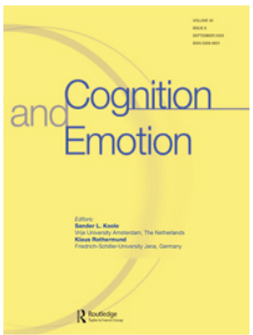

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The role of negative emotional granularity and interoceptive sensibility in neuropathic and nociplastic chronic pelvic pain

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ABSTRACT

Pain has been acknowledged as consisting of both emotional and sensory components. Within constructivist theories of emotion, the ability to make subtle distinctions among emotional states – known as emotional granularity (EG) – has been proposed as a downstream mechanism in chronic pain. However, its role remains empirically unexplored. In the current study, we investigated the concurrent and prospective associations between negative EG and pain intensity, and whether dispositional interoceptive sensibility (IAs) – the tendency to focus on internal sensations – moderates this relationship. Sixty-eight women with neuropathic and nociplastic chronic pelvic pain completed diary entries at each pain episode over one month. We assessed integral EG (across broad emotion families), between- and within-category EG (across and within the anger, fear, and sadness families), using multilevel models to predict concurrent and subsequent pain. Our results showed that higher integral and between-category EG were associated with lower concurrent pain among women with average-to-low IAs. Within-category EG showed no concurrent relationship with pain intensity. Prospectively, fear-specific EG in high-IAs participants was associated with heightened subsequent pain. Overall, our findings suggest that the association between EG and pain intensity is conditional on EG type, temporal frame, and interoceptive focus, thus highlighting its potential role in pain chronification.

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Emotional granularity; emotion differentiation; interoception; chronic pain; event-contingent design

1. Introduction

Emotional granularity (EG), also known as emotion differentiation, reflects the ability to make subtle, fine-grained distinctions among emotional states (Barrett et al., 2001). It reflects how individuals represent and describe their feelings in a given context. While some people can accurately and precisely articulate their current emotional states, others tend to report their experiences in more global or undifferentiated terms.

From a constructionist perspective (Barrett, 2017b), grounded in the idea of a Bayesian brain, emotions are not fixed biological reactions but situated constructions and emergent products of the brain's predictive regulation of the body. However, the brain's primary function is not to produce emotions but to regulate the *internal milieu* of the organism through a dynamic, anticipatory process known as allostasis. Through allostasis, the brain predicts and fulfils the body's physiological needs before they arise by managing energy resources such as glucose, oxygen, and hormonal activity (Kleckner et al., 2017).

This predictive regulation relies on interoception (Kleckner et al., 2017), the perception and integration of autonomic, hormonal, visceral and immunological

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homeostatic signals that collectively reflect the body's physiological state (Craig, 2014). Interoceptive sensations generate a foundational and ever-present state known as *core affect* (Lindquist et al., 2016), a neurophysiological state defined in two dimensions (Russell, 2003): valence (pleasant–unpleasant) and arousal (activated–deactivated). Core affect forms the affective backdrop of all individuals' experiences. To make sense of core affect, the brain draws upon conceptual knowledge to interpret and categorise bodily sensations felt in a particular moment within a specific context (Barrett et al., 2015). Depending on contextual cues, external sensory information, past experiences, and the individual's conceptual repertoire, the same bodily sensations as represented by the core affect may thus be categorised in different ways (Barrett, 2017b; Lindquist & Barrett, 2008), for instance as a physical (e.g. feeling hungry, sick) or an emotional state (e.g. feeling angry, relieved). Categorisation can also include connotations of a motivational drive (e.g. feeling determinate, brave) or of a cognitive evaluation (e.g. feeling safe, confused). This process, known as situated conceptualisation (Barrett et al., 2014), is rooted in predictive coding: The brain continuously generates predictions on the basis of prior experience to anticipate and interpret sensory and interoceptive inputs. Mismatches between expected and actual inputs (prediction errors) update and refine the internal models of the brain and guide future responses.

Within this framework, emotional experiences are actively constructed through the categorisation of core affective states. Whether core affect is interpreted as an emotion or something else depends on how the brain applies its conceptual knowledge within a specific context (Lindquist et al., 2015). Therefore, constructing a granular emotional experience depends on the precision of the brain's predictive models, the efficiency of allostatic regulation, and the richness of the individual's conceptual repertoire. The ability to label and differentiate emotions with high granularity reflects a finely attuned integration of interoceptive signals and a well-developed conceptual system. Together, they allow for nuanced categorisation of affective experiences in contextually appropriate and energy-efficient ways.

1.1. A predictive coding perspective on chronic pain

According to the most recent definition by the International Association for the Study of Pain (2012), pain is described as "An unpleasant *sensory and emotional* experience associated with, or resembling that associated with, actual or potential tissue damage". To further clarify this definition, the International Association for the Study of Pain specifies that (*a*) pain is always a subjective experience affected by biological, psychological, and social factors; (*b*) pain and nociception (defined as the neural process of encoding noxious sensory inputs) are distinct phenomena; (*c*) pain cannot be inferred solely from activity in sensory neurons; and (*d*) individuals learn the concept of pain through life experiences.

A growing body of evidence suggests that pain is a brain-based construction generated by a core predictive system (Lersch et al., 2023; Ongaro & Kaptchuk, 2018). When the brain receives nociceptive signals, it predicts and regulates pain experiences while simultaneously managing the body's energy resources in preparation for potential damage. However, prediction errors can occur: The brain may generate pain prediction in the absence of any actual or potential tissue damage (Roy et al., 2014). When these errors go uncorrected, the brain fails to update internal models and continues to allocate resources to address a threat that is not physically present (Poublan-Couzardot & Talmi, 2024).

Predictive coding theories have hypothesised that such persistent misallocations may underlie cases of chronic pain in the absence of damage to or threat of damage to nonneural body tissues (Eckert et al., 2022). This is observed in neuropathic chronic pain, which is caused by a lesion or disease of the somatosensory nervous system, and in nociplastic chronic pain, which arises from altered nociception despite no clear evidence of peripheral tissue damage or somatosensory system injury (International Association for the Study of Pain, 2012). Patients may also present with a combination of both nociceptive and nociplastic pain. In these cases, individuals may have experienced intense nociceptive input in the past and continue to predict pain after tissue recovery by disregarding corrective prediction errors (Apkarian et al., 2013).

Within a predictive coding account, a heightened prediction of pain would lead to intensified pain perception as patients with chronic pain tend to infer pain as the most likely cause of harmless body sensation (Hechler et al., 2016). It is also possible that the brain prioritises and amplifies the salience of nociceptive inputs even when these signals are weak owing to their allostatic relevance, thus

making aberrant predictions about internal bodily sensations (Moseley & Vlaeyen, 2015). In all these scenarios, individuals may come to chronically experience pain as real and intense, despite the absence of an identifiable organic origin.

1.2. The potential role of emotional granularity in chronic pain

Pain and emotion are acknowledged as substantially intertwined phenomena and several perspectives have been proposed to account for the complexity of their relationship (for a discussion, see Gilam et al., 2020; Poublan-Couzardot & Talmi, 2024). Emotions (particularly negative emotions) are known to significantly alter pain perception (Bushnell et al., 2013; Wiech & Tracey, 2009). Also, feelings of depression, anxiety, frustration, and fear are highly prevalent in chronic pain patients (Lerman et al., 2015).

Within a constructionist approach, the experience of pain and emotion are conceived "as two sides of the same coin" (Barrett, 2017a, p. 16) and based on similar underlying mechanisms. For instance, it has been shown that the brain networks involved in nociceptive processing overlap with those that support emotional experience (Woo et al., 2015). Recent evidence has even suggested that nociception may be conceptualised as a form of interoception (Craig, 2014; Damasio & Carvalho, 2013). Overall, the construction of emotional and pain experiences appears to rely on common, dynamically interacting neural systems with shared ascending and descending pathways (Chang et al., 2015; Wager et al., 2013, 2015).

This is particularly relevant in chronic pain conditions where no clear nonneural structural damage is found (Jungilligens et al., 2022; Poublan-Couzardot & Talmi, 2024). In this case, constructionist perspectives on emotion advance the hypothesis that the experience of pain and emotion may reflect different interpretations of similar bodily sensations. Chronic neuropathic and nociplastic pain may, in part, stem from difficulties in distinguishing and categorising certain internal sensations as corresponding to either pain or other affective states, including discrete emotions (Barrett, 2017a). This may be due to aberrant interoceptive predictions that fail to be corrected by updated internal models (Barrett, 2017a; Poublan-Couzardot & Talmi, 2024). Individuals with low EG may be especially prone to this misclassification (Barrett, 2017a): A hypothesis is that low differentiators struggle to accurately distinguish the emotional from the sensory component of pain; or they may misinterpret emotional distress and bodily discomfort as pain (Tracey, 2010). Over time, this misclassification may reinforce maladaptive interoceptive predictions, contributing to the persistence of pain (Poublan-Couzardot & Talmi, 2024).

Existing research supports this idea. For instance, individual differences in constructs conceptually related to EG, such as alexithymia and emotional awareness, have been linked to chronic pain. Specifically, individuals with chronic pain often report reduced emotional awareness compared to matched control groups (Lumley et al., 2021; Smith et al., 2020; Zunhammer et al., 2015). Likewise, people with chronic pain often show higher levels of alexithymia, a trait characterised by difficulties in the identification and description of one's emotions (Aaron et al., 2019). Moreover, higher levels of difficulty in identifying feelings have been associated with higher levels of pain intensity in people with chronic pain, suggesting that the ability to accurately identify and reflect on one's emotional state may offer a protective function (Aaron et al., 2019; Neumann et al., 2025). These findings seem to support the notion that impairments in the conceptualisation and differentiation of internal affective states may contribute to the miscategorisation and persistence of chronic pain.

1.3. Interoception as a shared mechanism in emotional granularity and chronic pain

Being multidimensional in nature, interoception encompasses three distinct components: Interoceptive accuracy (IAc), which refers to the objective accuracy (or precision) of one's ability to detect bodily signals (e.g. heart rate); interoceptive sensibility (IAs), which reflects self-reported attention and perceived sensitivity to internal sensations; and interoceptive awareness (IAw), which is defined as the meta-cognitive ability to accurately gauge bodily signals (Garfinkel et al., 2015). Within a Bayesian approach to interoception (Barrett & Simmons, 2015), IAc is thought to reflect precision in interoceptive systems, which depends on the relative weight assigned to prior representations and actual bodily sensations (Ainley et al., 2016). While IAc has been usually assessed through objective performance on tasks such as heartbeat perception, IAs refers to the subjective domain (i.e. individual's own perception of bodily awareness) and has been measured using various self-report questionnaires (Van Bael et al., 2024). IAc and

IAs are often unrelated, so that people may believe to be highly aware of their body sensations but perform poorly on accuracy tasks.

Of note, contemporary models organise interoceptive processes as comprising multiple hierarchical levels suggesting that the perception of internal bodily sensations depends on the nature of afferent signals, their central representation, and higher-order cognitive processes such as attention and categorisation (Garfinkel & Eccleston, 2025; Greenwood & Garfinkel, 2024). IAc would lie at a lower level of this hierarchy than IAs and IAw, which are thought to reflect more trait-like beliefs pertaining to interoceptive processing (Garfinkel & Eccleston, 2025). These predictive coding models have also suggested that interoception – in all its hierarchically levels – is a key factor for the understanding of pain (Garfinkel & Eccleston, 2025; Horsburgh et al., 2024) and emotion (Barrett, 2017b; Craig, 2014; Greenwood & Garfinkel, 2024; Seth & Friston, 2016).

Concerning pain, research has shown that patients with chronic pain tend to exhibit reduced IAc and increased IAs levels compared to control groups (for a review and meta-analysis, see Horsburgh et al., 2024), suggesting a mismatch between subjective interoceptive focus and objective performance. In other words, chronic pain sufferers appear to be more attuned to internal sensations while demonstrating a reduced ability to detect them accurately (Horsburgh et al., 2024). Of note, however, differences in IAs seem to largely depend on the type of measure employed, so that patients with chronic pain tend to obtain higher scores on measures that reflect noticing and paying attention to bodily sensations and lower on those relating to the management and regulation of those sensations. However, evidence concerning the relationship between interoceptive measures (IAc and IAs) and pain intensity in patients with chronic pain is still mixed and inconclusive (Horsburgh et al., 2024).

In parallel, research has investigated the role of interoception in emotional experience. A first bulk of studies have examined the link between IAc and the intensity of affective and emotional experience: While some studies have found that higher levels of IAc are related to more intense emotions and higher arousal focus, other studies have failed to find any association (for a meta-analysis, see Parrinello et al., 2022). Far less evidence exists concerning IAs: In a recent study using ecological momentary assessment (MacVittie et al., 2025), greater in-the-moment IAs was associated with the experience of more intense emotions. A second line of research has examined the relationships between interoception and emotion identification difficulties. For example, several studies have examined the hypothesis that alexithymia may be associated with interoceptive impairments across multiple bodily domains and with IAc and IAs dimensions of interoception (Brewer et al., 2016; Murphy et al., 2018). In a recent meta-analysis (Trevisan et al., 2019), alexithymia showed significant associations with IAs, but not with IAc, suggesting that objective detection of internal signals and subjective awareness of bodily sensations may play separate roles in emotional experience (MacVittie et al., 2025). Notably, the associations between IAs and alexithymia have been shown to vary depending on the specific aspect of IAs being measured, with alexithymia being positively related to measures of heighted attention and negatively related to measures of subjectively perceived accuracy and self-regulation (Gaggero et al., 2021; Van Bael et al., 2024). Finally, individuals with high alexithymic traits have been shown to misattribute vague or poorly differentiated internal sensations to somatic causes rather than emotional ones (Scarpazza et al., 2015), supporting the idea that difficulties in constructing nuanced emotional experiences may stem from imprecise or disrupted interoceptive predictions.

Although theoretical models suggest that higher levels of EG may reflect better differentiation of bodily sensations, only one study (Ventura-Bort et al., 2021) has so far directly tested this relationship. This study found that granularity for negative emotions experienced in ecological contexts was related to measures reflecting both IAs and emotional conceptualisation. Specifically, EG was positively related to self-reported "sensibility" (tapping the believes of one's accuracy in detecting internal sensations and emotional states) but negatively associated with self-reported "monitoring" (tapping the tendency to focus attention on bodily signals and emotions). The authors speculate that individuals with lower EG may need to deploy more attentional resources to their internal states to differentiate between experienced emotions. Of note, theoretically, attention is acknowledged among the "psychological ingredients" from which emotions are constructed as it can strongly influence how an individual's current state is

interpreted, thereby shaping the quality and precision of predictive inferences and, ultimately, the nature of affective experience (Barrett, 2009; Barrett et al., 2004).

Overall, these preliminary results suggest that IAs may constitute a downstream mechanism in the construction of nuanced emotional experiences, although its contribution may vary depending on the specific features under consideration. This pattern of findings has important implications for the understanding of chronic pain: Failures in interoceptive processing may contribute not only to emotional undifferentiation but also to the misclassification of affective states as pain.

2. The present study

In the present study, we investigated the relationship between negative EG, IAs, and pain in a sample of women experiencing neuropathic and nociplastic chronic pelvic pain (CPP). CPP is defined as persistent or recurrent pain perceived in the pelvic region, typically lasting for more than six months (American College of Obstetricians and Gynecologists, 2020). It is estimated to affect up–27% of women worldwide. Notably, at least 30% of women with CPP present no identifiable organic pathology in pelvic tissues, underlying a condition of nociplastic or neuropathic CPP (Siqueira-Campos et al., 2022).

The absence of identifiable tissue damage (and thus of objective clinical markers) is frequently associated with the experience of social invalidation and with a sense of inadequacy. Many women report that their pain is neither recognised nor understood, describing it as profoundly limiting in its impact on daily functioning (Shallcross et al., 2018). Their narratives often reflect themes of helplessness, frustration, emotional exhaustion, and a perceived loss of control over both bodily states and interpersonal relationships (Niedenfuehr et al., 2023; Shallcross et al., 2018).

Given the substantial emotional burden associated with neuropathic and nociplastic CPP, our study focused on negative EG (i.e. granularity in relation to negative emotions). Also, we consider momentary granularity, that is, the short-term fluctuations in EG within individuals. Furthermore, we distinguished among different components of negative granularity. In addition to examining integral granularity (i.e. granularity assessed across distinct emotion clusters), we further differentiated into between-category and within-category granularity. Between-category granularity refers to the ability to distinguish emotions that belong to different emotional clusters, whereas within-category granularity pertains to distinctions among emotions within the same cluster (Erbas et al., 2019). In our study, we focused on the core negative emotion clusters of anger, fear, and sadness because of their prominence in the emotional experiences of women with CPP (Hawkey et al., 2022; Niedenfuehr et al., 2023; Shallcross et al., 2018). This focus allowed for a more fine-grained investigation of how different facets of EG relate to pain experience in women with neuropathic and nociplastic CPP.

Using an ecological, event-contingent design, we examined the within-person association of EG with momentary pain intensity. Specifically, we investigated this relationship both concurrently (i.e. by assessing how momentary granularity relates to pain at the same time point) and prospectively (i.e. by examining whether granularity reported in a prior episode is associated with subsequent momentary pain intensity). Both temporal perspectives are particularly relevant within the framework of the predictive processing model, which posits that chronic pain may result from persistent and aberrant interoceptive predictions. Finally, we examined whether dispositional IAs moderates the relationship between EG and pain intensity. Among the components of interoception, we focused on IAs, as prior research has linked IAs to emotion identification difficulties and EG (e.g. Van Bael et al., 2024; Ventura-Bort et al., 2021). Of note, IAs has been linked to central sensitisation symptoms, pain severity and depression in patients with CPP (Scarpina et al., 2025; Spinoni et al., 2024).

Based on a constructionist model of emotion (Barrett, 2017a, 2017b), we hypothesised that greater negative EG would be associated with lower pain intensity within individuals. This hypothesis was also informed by prior findings linking granularity-related constructs, such as alexithymia and emotional awareness, to pain intensity in patients with chronic pain (Aaron et al., 2019; Lumley et al., 2021). Existing research suggests that difficulties in identifying and differentiating emotions may exacerbate pain intensity – potentially, by interfering with the distinction between the emotional and sensory components of pain (Barrett, 2017a). Furthermore, we hypothesised that the relationship between EG and pain would be moderated by dispositional IAs. Our moderation hypothesis was based on prior work documenting a significant association between alexithymia and IAs

(Gaggero et al., 2021; Trevisan et al., 2019; Van Bael et al., 2024) and on preliminary evidence that suggests that negative EG is negatively associated with self-reported vigilance toward bodily signals and emotions (Ventura-Bort et al., 2021). Taken together, these findings seem to suggest that the potential association between higher EG and reduced pain intensity may depend on individual differences in IAs.

To isolate the specific contribution of EG, we used a statistical model that allowed us to control for the valence, arousal, and dominance of the reported affective states, as well as for the overall intensity of negative emotions. This approach ensured that any observed relationship between granularity and pain could not merely be attributed to individual differences in general affective tone or emotional intensity.

3. Method

The data used in the present study were collected as part of a larger study on affective experience and chronic pelvic pain. All protocols described below received ethical approval by Università Cattolica di Milano (protocol number 58–23).

3.1. Participants

The participants were women diagnosed with neuropathic and nociplastic CPP, with pelvic pain lasting at least six months and occurring on a minimum of three days per month. The exclusion criteria included ongoing treatment for more than two years, recent (within the last two weeks) initiation or change in hormone treatment, recent (within the last two months) pelvic or abdominal surgery, scheduled pelvic or abdominal surgery during the study period, current pregnancy, or childbirth within the previous two months.

The initial sample consisted of 84 participants. Two participants dropped out after the baseline assessment, and 14 participants reported fewer than three pain episodes during the month and were therefore excluded from the final analysis in accordance with the study criteria. The final sample consisted of 68 participants ($M = 27.98$ years, $SD = 6.69$ years, age range 18–44 years).

The average number of pain episodes per month was 7.51 ($SD = 5.32$, range 3–42 episodes per month). With respect to pelvic pain diagnosis, 57.4% of the participants reported comorbidities involving multiple conditions, with vulvodynia being the most frequently reported diagnosis. A full description of the sample characteristics is presented in Table 1.

3.2. Materials

3.2.1. Pain intensity

Momentary pain intensity was assessed at each pain episode with a single item on a 7-point Likert-type scale ranging from 0 (*very mild*) to 6 (*very intense).* Previous experience sampling studies investigating pelvic pain used a single item to assess pain intensity (Pâquet et al., 2018; Rosen et al., 2015). Furthermore, recent research suggests that single items have good predictive and concurrent validity in intensive longitudinal designs (Song et al., 2023).

3.2.2. Negative emotions

At each pain episode, participants provided ratings of their current emotional state on a 7-point Likert scale ranging from 0 (*not at all*) to 6 (*extremely*) across fourteen negative emotion words (*embarrassed, worn out, angry, frustrated, fed up, nervous, frightened, anxious, anguished, worried, sad, depressed, lonely, and demoralised*). These emotion labels were selected on the basis of two criteria. First, in accordance with the Circumplex Model of Affect (Russell, 1980), the labels were representative of both low-and high-arousal negative affect. Second, labels were conceptually grouped into three core emotional clusters: anger (*angry, frustrated, fed up, nervous*), fear (*frightened, anxious, anguished, worried*), and sadness (*sad, depressed, lonely, demoralised*). Two emotion items (*embarrassed* and *worn out*) did not clearly belong to these clusters but were nonetheless included on the basis of previous research highlighting their relevance in the context of CPP (Niedenfuehr et al., 2023; Toye et al., 2014).

3.2.3. Negative emotional granularity

Three types of EG indices were calculated. First, we computed a momentary EG index reflecting the extent of emotion differentiation at each measurement occasion. This integral granularity index was computed using the method proposed by Erbas et al. (2022) and implementing the equation via the *emodiff* package in *R v.4.4.1*. Second, for each person and each time point, we computed separate momentary indices of granularity within and between three core negative emotion clusters (i.e. anger, fear, and sadness). Within-category granularity indices were computed via the same procedure used for the integral EG index, but considering the three specific subsets of emotion items. To derive

Table 1. Sample characteristics.

		Count	%
Education	Master's Degree	22	32.4
	High School	20	29.4
	Bachelor's Degree	13	19.1
	Advanced Postgraduate Education	10	14.7
	Middle School	1	1.5
Occupation	Working	34	50.0
	Studying	22	32.3
	Studying and Working	10	14.7
	Unemployed	1	1.5
Civil Status	Unmarried	56	82.3
	Married	8	11.8
	Divorced	3	4.4
No. Children	Zero	60	88.2
	One	5	7.3
	Two	2	2.9
Diagnosis	Vulvodynia	53	73.6
	Vulvar Vestibulitis/Vestibulodynia	37	51.4
	Pelvic Floor Hypertonia	33	45.8
	Pudendal Neuropathy	18	25.0
	Endometriosis	9	12.5
	Adenomyosis	7	9.7
	Clitoralgia/Clitorodynia	4	5.6
	Dyspareunia	4	5.6
	Interstitial Cystitis	2	2.8
	Dysmenorrhea	2	2.8
	Lichen	1	1.4
	Vaginismus	1	1.4
	Vulvostenia	1	1.4
Symptoms Onset	More than a Year	62	91.2
	Less than a Year	5	7.3
Pain Type	Spontaneous	43	63.2
	Provoked	24	35.3
Treatment	Both Pharmacological and Nonpharmacological	31	45.6
	Only Pharmacological	20	29.4
	Only Nonpharmacological	12	17.6
	None	4	5.9

Note: Advanced postgraduate education includes professional specialisation programmes and doctoral studies. Spontaneous pain refers to pain that occurs without any apparent external stimulus or trigger. Provoked pain refers to pain that is elicited or intensified by a specific stimulus or activity, such as touch, pressure, movement, or temperature. The percentages in the "Diagnosis" category exceeded 100% due to diagnostic comorbidities, as more than half of the participants (54.4%) reported having more than one diagnosis. Pharmacological treatments included tricyclic antidepressants (e.g. amitriptyline/Laroxyl), SNRIs (e.g. duloxetine/Cymbalta), gabapentinoids (e.g. pregabalin/Lyrica, gabapentin), hormonal therapies (e.g. oral contraceptives, vaginal rings), benzodiazepines (e.g. diazepam, Ansiolin), muscle relaxants, topical preparations (e.g. creams with amitriptyline, gabapentin, testosterone, estrogens), and various adjuncts, such as nutraceuticals, probiotics, and anti-inflammatory agents. Local (vaginal) administration was frequently reported for some treatments, particularly benzodiazepines and compounded creams. Nonpharmacological treatments included pelvic floor rehabilitation (e.g. physiotherapy, manual therapy, biofeedback, TENS, radiofrequency, electroporation, dilators, and self-massage), psychological support (e.g. psychotherapy), nutritional interventions (e.g. functional nutrition plans, supplementation with magnesium, vitamin D, omega-6), topical nondrug treatments (e.g. CBD- or CBG-based creams and oils), and complementary therapies (e.g. acupuncture, ozone therapy). Pelvic physiotherapy was the most frequently reported intervention and was often conducted by specialised physiotherapists or midwives.

the between-category index, we first averaged the emotion ratings within each cluster and then applied the same calculation procedure to these cluster-level means. Further details regarding the computation of the EG indices are provided in the Supplemental Material.

3.2.4. Affect

The Self-Assessment Manikin (Bradley & Lang, 1994) was used to assess the momentary affective state along the three core dimensions of the Circumplex Model of Affect (Russell, 1980): valence (ranging from unpleasant to pleasant), arousal

(ranging from low to high activation), and dominance (ranging from feeling controlled to feeling in control). The participants rated each dimension via a single nonverbal pictorial item on a 9-point Likert scale, with higher scores indicating greater levels of the corresponding affective quality.

3.2.5. Interoceptive sensibility

The *Body Awareness* section of the Porges Body Perception Questionnaire–Short Form (Cabrera et al., 2018; Cerritelli et al., 2021) was used to assess dispositional interoceptive sensibility. It consists of 26 items on bodily sensations. The BPQ measures the perceived sensitivity to neutral and negative bodily sensations (Desmedt et al., 2022), but it has been also considered as a measure of interoceptive attention (Murphy et al., 2019) capturing maladaptive attention, characterised by anxiety-driven hypervigilance toward bodily sensations and somatisation (Trevisan et al., 2021). The participants rate how often they are aware of each sensation via a 5-point Likert scale (from 1 = *never* to 5 = *always*). In this study, reliability was good (ω = 0.905; Cronbach's α = 0.902).

3.3. Procedure

Participants were recruited through healthcare professionals (e.g. gynecologists, midwives, physiotherapists) working in hospitals or specialised clinics for CPP. Recruitment was also facilitated through patient advocacy associations and online services dedicated to individuals with CPP. Participation in the study was voluntary and without any monetary compensation.

The study employed a repeated-measures design including three assessment points scheduled at one-month intervals. Between the first and second assessment, participants were asked to complete a one-month experience sampling protocol using an event-contingent design. The present study examines the data gathered during the experience sampling period only. To ensure transparency, a comprehensive description of the full study procedure is provided in the Supplemental Materials.

First, the participants were invited to attend an initial session in which the researchers explained the objectives and structure of the study, confirmed eligibility, and obtained informed consent. The participants then completed an online baseline questionnaire including measures of sociodemographic information, medical history, interoceptive sensibility, and additional measures not analysed in the present study.

During the initial session, the researchers also provided the participants with instructions to complete the one-month experience sampling protocol. In more detail, they were instructed to complete an online diary each time they experienced a pain episode. For each episode, the participants were asked to rate the current pain intensity, their affective state (i.e. valence, arousal, and dominance), and a fixed set of fourteen emotion labels. The participants were also invited to provide an open-ended narrative describing the pain episode and ratings of self-generated affective adjectives, which however were not included in the present analyses. To control for potential confounding factors, two versions of the diary protocol were used: One version asked the participants to rate affect and emotion measures before providing the open-ended narrative, while in the second version the participants freely described the pain episode before completing the emotion measures. The participants were randomly assigned to one of the two versions to counterbalance potential order effects.

3.4. Transparency and openness

Study materials, data, and analysis codes are available at https://osf.io/xsqgc/overview. This study was preregistered on the Open Science Framework (OSF) before data collection[1] (https://osf.io/deavf/overview).

3.5. Data analytic plan

Mixed effects models were used to investigate how momentary EG relates to pain intensity both concurrently (using contemporaneous mixed effects models) and prospectively (using prospective, i.e. time-lagged, mixed effects models). Moreover, we tested whether dispositional IAs moderates this association. All the analyses were conducted using the following three types of EG indices: Integral, between-category, and within-category (anger-related, fear-related, and sadness-related) granularity. In all models, we controlled for the contribution of time, affective state (valence, arousal, and dominance), and intensity of negative emotions. Moreover, to directly address the relative contribution of integral and between- versus within-category granularity, additional models were estimated in which within-category EG indices were included as covariates when testing the effects of integral and between-category EG. This approach allowed us to examine whether the observed associations with pain intensity remained significant when controlling for shared variance with within-category granularity,

thereby providing a more stringent test of their unique predictive value.

The analyses were conducted in *R v.4.4.1*. To fit linear mixed-effects models, we used *lme4* (Bates et al., 2015), and calculated *p* values for the model coefficients via *lmerTest* (Kuznetsova et al., 2017). To prevent confounding within- and between-person effects, diary-level independent variables were person-mean centred within participants, whereas IAs was grand-mean centred (Hamaker & Muthén, 2020). Furthermore, the independent variables were standardised, allowing for direct comparability of coefficient estimates. Following recent recommendations (Wang et al., 2019), we adopted a clusterwise standardisation approach for diary variables and global standardisation for IAs.

3.5.1. Contemporaneous mixed effects models

We began by exploring the within-person and between-person variability in both EG indices and pain intensity by calculating the intraclass correlation coefficient (ICC). Most of the variability in EG was observed within individuals over time rather than between individuals (between individuals ICC $\leq$ 0.001). By contrast, with respect to pain intensity, between-person variability accounted for a large portion of the total variance (ICC = 0.39). This preliminary step was necessary to specify the models in our subsequent analyses, which require between-person variability in the dependent variable and for which random slopes of the independent variable depend on within-person variability.

To investigate the concurrent association between EG and pain intensity, we specified a two-level multilevel model with random intercepts,[2] in which pain episodes (Level 1) were nested within participants (Level 2). Momentary pain intensity was modelled as a function of momentary EG. In addition, we included IAs as a Level 2 moderator of the within-person (Level 1) association between granularity and pain. The model was specified as follows:

Level 1 (pain episodes):

$$\text{Pain}_{\text{time(t),person(p)}} = \beta_{0p} + \beta_{1p} \cdot (EG_{tp}) + \beta_{2p} \cdot (EG_{tp} \cdot IAs_p) + \epsilon_{tp}$$

Level 2 (persons):

$$\beta_{0p} = \gamma_{00} + \gamma_{01} \cdot (IAs_p) + u_{0p}$$

We then estimated a second model by including momentary affect (valence, arousal, and dominance) and momentary intensity of negative emotions as additional independent variables (covariates). The equation for this extended model is reported in the Supplemental Material.

Different models were run for each of the five indices of momentary EG (integral, between-category, anger-related, fear-related, and sadness-related granularity). In sum, for each operationalisation of granularity, two separate models were estimated: (1) a baseline model testing the association between granularity and pain intensity, as well as the moderation by IAs; (2) a second model including covariates to control for affect and intensity of negative emotions.

Finally, in all the models, a covariate indexing time within the experience sampling protocol was included to control for a potential confounding effect of time. This variable was defined as a continuous, standardised measure of the time elapsed at the moment of each entry relative to the start of the data collection period.

3.5.2. Prospective mixed effects models

We specified a prospective two-level multilevel model with random intercepts, nesting pain episodes within participants. Pain at each pain episode (*t*) was predicted by EG at the previous pain episode (*t*−1) while simultaneously adjusting for the level of pain at the previous episode (*t*−1). We included IAs as moderator of the prospective effect of granularity on momentary pain. Since we used an event-contingent design, our observations were not equally spaced over time. To address this issue, we adjusted each within-person variable by multiplying it by a Delta Time (Δt) term, which represents the time interval between consecutive observations.[3] This approach allowed us to appropriately account for the variability (both within the same participant and across participants) in the time intervals between assessments and to control for potential time-related confounding factors. We specified the model as follows:

Level 1 (pain episodes):

$$\begin{aligned}\text{Pain}_{\text{time(t),person(p)}} = \beta_{0p} &+ [(\beta_{1p} + \beta_{2p} \cdot \Delta t_{tp}) \cdot EG_{t-1p}] \\ &+ [(\beta_{3p} + \beta_{4p} \cdot \Delta t_{tp}) \cdot (EG_{t-1p} \cdot IAs_p)] \\ &+ [(\beta_{5p} + \beta_{6p} \cdot \Delta t_{tp}) \cdot \text{Pain}_{t-1p}] + \epsilon_{tp}\end{aligned}$$

Level 2 (persons):

$$\beta_{0p} = \gamma_{00} + \gamma_{01} \cdot (IAs_p) + u_{0p}$$

Following the same logic of contemporaneous analyses, we estimated a second lagged model by

including lagged affect (i.e. the level of valence, arousal and dominance reported at the previous pain episode, $t-1$) and lagged negative emotions (i.e. the negative emotions reported at the previous pain episode, $t-1$) as covariates. The model equation for this extended model is presented in the Supplemental Material.

Once again, the models were independently estimated for each of the five indices of lagged EG (integral, between-category, anger-related, fear-related, and sadness-related granularity). In sum, for each index, we estimated two multilevel models to examine the prospective association of EG with pain and the incremental contribution of the covariates.

4. Results

The descriptive statistics and within- and between-person correlations are shown in Table 2.

To verify that the order of the questions (i.e. the two versions of the diary protocol) did not introduce systematic differences in the results, we conducted a set of linear mixed-effects models with random intercepts. No significant order effects were observed, indicating that the order in which questions were answered did not systematically bias self-reports. These results are reported in the Supplemental Material (Table S1).

With respect to the contemporaneous and prospective mixed effects models, the inclusion of time as an additional covariate did not significantly impact the parameter estimates or alter the overall pattern of the results. Thus, for clarity and ease of interpretation, the results reported below are based on models estimated without including this covariate. The full models, which include time into the study as a covariate, are provided in the Supplemental Material (Tables S8–S15).

4.1. Contemporaneous mixed effects models

4.1.1. Association between momentary integral EG and momentary pain intensity, with IAs as a moderator

As shown in Table 3, momentary integral EG was a significant predictor of momentary pain intensity. However, when covariates were included in the model, this association was no longer significant.

The interaction between EG and IAs was significant, indicating that the relationship between momentary integral granularity and pain varied as a function of IAs. As depicted in Figure 1, simple slope analyses indicated that the relationship between granularity and pain was significant and negative at low (−1 *SD*: $B[SE] = -0.29[0.07]$, $t = -3.88$, $p < .001$) and average (*M*: $B[SE] = -0.11[0.05]$, $t = -2.03$, $p = 0.04$) IAs levels. By contrast, this association was not significant at high levels of IAs (+1 *SD*: $B[SE] = 0.07[0.07]$, $t = 0.95$, $p = 0.34$). When covariates were added into the model, the link between granularity and pain remained statistically significant only at low levels of IAs (−1 *SD*: $B[SE] = -0.19[0.07]$, $t = -2.88$, $p < .001$; *M*: $B[SE] = -0.09[0.05]$, $t = -1.83$, $p = 0.07$; +1 *SD*: $B[SE] = 0.02[0.07]$, $t = 0.29$, $p = 0.77$).

When added into the model as covariates, momentary valence, dominance, and intensity of negative emotions were all significantly associated with pain intensity.

The inclusion of within-category EG indices as additional covariates did not meaningfully alter the parameter estimates, standard errors, or *p*-values associated with the main effects of interest. Notably, the main effect of integral EG on pain intensity reached statistical significance when fear-related EG was included as a covariate ($B[SE] = -0.14[0.06]$, $t = -2.25$, $p = 0.024$, when fear-related EG was included as a stand-alone within-category covariate; $B[SE] = -0.22[0.10]$, $t = -2.05$, $p = 0.041$, when all within-category EG indices were simultaneously entered into the model). Similarly, regarding the interaction between integral EG and IAs, simple slope analyses indicated that this association was significant at both low and average levels of IAs when fear-related EG was included as a covariate – either as a stand-alone within-category covariate (−1 *SD*: $B[SE] = -0.26[0.08]$, $t = -3.38$, $p < .001$; *M*: $B[SE] = -0.14[0.06]$, $t = -2.25$, $p = 0.02$; +1 *SD*: $B[SE] = -0.02[0.08]$, $t = -0.25$, $p = 0.80$) or alongside all other within-category EG indices (−1 *SD*: $B[SE] = -0.33[0.12]$, $t = -2.84$, $p < .001$; *M*: $B[SE] = -0.22[0.11]$, $t = -2.05$, $p = 0.04$; +1 *SD*: $B[SE] = -0.10[0.12]$, $t = -0.90$, $p = 0.37$); by contrast, when fear-related EG was not included as a covariate the interaction remained significant only at low levels of IAs. Full results for these models are reported in Table S16 in the Supplemental Material.

4.1.2. Association between momentary between-category EG and momentary pain intensity, with IAs as a moderator

As shown in Table 4, momentary between-category EG alone did not significantly predict momentary

Table 2. Descriptives and within- and between-person correlations.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. EG (Integral)	–	0.96***	0.68***	0.65***	0.72***	−0.09†	−0.09*	−0.07	−0.12**	−0.06	−0.01	0.04	−0.02	−0.08†	–
2. EG (Between)	−0.11	–	0.71***	0.72***	0.73***	−0.09*	−0.09*	−0.07	−0.11*	−0.07	0.00	0.07	−0.02	−0.08	–
3. EG (Anger)	−0.04	−0.02	–	0.44***	0.47***	0.02	0.02	0.05	−0.02	0.02	−0.06	0.09*	0.03	−0.03	–
4. EG (Fear)	0.26†	−0.33*	−0.17	–	0.49***	−0.06	−0.05	−0.07	−0.10*	0.01	0.00	0.04	−0.02	−0.02	–
5. EG (Sadness)	0.00	−0.28*	−0.12	0.24	–	−0.04	−0.04	−0.06	−0.04	−0.03	0.03	0.06	−0.01	−0.06	–
6. Negative Emotions	−0.06	−0.07	−0.09	0.19	0.04	–	0.98***	0.81***	0.81***	0.84***	−0.61***	0.31***	−0.48***	0.46***	–
7. Negative Emotions (Between)	0.11	0.02	0.16	−0.18	0.12	−0.14	–	0.82***	0.83***	0.85***	−0.62***	0.32***	−0.48***	0.44***	–
8. Negative Emotions (Anger)	0.02	−0.04	−0.08	0.37**	0.05	−0.15	−0.12	–	0.57***	0.57***	−0.56***	0.39***	−0.36***	0.42***	–
9. Negative Emotions (Fear)	0.11	−0.24†	−0.14	0.12	0.15	−0.09	0.06	0.05	–	0.61***	−0.48***	0.29***	−0.39***	0.37***	–
10. Negative Emotions (Sadness)	−0.04	−0.09	0.04	−0.23	0.07	0.05	0.08	0.01	0.12	–	−0.55***	0.16***	−0.46***	0.38***	–
11. Valence	0.06	0.01	0.09	0.09	−0.18	−0.16	−0.02	0.10	0.22	0.02	–	−0.25***	0.52***	−0.40***	–
12. Arousal	0.07	−0.04	0.15	0.08	0.07	0.23	0.00	0.05	−0.11	−0.11	−0.25*	–	−0.20***	0.19***	–
13. Dominance	0.02	−0.05	−0.09	−0.24	−0.03	−0.08	0.00	−0.35**	0.18	0.09	−0.12	0.16	–	−0.30***	–
14. Pain	−0.13	0.04	0.05	0.00	−0.04	−0.10	−0.07	−0.05	−0.08	−0.07	−0.04	−0.08	−0.24†	–	–
15. IAs	−0.04	−0.16	0.03	−0.01	0.08	−0.18	0.16	0.03	−0.15	−0.15	−0.04	−0.14	−0.01	0.12	–
M	−4.22	−1.74	−1.97	−2.03	−1.91	3.18	3.27	3.84	2.91	3.07	3.67	5.00	3.63	3.66	2.81
SD_{Within}	5.41	2.27	2.36	2.36	2.55	0.76	0.80	0.97	0.89	0.93	1.27	1.48	1.45	1.04	–
$SD_{Between}$	2.29	0.44	0.61	0.56	0.60	1.24	1.27	1.31	1.40	1.36	1.12	1.24	1.23	0.98	0.68

Note: Within-person correlations are presented above the diagonal, between-person correlations are presented below the diagonal. ***$p < .001$; **$p < .01$; *$p < .05$; †$.05 < p < .06$. EG (Integral) = Integral Momentary Emotional Granularity. EG (Between) = Momentary Between-category Emotional Granularity. EG (Anger) = Momentary Anger-related Emotional Granularity. EG (Fear) = Momentary Fear-related Emotional Granularity. EG (Sadness) = Momentary Sadness-related Emotional Granularity. Negative Emotions (Between) = Mean of the cluster-specific average negative emotions ratings for anger, fear, and sadness. Negative Emotions (Anger) = Anger-related Emotions. Negative Emotions (Fear) = Fear-related Emotions. Negative Emotions (Sadness) = Sadness-related Emotions. IAs = Dispositional Interoceptive Sensibility.

Table 3. Multilevel concurrent models of momentary pain intensity predicted by momentary integral negative emotional granularity and its interaction with interoceptive sensibility (*Model a*) while controlling for momentary affective valence, arousal and dominance and momentary negative emotions (*Model b*).

	Model a				Model b			
	Est. (SE)	95% CI	*t*	*p*	Est. (SE)	95% CI	*t*	*p*
Intercept	**3.66 (0.12)**	**[3.44, 3.88]**	**31.51**	**<.001**	**3.65 (0.12)**	**[3.42, 3.87]**	**30.40**	**<.001**
Momentary EG (Integral)	**−0.11 (0.05)**	**[−0.21, −0.01]**	**−2.01**	**0.044**	−0.08 (0.05)	[−0.17, 0.01]	−1.74	0.083
IAs	0.25 (0.13)	[−0.01, 0.50]	1.93	0.058†	0.24 (0.13)	[−0.02, 0.49]	1.82	0.074
Momentary EG (Integral) × IAs	**0.18 (0.05)**	**[0.07, 0.28]**	**3.42**	**<.001**	**0.11 (0.05)**	**[0.01, 0.20]**	**2.25**	**0.025**
Momentary Valence					**−0.20 (0.06)**	**[−0.33, −0.08]**	**−3.14**	**0.002**
Momentary Arousal					0.03 (0.05)	[−0.07, 0.12]	0.62	0.535
Momentary Dominance					−0.11 (0.06)	[−0.23, 0.00]	−1.95	0.051†
Momentary Negative Emotions					**0.31 (0.06)**	**[0.18, 0.43]**	**4.79**	**<.001**

Note: Boldface indicates significant effects with $p < 0.05$. †indicates marginally significant effects. *CI* = confidence interval. CIs were estimated using 2,500 bootstrap resamples. Momentary EG (Integral) = Momentary Integral Emotional Granularity. IAs = Interoceptive Sensibility.

pain intensity, whereas its interaction with IAs was statistically significant. Simple slope analyses indicated that the relationship between granularity and pain was significant and negative at low levels of IAs (−1 *SD*: *B*[*SE*] = −0.27[0.07], $t = -3.66$, $p < .001$). By contrast, this association was not significant at average (*M*: *B*[*SE*] = −0.09[0.05], $t = -1.63$, $p = 0.10$) or high (+1 *SD*: *B*[*SE*] = 0.10[0.08], $t = 1.31$, $p = 0.19$) levels of IAs. When covariates were added into the model, the simple slope remained significant only at low levels of IAs (−1 *SD*: *B*[*SE*] = −0.17[0.07], $t = -2.54$, $p = .01$; *M*: *B*[*SE*] = −0.07[0.05], $t = -1.40$, $p = 0.16$; +1 *SD*: *B*[*SE*] = 0.04[0.07], $t = 0.57$, $p = 0.57$).

When included in the model, momentary valence, dominance, and negative emotions were all significant predictors of pain intensity.

The inclusion of within-category EG indices as additional covariates did not meaningfully alter the parameter estimates, standard errors, or *p*-values associated with the main effects of interest. This suggests that the observed associations are robust even when

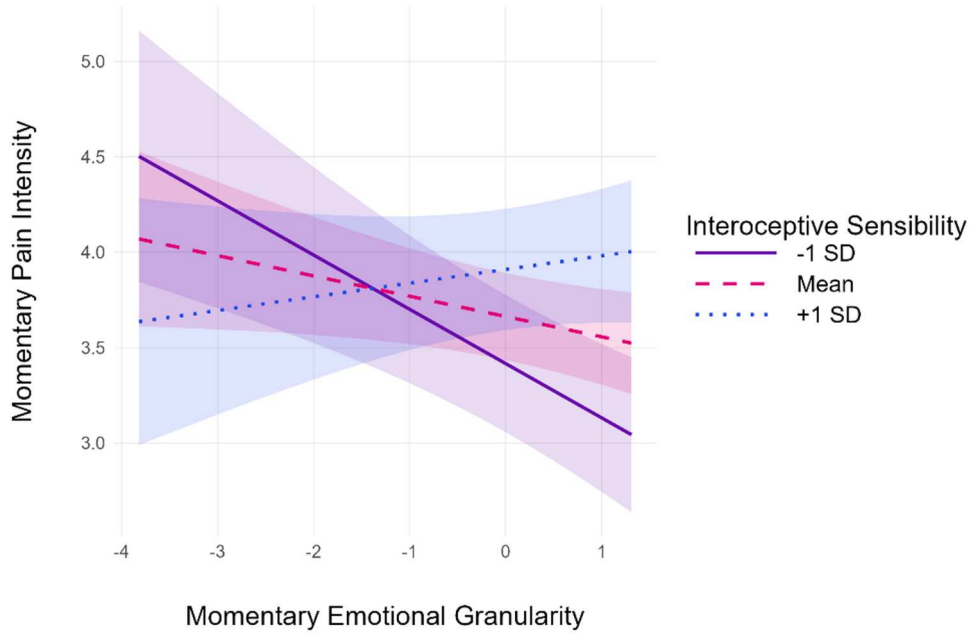

Figure 1. The contemporaneous association between emotional granularity and pain intensity, moderated by interoceptive sensibility.

Note: Lines indicate slopes for different levels of IAs; bands indicate a 95% confidence interval. The solid line represents the trajectory for an individual scoring one standard deviation below the mean score of IAs, the dashed line represents the trajectory at the mean score of IAs, and the dotted line represents the trajectory for one standard deviation above the mean score of IAs. Immagine che contiene testo, schermata, linea, diagramma.

Table 4. Multilevel concurrent models of momentary pain intensity predicted by momentary between-category emotional granularity and its interaction with interoceptive sensibility (*Model a*) while controlling for momentary affective valence, arousal and dominance and momentary negative emotions (*Model b*).

	Model a				Model b			
	Est. (SE)	95% CI	*t*	*p*	Est. (SE)	95% CI	*t*	*p*
Intercept	**3.64 (0.12)**	**[3.40, 3.88]**	**30.25**	**<.001**	**3.63 (0.12)**	**[3.39, 3.87]**	**29.48**	**<.001**
Momentary EG (Between)	−0.09 (0.05)	[−0.19, 0.02]	−1.59	0.113	−0.06 (0.05)	[−0.16, 0.03]	−1.30	0.194
IAs	0.24 (0.13)	[−0.01, 0.50]	1.85	0.069	0.24 (0.13)	[−0.03, 0.48]	1.77	0.082
Momentary EG (Between) × IAs	**0.18 (0.05)**	**[0.08, 0.29]**	**3.53**	**<.001**	**0.11 (0.05)**	**[0.01, 0.21]**	**2.21**	**0.028**
Momentary Valence					**−0.18 (0.07)**	**[−0.32, −0.06]**	**−2.84**	**0.005**
Momentary Arousal					0.03 (0.05)	[−0.07, 0.14]	0.65	0.519
Momentary Dominance					**−0.12 (0.06)**	**[−0.23, −0.01]**	**−2.08**	**0.038**
Momentary Emotions (Between)					**0.30 (0.07)**	**[0.17, 0.43]**	**4.58**	**<.001**

Note: Boldface indicates significant effects. *CI* = confidence interval. CIs were estimated using 2,500 bootstrap resamples. Momentary EG (Between) = Momentary Between-category Emotional Granularity, computed using three negative emotion clusters: Anger, fear, and sadness. Momentary Emotions (Between) = Mean of the cluster-specific average momentary ratings for anger, fear, and sadness. IAs = Interoceptive Sensibility.

controlling for within-category EG. Full results for these models are reported in Table S17 in the Supplemental Material.

4.1.3. Associations between momentary anger-related, fear-related, and sadness-related EG and momentary pain intensity, with IAs as a moderator

As shown in Table 5, momentary within-category EG was not a significant predictor of momentary pain intensity for any of the three emotion clusters (i.e. anger, fear, and sadness). Also, for all three clusters, the interaction between granularity and IAs was statistically significant in the baseline model but became nonsignificant when including covariates. Simple slope analyses are reported in the Supplemental Material (Table S2).

Concerning covariates, both affect valence and cluster-related intensity of negative emotions were significantly associated with pain levels in all three emotion clusters. By contrast, the effect of affect dominance was significant for the anger and fear clusters only.

4.2. Prospective mixed effects models

4.2.1. Association between lagged integral EG and momentary pain intensity, with IAs as a moderator

Integral EG measured at one time point did not significantly predict subsequent pain intensity, nor did the interaction between EG and IAs. In addition, lagged pain intensity was not significantly associated with current pain.

When covariates were added to the model, only lagged arousal was positively associated with current pain intensity, $B(SE) = 0.12(0.06)$, $CI[0.01, 0.25]$, $t = 2.00$, $p = 0.046$. This relationship indicates that greater levels of arousal experienced in a pain episode predict greater pain intensity in the subsequent episode. Notably, the interaction between lagged arousal and Δt was not significant, suggesting that the effect of lagged arousal was not dependent on the time elapsed between pain episodes.

The models' results are reported in the Supplemental Material (Table S3).

4.2.2. Association between lagged between-category EG and momentary pain intensity, with IAs as a moderator

In the baseline model, no associations reached statistical significance. Likewise, when covariates were added to the model, none of the main effects or interactions were significant. The models' results are reported in the Supplemental Material (Table S4).

4.2.3. Associations between lagged anger-related, fear-related, and sadness-related EG and momentary pain intensity, with IAs as a moderator

Concerning the anger-related cluster, lagged EG did not predict momentary pain intensity. However, the three-way interaction between lagged granularity, Δt, and IAs was statistically significant ($B[SE] = -0.1[0.05]$, $CI[-0.22, -0.01]$, $t = -2.05$, $p = 0.041$), suggesting that the joint effect of previous granularity and IAs on subsequent pain intensity depended also on the time elapsed between two consecutive pain episodes. However, this interaction effect became nonsignificant once covariates were added to the model. Additionally, both lagged arousal,

Table 5. Multilevel concurrent models of momentary pain intensity predicted by momentary anger-, fear-, and sadness-related emotional granularity and its interaction with interoceptive sensibility (*Model a*) while controlling for momentary affect valence, arousal and dominance and for momentary cluster-related emotions (*Model b*).

	Model a				Model b			
	Est. (SE)	95% CI	*t*	*p*	Est. (SE)	95% CI	*t*	*p*
Emotion Cluster: Anger								
Intercept	**3.72 (0.12)**	**[3.49, 3.96]**	**31.28**	**<.001**	**3.71 (0.12)**	**[3.47, 3.96]**	**29.83**	**<.001**
Momentary EG (Anger)	−0.03 (0.06)	[−0.14, 0.07]	−0.59	0.556	−0.06 (0.05)	[−0.16, 0.04]	−1.23	0.220
IAs	0.24 (0.13)	[−0.02, 0.48]	1.86	0.068	0.23 (0.13)	[−0.03, 0.50]	1.73	0.089
Momentary EG (Anger) × IAs	**0.15 (0.05)**	**[0.05, 0.26]**	**2.89**	**0.004**	0.05 (0.05)	[−0.04, 0.16]	1.09	0.276
Momentary Valence					**−0.20 (0.07)**	**[−0.33, −0.07]**	**−2.96**	**0.003**
Momentary Arousal					−0.00 (0.05)	[−0.11, 0.10]	−0.01	0.994
Momentary Dominance					**−0.16 (0.06)**	**[−0.27, −0.04]**	**−2.57**	**0.011**
Momentary Emotions (Anger)					**0.32 (0.07)**	**[0.19, 0.45]**	**4.81**	**<.001**
Emotion Cluster: Fear								
Intercept	**3.68 (0.12)**	**[3.44, 3.92]**	**30.18**	**<.001**	**3.67 (0.13)**	**[3.44, 3.91]**	**29.00**	**<.001**
Momentary EG (Fear)	−0.00 (0.06)	[−0.11, 0.11]	−0.08	0.938	0.01 (0.05)	[−0.09, 0.11]	0.22	0.828
IAs	0.25 (0.13)	[0.00, 0.51]	1.96	0.055†	0.25 (0.13)	[−0.02, 0.51]	1.85	0.070
Momentary EG (Fear) × IAs	**0.14 (0.05)**	**[0.04, 0.25]**	**2.71**	**0.007**	0.09 (0.05)	[−0.01, 0.19]	1.87	0.062
Momentary Valence					**−0.27 (0.06)**	**[−0.40, −0.15]**	**−4.34**	**<.001**
Momentary Arousal					0.09 (0.05)	[−0.01, 0.19]	1.74	0.083
Momentary Dominance					**−0.17 (0.06)**	**[−0.30, −0.05]**	**−2.83**	**0.005**
Momentary Emotions (Fear)					**0.15 (0.06)**	**[0.03, 0.27]**	**2.40**	**0.017**
Emotion Cluster: Sadness								
Intercept	**3.68 (0.12)**	**[3.44, 3.93]**	**29.64**	**<.001**	**3.66 (0.13)**	**[3.41, 3.91]**	**29.03**	**<.001**
Momentary EG (Sadness)	−0.07 (0.06)	[−0.18, 0.04]	−1.22	0.224	−0.05 (0.05)	[−0.15, 0.05]	−1.00	0.316
IAs	0.23 (0.13)	[−0.04, 0.48]	1.71	0.092	0.23 (0.14)	[−0.03, 0.51]	1.69	0.097
Momentary EG (Sadness) × IAs	**0.13 (0.05)**	**[0.02, 0.23]**	**2.35**	**0.019**	0.08 (0.05)	[−0.02, 0.17]	1.53	0.128
Momentary Valence					**−0.25 (0.07)**	**[−0.38, −0.12]**	**−3.84**	**<.001**
Momentary Arousal					0.07 (0.05)	[−0.03, 0.17]	1.29	0.200
Momentary Dominance					−0.11 (0.06)	[−0.23, 0.02]	−1.75	0.082
Momentary Emotions (Sadness)					**0.25 (0.06)**	**[0.13, 0.38]**	**3.94**	**<.001**

Note: Boldface indicates significant effects. †indicates marginally significant effects. *CI* = confidence interval. CIs were estimated using 2,500 bootstrap resamples. Momentary EG (Anger) = Momentary Anger-related Emotional Granularity. Momentary EG (Fear) = Momentary Fear-related Emotional Granularity. Momentary EG (Sadness) = Momentary Sadness-related Emotional Granularity. Momentary Emotions (Anger) = Momentary Anger-related Emotions. Momentary Emotions (Fear) = Momentary Fear-related Emotions. Momentary Emotions (Sadness) = Momentary Sadness-related Emotions. IAs = Interoceptive Sensibility.

B(*SE*) = 0.17(0.07), *CI*[0.04, 0.31], *t* = 2.60, *p* = 0.010, and lagged intensity of anger-related emotions, *B*(*SE*) = −0.18(0.08), *CI*[−0.34, −0.01], *t* = −2.10, *p* = 0.036, were significant predictors of momentary pain.

Concerning the fear-related cluster, the interaction between lagged fear-related granularity and IAs became significant after including the covariates, *B*(*SE*) = 0.12(0.06), *CI*[0.00, 0.24], *t* = 2.06, *p* = 0.041, but only at very high levels of interoception (−2 *SD*: *B*[*SE*] = −0.23[0.13], *t* = −1.71, *p* = 0.09; *M*: *B*[*SE*] = 0.02[0.06], *t* = 0.33, *p* = 0.74; +2 *SD*: *B*[*SE*] = 0.27[0.14], *t* = 1.95, *p* = 0.05). Lagged arousal also emerged as a significant predictor of pain intensity, *B*(*SE*) = 0.16(0.06), *CI*[0.03, 0.29], *t* = 2.52, *p* = 0.012.

Concerning the sadness-related cluster, no associations were statistically significant, either in the baseline model or after covariate adjustment.

The models' results are reported in the Supplemental Material (Tables S5–S7).

5. Discussion

The present study aimed to investigate the within-person association between negative EG and pain intensity across different levels of dispositional IAs, both concurrently and prospectively. We explored this relationship via multiple indices of granularity: Integral granularity, between-category granularity, and three within-category indices focused on the emotion clusters of anger, fear, and sadness. We hypothesised that, across both concurrent and prospective temporal patterns and granularity indices, higher EG would be associated with lower pain intensity, with dispositional IAs moderating this relationship. The results partially supported our hypotheses.

5.1. Momentary emotional granularity is related to lower concurrent pain: when and for whom it matters

When examining momentary predictors of pain, higher momentary integral granularity was

significantly related to lower momentary pain within participants. However, this association became non-significant after controlling for the affective tone and the intensity of negative emotions. The interaction between momentary granularity and dispositional IAs remained significant even after adjusting for all covariates, so that granularity was associated with lower pain intensity among participants with low IAs. Moreover, consistent with prior research (Bushnell et al., 2013; Mohr et al., 2012; Müller, 2011; Wiech & Tracey, 2009), lower affect dominance, heightened unpleasantness, and intensity of negative emotions were also associated with greater pain intensity.

Notably, when adding fear-related EG to the model alongside the other covariates, the negative association between integral EG and pain intensity remained significant. Also, when accounting for fear-related EG in the model, the interaction between momentary granularity and dispositional IAs remained significant even among participants with average levels of IAs. These results may suggest a suppression effect, whereby the effect of integral EG is partially masked by the variance shared with fear-related EG. The salience of fear in chronic pain populations (Alappattu & Bishop, 2011) could explain the substantial shared variance between integral and fear-related EG in the present sample.

The novel contribution of this study lies in identifying above-average fluctuations in negative EG as being related to lower pain intensity, particularly for chronic pain sufferers with average-to-low levels of IAs, beyond the concurrent affective and emotional experience. Negative emotions are known to significantly influence how pain is perceived (Bushnell et al., 2013; Wiech & Tracey, 2009), and research has shown that emotional and pain experience share overlapping neural circuits and interoceptive predictive processes (Chang et al., 2015; Wager et al., 2013, 2015; Woo et al., 2015). On the one hand, greater EG may facilitate a clearer conceptualisation of interoceptive signals, thereby minimising the overlap between the emotional and sensory component of pain experiences. In other words, EG may help prevent vague or poorly defined bodily discomfort from being mistakenly perceived as intense pain. Furthermore, a greater ability to accurately identify and label emotional states may enable individuals to reduce the emotional amplification of pain and thus to assign more appropriate meaning and intensity to their pain experiences. On the other hand, our results indicate that the association of momentary EG with lower pain experience was limited to participants scoring low in dispositional IAs – or average-to-low when fear-related EG was accounted for in the model. In other words, low (and, to some extent, average) levels of IAs may allow individuals to disentangle emotional from physical sensations during pain episodes and to assign appropriate meaning and intensity to their pain experiences; by contrast, heightened levels of (perceived) attentional focus on interoceptive signals seem to interfere with the association between momentary EG and reduced pain perception. Of note, some recent studies have claimed that higher scores on the Awareness scale of the BPQ (that we used to measure IAs) reflect maladaptive IAs, characterised by heightened attentional focus on internal signals and increased sensitive perception of uncomfortable bodily sensations (Gaggero et al., 2021; Trevisan et al., 2021; Van Bael et al., 2024). Also, it has been argued that pain perception in individuals with chronic pain may be fuelled by the tendency to attend to sensory cues that are consistent with their predictions of pain (Hechler et al., 2016).

Similar results emerged when examining the relationship between momentary between-category granularity and pain intensity. We found that, just as the ability to differentiate negative emotional states in general, the ability to differentiate emotions among distinct clusters was also associated with lower concurrent pain intensity in women with low IAs. This similarity may not be surprising given the observed strong and highly significant within-person correlation between the integral and between-category granularity indices. This convergence has also been reported in prior research on dispositional EG (Erbas et al., 2019). Our results seem to suggest that the overlap between integral and between-category granularity may not be limited to the dispositional level, but may also extend to momentary, state-level EG.

By contrast, when examining within-category granularity in relation to the anger, fear, and sadness clusters, the analyses revealed a different pattern of results. After adjusting for covariates, neither within-category granularity alone nor its interaction with IAs were associated with pain. Nonetheless, the intensity of negative emotions emerged as a robust predictor: Emotions within the anger, fear, and sadness clusters were all significantly related to

higher levels of pain. For all three clusters, perceived unpleasantness was also a significant predictor of pain intensity. Conversely, affect dominance was a significant predictor of pain only for the anger and fear clusters, consistent with the fact that both are typically considered high-arousal emotion families compared to sadness.

These findings suggest that the ability to better differentiate emotions within a single emotional cluster in a certain moment is not related to the concurrent pain perception, independently from individuals' levels of IAs. Affective qualities (unpleasantness and dominance) and the intensity of cluster-related negative emotions appear to be more relevant than the within-category precision of emotional labelling in the experience of pain. Granularity can vary across time and context (Erbas et al., 2018; Hoemann et al., 2023). Thus, it is plausible that it plays different roles depending on the level of emotional specificity. One hypothesis is that when individuals focus on differentiating between similar emotions within a single cluster, this detailed discrimination may inadvertently amplify the salience of negative emotions linked to that cluster. This may explain why the strength of negative emotions, rather than the precision of emotion differentiation, accounted for pain perception in our results. This finding stands in contrast to the pattern of results observed for integral EG, which was associated with lower pain intensity among individuals with low IAs. In such cases, attention is not narrowly focused on a single emotional cluster but spans multiple clusters. This suggests that the adaptive value of EG may depend on its scope – whether it is applied narrowly within a single cluster or broadly across multiple emotional domains.

Overall, our findings emphasise the importance of the level at which granularity is assessed. Distinguishing across negative emotion categories may help individuals with low IAs to decouple emotional distress from pain perception, thereby reducing pain intensity. By contrast, the momentary ability to differentiate emotions focusing on a single emotion category seems to be unrelated to pain intensity, possibly because this focus may lead individuals to amplify emotional discomfort and its impact on pain perception. Future studies could further examine the adaptive function of granularity in the context of chronic pain testing the hypothesis that its adaptive implications may depend not only on the ability to finely differentiate emotions per se but also on the degree of specificity with which those emotions are examined.

5.2. Prospective risk factors for pain chronification: hidden costs of feeling too much

When prospective predictors of pain were examined, EG alone did not exhibit a significant main effect on pain intensity. Our hypothesis was thus not confirmed. Also, the interaction between indices of granularity calculated across integral or between-category metrics and IAs was nonsignificant. When considering granularity indices within emotion categories, a different pattern of results was observed depending on the emotion cluster. Momentary granularity within the sadness cluster showed no significant effects on pain intensity. In a similar way, when examining the anger cluster, the interaction of granularity and IAs became nonsignificant after controlling for covariates. In contrast, granularity within the fear cluster significantly predicted future pain intensity among individuals with high levels of IAs. Thus, for individuals reporting a heightened perceived sensitivity to and sustained attentional focus on interoceptive signals, momentary increases in fear-related granularity (relative to their individual mean) were associated with greater subsequent pain intensity.

This finding is consistent with the Fear-Avoidance Model, one of the most prominent models in the field of CPP (Alappattu & Bishop, 2011). According to this model, fear-related emotional processing is a central mechanism in the maintenance and exacerbation of chronic pain: Pain-related fear fosters hypervigilance toward bodily sensations and avoidance behaviours, which in turn contribute to pain chronification. These dynamics are also supported within predictive coding accounts of chronic pain, which conceptualise pain persistence as a function of maladaptive priors that are continually reinforced through aberrant interpretations of internal bodily signals across hierarchical interoceptive levels (Garfinkel & Eccleston, 2025; Ongaro & Kaptchuk, 2018). Within this integrative theoretical framework, individuals scoring high in IAs may be especially vulnerable to experiencing pain-related fear due to their heightened attentional focus on internal signals. In this context, pain experiences may reinforce fear and validate the perceived need for continuous monitoring of bodily states, thereby contributing to a self-perpetuating cycle that sustains pain. Consistent with this

idea, our findings showed that when individuals with high IAs exhibited heightened fear-related granularity while experiencing pain, this increased differentiation was associated with greater pain in subsequent pain episodes. Thus, we speculate that, in individuals suffering from chronic pain, identifying and focusing on fear-related emotions while experiencing pain may reinforce maladaptive beliefs linking interoceptive sensations to threat or actual harm. This process may, in turn, intensify pain-related fear, ultimately exerting a prospective impact on future pain experiences. In other words, fear-related EG may contribute to a self-reinforcing cycle that promotes the persistence and chronification of pain in individuals with heightened attentional focus on bodily sensations.

Of note, past pain intensity did not emerge as a significant predictor of subsequent pain experiences. Although this finding may appear counterintuitive through the lens of predictive coding models, this apparent discrepancy may become more theoretically coherent if one considers the role of arousal rather than pain intensity per se – in our results, individuals who experienced high arousal during prior pain episodes were more likely to report elevated pain in subsequent episodes. Arousal, as an index of the intensity of an affective experience, represents a generalised state of physiological activation, which (according to the TCE) may be categorised and interpreted in different ways (e.g. as pain, emotion, or another affective state) depending on the context and the individuals' history. Of note, affective arousal is increasingly recognised as a key driver of pain persistence and chronicity (Ciuffini et al., 2023; Ravn et al., 2018). Consistent with this view, our results indicated that individuals who experienced high arousal during prior pain episodes were more likely to report elevated pain in subsequent episodes, regardless of lagged intensity of negative emotions and of pain. Because arousal reflects a dimension of general physiological state, these findings suggest that the persistence of pain over time may be driven by a more global affective activation rather than by the specific content of the experience (i.e. pain or emotional intensity) per se. This result aligns with the view that the boundaries between pain and emotion are often blurred in the immediate experience of pain, highlighting their shared affective mechanisms (Craig, 2003).

Interestingly, arousal was significantly associated with subsequent but not with concurrent pain levels. Conversely, the affective components of unpleasantness and dominance, and the intensity of negative emotions exhibited a reverse pattern of associations (i.e. they were associated with concurrent pain experience, but they did not emerge as significant predictors in the prospective models). These discrepancies seem to suggest that different affective mechanisms may occur depending on the temporal perspective. In the here and now, the experience of pain appears to be more closely linked to affective features that are either tied to evaluative dimensions of the situation or the emotional salience of the moment – captured by affect unpleasantness and dominance, and emotional intensity, which reflect the appraisal and meaning of the ongoing experience. Arousal, by contrast, seems to function as an undifferentiated affective activation that shapes anticipatory mechanisms involved in pain chronification. Within a predictive coding framework, this undifferentiated affective activation may become embedded in prior beliefs about bodily threat, biasing the interpretation of future interoceptive input and reinforcing pain expectations (Norton & Asmundson, 2003). These findings highlight the need to distinguish between immediate correlates of pain and the longer-term affective mechanisms that underpin pain persistence.

5.3. The conditional role of dispositional interoceptive sensibility on pain

Dispositional IAs did not emerge as a stand-alone predictor of pain, neither concurrently nor prospectively for any of the granularity indices examined. This finding is particularly noteworthy in light of previous literature suggesting that individuals with chronic pain tend to report elevated levels of IAs, both within the context of CPP (Scarpina et al., 2025; Spinoni et al., 2024) and chronic pain more broadly (Horsburgh et al., 2024). Furthermore, IAs has been associated with both hypersensitivity in acute CPP processing (Scarpina et al., 2025) and with pain severity in patients with chronic pain (Aaron et al., 2019), even though evidence regarding the relationship between IAs and pain intensity is still mixed and overall inconclusive (Horsburgh et al., 2024). Notably, however, this body of research has rarely considered the potential role of emotional components – that are intertwined with the sensory ones in the experience of pain (Bushnell et al., 2013; Craig, 2003; Wiech & Tracey, 2009). In this regard, our results seem to indicate that when within-person fluctuations in emotional and affective

correlates of pain are taken into account, the predictive power of dispositional IAs alone becomes considerably less central in explaining pain intensity. It is thus possible that the inconsistent association between dispositional IAs and pain intensity observed in prior studies may, at least in part, reflect shared variance with affective processes that have not been explicitly modelled. While IAs remains a relevant trait-level factor in the construction of pain experience, our findings seem to underscore the importance of considering IAs as part of a broader, interactive system of affective and emotional processes.

From a theoretical standpoint, it has been posited that interoceptive processing at multiple hierarchical levels underlies the construction of affective experiences (Craig, 2003; Garfinkel & Eccleston, 2025; Greenwood & Garfinkel, 2024; Horsburgh et al., 2024; Poublan-Couzardot & Talmi, 2024). Dispositional IAs is characterised by heightened (self-reported) attentional focus and sensitivity to internal sensations and represents a higher-level interoceptive domain that influences how bodily signals are attended to, interpreted, and categorised (Garfinkel & Eccleston, 2025; Zacharioudakis et al., 2020). Consistent with this conceptualisation, theoretical accounts and prior empirical evidence have emphasised the role of both interoceptive and exteroceptive attentional processes in affect categorisation (Barrett, 2009; Barrett et al., 2004; Potthoff et al., 2023; Ventura-Bort et al., 2021). Pain – as an affective state comprising both sensory and emotional components (Bushnell et al., 2013; Craig, 2003; Wiech & Tracey, 2009) – can be thus understood as emerging from ongoing higher-order constructive processes (Barrett, 2017a) that include individual differences in IAs (influencing the allocation of attention to internal signals) alongside within-person variation in EG (which reflects higher-order conceptual processes involved in the differentiation and categorisation of these signals).

Our findings tentatively support this view, suggesting that IAs is not directly associated with pain but rather shapes pain experience indirectly through its interaction with EG. A speculative hypothesis is that the association of IAs with pain intensity is overshadowed by emotional functioning and affective correlates. It may also be that the precision with which affect is categorised modulates the degree to which IAs translates into pain experience. These hypotheses, however, remain tentative and at a speculative level since affective categorisation processes were not directly operationalised in the present study, but were inferred from a single conditional pattern – specifically, the negative conditional association between EG and pain intensity. Thus, direct empirical examination in future research is needed.

5.4. Limitations and future directions

The present study represents (to the best of our knowledge) the first attempt to investigate the role of EG in chronic pain experiences. Despite its novel contribution, some limitations should be considered when interpreting the findings.

A key limitation concerns the variability in the number of pain episodes reported by participants, which may have influenced the reliability of the momentary granularity indices. Because the denominator in the index formula was computed on the basis of variance across all observations, the stability of the momentary granularity estimate ultimately depends on the total number of measurements per participant. Although momentary granularity is calculated on a moment-by-moment basis, its reliability hinges on variance estimates aggregated across all available measurement occasions. The ecological, event-contingent design of the study (in which the measurement is inherently linked to the number of pain episodes) limited our possibility to strictly control or standardise the frequency of pain episodes across participants. Although inclusion and exclusion criteria were employed to minimise heterogeneity, the high intraindividual variability in chronic pain (Sundström et al., 2025) prevented us to reach full consistency in the number and frequency of pain episodes across participants. Despite this limitation, we chose not to extend the assessment period to reduce participant burden (e.g. from one to two months). Future research could address this limitation by increasing the number of pain episodes monitored, ideally selecting samples characterised by more frequent pain events.

A related limitation is the sample size. Although sixty-eight participants with at least three observations each are considered adequate on the basis of simulation studies (Maas & Hox, 2005), a larger sample would increase the statistical power and improve the robustness of the results. Thus, replication with larger samples is needed.

The absence of physiological measures represents an additional limitation. Integrating objective assessments of IAc alongside self-reported data of IAs

would provide a more comprehensive understanding. The scientific literature indicates that IAs and IAc are related differently to chronic pain experiences (Horsburgh et al., 2024). Future studies could explore whether the association between EG and pain varies depending on IAc levels. Furthermore, given that our results showed a significant prospective link between self-reported arousal and subsequent pain experiences, measuring physiological arousal during pain episodes would offer an objective counterpart to subjective reports.

Finally, this study focused primarily on within-person dynamics, in line with recent calls and growing evidence highlighting the importance of capturing intraindividual variability in chronic pain (Sundström et al., 2025). Future research investigating between-person associations could provide further insights. Our focus on within-person fluctuations allowed for a detailed exploration of the interplay between affect, emotional components, and pain. However, within- and between-person approaches are not mutually exclusive, and both can yield complementary insights. Exploring between-person patterns may further enhance the understanding of chronic pain mechanisms. In particular, investigating dispositional granularity alongside dispositional variables such as interoceptive ability could provide additional valuable contributions to this field.

6. Conclusions

Overall, our findings suggest that within-person EG is a dynamic, conditional factor in the experience of neuropathic and nociplastic CPP. Moment-to-moment, broader (integral and between-category) granularity was associated with lower concurrent pain intensity, particularly among women with average-to-low levels of dispositional IAs. Moreover, we found that finer fear-specific granularity prospectively amplified pain in participants with high IAs, and this result is consistent with fear-avoidance and predictive-processing accounts of pain chronification. These findings suggest that the role of granularity in the experience of pain may depend on an individual's habitual focus on bodily signals, but also on the scope of differentiation. While preliminary, these findings also suggest that interventions that foster cross-category emotion labelling, particularly when combined with strategies to temper hypervigilant interoceptive focus, may be a promising intervention strategy for alleviating CPP. Nonetheless, further studies with larger samples and more measurement occasions are warranted to replicate and extend these within-person effects.

Notes

1. We initially hypothesised that the association between emotional granularity and pain intensity would be stronger among individuals with neuropathic and nociplastic CPP compared to those with CPP due to tissue damage. However, this hypothesis could not be tested, as our final sample included only participants with neuropathic and nociplastic CPP. This limitation was due to difficulties in recruiting participants with organic CPP who met the study's inclusion criteria and were available for intensive longitudinal data collection. A second difference between the present study and the preregistration is that this latter focused on prospective hypotheses only. To capture potential moment-to-moment dynamics that may not be evident in prospective models alone, we decided to add a non-preregistered objective to examine concurrent associations. Third, although IAs was mentioned in the preregistration as an exploratory variable, the specific moderation hypothesis tested in this study was developed post hoc in light of new literature published after preregistration (Poublan-Couzardot & Talmi, 2024). Finally, preregistered hypotheses regarding changes in granularity and pain over time are not addressed here and will be examined in a separate manuscript.
2. We did not include random slopes for emotional granularity in our model, since the ICC for all emotional granularity indices was extremely low ($ICC \leq 0.001$). This suggests that almost all the variance in granularity occurred within individuals across time. Given this lack of meaningful between-person variation, including random slopes for emotional granularity would not substantially improve model fit and would introduce unnecessary complexity, contrary to the principle of parsimony guiding our modeling choices.
3. To compute Δt, we first scaled the time variable (which represents the date and time of each participant's entry) by converting the date and time of each participant's entry into a continuous scale spanning a 1-month period. This transformation resulted in a range from 0 to 30, with each decimal value representing a specific date and time within that period. We then calculated Δt by subtracting the scaled time of the previous measurement from the next measurement.

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Disclosure statement

No potential conflict of interest was reported by the author(s).

Data availability statement

Study materials, data, and analysis codes are available at https://osf.io/xsqgc/files/osfstorage.

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