intensity was individually determined as moderate before the experiment. Nonpainful (vibrotactile) stimuli were delivered by tactors (Engineering Acoustics, Inc.) for 300 ms with instantaneous rise and fall time. Visual target stimuli consisted of black squares (1.1 x 1.1 cm), presented at the left or right side of the screen on a white background.

Procedure

Upon arrival in the laboratory, participants were seated in a comfortable chair and instructed to sit quietly and relax to monitor heart rate. They were then asked to complete the questionnaires. Next, they were seated in front of a computer screen and moderate pain intensity was individually determined by administering electrocutaneous stimuli of increasing intensity at either location of the arm (starting with 0.5 mA). Participants rated the intensity of the electrocutaneous stimulus until indicating moderate pain. This last stimulus was used for the distraction task as stimulus of “high pain intensity”. The stimulus of “low pain intensity” was generated by multiplying this intensity by 0.89 (Arntz & Lousberg, 1990). This procedure resulted in an overall mean of 2.2 mA for the high pain stimulus (same for left and right). Finally, participants performed the distraction task.

Data reduction and analysis

To calculate vagally mediated HRV-indices, sequential interbeat intervals were analysed with the software ARTiiFACT (Kaufmann, Sütterlin, Schulz, & Vögele, 2011). All signals were visually inspected for artefacts. Firstly, measurement artefacts were identified by applying a distribution-related threshold criterion. Erroneous beats were deleted and substituted by cubic spline interpolation of neighbouring intervals. Spectral frequency

measures were derived using Fast Fourier Transformation. The high frequency band was defined as 0.14 to 0.4 Hz and expressed in power [ms^2] as recommended by the Task Force (1996). The criterion for outliers in HRV measures was defined as values deviating more than 3 *SD* from the sample mean (cf. Koval et al., 2013). We used normalized respiratory sinus arrhythmia (RSA_{norm}), which is proposed as a procedure to normalize HRV power in the high frequency band with mean interbeat interval (Hayano et al., 1990). RSA_{norm} was found to be less affected by sympathetic activity than uncorrected HRV parameters (for a review see Grossman & Taylor, 2007). RSA_{norm} can be considered as indicator for vagal cardiac activity and is, therefore, used as physiological marker of self-regulatory capacity.

Three participants were excluded because they did not experience moderate pain in the focus trials (< 20 on a scale from 0 to 100). One participant was excluded because the distraction index deviated by more than 4 *SD* from the group mean. Two participants were excluded because of outlying HRV-data (> 3 *SD* from the mean). The final sample consisted of 39 participants (31 women; age $M = 22.5$, $SD = 3.7$).

Statistical analyses were performed with SPSS statistical software, version, 24.0 for Windows (SPSS, Chicago, IL). Analyses were performed on the response latencies, pain intensity ratings and pain unpleasantness ratings of the electrocutaneous stimuli. Descriptive statistics, correlation analyses, multivariate repeated-measures ANOVAs, and linear regression analyses were performed to test the hypotheses (2 tailed). HRV and self-control were entered simultaneously in the regression analyses as both the average variance inflation factors and tolerance statistic did not indicate a potential problem or indication for bias of the regression.

Results

Correlational analyses

Correlations between variables are listed in Table 12. We found a negative association between negative affect and self-control, indicating that higher levels of negative affect relate to lower self-control. Of particular importance were the correlations with the distraction index and the attentional interference index. We did not find any significant association with these indices.

Table 12

Means (M), Standard Deviations (SD) and Pearson Correlation Coefficients for All Measures

	<i>M</i>	<i>SD</i>	2	3	4	5	6	7	8
1. Anxiety sensitivity (ASI)	25.00	9.63	.298 [†]	-.014	.304 [†]	-.162	-.176	-.200	-.040
2. Pain Catastrophizing (PCS)	20.59	8.78		-.004	-.014	-.051	-.217	-.188	.035
3. PA (PANAS)	27.87	5.43			.074	-.134	.031	-.187	-.313 [†]
4. NA (PANAS)	13.62	4.42				-.345 [*]	.253	-.117	-.060
5. Self-control (BSCS)	41.41	5.80					-.044	.115	.309 [†]
6. HRV (RSAnorm)	0.75	0.30						-.093	-.125
7. distraction index	1.67	4.19							.092
8. interference index	66.94	104.25							

Note. ASI = Anxiety Sensitivity Index, PCS = Pain Catastrophizing Scale, PANAS = Positive And Negative Affect Schedule, PA = positive affect, NA = negative affect, BSCS = Brief Self-Control Scale, HRV = heart rate variability, RSAnorm = normalized respiratory sinus arrhythmia, [†] $p < .10$, ^{*} $p < .05$.

Distraction efficacy

Analyses were performed on trials during which a correct response was given and on the ratings of the electrocutaneous stimuli only. The efficacy of distraction was examined by comparing the pain ratings on focus trials and distraction trials for high and low pain stimuli with a higher index indicating that distraction was more efficacious. We conducted a two (focus, distraction) by two (high, low intensity) multivariate repeated-measures ANOVAs with pain ratings (intensity, unpleasantness) as dependent variables. Firstly, analyses revealed overall significant effects for condition (focus vs. distraction), $F(2,37) = 3.45$, $p < .05$, $\eta^2 = .16$, and stimulus intensity (high pain vs. low pain), $F(2,37) = 24.03$, $p < .001$, $\eta^2 = .57$, on self-reported pain ratings. In a next step, we looked separately at pain intensity ratings and pain unpleasantness ratings.

Regarding pain intensity ratings, the analysis revealed a significant effect for condition (focus vs. distraction), $F(1,38) = 6.23$, $p < .05$, $\eta^2 = .14$, and for stimulus intensity (high pain vs. low pain), $F(1,38) = 35.77$, $p < .001$, $\eta^2 = .49$. There was no interaction effect, $F(1,38) = 0.01$, $p = .909$, $\eta^2 = .00$. Regarding pain unpleasantness ratings, the analysis revealed a non-significant effect for condition (focus vs. distraction), $F(1,38) = 2.10$, $p = .155$, $\eta^2 = .05$, and a significant effect for stimulus intensity (high pain vs. low pain), $F(1,38) = 9.29$, $p < .001$, $\eta^2 = .57$. There was no interaction effect, $F(1,38) = 0.16$, $p = .691$, $\eta^2 = .00$. Results are illustrated in Figure 4.

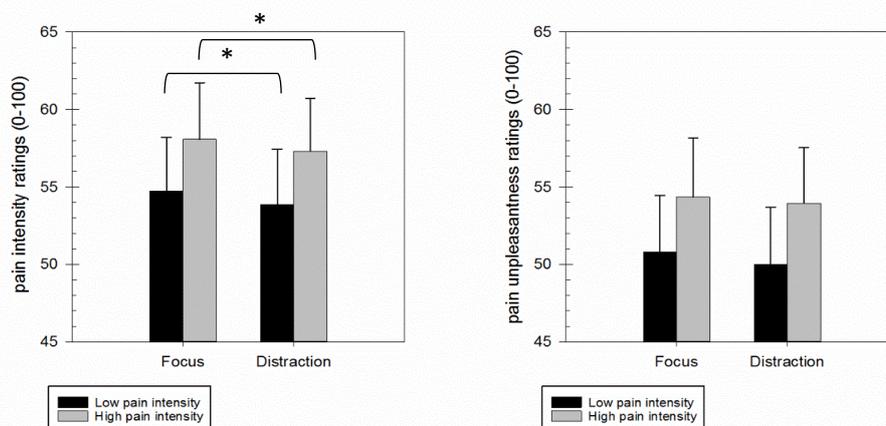


Figure 4. Distraction efficacy for high and low pain intensity stimuli.

Attentional interference by pain

Analyses were carried out on the reaction times in the distraction trials with correct responses only (98.5%). Attentional interference was examined by comparing the reaction times between distraction trials with high and low pain intensity, and vibrotactile stimuli. A one-way repeated-measures ANOVA with contrasts was run. Mean reaction times were 518.08 ms ($SD = 172.91$) for trials with high pain intensity, 493.87 ms ($SD = 140.54$) for trials with low pain intensity and 451.14 ms ($SD = 95.24$) with vibrotactile stimuli. Mauchly's test indicated that the assumption of sphericity had been violated, $\chi^2(2) = 22.68, p < .001$, therefore the Greenhouse-Geisser corrected tests are reported ($\epsilon = .69$). The results show that reaction times were significantly affected by the type of trial, $F(1.37, 52.12) = 13.67, p < .001, \eta^2 = .26$. Planned contrast analyses revealed that reaction times were significantly higher in trials with high pain than with low pain intensity [$F(1,38) = 6.56, p < .05$] and vibrotactile stimuli [$F(1,38) = 16.08, p < .001$]. Results are illustrated in Figure 5.

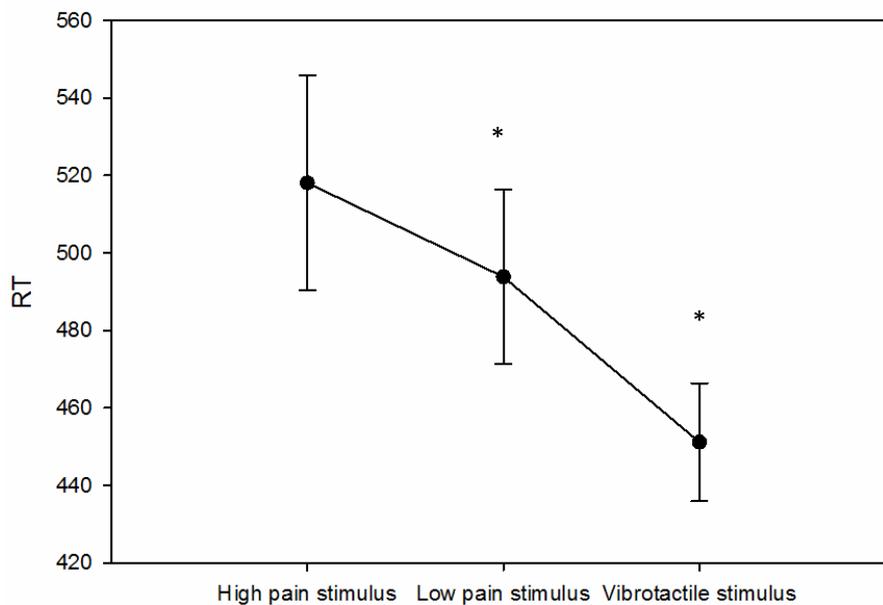


Figure 5. Attentional interference by pain measured with reaction times (RT) in distraction trials with high and low intensity pain stimuli, and vibrotactile stimuli.

Predictability of distraction efficacy and attentional interference by heart rate variability

Separate regression analyses were carried out to investigate the predictive power of HRV and self-control for distraction efficacy and attentional interference. No control variables were entered. The models were not significant ($R^2 = .02$, $p = .681$ for distraction efficacy and $R^2 = .11$, $p = .129$ for attentional interference) and are shown in Table 13.

Table 13

Regression Analyses Explaining Distraction Index and Attentional Interference Index, with Standard Errors and 95% Confidence Intervals Reported in Parentheses

Distraction index		<i>b</i>	<i>SE B</i>	β	<i>p</i>
	HRV	-1.22 (-5.83, 3.40)	2.27	-.09	.596
	Self-control	0.08 (-0.16, 0.32)	0.12	.11	.504
Attentional interference index					
	HRV	-38.31 (-147.91, 71.92)	52.04	-.11	.483
	Self-control	5.45 (-0.29, 11.20)	2.83	.30	.062

Note. HRV = heart rate variability.

As the correlation between PA and international interference was marginally significant ($p < .10$), we carried out an additional regression analysis in which we controlled for PA by entering it in a first step. There was no significant effect when adding HRV and self-control in a second step ($\Delta R^2 = .08$, $p = .184$), as shown in Table 14.

Table 14

Regression analysis explaining attentional interference index when controlling for positive affect, with standard errors and 95% confidence intervals reported in parentheses

Attentional interference index		<i>b</i>	<i>SE B</i>	β	<i>p</i>
Step 1					
	PA	-6.01 (-12.09, 0.06)	3.00	-.31	.052
Step 2					
	PA	-5.26 (-11.28, 0.76)	2.96	-.27	.085
	HRV	-35.95 (-142.55, 70.66)	52.51	-.11	.498
	Self-control	4.80 (-0.84, 10.43)	2.78	.27	.093

Note. $R^2 = .10$ for step 1, PA = positive affect, HRV = heart rate variability.

Discussion

The aims of the current study were (1) to assess distraction efficacy, (2) to assess attentional interference by pain, and (3) to investigate the predictive value of self-regulatory capacity as indexed by HRV and self-reported self-control for distraction efficacy and attentional interference. Firstly, participants reported lower pain during the distraction trials compared with the focus trials, indicating that the distraction was efficient. Secondly, reaction times in the distraction trials were significantly longer when a stimulus of high pain intensity was presented than when a stimulus of low pain intensity or a vibrotactile stimulus was presented, indicating attentional interference by pain. Thirdly, neither HRV nor self-reported self-control predicted distraction efficacy or attentional interference by pain.

In line with our expectations and with previous research (Petrovic et al., 2000; Valet et al., 2004; Van Ryckeghem et al., 2012), participants reported less pain during trials in which they were asked to localize the visual stimulus than during trials in which they were asked to focus on the somatosensory stimulus. The decrease in pain ratings during a distraction task has previously been shown to relate to the intensity aspect rather than to the affective aspect of pain (Verhoeven et al., 2010, 2011). The neurocognitive model of attention to pain distinguishes between the involuntary bottom-up capture of pain and top-down modulation processes (Legrain et al., 2009). One way of investigating top-down modulation of pain is distraction, which was found to be accompanied by altered activity in pain-related brain areas (Petrovic et al., 2000). According to the neurocognitive model of attention (Legrain et al., 2009), the modulation processes depend on working memory. On the one hand, the efficacy of a distraction task depends on the degree to which task-relevant features are distinct from pain-related features (attentional set hypothesis, e.g. Van Ryckeghem et al., 2013). On the other hand, the degree to which a task demands attention determines the extent of pain reduction during the task (attentional load hypothesis, e.g. Romero, Straube, Nitsch, Miltner, & Weiss, 2013). Distraction has been shown to be less efficacious for pain stimuli high in intensity as these draw more attention and interrupt more easily ongoing tasks (Eccleston & Crombez, 1999; Seminowicz & Davis, 2007; Van Ryckeghem et al., 2012). Although not the focus of the current study, we did not find an interaction between stimulus intensity (high vs. low pain intensity) and condition (distraction vs. focus). This may be explained by the relatively small difference in intensity between the two electrocutaneous painful stimuli.

We were also able to demonstrate attentional interference by pain as indicated by delayed reaction times in the presence of pain stimuli. The interruptive characteristic of pain has been described previously (Crombez, Eccleston, Baeyens, & Eelen, 1996, 1998; Eccleston & Crombez, 1999; Vancleef & Peters, 2006). It alerts the individual about a potential threat. Research suggests that the degree to which attention is captured by pain depends on the intensity of pain, i.e. it increases when pain is more intense (Eccleston & Crombez, 1999; Van Ryckeghem et al., 2012). Interestingly, we found significant differences in reaction times not only between the trials in which high pain intensity stimuli and non-painful vibrotactile stimuli were presented, but between all three types of trials. This finding confirms that more intense pain interrupts ongoing processes more easily and suggests that attentional interference by pain may be susceptible to slight changes in pain intensity.

Contrary to our hypotheses, we did not find a predictive effect of self-regulatory capacity on distraction efficacy and attentional interference. We expected to find vagal activity and self-reported self-control to influence attentional selection based on previous findings demonstrating the influence of inhibitory control in attention regulation (Hansen, Johnsen, & Thayer, 2003; Johnsen et al., 2003; Kaufmann et al., 2012). Verhoeven and colleagues (2011) investigated the role of executive functioning, i.e. inhibition, task switching and working memory, for distraction efficacy. Although general executive functioning abilities were unrelated to distraction efficacy, participants with better inhibition abilities endured pain for a longer time. The authors suggest that inhibition abilities may be of importance for the efficient engagement with a distraction task rather than for the decrease of pain experience during the distraction task. The present findings confirm that inhibitory capacity, as indexed by HRV and self-reported self-control, does not relate to distraction efficacy. In contrast to Verhoeven and colleagues (2011), however, we did not find an association between inhibitory capacity and attentional interference by pain, i.e. task performance.

Several factors may explain this null finding. Firstly, the current sample consisted only of students who were generally fit and healthy. As HRV depends on developmental (e.g. age) and physiological factors (e.g. cardiorespiratory fitness), this may have restricted the range of HRV, thereby masking meaningful associations. Secondly, HRV may not be sensitive enough as a measure to index a cognitive trait, i.e. general inhibitory capacity. Thirdly, other factors may have played a more important role. For example, attentional bias towards pain-related information reduces the efficacy of a distraction technique (Van

Ryckeghem et al., 2012), whereas motivation has been shown to increase engagement in a distraction task (Verhoeven et al., 2010).

To complement the physiological measure of self-regulatory capacity, we also assessed self-control via questionnaire, but with similar null findings. One explanation for this lack of associations may relate to the assessment via a general questionnaire (BSCS). It is possible that the BSCS (Tangney et al., 2004) lacks specificity to the experimental setup with a specific task and, thus, the association with the ability to inhibit the predominant response of attending to pain and to resist being interrupted by pain, may have been masked. Ajzen (1988) described this problem of lacking compatibility between measurements as having a detrimental effect on the size of associations between measurements. The lack of association between the psychophysiological index and self-reported self-regulation does not surprise. According to the neurovisceral model of integration (Thayer & Lane, 2000, 2009), HRV reflects the flexibility and adaptability of an organism, which depends on prefrontal inhibitory functioning. Questionnaires, however, represent individual (meta-cognitive) beliefs that are commonly biased by social desirability and other factors (Van de Mortel, 2008). Thus, it is unlikely that self-regulatory capacity as assessed by HRV and self-report reflect the same aspects of one construct.

This study has some limitations. Firstly, this study was performed in a well-controlled environment using experimentally induced pain and one should be cautious in generalizing the results to other settings. Secondly, our sample was homogenous and consisted only of healthy students. Results cannot necessarily be generalized to other populations. Thirdly, the sample mainly consisted of women, so that we could not investigate gender differences. Fourthly, future studies may assess self-reported self-control in the specific context of pain.

To conclude, self-regulatory capacity does not seem to be related to the efficacy of distraction or attentional interference. It may be useful to investigate inhibitory indicators which are more specific to the regulation of pain, consider factors such as attentional bias and motivation, and extend research to natural contexts.

Acknowledgment

This research was supported in part by the grant “Self-Regulation and Pain” (SEREPA; University of Luxembourg) to the senior author.

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GENERAL DISCUSSION

In this general discussion, the main findings are highlighted and integrated, and clinical implications and limitations are discussed. Finally, possible avenues for future research are proposed.

The purpose of the current project was to investigate the role of self-regulatory capacity for chronic pain, such as in fibromyalgia (FM). This condition is well suited to investigate the role of self-regulatory capacity, as it is characterised not only by physical complaints, but also by daily challenges associated with psychological distress, fatigue and cognitive disturbances (Choy et al., 2009; Mease et al., 2007, 2009).

Previously, it has been suggested that self-regulatory capacity plays a crucial role in the adaptation to daily pain, but evidence on the potential mechanisms involved is scarce. To provide a broad perspective, studies 1 and 2 focussed on the regulation of emotional processes, using diary designs in clinical populations providing the advantage of high ecological validity. Studies 3 and 4 employed experimental settings to allow for a more in- depth analysis of mechanisms related to the role of regulatory capacity in attentional processes.

Main findings

The primary aim of **study 1** was to investigate the role of affective instability for pain and daily pain outcomes in chronic pain patients. Affective instability is conceptualized as frequent fluctuations in mood over time (Jahng, Wood, & Trull, 2008) and has been related to ill-health (Ebner-Priemer & Trull, 2009; Houben, Noortgate, & Kuppens, 2015). Patients completed questions regarding daily pain severity, disability, cognitive complaints as well as positive affect (PA) and negative affect (NA) every evening on 14 consecutive days. We used an innovative measure of affective instability, i.e. the mean square of successive differences (MSSD), incorporating both variability (i.e. average magnitude) and temporal dependency (i.e. average frequency) of affective changes (Jahng et al., 2008). This study is the first to demonstrate the role of NA instability in the adjustment to chronic pain. The findings show that not only the intensity, but also the temporal variability of NA is related to daily pain outcomes. This means that chronic pain patients who experience higher levels of NA instability report poorer daily functioning in general, i.e. more disability and cognitive complaints. On days where they experience higher than usual levels of pain, they even experience greater declines in general functioning. The distinction between intensity and temporal variability completes the

picture of dynamically fluctuating emotions in daily life and demonstrates the importance of broadening the perspective to better understand the daily emotional experiences of chronic pain patients.

Study 2 builds on these findings and investigates a possible underlying mechanism by adding a psychophysiological index of regulated emotional responding, i.e. vagal cardiac control as indexed by HRV. In addition to the replicating the findings of study 1, this study aimed to compare affective instability between FM patients and healthy controls, and to investigate the link between vagal activation and dysregulated emotion, i.e. affective instability. Firstly, and in line with studies demonstrating the relationship between NA instability and psychopathology (Thompson et al., 2012; Trull et al., 2008) as well as psychological well-being (Houben et al., 2015), FM patients showed higher levels of NA instability than healthy controls. It may well be that persistent pain and emotion dysregulation share underlying mechanisms serving as a driver for problems of emotion and pain regulation (Flink, Boersma, & Linton, 2013; Linton, 2013). Secondly, we found an association between vagal activation and NA instability in both patients and healthy controls, indicating that HRV may serve as a physiological indicator of affective instability (Koval et al., 2013). It is therefore reasonable to assume that vagal activation in patients is mostly associated with the down-regulation of NA. Thirdly, findings from study 1 on the role of NA instability in daily pain outcomes were only partially replicated. The lack of moderation effects of affective instability between daily pain and daily pain outcomes may be due to a lack in statistical power. In summary, these findings suggest that targeting NA instability in the psychological treatment of chronic pain may be beneficial to provide patients with tools to better adapt to their conditions.

With **study 3**, the focus moved from the regulation of emotions to the regulation of attention, which is typically associated with the processing of pain. This study aimed to investigate hypervigilance in FM. Hypervigilance constitutes a top-down process, which facilitates the involuntary capture of attention by pain and is, therefore, suggested as an underlying mechanism for symptom perception. Based on the generalized hypervigilance hypothesis (GHH; Hollins et al., 2009; McDermid, Rollman, & McCain, 1996), we expected FM patients to be hypervigilant to internal bodily signals as reflected in interoceptive accuracy. Moreover, we hypothesized hypervigilance to reflect a deficiency in inhibitory control (Carrillo-de-la-Peña, Vallet, Pérez, & Gómez-Perretta, 2006) and therefore to be inversely associated with HRV. Participants were asked to silently count their heartbeats to calculate an index of interoceptive accuracy. In line with previous research (Cohen et al., 2000; Raj, Brouillard, Simpson, Hopman, & Abdollah, 2000), groups differed in HRV in

that FM patients showed lower HRV. Patients reported higher body vigilance than healthy controls, but there were no group differences in interoceptive accuracy. These results do not support the GHH (Hollins et al., 2009; McDermid et al., 1996), but suggest that hypervigilance is not a general characteristic, but a dynamic process which is activated under specific conditions. For example, hypervigilance may only occur in the context of pain or threat (Crombez, Van Damme, & Eccleston, 2005).

Study 4 focussed on distraction as an inhibiting attentional mechanism, which is one of the most frequently used coping strategies. Previous research suggests that distraction efficacy depends on underlying mechanisms such as pain catastrophizing (Verhoeven et al., 2010) or inhibitory control (Friedman & Miyake, 2004; Nigg, 2000). This study aimed to validate HRV as an index of self-regulation capacity in an experimental setting involving a distraction paradigm. Participants were asked to localize either a visual (distraction trial) or a somatosensory (electrocutaneous or vibrotactile) stimulus (focus trial), while both stimuli were presented in each trial. Participants were then asked to rate pain intensity and pain unpleasantness of the stimuli. The aims of the study were threefold. Firstly, we expected that pain ratings would be lower when attention was directed away from pain than when it was directed towards pain. Secondly, we expected reaction times for localizing the visual signal to be longer in the presence of pain. Thirdly, we hypothesized that self-regulatory capacity would be positively associated with distraction efficacy and negatively associated with attentional interference. Results showed an effect of the distraction task on self-reported pain intensity, but not pain unpleasantness, which is in line with previous studies (Verhoeven et al., 2010, 2011). Further we found attentional interference indicated by delayed reaction times in the presence of pain. Interestingly, the delay increased with pain intensity. Contrary to our expectations, we did not find an association between self-regulatory capacity as indexed by HRV or self-report and distraction efficacy or attentional interference. These findings suggest that the indicators of self-regulation capacity used in this study may lack sensitivity or specificity in this controlled experimental setting and motivate future research to assess indicators for regulation in the context of pain.

Integrative discussion

The purpose of the current project was to adopt a self-regulatory perspective in the investigation of emotion and attention regulation involved in the experience of pain and consequently the adaptation to pain. Findings expand on previous research in a number of ways.

The disentanglement of pain and emotions remains a challenge for research, and the question of their relationship is an intriguing scientific problem, which has generated abundant research. The challenge to better understand the dynamic interplay between emotions and pain may require the shift from a static (i.e. questionnaire) to a more flexible and dynamic (i.e. diary methodology, ecological momentary assessment) approach. In the past two decades, research linking various patterns of short-term emotional change to adaptive or maladaptive psychological functioning has surged. Numerous studies have shown that not only how one feels on average, but also how one's emotions fluctuate across time is crucial for mental health and well-being (e.g. Selby et al., 2012; Thompson et al., 2012; Trull et al., 2008). Interestingly, specific patterns of emotional fluctuations across time have been identified as characteristic for psychological well-being or psychopathology, one of which is NA instability (Houben et al., 2015). This dynamic approach has provided insight into what constitutes normative emotional development, has helped to identify risk factors and has improved the understanding of mental disorders (Ebner-Priemer & Trull, 2009; Houben et al., 2015; Maciejewski, Lier, Branje, & Koot, 2015; Selby et al., 2015). The current results add to these findings by expanding research towards a population predominantly characterized by physical complaints. The results confirm the notion that temporal variability captures another aspect than average levels of emotions in that mean levels of PA and NA did not relate to instability levels of PA and NA. Measures of affective instability add to average levels of emotions by capturing the variability (i.e. average magnitude) and temporal dependency (i.e. average frequency) of emotions. Moreover, we demonstrated that (1) NA instability relates to daily disability and cognitive complaints, (2) NA instability moderates the association between daily pain and its outcomes, (3) NA instability is increased in chronic pain patients compared with healthy controls, and (4) NA instability is related to HRV in FM patients and healthy controls. These results extend previous findings by demonstrating that NA instability is crucial not only for mental but also physical health. There is further evidence from the current data that HRV and affective instability, particularly NA instability, may constitute complementary indices of emotion regulation capacity. HRV was proposed as an indicator

for the degree to which cardiac activity can be modulated by prefrontal inhibition to meet changing situational demands, thus, reflecting behavioural and cognitive flexibility as well

as adaptive emotion regulation (Appelhans & Luecken, 2006; Thayer & Lane, 2000, 2009). Higher levels of depression, anxiety and stress may point to a higher need for the down-regulation of NA in chronic pain patients, therewith explaining the lack of association between HRV and PA instability. In summary, these results underline the importance of taking into account the dynamic nature of emotions in order to capture the complexity of emotional experiences and their impact on daily general functioning of chronic pain patients. These findings may further contribute to the development of more tailored therapeutic strategies by targeting the regulation of NA.

While studies 1 and 2 addressed new research questions in regard to the role of regulatory capacity in daily general functioning and emotional functioning of chronic pain patients, study 3 investigated the role of self-regulatory capacity in hypervigilance, a top-down attentional process related to symptom perception in FM. The aetiology of FM is not fully understood, but clinical observations suggest that patients are characterized by excessively attending to pain-related information, i.e. are hypervigilant to pain stimuli. In spite of extensive research on this potential top-down facilitation mechanism, current results on the question whether patients are indeed hypervigilant are inconsistent. In line with the assumption of generalized hypervigilance in FM there is evidence for increased levels of body and pain vigilance (on self-report measure), and decreased pain thresholds and tolerance levels for experimentally induced pain and innocuous external stimuli (Crombez, Eccleston, Van den Broeck, Goubert, & Van Houdenhove, 2004; Hollins et al., 2009; Kosek, Ekholm, & Hansson, 1996; McDermid et al., 1996; Peters, Vlaeyen, & van Drunen, 2000). Nevertheless, there are some methodological problems with this research. Most results rely on self-report measures, which are prone to be biased by non-attentional factors. These measures may therefore reflect physical complaints rather than detect excessive body vigilance (Van Damme et al., 2009). Also, experimental studies have primarily focused on the perception of external stimuli which may not be directly relevant for bodily perception. Symptom perception may be more closely related to the perception of internal signals, but research in this area is few and far between. The results of study 3 suggest that there are no differences in interoceptive accuracy between FM patients and healthy controls. We have provided some arguments why a more dynamic view of hypervigilance should be adopted, as it seems context dependent and modality-specific rather than a general and stable characteristic of FM patients. Moreover, it may be

reasonable to assume that hypervigilance may be related to a tendency to over-report internal signals¹. For example, Brown and colleagues (2012) have reported a link between physical symptom reporting and the tendency to experience somatosensory distortion. In this study, participants completed a somatic signal detection task in which somatosensory distortion was measured by the false alarm rate, i.e. the frequency of illusory touch experiences. Thus, it may be useful to not only calculate the absolute accuracy of signal detection, but to also examine false alarms, i.e. counting heartbeats where there are none. An over-report would indicate a bias to interpret noise as behaviourally relevant signal. It may also be worth to consider other measures of accurate bodily perception. As such, measuring nonspecific skin conductance fluctuations is another innovative approach to bodily signal detection allowing to assess false reports (Krautwurst, Gerlach, Gomille, Hiller, & Witthöft, 2014). These are indicators of phasic autonomic arousal based on the activity of the sympathetic nervous system (Andor, Gerlach, & Rist, 2008). The perception of physiological changes may closely relate to somatosensory amplification. Krautwurst and colleagues (2014) reported a bias to overestimate nonspecific skin conductance fluctuations in individuals with increased health anxiety. This approach may help to answer the question whether hypervigilance leads to perceptual amplification through a reinterpretation of noisy signals, and whether health anxiety may modulate this relationship.

In line with previous studies, we found lower HRV in FM patients (Cohen et al., 2000; Raj et al., 2000) compared with healthy controls. This finding can be expanded according to Johnsen and colleagues (2003), who found that inefficient attentional regulation, and more specifically hypervigilance, is associated with lower HRV. These different factors may contribute to a downward spiral, gradually complicating patients' life and impeding their well-being. One must, however, be cautious with implying causal relationships as our results are based on cross-sectional analyses.

In contrast to studies 1-3, which were clinical studies, study 4 used a purely experimental design with healthy students, and focussed on self-regulatory capacity as one possible underlying mechanism for distraction efficacy. Distraction can be defined as an attentional strategy to cope with pain in which attention is directed away from pain. It is one of the most commonly used coping strategies of chronic pain patients (Johnson, 2005), and found in diverse treatment programs for acute and chronic pain (Elomaa, Williams, & Kalso, 2009; Morley, Shapiro, & Biggs, 2004). We found that distraction was efficacious as participants indicated lower pain intensity in distraction trials compared with focus trials. As findings regarding the efficacy of distraction are, however, inconsistent, theoretically

¹We thank an unknown reviewer for this comment.

driven research is important. Several factors have been suggested as possible underlying mechanisms, such as inhibitory control (Friedman & Miyake, 2004; Nigg, 2000). In a previous study inhibition capacity was associated with lower attentional interference, but not related to the pain-reducing effects of distraction (Verhoeven et al., 2011). We could not confirm this relationship. Several factors may account for this null finding. For example, HRV may lack sensitivity as a measure to reflect general inhibitory capacity. Alternatively, or additionally, other factors such as motivation (Verhoeven et al., 2010) may have played a more important role. Noteworthy, however, is that numerous studies point to the direct link between HRV and pain experience by reporting pain-reducing effects achieved with HRV biofeedback training (Hallman, Olsson, von Schéele, Melin, & Lyskov, 2011; Hassett et al., 2007; Sowder, Gevirtz, Shapiro, & Ebert, 2010; Tan et al., 2009).

Generally, the findings of the current project correspond to the interactive view of the adjustment to pain by the dynamic interaction of self-regulatory capacity and self-regulatory demands as well as pain, as proposed by Solberg Nes and colleagues (2009). The authors suggest that excessive self-regulatory demands, such as stress or pain, can fatigue self-regulatory capacity. We indeed found decreased levels of HRV in FM patients as compared to healthy individuals, which is in line with previous research (Cohen et al., 2000; Raj et al., 2000). The question of causality between vagal activation and pain experience, however, cannot be answered with these results. It is also possible that pain conditions affect the cardiovascular system and thereby lead to changes in HRV. Future research should be encouraged to target this question and shed light on putative mediating mechanisms. The results of studies 1 and 2 illustrate the relationship between daily pain and the capacity to regulate emotions. They indicate that pain interacts with levels of NA instability to affect daily general functioning as measured by disability and cognitive complaints. Moreover, higher levels of NA instability in FM patients indicate problems in satisfactorily meeting the demands of regulating NA (Carpenter & Trull, 2013; Selby et al., 2015). The results of studies 3 and 4 add to the picture by illustrating how pain interferes with the capacity to regulate attention either by reporting excessive focus on bodily sensations (study 3) or by being interrupted in a visual task (study 4). The model further elaborates the role of executive functions, which is outside the scope of the current project.

Clinical implications

The current findings may have several implications for the treatment of chronic pain patients. Firstly, it may be an important strategy for clinicians to target the regulation of affect, specifically NA, to support patients in acquiring new tools to better adapt to their conditions. One could think of several ways to do so. For patients who report deficits in certain regulation skills, clinicians could include the training of these skills. One example of such an intervention could be based on the Affect Regulation Training, which is a module-based non-disorder-specific intervention to improve emotion regulation skills (Berking & Whitley, 2014). The authors stress the importance of targeting general emotion regulation skills and report that enhanced emotion regulation skills facilitate treatment gains in other areas (Berking et al., 2008). The training is a transdiagnostic intervention, in which specific emotion regulation skills are conceptualized based on the available literature. The training approach is based on a model that conceptualizes adaptive emotion regulation as a situation-dependent interaction of several emotion regulation skills (Berking & Schwarz, 2014):

1. be aware of emotions

This skill is a fundamental advantage as it allows for automatized regulation processes which are likely to dominate the daily routine of regulation (Koole & Rothermund, 2011). If, however, undesired affective states cannot be regulated by implicit processes, an individual resorts to effortful attempts to regulate. The awareness of emotional states facilitates the use of conscious regulation strategies. It may be reasonable to assume that chronic pain patients encounter difficulties in disentangling negative affective states from pain experiences so that it may already be a challenge for them to be aware of whether they are in pain or experience NA.

2. identify and label emotions

The authors describe this skill as the match of emotional experiences with appropriate semantic categories (e.g. "What I feel now is sadness"). An individual can then build knowledge about this state (e.g. nature and purpose of the emotion) and on potentially effective regulation strategies (e.g. distraction, reappraisal) to facilitate adaptive regulation. Chronic pain patients could learn to better differentiate between the numerous NA states they experience in everyday life.

3. identify relevant maintaining factors

This skill refers to the development of an inner working model to explain why an affective state was cued and why it is maintained. The purpose of understanding factors that initially elicit and maintain affective states helps is threefold. Firstly, one can give meaning to an aversive experience, thereby making it easier to bear. Second, one can clarify whether the emotion can be changed. Third, one can identify targets to effectively modify the emotion. In case of persistent pain, one main objective would be to identify pain as eliciting and maintaining factor for NA. If being able to differentiate between NA cued by pain or by other situations, patients could then more easily opt for an appropriate regulation strategy.

4. modify affective states actively

An individual who is able to effectively change the features of an emotion (e.g. intensity, duration) benefits from emotional self-efficacy reducing anxiety and avoidance tendencies. These modification skills are commonly found in behavioural therapy for depression (e.g. positive activities). This skill could be crucial for chronic pain patients as their pain experiences are generally accompanied by NA (Potter, Zautra, & Reich, 2000). Emotions like hopelessness and frustration could be targeted to allow for patients to engage in positive activities again.

5. accept and tolerate NA states

This skill is an advantage in situation in which emotions cannot be modified (Hayes, Strosahl, & Wilson, 1999) and reduces the risk to engage in maladaptive affect regulation (Wupperman, Neumann, & Axelrod, 2008). Chronic pain patients may easily get frustrated because they cannot change the cause of their NA. It may be an important strategy for them to learn a more tolerating and accepting attitude to replace the desire to control pain.

6. confront emotionally distressing situations

Approaching situations that may cue NA states is often necessary to accomplish personally relevant goals (Hayes et al., 1999). By doing so, individuals can further improve existing regulation skills and even develop new ones. This skill may be particularly important for chronic pain patients to overcome NA associated with physical activity for example.

7. support oneself in distressing situations

Self-soothing strategies are crucial to reduce self-criticism that is often the cause of NA states. This is an important aspect to better deal with disappointment and stress. Patients learn how to provide compassionate self-support and can thereby reduce a vicious circle of NA states, non-achievement of set goals and self-blame. They could thereby learn how to prevent too intense and too frequent fluctuations of emotions and thereby reduce helplessness when faced to their NA. This may be an important first step to support them in self-efficacy and reduce physical and cognitive consequences of pain.

Interestingly, the authors developed a questionnaire to evaluate these emotion regulation skills (Berking & Znoj, 2008). Psychotherapy patients seem to benefit from this training and particularly the skills to tolerate and actively moderate NA (Berking et al., 2008), which could be of relevance also for chronic pain patients. Overall, this training provides a promising transdiagnostic approach, which can be considered an adjunct intervention. According to the findings of the current project, the administration of daily mood assessments during the first weeks of treatment would be a useful tool for clinicians to develop a more accurate picture of the emotional fluctuations and improve treatment planning (Solhan, Trull, Jahng, & Wood, 2009).

In case that patients demonstrate rigid overregulation, mindfulness-based trainings (Grossman, Tiefenthaler-Gilmer, Raysz, & Kesper, 2007; Rosenzweig et al., 2010) or Acceptance and Commitment Therapy (ACT; Hayes et al., 1999; McCracken, Vowles, & Eccleston, 2005; Vowles, McCracken, & O'Brien, 2011) may be promising approaches. These approaches are not completely separable from the Affect Regulation Training (Berking & Whitley, 2014), but they focus more on skills related to awareness, acceptance and self-soothing.

Mindfulness-based stress reduction (MBSR) is a group intervention that appears to be a promising approach for the treatment of chronic pain (Grossman, Niemann, & Schmidt, 2004). It consists of an intensive training in mindfulness meditation and its applications for daily life, such as coping with stress, pain and illness (Kabat-Zinn, 1990). The purpose is for patients to progressively acquire a mindful awareness of internal states, which is non-judgemental and nonreactive (Grossman et al., 2007). This may be particularly useful in regard of NA states, which are intertwined with persistent pain. Studies investigating early treatment effects and follow-up outcomes demonstrate improvements in pain intensity, somatic complaints, psychological distress, well-being and quality of life, sleep quality and fatigue (Grossman et al., 2007; Kaplan & Goldenberg,

1993; Pradhan & Baumgarten, 2007; Rosenzweig et al., 2010; Sephton, Salmon, & Weissbecker, 2007). The beneficial effects of MBSR interventions are primarily mediated through the self-regulation of attention, which aim to modulate the sensory and affective components of pain via central nervous system pathways (Cahn & Polich, 2006; Creswell, Way, Eisenberger, & Lieberman, 2007; Kingston, Chadwick, Meron, & Skinner, 2007; Lutz, Slagter, Dunne, & Davidson, 2008). Moreover, mindfulness meditation promotes muscle relaxation and enhances body awareness, which possibly leads to improved self-care (Greeson, 2009; Rosenzweig et al., 2010) and reduces physical complaints. Finally, mindfulness aims to reduce reactivity to distressing thoughts and feelings and may act as a buffer against stress-related mood dysfunction (Baar, 2003; Garland, Gaylord, & Park, 2009).

Similarly, acceptance-based treatments for chronic pain patients differ from usual treatments as they target on the reduction of distressing and disabling effects of pain rather than on reducing pain itself (McCracken et al., 2005). As chronic pain is typically aversive and difficult to control, patients may rather learn to face uncomfortable thoughts and feelings and to focus their efforts on behaviours that improve functioning (McCracken et al., 2005). Overall effects have been investigated in a meta-analysis, which revealed small to medium effects sizes on physical and mental health that are comparable to Cognitive Behavioural Therapy (Veehof, Oskam, Schreurs, & Bohlmeijer, 2011). More specifically, numerous studies show short-term and long-term effects such as improvements in emotional and physical functioning, i.e. physical performance measures (Vowles et al., 2011; Vowles & McCracken, 2008), reduction of sick leave and healthcare (Dahl, Wilson, & Nilsson, 2004; McCracken & Vowles, 2008) and changes in psychological distress and disability (McCracken et al., 2005), even when controlling for changes in pain intensity and catastrophic thinking (Vowles, McCracken, & Eccleston, 2007). ACT is not only implemented in treatment programs for chronic pain, but shows comparable effects in the treatment of mental disorders such as depression, anxiety or psychosis (Powers, Zum Vörde Sive Vörding, & Emmelkamp, 2009). The overall goal of ACT is to achieve psychological flexibility (Hayes et al., 1999), which has been related to resilience and good mental health (Kashdan & Rottenberg, 2010). Consequently, it may be reasonable to focus on different aspects of psychological flexibility to increase mental health and the adjustment to chronic pain conditions. One crucial facet of psychological flexibility has been addressed before and refers to emotional flexibility, i.e. producing adequate responses to emotion-eliciting events (Waugh, Wager, Fredrickson, Noll, & Taylor, 2008;

Westphal, Seivert, & Bonanno, 2010). Likewise, it may be useful to address the reduced physiological flexibility as indicated by lower levels of HRV in FM patients.

HRV could be targeted by a behavioural intervention such as biofeedback training. The central focus of HRV biofeedback is to increase the amplitude of respiratory sinus arrhythmia by breathing at one's individual resonant frequency (Lehrer, Vaschillo, & Vaschillo, 2000). Feedback usually is given in the form of visual signals derived from electrocardiographic recordings. Some studies have demonstrated that improving suboptimal HRV had (1) immediate effects on HRV, and (2) delayed effects on other cardiac parameters such as blood pressure and baroreflex as well as psychological effects such as facilitated adjustment to the chronic pain condition (Hallman et al., 2011; Hassett et al., 2007; Nolan et al., 2005). HRV biofeedback trainings rely on a technique which has no side effects and is easily learned and accepted by patients (Hassett et al., 2007). It would certainly be a gain to measure HRV in clinical practice to identify patients who would benefit most from this complementary intervention.

An interesting additional integrative approach may be based on physical self-regulation (Sauer, Burris, & Carlson, 2010). The authors proposed an integrative intervention aiming at the improvement of self-regulatory strength and leading to decreases of both physical and psychological symptoms. The intervention includes guidelines for improving sleep, the change of behavioural factors associated with long-term pain (e.g. monitoring and reducing muscle para-function, relaxation), instructions related to physical activity, diet and fluid intake as well as diaphragmatic breathing entrainment (i.e. number of breaths per minute and solely rely on diaphragmatic function during inspiration). This intervention may help patients to have a set of diverse tools at their disposal, aiming at overall better adaptation to pain.

Limitations

The current findings make a significant contribution to present research investigating the underlying mechanisms in the adaptation to pain. Nevertheless, the inclusion of further measures would have completed these studies. In the diary studies, for example, we did not include a measure of external events independent of subjective states (Zautra & Sturgeon, 2016). Such events, however, can affect the experience and regulation of pain, PA and NA, cognitions and disablement (Zautra & Sturgeon, 2016). Assessing these events would have shed light on the role of external factors contributing

to emotional experiences and the adjustment to daily pain. It may also be useful to integrate putative mediating variables such as emotion regulation strategies used, indicators of failed regulation such as perseverative cognitions (i.e. worry and rumination) or other variables known to influence levels of pain-related disability (e.g. pain-related fear) to gain a more complete understanding of underlying processes. We only focused on daily reports, but methods like the Day Reconstruction Method (DRM; Kahneman, Krueger, Schkade, Schwarz, & Stone, 2004) or ecological momentary assessment (EMA) would allow to take into account within-day fluctuations of affect. The DRM is an ecological instrument to assess various experiences during a given day through a systematic reconstruction conducted on the following day (Kahneman et al., 2004). Based on previous research, which has indicated similar affect profiles recorded with the DRM and EMA (Dockray et al., 2010; Kim, Kikuchi, & Yamamoto, 2013), the DRM presents itself as a valuable instrument which would allow for the assessment of short-term emotion dynamics.

It may further be of use to extend the present findings regarding the internal bodily signal perception by other measures. For example, it would be important to integrate the perception of signals originating from other sensory systems to target the question of modality-specificity of hypervigilance. Also, we only assessed hypervigilance in a general context. It could be useful to either expand the experimental setting to investigate the impact of contextual features of the signals (e.g. threat) or to overcome the question of context-dependency by investigating hypervigilance in more naturalistic situations.

A general concern refers to the fact that self-regulatory capacity was primarily measured using HRV (although in one study we also assessed self-report). Particularly in regard of the role of self-regulatory capacity in distraction efficacy, it may be interesting to implement a behavioural measure of inhibitory capacity (e.g. motor response inhibition). Demands on self-regulatory capacity refer to many different domains of behaviour (e.g. physical activity, social interaction) and to many different levels of an organism (e.g. physiology, cognition) and thereby involve numerous processes (e.g. goal selection, decision-making). Thus, it may be necessary to capture the capacity to self-regulate in a more comprehensive manner by systematically assessing different measures. Suggestions are outlined in the next paragraph. It should further be noted that HRV is susceptible to the effects of medication, which is a concern, especially in clinical populations. Chronic pain patients commonly take different kinds of medication, which could have a long-term effect on the cardiovascular system and bias measures that are

based on the heart rate. Finally, the findings of the current project are mostly restricted to FM. These patients are particularly suitable for this project as the lack of a medical explanation may put a comparatively high strain on self-regulatory capacity, as opposed to explained chronic pain. Also, the fact that this condition is characterized by high levels of psychological distress (Choy et al., 2009; Mease et al., 2007, 2009) highlights the relevance of investigating the regulation of emotions. We can, however, not generalize findings to other pain conditions.

Challenges for future research

Although the current project provides innovative and broad insights into the role of self-regulatory capacity in pain, it raises new questions and can thereby guide future research.

Firstly, we only assessed emotional fluctuations between days. Future research should consider time windows, which are closer to the actual pain experience (Zautra & Sturgeon, 2016). This approach would allow for meaningful time-lag analyses giving more insight into temporal relationships between emotions and pain experiences. Moreover, these analyses could help to identify particularly vulnerable periods for chronic pain patients (Zautra & Sturgeon, 2016). As mentioned above, EMA or DRM (Kahneman et al., 2004) are methods which would allow for the assessment of within-day fluctuations and temporal relationships between pain experiences and emotional states.

Secondly and based on the psychometric properties of HRV, future research could aim to minimize occasion-specific variance by including several assessments of heart rate. Bertsch and colleagues (2012) performed structural equation modelling to estimate the proportions of situational factors and consistent trait variance. They report that the portion of trans-situational consistent trait variance is approx. 49% for a single measurement and increases to 66% and 75% across two or three occasions, respectively. The authors suggest an aggregation over at least two measurements to interpret vagally mediated HRV as a stable biomarker reflecting a trans-situational consistent trait of adaptability to situations.

Thirdly, and as mentioned in the limitations, there may be a need to capture self-regulatory capacity more systematically with complementary indicators. We focused on HRV as a physiological index for the reciprocal inhibitory cortico-subcortical neural circuit reflecting the adaptability of an organism to changing environmental demands (Thayer &

Lane, 2000, 2009). We further investigated affective instability as an experiential reflection of emotion regulation (Carpenter & Trull, 2013; Selby et al., 2015). The advantage of HRV certainly relates to the fact that it refers to a capacity, which is not principally affected by motivational or volitional factors. Consequently, it can be distinguished from measures of performance that require the use of regulatory strategies (e.g. Stroop task). A measure which assesses the actual use of self-regulatory strategies rather than the theoretically useable capacity to regulate, could therefore be more sensitive in regard of general daily functioning. It may be reasonable to assume that patients can adapt to their levels of regulatory capacity and learn to better apply regulation strategies, i.e. compensate their deficits. The use of experimental tasks (e.g. go/nogo) would allow for the comparison between performance and capacity. Motivational components should additionally be assessed as these may influence performance and systematically relate to levels of depression (Jeong & Cranney, 2009). Such experimental performance tasks yield a behavioural measure that reflects motor response inhibition. To complement the picture, future research could aim to assess meta-cognitive aspects of self-regulatory capacity. Such a measure could shed light on the evaluation of one's regulatory capacity, the use of regulatory strategies and the success of regulatory efforts. A challenge for future research would consist in combining the different indicators and gain a better understanding of how these relate to each other and which components seem to have the highest impact on the adaptation to pain.

Fourthly, there is a need for experimental research on self-regulatory capacity in clinical and non-clinical populations. As stated in the limitations, self-regulation involves several mechanisms, which act at different levels, e.g. decision-making, proactive coping, goal setting, self-monitoring and self-efficacy (Baumeister, 1998; Karoly, 1993). This complexity is beyond the scope of this research but certainly challenges future research. Experimental studies should target the systematic investigation of particular mechanisms in experimental designs before integrating them into complex field studies.

Finally, future research could target the question whether self-regulatory deficits are relevant only for a subset of patients. It may well be that perseverative cognitions, such as worry or rumination, could be likely candidates to mediate the effects between self-regulatory demands, such as pain or stress, and health as well as general functioning (Brosschot, Pieper, & Thayer, 2005). Evidence suggests that perseverative cognition relates to lower HRV and ineffective recovery from stress (Brosschot, Gerin, & Thayer, 2006; Brosschot, Van Dijk, & Thayer, 2007). Patients showing a tendency to ruminate may

particularly be at risk for regulatory deficits. Future studies could therefore not only look at baseline HRV, but also investigate the reactivity and recovery of HRV during stress in an experimental setting. This strategy could shed light on whether these features of HRV are more sensitive and could help to identify patients at risk.

Concluding remark

In summary, the current project sheds light on the regulation of emotional and attentional processes, which relate to the experience of and adaptation to pain. More precisely, we applied an innovative approach to measure daily emotional fluctuations rather than average levels of affect in order to investigate their effect on daily pain outcomes. We demonstrated that NA instability relates to HRV and is of relevance for general functioning of chronic pain patients. The experimental results, however, did not show a predictive value of self-regulatory capacity for attentional processes supposedly involved in symptom perception and pain coping. Findings may motivate future research to investigate different aspects of self-regulatory capacity in natural and experimental settings, and provide concrete clinical implications for the treatment of chronic pain.

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**ATTACHMENT:
DATA STORAGE FACT SHEETS**

Data Storage Fact Sheet Chapter 1

% Data Storage Fact Sheet (version september, 10, 2016)
 % Data Storage Fact Sheet <PhD Silke Rost, Chapter 1>
 % Author: Silke Rost
 % Date: 10/09/2016

1. Contact details

=====

1a. Main researcher

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 - e-mail: geert.crombez@ugent.be

1c. Responsible Staff Member: Promotor University of Luxembourg (ZAP)

- name: Claus Vögele
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 - e-mail: claus.voegele@uni.lu

If a response is not received when using the above contact details, please send an email to data.pp@ugent.be or contact Data Management, Faculty of Psychology and Educational Sciences, Henri Dunantlaan 2, 9000 Ghent, Belgium.

2. Information about the datasets to which this sheet applies

=====

* Reference of the publication in which the datasets are reported:

Rost, S., Van Ryckeghem, D., Koval, P., Sütterlin S., Vögele C. & Crombez G. (2016). Affective instability in patients with chronic pain: a diary approach. Pain 157: 1783-1790.

* Which datasets in that publication does this sheet apply to?:

All datasets reported in publication and PhD dissertation chapter.

3. Information about the files that have been stored

=====

3a. Raw data

* Have the raw data been stored by the main researcher? [x] YES / [] NO
 If NO, please justify:

* On which platform are the raw data stored?
 - [] researcher PC
 - [] research group file server
 - [x] other (specify): Dimitri van Ryckeghem PC

* Who has direct access to the raw data (i.e., without intervention of another person)?

- main researcher
- responsible ZAP
- all members of the research group
- all members of UGent
- other (specify): Dimitri van Ryckeghem (dimitri.vanryckeghem@ugent.be)

3b. Other files

* Which other files have been stored?

- file(s) describing the transition from raw data to reported results. Specify:

SPSS syntax files for transition from raw data to a data-file that can be used for analyses:

affect.sps

new CogDys variable.sps

- file(s) containing processed data. Specify:

SPSS (one Level 1 file with the diary data and one Level 2 file with the questionnaire data) and MDM data files for analyses:

MSSD_NA.sav

MSSD_PA.sav

NA_min4_L2.sav

PA_min_L2.sav

masterfile_level1_final_subjects.sav

masterfile_level2_final_subjects.sav

submission.mdm

newcogdys.mdm

depr.mdm

age.mdm

- file(s) containing analyses. Specify:

SPSS and html output files from the multilevel analyses:

descriptives and correlations.spv

disability_NA_0.html

disability_NA_1.html

disability_NA_2.html

disability_NA_3.html

disability_NA_4.html

disability_NA_STAI.html

disability_PA_2.html

disability_PA_3.html

disability_PA_4.html

disability_PA_STAI.html

- files(s) containing information about informed consent
- a file specifying legal and ethical provisions
- file(s) that describe the content of the stored files and how this content should be interpreted. Specify: ...
- other files. Specify:

The protocol of the project was stored online and is freely available on the Ghent University Academic bibliography: <http://hdl.handle.net/1854/LU-3050986>

* On which platform are these other files stored?

- individual PC
- research group file server
- other: ...

* Who has direct access to these other files (i.e., without intervention of another person)?

- main researcher
- responsible ZAP
- all members of the research group
- all members of UGent
- other (specify): ...

4. Reproduction

=====

* Have the results been reproduced independently?: YES / NO

* If yes, by whom (add if multiple):

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- address: 115 Victoria Parade Fitzroy Victoria 3065
- affiliation: KU Leuven & Australian Catholic University
- e-mail: peter.koval@acu.edu.au

Data Storage Fact Sheet Chapter 2

% Data Storage Fact Sheet (version september, 10, 2016)
 % Data Storage Fact Sheet <PhD Silke Rost, Chapter 2>
 % Author: Silke Rost
 % Date: 10/09/2016

1. Contact details

1a. Main researcher

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 - e-mail: silke.rost@uni.lu or silke.rost@ugent.be

1b. Responsible Staff Member (ZAP)

- name: Geert Crombez
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 - e-mail: geert.crombez@ugent.be

1c. Responsible Staff Member: Promotor University of Luxembourg (ZAP)

- name: Claus Vögele
 - address: 11, porte des sciences, 4366 Esch-sur-Alzette, Luxembourg
 - e-mail: claus.voegel@uni.lu

If a response is not received when using the above contact details, please send an email to data.pp@ugent.be or contact Data Management, Faculty of Psychology and Educational Sciences, Henri Dunantlaan 2, 9000 Ghent, Belgium.

2. Information about the datasets to which this sheet applies

* Reference of the publication in which the datasets are reported:

Rost, S., Van Ryckeghem, D., Koval, P., Sütterlin, S., Vögele, C. & Crombez, G. (in prep). Altered regulation of negative affect in fibromyalgia: a diary study.

* Which datasets in that publication does this sheet apply to?

All datasets reported in publication and PhD dissertation chapter.
 All raw data was integrated in two datasets. The first contains diary data (Level 1 data).
 The second contains all Level 2 data: (1) questionnaire data, and (2) HRV data.

3. Information about the files that have been stored

3a. Raw data

* Have the raw data been stored by the main researcher? YES / NO

If NO, please justify:

* On which platform are the raw data stored?

- researcher PC
- research group file server

* Who has direct access to the raw data (i.e., without intervention of another person)?

- main researcher
- responsible ZAP
- all members of the research group
- all members of UGent

3b. Other files

* Which other files have been stored?

- file(s) describing the transition from raw data to reported results. Specify:

Excel-files containing processed HRV data, SPSS syntax files for transition from raw data to a data-file that can be used for analyses, SPSS data-files including information about processing of diary data, word-document mentioning excluded data sets:

CHR.xlsx

GEZ.xlsx

healthy_subjects_EOD_cleaning_data.sav

chronic pain_EOD_cleaning_data.sav

exclusion of participants ASEFI.docx

affect.sps

restructure data.sps

PA_NA.sps

questionnaires_alpha.sps

log transform.sps

syntax_within-person reliability.sps

- file(s) containing processed data. Specify:

SPSS and MDM data files for analyses:

MSSD_NA.sav

MSSD_PA.sav

NA_L2.sav

PA_L2.sav

level1_final sample.sav

level2_final sample.sav

NA_restructured.sav

PA_restructured.sav

SSD_depr.mdm

level1_CHR_final sample.sav

level2_CHR_final sample.sav

daily outcomes_HRV.mdm

- file(s) containing analyses. Specify:

SPSS output files and html output files (from multilevel analyses) containing the results of the study:

within-person reliability PA_NA.spv

descriptives.spv

NA_SSD_unconditional.html

NA_SSD_MeanGroupHF.html

NA_SSD_MeanGroupRmssd.html

PA_SSD_unconditional.html

PA_SSD_MeanGroupHF.html

PA_SSD_MeanGroupRmssd.html

disability_0.html

disability_1.html

disability_NA2.html
 disability_NA3.html
 disability_NA4.html
 disability_NA_anx.html
 disability_NA_depr.html
 disability_PA2.html
 disability_PA3.html
 disability_PA4.html
 disability_PA_anx.html
 disability_PA_depr.html
 distractibility_0.html
 distractibility_1.html
 distractibility_NA2.html
 distractibility_NA3.html
 distractibility_NA4.html
 distractibility_NA_anx.html
 distractibility_NA_depr.html
 distractibility_PA2.html
 distractibility_PA3.html
 distractibility_PA4.html
 distractibility_PA_anx.html
 distractibility_PA_depr.html

- files(s) containing information about informed consent
- a file specifying legal and ethical provisions

ethical committee approvals from the University Hospital Ghent and the University of Luxembourg

- file(s) that describe the content of the stored files and how this content should be interpreted. Specify: ...
- other files. Specify:

The protocol of the project was stored online and is freely available on the Ghent University Academic bibliography: <http://hdl.handle.net/1854/LU-5686902>

* On which platform are these other files stored?

- individual PC
- research group file server
- other: ...

* Who has direct access to these other files (i.e., without intervention of another person)?

- main researcher
- responsible ZAP
- all members of the research group
- all members of UGent
- other (specify): ...

4. Reproduction

* Have the results been reproduced independently?: YES / NO

* If yes, by whom (add if multiple):

- name:
- address:
- affiliation:
- e-mail:

Data Storage Fact Sheet Chapter 3

% Data Storage Fact Sheet (version september, 10, 2016)
 % Data Storage Fact Sheet <PhD Silke Rost, chapter 3>
 % Author: Silke Rost
 % Date: 10/09/2016

1. Contact details

1a. Main researcher

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 - address: Henri Dunantlaan 2, 9000 Gent, Belgium
 - e-mail: geert.crombez@ugent.be

1c. Responsible Staff Member: Promotor University of Luxembourg (ZAP)

- name: Claus Vögele
 - address: 11, porte des sciences, 4366 Esch-sur-Alzette, Luxembourg
 - e-mail: claus.voegel@uni.lu

If a response is not received when using the above contact details, please send an email to data.pp@ugent.be or contact Data Management, Faculty of Psychology and Educational Sciences, Henri Dunantlaan 2, 9000 Ghent, Belgium.

2. Information about the datasets to which this sheet applies

* Reference of the publication in which the datasets are reported:

Rost, S., Van Ryckeghem, D., Schulz, A., Crombez, G. & Vögele, C. (in revision).
 Generalized hypervigilance in fibromyalgia: Normal interoceptive accuracy, but reduced self-regulatory capacity.

* Which datasets in that publication does this sheet apply to?:

All datasets reported in publication and PhD dissertation chapter.
 All raw data was integrated in one dataset including (1) questionnaire data, (2) HRV data, and (3) data from the heartbeat tracking task.

3. Information about the files that have been stored

3a. Raw data

* Have the raw data been stored by the main researcher? YES / NO
 If NO, please justify:

* On which platform are the raw data stored?
 - researcher PC
 - research group file server

- [] other (specify): ...

* Who has direct access to the raw data (i.e., without intervention of another person)?

- [x] main researcher
- [x] responsible ZAP
- [] all members of the research group
- [] all members of UGent

3b. Other files

* Which other files have been stored?

- [x] file(s) describing the transition from raw data to reported results. Specify:

Excel-files containing processed HRV data and data from the heartbeat tracking task,
SPSS syntax files for transition from raw data to a data-file that can be used for
analyses, word-document mentioning excluded data sets:

CHR.xlsx

GEZ.xlsx

CA_ASEFI_mitNull.xlsx

exclusion of participants ASEFI.docx

Syntax_Cronbachs Alpha.sps

- [x] file(s) containing processed data. Specify:

SPSS data file for analyses:

Hypervigilance_final sample.sav

- [x] file(s) containing analyses. Specify:

SPSS output files containing the results of the study:

correlation.spv

descriptives.spv

regression.spv

t-test IA_MANOVA HRV.spv

- [x] files(s) containing information about informed consent

- [x] a file specifying legal and ethical provisions

ethical approvals from the University Hospital Ghent and the University of Luxembourg

- [] file(s) that describe the content of the stored files and how this content should be
interpreted. Specify: ...

- [x] other files. Specify:

The protocol of the project was stored online and is freely available on the Ghent
University Academic bibliography: <http://hdl.handle.net/1854/LU-5686902>

* On which platform are these other files stored?

- [x] individual PC
- [] research group file server
- [] other: ...

* Who has direct access to these other files (i.e., without intervention of another person)?

- [x] main researcher
- [x] responsible ZAP
- [] all members of the research group

- all members of UGent

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=====

* Have the results been reproduced independently?: YES / NO

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- name:

- address:

- affiliation:

- e-mail:

Data Storage Fact Sheet Chapter 4

% Data Storage Fact Sheet (version september, 10, 2016)
 % Data Storage Fact Sheet <PhD Silke Rost, Chapter 4>
 % Author: Silke Rost
 % Date: 10/09/2016

1. Contact details

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1c. Responsible Staff Member: Promotor University of Luxembourg (ZAP)

- name: Claus Vögele
 - address: 11, porte des sciences, 4366 Esch-sur-Alzette, Luxembourg
 - e-mail: claus.voegel@uni.lu

If a response is not received when using the above contact details, please send an email to data.pp@ugent.be or contact Data Management, Faculty of Psychology and Educational Sciences, Henri Dunantlaan 2, 9000 Ghent, Belgium.

2. Information about the datasets to which this sheet applies

* Reference of the publication in which the datasets are reported:

Rost, S., Van Ryckeghem, D., Crombez, G. & Vögele, C. (in prep). An experimental investigation of the role of self-regulatory capacity in the distraction from pain

* Which datasets in that publication does this sheet apply to?:

All datasets reported in publication and PhD dissertation chapter.
 All raw data was integrated in one dataset including (1) output from the distraction task (self-reports, reaction times), (2) questionnaire data, and (3) HRV data.

3. Information about the files that have been stored

3a. Raw data

* Have the raw data been stored by the main researcher? YES / NO

If NO, please justify:

* On which platform are the raw data stored?

- researcher PC

- research group file server
- other (specify):

* Who has direct access to the raw data (i.e., without intervention of another person)?

- main researcher
- responsible ZAP
- all members of the research group
- all members of UGent

3b. Other files

* Which other files have been stored?

- file(s) describing the transition from raw data to reported results. Specify:

Excel-files containing processed HRV data and data from the distraction task, SPSS syntax files for transition from raw data to data that can be used for analyses:

Syntaxe.sps
 RSAnorm.sps
 SP.xlsx
 HRV outliers.docx
 final_aandachtstaak_cleaned.xlsx
 final_andaachtstaak_cleaned_task interference.xlsx
 final_andaachtstaak_pain_experience.xlsx

- file(s) containing processed data. Specify:

SPSS file containing the complete dataset for analyses:
 SEREPA3_final.sav

- file(s) containing analyses. Specify:

SPSS output files containing the results of the study:
 descriptives and correlations_39.spv
 distraction efficacy overall.spv
 attentional interference.spv
 attentional interference control PA.spv
 HRV_distraction efficacy and attentional interference.spv
 Means and SE for graphs.spv

- files(s) containing information about informed consent

- a file specifying legal and ethical provisions

ethical approval from the University of Luxembourg

- file(s) that describe the content of the stored files and how this content should be interpreted. Specify: ...

- other files. Specify: ...

* On which platform are these other files stored?

- individual PC
- research group file server
- other: ...

* Who has direct access to these other files (i.e., without intervention of another person)?

- main researcher
- responsible ZAP

- all members of the research group
- all members of UGent

4. Reproduction

=====

* Have the results been reproduced independently?: YES / NO

* If yes, by whom (add if multiple):

- name:
- address:
- affiliation:
- e-mail: