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ORIGINAL ARTICLE

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Looking for trouble: A common threat-detection mechanism underlying pain, fear, and anxiety

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I put forward a novel cognitive architecture for pain, fear, and anxiety, according to which these three capacities are underpinned by a common *threat-detection mechanism*. This mechanism takes information about potential threats as input, assesses whether the threat is actual and, if it deems it is, outputs a threat representation. If this is correct, pain, fear, and anxiety turn out to be different manifestations of the same cognitive mechanism. I defend my proposal by discussing a large swath of convergent evidence from different quarters of the sciences of the mind.

KEYWORDS
anxiety, cognitive architecture, fear, pain, threat

1 | INTRODUCTION

Everyone believes that pain, fear, and anxiety are different capacities. And everyone is right. One could therefore be forgiven for assuming that pain, fear, and anxiety are underpinned by distinct cognitive mechanisms. In fact, this assumption must have seemed so obvious that most philosophers and cognitive scientists have taken it as given. But the assumption is false. In this article, I show that if we take a synoptic look at pain, fear, and anxiety, it becomes apparent that these capacities share a number of surprising features, and I argue that the best explanation of this fact is that pain, fear, and anxiety are underpinned by a common cognitive mechanism, whose function is to detect threats. Accordingly, one can summarise my proposal as follows: Pain, fear, and anxiety, although different capacities, are underscored by a common *threat-detection mechanism*.

My account will become clear as we go along, but let me spell out a few things at the outset. The words "pain", "fear", and "anxiety" can be used to refer both to capacities and to mental states. In this article, I put forward a novel cognitive architecture of the *capacities* for pain, fear, and anxiety. As such, I will normally use "pain", "fear", and "anxiety" in the capacity sense. To denote the outputs of these capacities—that is, pain, fear, and anxiety *qua mental states*—I will instead typically use "pain/fear/anxiety experience" or "feeling pain/fear/anxiety".[1]

It is important to note that "pain", "fear", and "anxiety" each refer to capacities of a varying nature. My focus is on some of their most basic forms. When I talk of fear and anxiety, I have in mind *environmental* fear and anxiety (e.g., being afraid of a big animal, or feeling anxious when walking in a park at night), rather than their social (e.g., fear of rejection) or existential (e.g., Kierkegaardian angst) counterparts (see Kurth, 2018 for a more in-depth taxonomy).[2] As for pain, I will concentrate on *acute* (rather than chronic), *somatic* (rather than visceral), *nociceptive* (rather than neuropathic) pain (cf. Loeser & Treede, 2008).[3] You cut your finger; you twist your ankle; you put your foot into scalding water. Ouch, ouch, and ouch again! This is what I mean by "pain".[4]

Even more specifically, I will be concerned with the *simplest* types of acute, somatic, nociceptive pain and environmental fear/anxiety—where "simplest" indicates their relative cognitive unsophistication. For example, I will discuss fear of predators, but I will put to one side things like being afraid that your car will catch fire unless you fix its exhaust system. The reason for this is twofold. First, these *cognitively minimal* forms of acute, somatic, nociceptive pain and environmental fear/anxiety are the ones on which we possess the largest set of scientific data. Second, I consider this to be sound methodological advice: Start accounting for the most basic forms of a phenomenon and, once this explanatory goal has been achieved, move up to the more complex cases.

And here goes the final clarification. Pain, fear, and anxiety are each underpinned by a multiplicity of mechanisms. When I say that a common cognitive mechanism underpins them, I do not mean that they have the same overall total cognitive architecture. I just mean that they share *at least one* cognitive mechanism, whose function is to detect threats. To argue for this, I proceed as follows: I first show that pain, fear, and anxiety each perform a threat-detection function (Sections 2 and 3); I then argue that there is one mechanism underscoring this function (Sections 4–6).

2 | REPRESENTING THREATS

Pain, fear, and anxiety, I propose, are *threat-detection capacities*. They receive information about potential threats as input, and they assess whether the threat is actual. If they deem it is, they produce a threat representation as output. I argue for this in two steps: In this section, I focus on their output; in Section 3, I turn to their input.

Many philosophers maintain that the outputs of pain, fear, and anxiety are mental representations (de Vignemont, 2024; Mendelovici, 2013; Tye, 1995), and so do I. But what do they

[1] I say "normally" and "typically" because, on some occasions, the context should suffice to clarify what I mean.

[2] I expand on the notions of environmental fear/anxiety in Section 2.

[3] These criteria are meant to rule out phenomena like arthritic pain (which is chronic), menstrual pain (which is visceral), and diabetic pain (which is neuropathic).

[4] My proposal might also apply to other forms of pain, fear, and anxiety, but this is beyond the remit of this article.

represent? I propose that pain, fear, and anxiety experiences all have a content of the form *O is a threat*. To argue for this, I adopt the following standard methodology: I will assign content *C* to mental state *M* on the basis that assigning *C* to *M* best explains the role *M* plays in a creature's cognitive economy (Shea, 2018).

2.1 | What pain represents

Suppose you feel pain when you put weight on your right ankle. The main effects of this experience include[5]:

i. *Behavioural responses*: You engage in protective (e.g., not walking on your ankle) and restorative (e.g., massaging the ankle) behaviours (Seymour et al., 2023).
ii. *Cognitive responses*: Your pain experience captures and consumes your attention, at the expense of other cognitive processes (Eccleston & Crombez, 1999).
iii. *Communicative responses*: You groan, grimace, and moan. You also make pain utterances such as "My ankle hurts!" (de C Williams, 2023; Wiggleton-Little, 2024).

What ascription of content best explains the fact that your pain experience results in (i) to (iii)? A well-known proposal is that pain experiences represent the presence of *damage* in the body (Tye, 1995). This proposal has a lot going for it. If your pain experience represents that your ankle is damaged, we can explain why you stop walking on your ankle (you do not want to worsen the damage) and perform restorative behaviours (you want to fix the damage). We can also make sense of why your pain experience grabs your attention at the expense of other cognitive processes (taking care of the damage is the most urgent thing to do—the rest can wait), and why you engage in the communicative behaviour you do (if your body is damaged, crying for help is a sensible thing to do).

Should we conclude that pain experiences represent bodily damage? Klein (2015b) argued, convincingly in my view, that we should not (see also Casser, 2021; Corns, 2014). His argument goes like this. Many agree that a necessary condition for mental states of type *M* to represent property *P* is that instances of *M* are reliably caused by instances of *P* (Adams & Aizawa, 2021). There are however numerous instances of pain that are not caused by bodily damage. You immerse your foot in very hot, but not scalding, water; you stretch your leg a bit too far; you sit in an uncomfortable position. In these cases (and the list could go on *ad nauseam*), you have a pain experience, but no bodily damage is to be found. Therefore, pain experiences do not represent bodily damage.

Here is a way out. Bodily damage is a threat. Therefore, when you feel pain after breaking an ankle, your pain experience has been caused by a threat. But a bodily condition can be a threat even if there is no damage—it is enough that the bodily condition is *in the neighbourhood of damage*, where this notion picks out proximity to damage along a variety of dimensions, ranging from temporal proximity to physiological proximity. All the cases discussed by Klein belong to this category. Consider thermal pain. A skin temperature of just above 43°C typically brings about a pain experience in human adults. Non-vulnerable adults are unlikely to get a burn injury from this temperature. However, a mere one-degree Celsius rise would normally

[5]In characterising the effects of pain/fear/anxiety experiences, I focus on the core effects discussed in the cognitive sciences.

suffice for tissue damage to occur (Martin & Falder, 2017).[6] Accordingly, if we hypothesise that, rather than damage to the body, pain experiences represent that *a certain bodily condition is a threat*, we do not face the "causal problem" any longer.[7]

But can the idea that a pain experience represents a bodily condition as a threat also explain (i) to (iii)? It can. Why do you stop extending your leg when you feel pain? Because you have received the information that your muscles are under threat. Moreover, since it is important to know whether your body is threatened, it should not come as a surprise that your pain experience captures your attention. By the same token, it is useful to let other people know that your body is under threat—hence, your groaning and grimacing. I conclude that pain experiences represent threats.

2.2 | What fear represents

The idea that fear experiences represent threats is widespread (see Adolphs, 2013 for an overview). And with good reason. You are walking through a forest, when you notice a tiger and become afraid. Here are the likely effects of your experience:

i* *Behavioural responses*: You can fight the tiger, flee, or freeze (Adolphs, 2013).
ii* *Cognitive responses*: You become hyper-vigilant. Your attention is drawn towards the tiger, and you carefully check what it is doing (Davis & Whalen, 2001).
iii* *Communicative responses*: You can call for help or signal the presence of the tiger to other people (Scarantino et al., 2022).

The hypothesis that your fear experience represents the tiger *as a threat* can account for (i*). Animal models suggest that when faced with a predator, one's fear behaviour is modulated by distance (Fanselow & Lester, 1988): If the predator is far away, freezing is the likely response; if it is approaching, fear typically results in fleeing; when avoidance seems impossible, defensive attacking often ensues. This is what one would expect if fear experiences represented their object as a threat—all these behaviours are contextually appropriate strategies for threat reduction. The same point applies to (ii*) and (iii*). If your fear experience represents the tiger as a threat, we can make sense of why it results in you being hyper-vigilant, and why you are likely to signal the presence of the tiger to other people.

Both pain experiences and fear experiences have a content of the form *O is a threat*. There are, however, two important differences between the content of these experiences. First, these experiences often ascribe the property *being a threat* to different types of objects: While pain experiences always represent a bodily condition as a threat, many fear experiences ascribe the property of being a threat to a *worldly object* (a tiger, a snake, an assailant, you name it). This does

[6]There is a reason why pain experiences have this causal profile. While it is important for an organism to know that its body has been damaged, it is equally important for the organism to prevent bodily damage. Pain experiences perform this *preventative role* by being causally sensitive to bodily conditions that are dangerously close to damage.
[7]Klein offers a different solution: Pain experiences do not represent at all; rather, they command to protect a certain body part (see Barlassina & Hayward, 2019; Martínez, 2011 for alternative ways of developing an imperativist account of pain). While I have a lot of sympathy for the idea that pain experiences have imperatival content, I think that Klein's suggestion that they *only* have such content is hasty. Klein reaches this conclusion by arguing that there is no "nontrivial property that pain-causes have in common" (Klein, 2015b, p. 35). If I am right, *being a threat* is such a property.

not mean that one cannot be afraid of being in such-and-such a bodily condition.[8] The point is rather that while pain experiences are invariably body-directed, fear experiences need not be so.

But what about those fear experiences that are indeed body-directed? Yesterday, I was afraid of having a back injury; today, I have a backache. Do these experiences have the same content? The answer is still "No"—and it is here that the second difference in content becomes relevant. Even if it is true that my fear experience and my pain experience ascribe the same property (i.e., *being a threat*) to the same object (i.e., the condition of my back), the fact remains that this object is represented in different ways: My backache carries a lot of fine-grained, sensory information about the state of my back; my fear experience does not.[9]

2.3 | What anxiety represents

You are walking in a park at night, when you hear footsteps behind you and start to feel anxious. Here is what is likely to happen:

i** *Behavioural responses*: You decide to walk close to the lamp posts. It is not the quickest route, but you do not want to walk where nobody can see you (Steimer, 2002).

ii** *Cognitive responses*: You are in a state of alertness and you "keep your eyes peeled" (Grupe & Nitschke, 2013).

If your anxiety experience represents somebody behind you as a threat, we can explain why you exert such caution.

Both fear experiences and anxiety experiences can represent somebody as a threat. Does this mean these experiences have the same content? There is a lot of controversy at this juncture and, given my aims, it is not necessary to settle the issue. Still, I think that the proposal made by Kurth (2018) is on the right track. According to him, while "fear concerns *clear* threats ... , anxiety ... concerns situations involving *unclear* threats" (Kurth, 2018, p. 33). In other words, fear experiences typically involve a representation of a *specific* threat (say, a snake nearby you), and therefore result in a series of prepotent responses geared to deal with that particular threat as effectively as possible (i.e., i* to iii*). Anxiety experiences instead typically represent an *uncertain* threat, and thus bring about a more generalised set of responses aimed at minimising risk (i.e., i**) and at gathering evidence (i.e., ii**).

2.4 | Taking stock

Pain, fear, and anxiety involve the production of a common representational output type, namely, threat representations. To be sure, there are differences in content between these representations: While pain experiences are always about one's *bodily condition*, fear experiences and anxiety experiences are typically directed at some *worldly object* (a *specific* one, in the case of

[8]In this regard, the word "environmental" in "environmental fear" (as well as in "environmental anxiety") is potentially misleading, since it suggests that one's fear (or one's anxiety) must be directed at the environment rather than at one's body. This is not, however, the notion of environmental fear/anxiety I have in mind. Rather, following Kurth (2018), I take environmental fear/anxiety to contrast with things like *social* fear/anxiety (e.g., being afraid of/anxious about losing one's social status) or *practical* fear/anxiety (i.e., fear of/anxiety about what the right thing to do is).

[9]The point applies, mutatis mutandis, to the relationship between pain experiences and anxiety experiences.

fear, or an *uncertain* one, in the case of anxiety). Still, what unites them is greater than what divides them: Pain experiences, fear experiences, and anxiety experiences all have a content of the form *O is a threat*. In the next section, I argue that the inputs to pain, fear, and anxiety exhibit a similar pattern of similarities.

3 | INFORMATION ABOUT POTENTIAL THREATS

Some things are *potential threats*: They are things that *might* constitute a threat, given the situation one is in. For example, in an environment containing a large number of vipers, any coiled object is a potential threat to me, since any coiled object could be a viper. In this section, I propose that information about potential threats constitutes the input to pain, fear, and anxiety. More precisely, I argue that pain processing is activated by inputs carrying information about potentially threatening *bodily conditions*, while fear and anxiety processing can be turned on both by information concerning *bodily threats* and by information about *worldly threats*.

Two terminological clarifications should help elucidate my proposal. First, we should keep inputs and stimuli distinct. *Stimuli* are the distal causes of pain, fear, and anxiety—for example, twisting your ankle, an approaching animal, or a sound coming from behind a bush. *Inputs* are instead proximal causes; that is, the neural signals that directly activate pain, fear, and anxiety processing. My claim can therefore be reformulated as follows: The inputs to pain, fear, and anxiety carry the information that a certain stimulus is a potential threat.

What does it mean that something carries information about something else? Here goes the second terminological clarification. I assume a widespread notion of information, according to which *A* carries information about *B* if and only if *A* reduces the uncertainty of *B* (Cover & Thomas, 2005). Why does smoke carry information that there is fire? Because the presence of smoke reduces the uncertainty of the presence of fire.

3.1 | Pain's input

A simple way in which *A* can reduce the uncertainty of, and therefore carry information about, *B* is by reliably co-varying with it. Why does smoke carry information that there is fire? Because when there is smoke, there (typically) is fire, and vice versa. Hence the question: Do the inputs to (acute, somatic, nociceptive) pain reliably co-vary with potentially threatening bodily conditions? The answer seems to be, "Yes, of course". After all, the causal chain that leads to this type of pain begins with nociceptors in the skin, muscles, and bones responding to stimuli and nociceptors are defined as the "specialised neurons that detect and respond to *potentially damaging* forms of energy" (Tracey, 2017, p. R123, emphasis added). Can I then conclude that pain takes information about potentially threatening bodily conditions as input? Not yet. Two hurdles need to be cleared first.

3.1.1 | The problem of episodic analgesia

Casser (2021, p. 369) correctly notices that *episodic analgesia* is a rampant phenomenon: "Temporary insensitivity to pain [exhibited, e.g., by wounded soldiers or by people admitted in an

emergency room] illustrate[s] just how commonly noxious stimuli fail to elicit pain sensations". Does this tell against the idea that pain is turned on by information about potential bodily threats? I do not think so. Episodic analgesia would be a problem for me if it had to do with pain processing in the brain failing to respond to threat-related inputs. But episodic analgesia does not work like this. Casser again: "[because of inhibitory influences] nociceptive signals caused by noxious stimuli might be prevented from reaching relevant brain regions, and therefore fail to elicit pain experiences in the organism" (Casser, 2021, p. 370). This point is important, so let me elaborate upon it.

The most influential theory of pain is the *gate control theory* (Melzack, 1996). It is called this because it posits the existence of a gate mechanism in the dorsal horn of the spinal cord that controls which nociceptive inputs are sent to the brain for pain processing. This input-control activity depends on two types of pathways. There are *ascending pathways*, which send information from the periphery of the body to the dorsal horn, with the latter integrating this multifarious information and modulating what nociceptive input is sent to the brain accordingly. Klein (2024, p. 1451) puts it nicely: "The spinal gate acts ... as a sort of volume knob ... Some inputs (sustained C-fibre firing, for example ...) increase the gain ... Other inputs turn down the volume". There are also *descending pathways* from the brain to the spinal cord, which allow for the top-down modulation of nociceptive input—for example, they allow for beliefs to influence what input is sent from the dorsal horn to the brain for pain processing.

We can thus elaborate on Casser's point as follows: Episodic analgesia is likely to be the result of descending, inhibitory modulation over the spinal gate. Suppose that a badly wounded person appraises that the best course of action is to run to the hospital, rather than to collapse in agony. If this information is sent from the brain to the dorsal horn, it can result in the latter "turning down the nociceptive volume". As a result, the brain does not get (enough) nociceptive input, and this is why it does not produce a pain response. Episodic analgesia is therefore not a counterexample to my claim that pain processing in the brain is turned on by information about potential threats.

3.1.2 | The problem of inflammatory pain

In *inflammatory pain*, nociceptors become sensitised following injury, leading to allodynia; that is, experiencing pain in response to normally innocuous stimuli (Neumann et al., 1996). Prima facie, this is a case in which nociceptive pain is turned on by something other than information about a potentially threatening bodily condition.

There are at least two answers to this challenge. The first one starts by noticing that inflammatory pain, if it is a type of nociceptive pain at all (Woolf, 2010), is a *sui generis* type anyway, in that it involves distinctive proinflammatory mediators (Kidd & Urban, 2001). Accordingly, one way to resist the argument from inflammatory pain would simply be to further restrict my account to acute, somatic, nociceptive, *non-inflammatory* pain. Since the latter is a well-established category—it corresponds to what pain scientists call "normal pain" (Urch, 2007)—this manoeuvre should not be taken as problematic.

The second response has it that, appearances notwithstanding, inflammatory pain *is* turned on by information about potential bodily threats. Suppose you have broken your nose, and your body has responded to this injury with neuroinflammation. This inflammatory process involves the sensitization of nociceptors, which can now be activated by normally innocuous stimuli. The key word here is "normally". Your nose is broken, and this means that what normally does

not constitute a threat can *now* be a threat—any normally innocuous stimulus can, in this situation, make your injury worse or hinder the healing process. Nociceptive activity in inflammatory pain is thus a response to a *current* potential threat.

All things considered, I am inclined to favour the second response over the first one, but I am fine if you prefer to take the other route. Either way, the argument from inflammatory pain does not impugn the claim that pain is activated by information concerning potentially threatening bodily conditions.

3.2 | The input to fear and anxiety

Do the inputs to fear and anxiety reliably co-vary with, hence carry information about, potential threats? Admittedly, fear and anxiety can occur in the absence of potentially threatening stimuli. Certain phobias are obvious examples—people suffering from *koumpounophobia* are afraid of clothing buttons (McRae et al., 2022). We can however put these cases to one side: These are dysfunctional forms of fear/anxiety, and they are dysfunctional exactly because they are activated by the wrong type of input.

What about properly functioning instances of fear and anxiety? Let us begin with fear. It is traditional to distinguish two types of fear-inducing stimuli (Blanchard & Blanchard, 1989): (i) innate fear stimuli and (ii) learned fear stimuli. In humans and other mammals, innate fear stimuli include: (A) predators; (B) aggressive conspecifics; and (C) lack of oxygen (Silva et al., 2016). I suppose I do not need to explain why all these are potential threats.

What about (ii)? Conditioned fear is the most extensively studied type of learned fear (Izquierdo et al., 2016), so I focus on it. In particular, I limit my discussion to classical cued conditioning, but nothing hinges upon this. In classical cued conditioning, a subject is presented with a neutral stimulus (called "conditioned stimulus", CS—e.g., the sound of a bell) paired with a threatening unconditioned stimulus (US—e.g., an electric shock). As a result, the subject forms a link in memory between the CS and the US, and the CS will now bring about a fear response in the subject (better: a conditioned fear response) even in the absence of the US. One might think that fear conditioning is an obvious counterexample to my claim that fear is activated by information about potential threats, in that CSs are non-threatening by design. But this would be obviously wrong: In virtue of being presented with the CS–US pairing, the subject has learned via association that the CS is predictive of the US—that is, they have learned that the CS is an indicator of a potential threat.

Fear is turned on by information about potential threats. The same is true of anxiety, since its prototypical stimuli are again things like predators and aggressive conspecifics (Blanchard et al., 1991; Blanchard, Hynd, et al., 2001). Does this mean that the inputs to fear and anxiety carry exactly the same information? This question mirrors that of Section 2.3, when we asked whether fear and anxiety have the same representational *output*. Yet again, it is unnecessary for me to settle on an answer, but I think that an idea along the lines of Kurth (2018) is likely to work in this case as well: While fear is activated by information concerning *specific* potential threats (say, a big animal nearby you), anxiety tends to be activated by information concerning *uncertain* potential threats (e.g., something lurking behind a bush). For example, when a cat is put into a mouse's cage, this engenders a fear response in the mouse (i.e., the mouse flees or freezes), but when the mouse just gets exposed to the cat's odour, this brings about anxiety-related behaviours like vigilance, exploration, and circumspection (Blanchard & Blanchard, 2008).

3.3 | Putting things together

If we put together what we have done in Sections 2 and 3, we can draw the following conclusion: While pain, fear, and anxiety are different capacities, they exhibit a striking functional similarity—they all take information about potential threats as input and they produce threat representations as output.[10] In other words, pain, fear, and anxiety are all threat-detection capacities. Receiving information about potential threats turns them on, and they then analyse this information to determine whether the threat is actual. If the assessment is positive, a representation of the form *O is a threat* is outputted.[11] The question now becomes, "How many threat-detection mechanisms are at work here?" It is to this question that I now turn.

4 | NEURAL REALISERS

A possibility—call it "the multiple mechanisms hypothesis"—is that pain, fear, and anxiety are each subserved by a distinct threat-detection mechanism. Possible ... but not actual. I instead endorse the *single mechanism hypothesis*, according to which there is *one* threat-detection mechanism common to pain, fear, and anxiety. In the next three sections, I make the case for this latter hypothesis by considering data from different quarters of the cognitive sciences.[12]

I begin in this section by discussing evidence concerning the neural realisers of threat-detection. The single mechanism hypothesis, but not the multiple mechanisms hypothesis, predicts that threat-detection for pain, fear, and anxiety relies upon *shared neural resources*. Is this prediction borne out by the data? I argue it is.

Let us start with fear and anxiety. Evidence from rodents, non-human primates, and humans indicates that the *central extended amygdala* (CEA)—a brain region encompassing the central nucleus of the amygdala (Ce) and the bed nucleus of the stria terminalis (BST)—plays a key role in threat-detection for *both* fear and anxiety (Tovote et al., 2015). This, however, only gets me so far, since it might be the case that different parts of the CEA have different functions vis-à-vis fear and anxiety. This is what has been proposed by Kurth (2018), who maintains that fear-related threat-detection is underpinned by the Ce, while anxiety-related threat-detection depends on the BST. Kurth argues for this claim as follows.[13] Fear is in the business of detecting specific threats, while the function of anxiety is to detect uncertain threats, and:

[10]We should not conceive of this input–output domain as a natural category "out there in the world". Rather, the threat domain is a *content domain*, that is, the domain pain, fear, and anxiety range over. As such, it "isn't delineated by mind-independent facts ... [but it is] simply a reflection of ... the cognitive mechanisms that are directed at [it]" (Margolis & Laurence, 2023, p. 3). In this respect, the best one can do to pinpoint the domain in question is to proceed via paradigm cases.

[11]An alternative way to think of the relation between threats and pain/fear/anxiety experiences would be to say that the content of these experiences reduces to the representation of a certain object, and that the property *being a threat* is manifested in the *attitude* one has towards this object (Deonna & Teroni, 2008). While I favour the *content view* over the *attitude view*, I am happy for somebody with different theoretical preferences to repack my proposal in an attitudinalist fashion.

[12]Elman and Borsook (2018) maintain that there is a common threat-detection mechanism underlying pain and fear, but a different one underlying anxiety. My arguments for the claim that there is a *single* threat-detection mechanism underlying pain, fear, and anxiety show that Elman and Borsook's *two mechanisms hypothesis* is also wanting.

[13]Kurth also considers lesion and neuropharmacological studies. I discuss them in Section 5.

(i) fMRI scans show that uncertain threats activate the BST (but not the Ce), while specific threats generate a response in the Ce (but not in the BST) (Alvarez et al., 2011; Somerville et al., 2013);
(ii) optogenetic studies indicate the existence of two distinct neural circuits in the CEA: A circuit in the Ce that responds to specific threats (but not to uncertain ones), and one in the BST that responds to uncertain threats (but not to specific ones) (Jennings et al., 2013; Kim et al., 2013).

If Kurth is right, the single mechanism hypothesis is in trouble. However, more recent evidence tells against (i) and (ii). Not only do fMRI scans in humans indicate that the Ce and the BST have a similar functional profile—crucially, they *both* respond to *both* specific and uncertain threats (Andreatta et al., 2015; Klumpers et al., 2015; Williams et al., 2015)—but many different experimental protocols also reveal that the Ce and the BST form a closely integrated circuit (Fox et al., 2015). Shackman and Fox summarise these lines of research as follows:

> A wide variety of evidence demonstrates that the CEA plays a crucial role in evaluating and responding to a range of threat-related cues and contexts. ... The Ce and BST have both proven sensitive to uncertain or temporally remote threats; ... both show ... responses to acute threat cues; and both show heightened activity during sustained exposure to novel or diffusely threatening contexts. In light of this evidence, the claim that the extended amygdala is strictly segregated into fear- and anxiety-related subdivisions is no longer tenable (Shackman & Fox, 2016, p. 8058).

The single mechanism hypothesis does not just predict that threat-detection for fear and anxiety relies upon the same neural resources. It also predicts that these resources are deployed for pain-related threat-detection. Is that the case? I think so. Simplifying quite a bit, the neural architecture of pain's ascending pathway has the following organisation (Mercer Lindsay et al., 2021; Veinante et al., 2013). The dorsal horn of the spinal cord gathers information from the periphery of the body and sends nociceptive information to the parabrachial nucleus (PB) and the thalamus. From these two regions, nociceptive information is sent to the somatosensory and the insular cortices, where a representation of the sensory features of pain (e.g., its location) obtains. These sensory representations are then relayed to the CEA, which also directly receives nociceptive signals from the PB. By receiving these sensory and nociceptive signals, and in virtue of also receiving information from cognitive and emotional centres, the CEA is ideally positioned to assess whether one's current bodily condition constitutes a threat.

To sum up, neuroscientific evidence appears to confirm a key prediction made by the single mechanism hypothesis, namely, that threat-detection for pain, fear, and anxiety is underpinned by the same neural resources.

5 | LACK OF DISSOCIATIONS

A key difference between the single mechanism and the multiple mechanisms hypotheses has to do with the patterns of threat-detection impairments that they predict. If the multiple mechanisms hypothesis is true, we should expect that the threat-detection processes for pain, fear, and anxiety dissociate (e.g., we should find cases in which pain-related threat-detection is impaired

while fear-related threat-detection is intact). In contrast, the single mechanism hypothesis predicts the existence of *associated impairments*—cases in which the threat-detection processes for pain, fear, and anxiety jointly (and selectively) collapse.

In this and the next section, I argue that the single mechanism hypothesis has the empirical upper hand, since there are no convincing instances of threat-detection dissociations (Section 5), while there are cases of joint impairments (Section 6).

5.1 | Fear versus anxiety

Let us start with fear and anxiety one more time. The strongest case for the dissociability of threat-detection for these two capacities comes again from Kurth (2018), who discusses two lines of evidence. The first one is Caroline and Robert Blanchard's work on rodents' defensive behaviours (Blanchard et al., 1993, 1997; Blanchard, Griebel, & Blanchard, 2001). The Blanchards operationalised *fear-related behaviours* in terms of rodents' responses to an approaching predator (i.e., fight, flight, freeze, and defensive vocalisation), while they operationalised *anxiety-related behaviours* as the responses to stimuli (e.g., a cat's odour) that signal the possible presence of a predator (i.e., avoidance, behavioural inhibition, and various types of risk assessment). On this basis, they investigated the effects of different compounds on these classes of behaviour. Kurth (2018, p. 39) summarises the results of this research as follows: "Work on rodents suggests that anxiety and fear are doubly dissociable. ... We see that when rats and mice are given panicolytics, the fear-related responses are blocked, though the anxiety-related behaviour remains unaffected. We also see the converse when anxiolytics are administered".

I have two issues with this way of presenting the results. First, the distinction between anti-anxiety and anti-panic drugs is controversial, since some of the drugs that the Blanchards label as anxiolytic—for example, a benzodiazepine like diazepam—are also used to treat panic disorders (Stein et al., 2010), while some of the drugs that they label as panicolytics are also used to treat generalised anxiety disorders (e.g., alprazolam, which is in fact another benzodiazepine [Ait-Daoud et al., 2018]). Second, and more importantly, it is not true that some drugs influence rodents' fear-related behaviours but not their anxiety-related behaviours, while others have the opposite effect. "Anxiolytic" drugs in fact modify some fear-related behaviours (e.g., chlordiazepoxide reduces defensive vocalisation and/or defensive attack in rats and mice—Blanchard et al., 1993; Griebel, Blanchard, Jung, & Blanchard, 1995), while "panicolytic" drugs affect some anxiety-related behaviours (e.g., odour risk assessment and behavioural inhibition in rats—Blanchard et al., 1993). It is also not the case that "panicolytic" drugs affect all rodents' fear-related behaviours at once (e.g., imipramine alters the fight-or-flight response, but not freezing [Griebel, Blanchard, Agnes, & Blanchard, 1995]), and the same applies, mutatis mutandis, to "anxiolytics" (Griebel, Blanchard, Jung, & Blanchard, 1995). Therefore, this evidence fails to support the existence of dissociable threat-detection mechanisms for fear and anxiety.

The second line of evidence considered by Kurth concerns the potentiation of the startle response. This is the reflex response to a sudden stimulus (e.g., a loud noise), and it can be enhanced in different ways. *Fear-potentiation startle* works as follows: A subject goes through a conditioning phase where a neutral stimulus (the conditioned stimulus, CS—say, a tone) is paired with a threatening one (say, a footshock); the subject is then presented with both a startle-inducing stimulus (SIS) and the CS. In both humans and non-human animals, the joint presentation of the SIS and the CS results in a greater startle amplitude compared to the

presentation of the SIS alone (Davis et al., 1993). *Light-enhanced startle* instead relies on the fact that rodents are nocturnal animals and find brightly lit environments anxiogenic. When put in this type of environment, they exhibit greater startle responses to a SIS (Groenink et al., 2008).

And now back to the alleged dissociability of threat-detection for fear and anxiety. Studies on rodents have shown that lesions of/pharmacological interventions (hereafter "interventions") to the central amygdala (Ce) block fear-potentiated startle but have no effect on light-enhanced startle (Campeau & Davis, 1995; Hitchcock & Davis, 1987; Walker & Davis, 1997). In contrast, the same interventions to the bed nucleus of the stria terminalis (BST) block light-enhanced startle but leave fear-potentiation startle intact (Gewirtz et al., 1998; Walker & Davis, 1997). Kurth interprets these findings as indicating that interventions to the Ce affect fear but not anxiety processing, while interventions to the BST have the opposite effect. Based on this, he concludes that "anxiety and fear ... engage distinct—and dissociable— ... pathways" (Kurth, 2018, p. 40).

Kurth's interpretation of the startle response experiments is not implausible, but there is a better one. Fear-potentiation startle involves a conditioning process, while light-enhanced startle exploits rodents' natural tendency to respond to bright lights with anxiety. It is therefore possible to interpret the findings discussed by Kurth as follows: Interventions to the Ce selectively affect *conditioned* responses to threats, while interventions to the BST selectively affect *unconditioned* responses to threats. But why should we favour the "conditioned versus unconditioned" interpretation over the "fear versus anxiety" one? We should do so because only the former can account for these other data: (A) lesions to the Ce disrupt fear-related responses other than the fear-potentiation startle, but only if these responses are *conditioned* (Gentile et al., 1986; Iwata et al., 1986); (B) lesions to the BST reduce startle responses to *specific* stressors—these responses therefore do not count as anxiety-related by Kurth's own lights—but only if such stressors are *unconditioned* (Gewirtz et al., 1998). I conclude that the data on the potentiation of the startle response do not provide support for the dissociability of the threat-detection processes for fear and anxiety.

5.2 | Pain versus fear/anxiety

Do pain, on the one hand, and fear/anxiety, on the other, dissociate? One might want to answer this question by considering whether people suffering from congenital insensitivity to pain (CIP) can feel fear and anxiety. To the best of my knowledge, however, no systematic investigation has been conducted on this issue. But there is no need to hold our breath. CIP results from a problem with nociception (e.g., non-functional nociceptors), which leads to the brain not receiving nociceptive information (Nahorski et al., 2014). Accordingly, even if it turned out that subjects with CIP could feel fear/anxiety, this dissociation would not constitute evidence for the hypothesis that there are different threat-detection mechanisms in the brain.

A comparison should get the point across. Suppose you maintain that there are different mechanisms in the brain dedicated to visual and auditory processing, respectively. To convince me that you are right, it would not do to merely tell me about a person who has damaged eyes but intact ears and therefore can hear but cannot see. This would not be a relevant piece of evidence because it simply points to the existence of independent input channels, without telling me anything about the organisation of perceptual processing in the brain. By the same token,

even if it were the case that subjects suffering from CIP can feel fear/anxiety, this would not lend further credence to the multiple mechanisms hypothesis.[14]

A prima facie more relevant case is that of patient SM. Following bilateral damage to the amygdala, SM does not undergo fear or anxiety when presented with threatening stimuli such as dangerous animals or visiting a "haunted house" (Feinstein et al., 2011). Moreover, this fear/anxiety impairment appears to be selective, in that SM experiences basic emotions other than fear/anxiety. However, SM does not seem to have a pain deficit, and one could therefore be tempted to conclude that she exhibits the type of pain versus fear/anxiety dissociation predicted by the multiple mechanisms hypothesis. This temptation should be resisted though, since it has subsequently been discovered that SM *can* feel fear and anxiety! In particular, SM responds with fear/anxiety to suffocation: After inhaling a high concentration of CO_2, SM reported high levels of fear and anxiety, and experienced a panic attack (Feinstein et al., 2013). Yet again, evidence for the dissociability of threat-detection for pain versus fear/anxiety is wanting.

5.3 | Taking stock

The multiple mechanisms hypothesis predicts the existence of cases in which the threat-detection processes for pain, fear, and anxiety dissociate. In this section, I argue that no such dissociations have been found. What about cases of associated impairments, that is, cases in which the threat-detection processes for pain, fear, and anxiety jointly (and selectively) collapse? If the single mechanism hypothesis is true, we should expect to see cases like that. In the next section, I discuss two cases of such associated impairments.

6 | ASSOCIATED IMPAIRMENTS

So-called pain asymbolia is a rare condition that has attracted significant philosophical attention. A major point of contention is whether pain asymbolia involves a specific pain deficit (Grahek, 2011) or a more general incapacity for bodily care (Bain, 2014; Klein, 2015a). I am dissatisfied with both of these hypotheses.

Asymbolics do exhibit a surprising pain deficit (Berthier et al., 1988; Schilder & Stengel, 1931): Upon receiving noxious stimuli, they report experiencing pain and they also have autonomic responses (e.g., increased heart rate), but they neither withdraw from these stimuli (they in fact sometimes approach them) nor do they wince or grimace—and when asked, they say they are not bothered by these stimuli, as corroborated by their calm demeanour during pain assessment. *Pace* Grahek, however, their deficit is not limited to pain: Asymbolics also fail to respond with fear or anxiety to threats such as being threatened with a knife, being verbally menaced, or almost being run over by a lorry (Berthier et al., 1988; Schilder & Stengel, 1931). The "specific pain deficit" hypothesis casts its net too narrowly.

The idea that asymbolia involves a bodily care impairment faces the opposite problem—it overgeneralises. There are many ways to care about one's body: Avoiding physical threats, avoiding contaminated objects, eating and drinking, carrying out bodily functions, and so on.

[14]The single mechanism hypothesis does not make any claim about the architecture of the input channels that *directly* feed into the threat-detection mechanism either. The existence of a common threat-detection mechanism underlying pain, fear, and anxiety is consistent with both a unique and multiple such input channels.

But asymbolics eat, drink, go to the toilet, and steer clear of filthy objects. Both Klein and Bain are aware of this issue. Klein (2015a) tackles it by drawing a distinction between temporally immediate and temporally distant threats, and by proposing that asymbolics' deficit is limited to the former. Bain (2014, p. 317) instead distinguishes different kinds of bodily care and suggests that asymbolics do not "care that one's body not be damaged (call this d-care), ... [while they] care that [their bodily] needs are met (n-care)". My reply is simple: Both proposals are extensionally inadequate. In the case of Klein's, the problem is that disgust responses as well as very intense thirst, hunger, or the urge to urinate demand "action now or in the very near future" (Klein, 2015a, p. 499), while some forms of pain (e.g., a mild backpain) and of fear/anxiety (e.g., being afraid of/anxious about flying *next week*) can be dealt with "in one's own time" (Klein, 2015a, p. 499). The issue with Bain's is that, as we saw in Section 2.1, not all pain experiences are responses to bodily damage (and the same applies to fear and anxiety experiences). An explanation of asymbolia in terms of a "d-care" deficit therefore lacks sufficient explanatory breadth. The pendulum has swung from overgeneralisation back to undergeneralisation.[15]

By contrast, the single mechanism hypothesis has the right explanatory size: If there is a mechanism in charge of performing threat-detection for pain, fear, and anxiety, we should expect that when the mechanism breaks down, one should not be able to respond to threats with pain, fear, or anxiety, while one's other capacities should remain intact. Asymbolia is thus best accounted for in terms of a breakdown of the threat-detection mechanism (the condition should therefore be relabelled "threat asymbolia"). Here is another way to see the issue. Human beings face several adaptive problems. For example, we need to eat, avoid pathogens, and respond to what I call "threats". We likely possess a host of capacities to address these challenges: disgust deals with pathogens (Curtis & de Barra, 2018); hunger motivates us to eat (Burnett et al., 2016); and—if I am right—pain, fear, and anxiety detect threats by engaging a common threat-detection mechanism. This explains why we respond to the sight of rotten food with disgust rather than with fear: This is a pathogen-ridden stimulus, and it therefore activates the disgust system rather than the threat-detection mechanism. It also explains why asymbolics eat and steer clear of filthy objects: They have intact hunger and disgust; it is only their threat-detection mechanism that is impaired.

This explanation also comports with what we know about the neural basis of this condition. Asymbolia is due to injuries to the insular cortex, which result in a disconnection between that brain area and the amygdala (Berthier et al., 1988). As we have seen in Section 4, the somatosensory and the insular cortices are responsible for elaborating the sensory aspect of pain, and they then send this information to the central extended amygdala (CEA). If the CEA is, as I have suggested, the neural realiser of the threat-detection mechanism, an insula-amygdala disconnection should produce an atypical pain experience with the usual sensory component of pain, but with no threat representation. And this would explain why asymbolics report being in

[15]There are further issues with the "lack of bodily care" view. Klein suggests that asymbolia should "resemble a kind of depersonalisation syndrome" (Klein, 2015a, p. 510), in that in both cases, "one's body becomes, as it were, just another object in the world" (Klein, 2015a, p. 510). He then argues that schizophrenia, which often involves depersonalisation, supports the "lack of bodily care" view, since schizophrenics exhibit a form of pain abnormality akin to that of asymbolics. I am not convinced. Schizophrenics have a different pain profile than asymbolics—they have increased sensitivity to acute pain and reduced sensitivity to prolonged pain (Lévesque et al., 2012). Also, they experience very intense fear and anxiety (Temmingh & Stein, 2015). Depersonalisation does not therefore seem to be the root cause of asymbolia. This point is further supported by de Vignemont (2015), who notices that subjects suffering from somatoparaphrenia deny ownership of some of their bodily parts—hence they represent these bodily parts as "just another object in the world"—but nonetheless feel pain in these "alien" parts.

pain but do not respond to it—why should they, if their pain experience does not carry the information that their body is under threat? By the same token, if asymbolics' fear and anxiety processing does not engage the CEA, it should not come as a surprise that their responses to threats are, to use the famous words of William James, "pale, colourless, [and] destitute of emotional warmth" (James, 1884, p. 190).

I want to conclude this section by briefly discussing the case of Joanne Cameron, another subject exhibiting a joint impairment in pain, fear, and anxiety. Cameron had been living a good life: She had a job she liked, a happy marriage, two grown-up children, and lots of positive emotions. It is negative experiences that do not come naturally to her (Habib et al., 2019; Levy, 2020). This is how she describes giving birth to her son, "My friends would come up to me and say ... 'If you're in pain, take everything they give you'. I went in thinking, 'As soon as it gets painful, I'll ask for the drugs'. But it was over before I knew it" (Levy, 2020). However, Cameron's pain insensitivity is peculiar. As we have seen in Section 5.2, "regular" pain insensitivity is caused by a deficit in nociception. But Cameron does not have any nociceptive deficit (Habib et al., 2019). More importantly, she cannot feel fear and anxiety either:

> I said to [Cameron], "Are you worried about what's going to happen today?" Because she was meeting our clinicians to have a skin biopsy and do quantitative sensory testing—pain-threshold tests. She said, "No. In fact, I'm never worried about anything" (Levy, 2020).

In the absence of the single mechanism hypothesis, Cameron's condition looks both unexpected and puzzling.[16] Why should somebody have a deficit like that? But a deficit *like that* is exactly what we should expect to see if there is a common threat-detection mechanism for pain, fear, and anxiety, and this mechanism is impaired. The capacity to both predict and explain Cameron's condition lends empirical support to the single mechanism hypothesis.

7 | CONCLUSION

In this article, I argued for two claims. First, pain, fear, and anxiety are threat-detection capacities: They are activated by information about potential threats; they then analyse this information and, if they take the threat to be actual, they output a threat representation. Second, I defended the hypothesis that these three capacities are underpinned by a common threat-detection mechanism.

Since threats pose a major adaptive problem, one might ask whether the mind/brain contains *other* threat-detection mechanisms. Legrain et al. (2011) answer this question in the affirmative and hypothesise the existence of a mechanism realised in the somatosensory and insular

[16]At least, it is puzzling at the *psychological level* of explanation. Some progress has been made with respect to the genetic and molecular bases of Cameron's condition (Habib et al., 2019; Mikaeili et al., 2023). Anandamide (ANA) is an endocannabinoid that plays an important role in pain, fear, and anxiety reduction. The activity of ANA is partly controlled by the FAAH enzyme, which is responsible for breaking down ANA once the latter has carried out its function. This means that the inhibition of the FAAH enzyme results in an abnormal presence of ANA, which in turn determines an excessive reduction in pain, fear, and anxiety. It turns out that Cameron has two FAAH-related genetic mutations, which resulted in the reduced expression of the FAAH enzyme, leading to Cameron's incapacity to feel pain, fear, and anxiety. The account I have proposed is not in alternative to this one—it is simply pitched at another level of explanation.

cortices, with the function of "processing sensory information that is the most susceptible to signal potential danger *in the proximal space*" (Legrain et al., 2011, p. 120, emphasis added). While my proposal is consistent with the existence of an additional *peripersonal threat-detection mechanism*, I think that the evidence for such a mechanism is lacking. The neural circuitry Legrain and colleagues are pointing to is nothing but the well-known Salience Network (SN), a mechanism whose function is to detect stimuli that "stand out" from others (Uddin, 2015)—for example, the SN is activated by seeing a red figure after having seen a series of blue figures (Downar et al., 2000). Legrain and collaborators initially got this right, when they presented their account in terms of *salience detection* (Legrain et al., 2011, pp. 118–19). Their mistake was to then couch "salience detection" as the detection of "events that are significant for the integrity of the body" (Legrain et al., 2011, p. 119).

One might respond that the SN is not a *mere* salience detection mechanism. It is *also* a peripersonal threat-detection mechanism, in that it is sensitive to peripersonal threatening stimuli as well. I do not find this response persuasive. To begin with, there is experimental evidence in favour of the claim that threatening stimuli do not activate the SN in virtue of being threatening, but simply in virtue of their physical salience (Baker et al., 2021). Moreover, even granting that in addition to physical salience, the SN is also sensitive to emotional salience (i.e., it is sensitive to threats *qua threats*), the case remains that it is not the SN that assesses whether a stimulus is a threat. Rather, as shown by Ince et al. (2023), it is the amygdala that performs this threat-detection analysis, and then "directly contributes to salience processing in the SN by enhancing sensory presentations of negative emotional events" (Ince et al., 2023, p. 119664).

But the amygdala (better: the central extended amygdala) is, I proposed, the neural realiser of the threat-detection mechanism underlying pain, fear, and anxiety. The following architecture thus emerges: First, the threat-detection mechanism underlying pain, fear, and anxiety analyses whether a stimulus is a threat; the threat representations outputted by this mechanism are then sent to, among other places, the SN; finally, the SN uses these threat representations to allocate attentional resources. But this means that the SN is not an additional threat-detection mechanism: The threat-detection job is entirely performed by the mechanism underlying pain, fear, and anxiety. That is the mechanism that is looking for trouble.

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DATA AVAILABILITY STATEMENT

There is no data available.

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