



The sensorimotor theory of pathological pain revisited

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ABSTRACT

Harris (1999) proposed that pain can arise in the absence of tissue damage because changes in the cortical representation of the painful body part lead to incongruities between motor intention and sensory feedback. This idea, subsequently termed the sensorimotor theory of pain, has formed the basis for novel treatments for pathological pain. Here we review the evidence that people with pathological pain have changes to processes contributing to sensorimotor function: motor function, sensory feedback, cognitive representations of the body and its surrounding space, multisensory processing, and sensorimotor integration. Changes to sensorimotor processing are most evident in the form of motor deficits, sensory changes, and body representations distortions, and for Complex Regional Pain Syndrome (CRPS), fibromyalgia, and low back pain. Many sensorimotor changes are related to cortical processing, pain, and other clinical characteristics. However, there is very limited evidence that changes in sensorimotor processing actually lead to pain. We therefore propose that the theory is more appropriate for understanding why pain persists rather than how it arises.

1. Introduction

When we move, our brain generates a prediction of the sensory outcome of the movement based on the motor command (Blakemore et al., 2000; Wolpert and Flanagan, 2001). The mismatch, or lack thereof, between the predicted and actual sensory feedback is an important signal to the central nervous system, allowing us to adjust our movements online, and determine whether our motion is self-generated or caused by outside forces (Fig. 1). Problems with the ability to represent intended movements, generate sensory predictions, or compare predicted and actual feedback are thought to contribute to a range of neurological and psychological abnormalities including auditory hallucinations and anosognosia (Frith et al., 2000).

Harris (1999) proposed that incongruence between motor intention and sensory feedback (specifically, tactile and proprioceptive) could underlie conditions characterised by pain in the absence of tissue pathology, referred to as the sensorimotor theory of pain (McCabe and Blake, 2007). Harris (1999) suggested that rather than being resolved,

sensorimotor incongruence can persist and give rise to the affective experience of pain, associated anomalous sensations, and physical symptoms such as dystonia. According to the sensorimotor theory of pain, a comparator process detects incongruence between motor intention and sensory feedback. Cortical reorganisation was the proposed reason for sensorimotor incongruence, specifically in the areas of primary somatosensory (S1) and motor (M1) cortices representing the painful body part, reviewed extensively elsewhere (Chang et al., 2018; Dahlberg et al., 2018; Di Pietro et al., 2013a, 2013b; Furuya and Hanakawa, 2016; Goossens et al., 2018; Kuner and Flor, 2017; Parker et al., 2016; Tanasescu et al., 2016; Upadhyay et al., 2018). The theory has been used to explain conditions such as focal hand (“violinists”) dystonia, repetitive strain injury, low back pain, phantom limb pain, and Complex Regional Pain Syndrome (CRPS; McCabe and Blake, 2007); and has shaped the development of several therapies. We review evidence for the sensorimotor theory of pain: specifically whether there are changes in sensorimotor processing in pathological pain and the extent to which these have been related to altered cortical processing and

Abbreviations: CRPS, Complex Regional Pain Syndrome; ICD-11, ICD-11.

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clinical characteristics including pain. We include evidence from all pathological pain conditions, which we have organised by ICD-11 categories (Nicholas et al., 2019; Scholz et al., 2019), or for categories that contain only a single condition we refer directly to the condition (e.g. fibromyalgia instead of widespread pain).

We have organised our review according to the possible causes of sensorimotor incongruence. Each of these possible causes generates expectations about neurological and behavioural phenomena in people experiencing pain (Table 1). Sensorimotor incongruence could arise due to problems with movements and/or sensations, leading to differences between the expected and actual sensory consequences of action. We therefore begin by evaluating evidence for motor (2.1) and sensory (2.2) changes in pathological pain. Sensorimotor incongruence could also arise due to changes in the cognitive representations of the body (2.3) and peripersonal space (i.e. the space immediately surrounding the body; 2.4), since these could lead to incorrect motor intentions and poor localisation of sensory input (e.g. touch). Furthermore, sensorimotor conflict is typically resolved by giving greater priority to the more reliable signal, and/or through adaptation (Kording and Wolpert, 2004). For there to be sustained sensorimotor conflict (leading to pain), people with pathological pain would need to have problems with these resolving processes. Thus, we consider evidence for problems with multisensory processing (2.5) and sensorimotor integration (2.6). Finally, we scrutinise the proposed causal relationship between sensorimotor conflict and pain by evaluating evidence that sensorimotor conflict leads to pain and that treatments targeting sensorimotor processing provide pain relief (2.7).

2. Review of evidence for the sensorimotor theory of pain

2.1. Motor functions

There are several processes that could interfere with motor functioning in pathological pain. In some cases, there could be functional changes to the painful body part (e.g. due to musculoskeletal damage) that would cause movement problems even if pain was completely relieved. Changes to movement might also occur if the person adapts or minimises their movements to prevent pain, even in the absence of tissue damage (Hasenbring et al., 2020; Hodges and Tucker, 2011; Lund et al., 1991). Over time, such adaptation could lead to changes in muscle morphology and strength, which can themselves lead to (more) pain (De

Pauw et al., 2016; Lee et al., 1999). Both biomechanical changes and muscle fatigue caused by disuse-related muscle atrophy could require adaptation of the sensorimotor system (Wolpert et al., 2011). According to Harris (1999), pathological pain could arise as a result of this adaptation. For instance, (painful) peripheral problems, such as spasm, could arise due to altered sensorimotor processing (Abbruzzese and Berardelli, 2003).

We review the evidence suggesting that people with pathological pain have difficulties with movement (2.1.1), and that they might imagine movements differently to people without pain (2.1.2). The latter would add to the evidence that motor representations are compromised (for review see Ladda et al., 2021), supporting Harris' proposed cortical origin of pain. If motor deficits contribute to sensorimotor incongruence, then motor deficits in people with pathological pain should relate to altered cortical processing (2.1.3), and pain (2.1.4).

2.1.1. Evidence for motor deficits in people with pathological pain

Problems with movement are highly prevalent in people with pathological pain. Up to 75% of people with chronic pain report that pain negatively impacts their ability to exercise, lift objects, or walk (Breivik et al., 2006). Experimental research has revealed evidence for motor deficits in people with CRPS, fibromyalgia, neuropathic pain, and musculoskeletal pain that would not necessarily manifest in a standard diagnostic assessment. For instance, people with carpal tunnel syndrome can have greater variability in precision pinch and reach-to-pinch movements (Gehrmann et al., 2008). People with CRPS have poorer reach-to-grasp movements (Maihöfner et al., 2007; Osumi et al., 2017b), slower finger tapping (Reid et al., 2017; Schilder et al., 2012), and slower initiation (Halicka et al., 2020a) and execution of movements (Vittersø et al., 2021a), although motor difficulties are not always found (Christophe et al., 2016a). Both gross and fine motor control can be disrupted in fibromyalgia (Pérez-de-Heredia-Torres et al., 2013), including impaired manual dexterity (Canny et al., 2009; Pérez-de-Heredia-Torres et al., 2013), and slower finger tapping (Gentile et al., 2020), although motor deficits are not always detectable at a group level (Rasouli et al., 2017). People with hand osteoarthritis can have impaired grip-force matching accuracy (Magni et al., 2021). People with low back pain often make slower lumbar movements (for reviews see Laird et al., 2014; Wernli et al., 2020). People with neck pain can have reduced range of movement, movement speed, and head positioning accuracy (for review see Hesby et al., 2019).

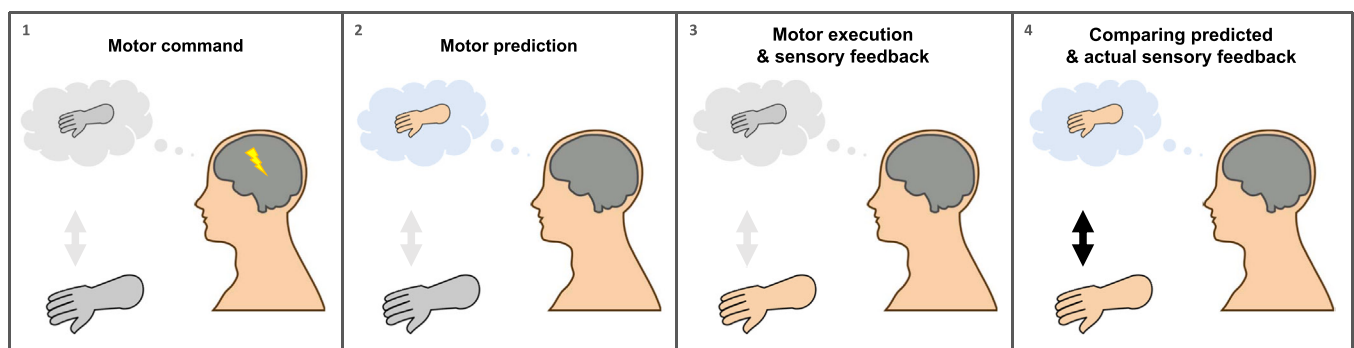


Fig. 1. Sensorimotor processing. A motor command is generated in the motor cortex (Panel 1) and transmitted to the neurons innervating the relevant muscles, whose discharge results in muscle contractions (for reviews, see Schwartz, 2016; Shadmehr et al., 2010; Stifani, 2014). A prediction/internal forward model of the sensory and motor consequences of the is generated (Panel 2), which compensates for delayed and noisy feedback (Shadmehr et al., 2010). Once executed (Panel 3), limb position and movement are monitored through sensory feedback (Shadmehr et al., 2010). This feedback, along with spinal and cortical reflexes (Kurtzer et al., 2008; Soechting and Lacquaniti, 1988), is used to correct for movement errors (Shadmehr et al., 2010). These information sources are integrated (Panel 4) and weighted by their reliability (Kording and Wolpert, 2004). Cognitive representations of the body's size and shape, as well as nearby space, are important for planning and executing movements (Shadmehr et al., 2010), locating sensations, and generating accurate motor intentions (Longo et al., 2010; Medina and Coslett, 2010; Serino and Haggard, 2010; Tamè et al., 2019). If any of the processes involved in sensorimotor functioning are compromised, they may present conflicting information to the nervous system. Such a conflict could be considered a sensorimotor incongruence, theorised to cause the affective experience of pain (Harris, 1999). However, under normal circumstances the nervous system deals with conflicting information by giving priority to the most reliable signals, which allows the sensorimotor system to learn and adapt (Wolpert et al., 2011).

Table 1

Topic organisation for considering the evidence for the sensorimotor theory of pain. Sensorimotor incongruence could arise due to problems with motor or sensory functions, body or spatial representations, multisensory processing, and/or multisensory integration. For each of these functions, we consider evidence for deficits in people with pathological pain, and that these deficits relate to altered cortical processing, and pain and other clinical characteristics. Finally, for sensorimotor incongruence to lead to pain, there should be evidence for causal links between these phenomena.

Section	Description
Motor functions	
2.1.1	Evidence for motor deficits in people with pathological pain
2.1.2	Evidence for difficulties with imagined movements in people with pathological pain
2.1.3	Evidence that motor (imagery) deficits relate to altered cortical processing
2.1.4	Evidence that motor (imagery) deficits relate to pain and other clinical characteristics
Sensory functions	
2.2.1	Evidence for sensory changes in people with pathological pain
2.2.2	Evidence that sensory changes relate to altered cortical processing
2.2.3	Evidence that sensory changes relate to pain and other clinical characteristics
Body representations	
2.3.1	Evidence for distorted body representations in people with pathological pain
2.3.2	Evidence for difficulties with updating body representations in people with pathological pain
2.3.3	Evidence that distorted body representations relate to altered cortical processing
2.3.4	Evidence that distorted body representations relate to pain and other clinical characteristics
Peripersonal spatial representations	
2.4.1	Evidence for distorted peripersonal space representations in people with pathological pain
2.4.2	Evidence for difficulties with updating peripersonal space representations in people with pathological pain
2.4.3	Evidence that distorted peripersonal space representations relate to altered cortical processing
2.4.4	Evidence that distorted peripersonal space representations relate to pain and other clinical characteristics
Multisensory processing	
2.5.1	Evidence for altered multisensory processing in people with pathological pain
2.5.2	Evidence that altered multisensory processing relates to altered cortical processing
2.5.3	Evidence that altered multisensory processing relates to pain and other clinical characteristics
Sensorimotor integration	
2.6.1	Evidence for altered sensorimotor integration in people with pathological pain
2.6.2	Evidence that altered sensorimotor integration relates to altered cortical processing
2.6.3	Evidence that sensorimotor integration relates to pain and other clinical characteristics
Causal links between sensorimotor incongruence and pain	
2.7.1	Evidence that incongruent sensory and motor information cause pain
2.7.2	Evidence that targeting sensorimotor processing reduces pain

2.1.2. Evidence for difficulties with imagined movements in people with pathological pain

Motor imagery is the process of imagining the execution of a movement, and involves making a prediction about the sensory outcome of the imagined movement (Kilteni et al., 2018). Changes in motor imagery provide clearer evidence that motor representations are altered (Jeannerod and Decety, 1995). Motor imagery can be altered in musculoskeletal pain, and neuropathic pain conditions, such as chronic low back pain (La Touche et al., 2019), spinal cord injury (Scandola et al., 2017), and phantom limb pain (Kikkert et al., 2017). These findings are consistent with the sensorimotor theory of pain in that they suggest that changes in the central representation of movements are related to pain in some conditions (see 3.1.4). Another potential source of sensorimotor incongruence is problems with accurately predicting the outcome of a movement, although this question has yet to be addressed

in the pain literature.

2.1.3. Evidence that motor (imagery) deficits relate to altered cortical processing

As the sensorimotor theory of pain proposes a cortical origin of pathological pain, motor deficits should be related to altered cortical processing if they are indeed contributing to sensorimotor incongruence and pain. Consistent with this idea, there is evidence of motor deficits that can be more directly attributed to central reorganisation. Motor performance in CRPS has been characterised as including bradykinesia (slowed movement execution) and hypokinesia (slowed movement initiation) of the affected limb - both movement disorders of central dysfunction (Galer et al., 1995). Many of the motor deficits in dystonia relate to altered sensorimotor processing, such as sensorimotor integration, oculomotor and head control, and limb control (for reviews see Avanzino et al., 2015; Conte et al., 2019; Desrochers et al., 2019). Furthermore, altered cortical activation during motor tasks has been found for people with CRPS (Azqueta-Gavaldon et al., 2020; Maihöfner et al., 2007), dystonia (for review see Conte et al., 2020), fibromyalgia (Eken et al., 2018; Gentile et al., 2020), knee osteoarthritis (Shanahan et al., 2015), phantom limb pain (for reviews see Makin and Flor, 2020; Scaliti et al., 2020), and shoulder instability (for review see Livett et al., 2021). The reorganisation of S1 representations, however, was not found to relate to motor performance of the CRPS-affected limb (Pfannmöller et al., 2019). In summary, across several pathological pain conditions, motor deficits can be associated with changes across M1, S1, the cerebellum, and the posterior parietal cortex: areas related to motor control, sensory processing, and sensory-motor integration.

Neuroimaging studies of motor imagery have found different patterns of activation in people with CRPS, dystonia, musculoskeletal pain, and neuropathic pain conditions relative to pain-free controls. Altered cortical activity during motor imagery has been observed for people with CRPS and dystonia (Gieteling et al., 2008), chronic low back pain (Vrana et al., 2015), and phantom limb pain (for review see Andoh et al., 2018). The patterns of altered activity vary somewhat between studies, which could be due to the different types of movements that participants were asked to imagine. Nonetheless, altered activity of somatosensory and motor areas is compatible with the idea of altered motor representations. Considered alongside the evidence from imaging studies of actual motor performance, these findings demonstrate that motor impairments and motor imagery in pathological pain conditions are associated with altered cortical activity, consistent with the sensorimotor theory of pain (Harris, 1999).

2.1.4. Evidence that motor (imagery) deficits relate to pain and other clinical characteristics

From people with musculoskeletal pain, and neuropathic pain there is evidence for a relationship between motor deficits and pain, and that motor deficits can precede pain. For people with carpal tunnel syndrome, pain severity is associated with motor impairment (Fernández-Muñoz et al., 2016). Altered motor performance is associated with an increased risk of low back pain (for reviews, see Meier et al., 2019; van Dieën et al., 2017) and is predictive of developing back pain (for review see Sadler et al., 2017), although the magnitude of this association is small (for meta-analysis see Christe et al., 2021). For people with phantom limb pain, pain intensity is correlated with slower phantom finger tapping, but not motor imagery (Kikkert et al., 2017). Furthermore, people who are able to move their phantom limb have a lower likelihood of experiencing phantom limb pain (Münger et al., 2020).

However, the relationship between motor impairment and pain is not ubiquitous. For instance, researchers have found no evidence that pain was related to motor deficits in CRPS (Schilder et al., 2012), or in fibromyalgia (Pérez-de-Heredia-Torres et al., 2013). Furthermore, motor impairments were not affected by the presence or absence of pain for people with recurrent low back pain (Shih et al., 2021). The latter findings could suggest that motor deficits are unrelated to pain, or that

they precede pain. Only the latter is consistent with the sensorimotor theory of pain (Harris, 1999). Studies investigating the causal link between pain and motor performance found that motor performance of people with CRPS improved following pain relief from a ketamine infusion (Schilder et al., 2013), or a nerve blockade (Osuni et al., 2017b). These findings suggest that in CRPS motor deficits are at least partly a *consequence* of pain. However, this does not preclude the possibility that motor deficits could arise first and lead to pain, and then pain could further exacerbate the motor deficits.

Evidence that generation of motor commands (without actually executing movements) can lead to pain and other symptoms was provided by findings that imagining movement of the affected hand and wrist increased swelling and pain in both people with CRPS and non-CRPS pain (Moseley et al., 2008b). Similarly, imagining movement of the foot increased pain in people with (but not those without) neuropathic pain after complete thoracic spinal cord injury (Gustin et al., 2008).

2.1.5. Interim summary

There is evidence across several studies and conditions for motor deficits in CRPS, fibromyalgia, neuropathic pain, and musculoskeletal pain, and that these are associated with cortical reorganisation (Duncan and Boynton, 2007), cortical processing (Patel et al., 2014), and pain, consistent with the sensorimotor theory of pain (Harris, 1999). Most of this evidence is not causal, however, and in some cases pain relief improves motor performance. Harris (1999) described a way by which motor deficits could lead to pain for some painful conditions. However, the theory does not preclude the opposite: that pain, once established, contributes to motor deficits. Harris (1999) did not stipulate the mechanisms involved in this process, which could include spasms (Abbruzzese and Berardelli, 2003). This process is also likely to involve an interaction between peripheral and central mechanism, although a detailed discussion of this topic is beyond the scope of the current review. Arguably stronger evidence for the sensorimotor theory of pain comes from studies showing that motor imagery is altered in CRPS, musculoskeletal pain, and neuropathic pain, can be specific to the painful limb/area, and that performing imagined movements exacerbates pain and other symptoms. These findings suggest that central motor representations may be altered and are causally linked to pain. Overall, there is strong evidence for motor deficits that are related to cortical reorganisation in people with pathological pain, and that these are at least related to, and perhaps lead to pain.

2.2. Sensory functions

During movement, sensory feedback is used to monitor joint angles and kinematics and thereby correct for movement errors (Shadmehr et al., 2010). Central to Harris' reasoning was the proposal that reorganisation of somatosensory cortex associated with (non-painful) sensory changes, when combined with cortical motor signals, leads to sensorimotor conflict and thereby the affective experience of pain. We therefore review evidence for changes in the quality, precision, accuracy, and reliability of sensory input in people with pathological pain. We focus our review of sensory changes on touch (2.2.1.1) and proprioception (2.2.1.2), and consider evidence linking sensory deficits to altered cortical processing (2.2.2), and to pain (2.2.3).

2.2.1. Evidence for sensory changes in people with pathological pain

2.2.1.1. Touch.

Tactile information is used to inform sensorimotor processing (Dijkerman and De Haan, 2007; Rao and Gordon, 2001). Many pathological pain conditions are accompanied by reduced accuracy and precision of sensory information and problems with filtering irrelevant information (Conte et al., 2012; Rocchi et al., 2016).

Reduced tactile sensitivity (i.e. mechanical detection thresholds) of

the affected area has been reported for people with CRPS (Gierthmühlen et al., 2012), fibromyalgia (Evdkimov et al., 2019; Üçeyler et al., 2013), knee osteoarthritis (Hochman et al., 2013), musculoskeletal pain (Geber et al., 2008), neuropathic pain (Krumova et al., 2012; Maier et al., 2010), peripheral nerve injury (Gierthmühlen et al., 2012; Maier et al., 2010), and whiplash associated disorder (Kosek and Januszewska, 2008). In CRPS, reduced tactile sensitivity and other sensory changes can extend to other areas of the body ipsilateral to the affected limb (Rommel et al., 2001), suggesting a central mechanism. However, not all studies find evidence of altered tactile sensitivity in people with fibromyalgia (for meta-analysis see Augière et al., 2021), and others show *heightened* sensitivity in people with knee pain (for review see De Oliveira Silva et al., 2019), CRPS, peripheral nerve injury, and polyneuropathy (Maier et al., 2010). Taken together, there is substantial evidence that detection of touch can be altered for the affected area, but that this can vary within and between pathological pain conditions.

People with dystonia, fibromyalgia, and headache/orofacial pain can show changes in temporal components of touch processing. Larger somatosensory temporal discrimination thresholds have been reported for people with dystonia (for reviews see Avanzino et al., 2018a; Avanzino et al., 2015; Conte et al., 2019; Desrochers et al., 2019), fibromyalgia (Gunendi et al., 2019), and migraine (Vuralli et al., 2016), although no abnormalities were found in tension-type headache (Vuralli et al., 2017). These findings therefore suggest that the filtering of irrelevant sensory information may be altered in dystonia, fibromyalgia, and headache/orofacial pain.

Two-point discrimination thresholds are larger, reflecting worse tactile acuity, in many pathological pain conditions, such as CRPS (Di Pietro et al., 2020; Plegier et al., 2006; Reischwich et al., 2012), fibromyalgia (Martínez et al., 2019), headache/orofacial: migraine (Luedtke et al., 2018), temporomandibular disorders (La Touche et al., 2020), musculoskeletal pain: Achilles tendinopathy (Debenham et al., 2016), arthritis (Catley et al., 2014), chronic neck pain (Harvie et al., 2018), frozen shoulder (Mena-del Horno et al., 2020), knee osteoarthritis (Stanton et al., 2013), low back pain (Catley et al., 2014), lower back pain in cerebral palsy (Yamashita et al., 2019), and neuropathic pain (Taylor et al., 2010). Occlusal tactile acuity is higher in people with temporomandibular disorder (Bucci et al., 2020). However, tactile acuity deficits are not always found in people with burning mouth syndrome (Grushka et al., 1987), CRPS (Halicka et al., 2020a), dystonia (Mainka et al., 2021), frozen shoulder (Breckenridge et al., 2020a), hand osteoarthritis (Magni et al., 2018), lateral elbow tendinopathy (Wiebusch et al., 2021), low back pain (Meier et al., 2021), phantom limb pain (Fuchs et al., 2021), or unilateral shoulder pain (Caseiro et al., 2021). Despite many studies to the contrary, overall, there is clear evidence to suggest that tactile acuity is altered in several pathological pain conditions (for meta-analyses see Adamczyk et al., 2018; Catley et al., 2014).

Spatial processing of touch can be altered in pathological pain. Spatial discrimination thresholds, indexed by a participant's ability to distinguish between smooth and grooved surfaces presented at different orientations, are higher for people with back pain (Zamorano et al., 2015), benign essential blepharospasm, cervical dystonia, and focal hand dystonia (Molloy et al., 2003). Furthermore, spatial touch processing is involved in locating tactile sensations and can be altered in CRPS, fibromyalgia, musculoskeletal pain, and neuropathic pain. People with CRPS (Förderreuther et al., 2004; Trojan et al., 2019), and chronic low back pain (Wand et al., 2013) were less accurate and/or less consistent when locating touch on their painful body part. Furthermore, referred sensations have been reported in CRPS (Cohen et al., 2013; Maihöfner et al., 2006; McCabe et al., 2003), fibromyalgia (Martínez et al., 2019), and neuropathic pain following complete spinal cord injury (Soler et al., 2010), but not low back pain (Wand et al., 2013). People with CRPS can also experience allochiria (Cohen et al., 2013), a type of referred sensation where touch to the affected limb is perceived in the analogous location on the opposite limb (Di Pietro et al., 2020; Echaliér

et al., 2020). People with CRPS also take longer to locate a touch on the fingers (Kuttikat et al., 2018), and do not use tactile information optimally when making predictions of the spatial location of stimuli (Brown et al., 2020).

Taken together, there is substantial evidence that tactile detection and temporal and spatial aspects of touch processing are degraded for people with CRPS, dystonia, fibromyalgia, headache/orofacial pain, musculoskeletal pain, and neuropathic pain, and they appear to use this information differently to pain-free individuals.

2.2.1.2. Proprioception. Proprioception is needed for accurate motor predictions (Sober and Sabes, 2005; Tuthill and Azim, 2018), body representations, and applying muscle force (Proske and Gandevia, 2012). Proprioceptive deficits have been reported in many pathological pain conditions (for a review see Tsay et al., 2015), including CRPS (Schouten et al., 2003; van de Beek et al., 2002), dystonia (Avanzino et al., 2020; Khosravani et al., 2020; Ozen et al., 2021), fibromyalgia (Bardal et al., 2012; Celenay et al., 2019), musculoskeletal pain: knee osteoarthritis (for meta-analysis see Van Tunen et al., 2018), lateral elbow tendinopathy (Wiebusch et al., 2021), scoliosis (Szczygiel et al., 2021), and whiplash associated disorder (for meta-analysis see Mazaheri et al., 2021). People with a history of distal radius fracture can also have proprioceptive deficits (Muurling et al., 2020). There is mixed evidence of proprioceptive deficits in other musculoskeletal conditions, such as chronic idiopathic neck pain (for meta-analysis see Stanton et al., 2016), Ehlers-Danlos Syndrome (Clayton et al., 2013; Rombaut et al., 2010; Sahin et al., 2008), low back pain (Korakakis et al., 2021; Laird et al., 2014; Meier et al., 2019; Tong et al., 2017), and shoulder pain (for review see Ager et al., 2019). In summary, there is evidence for proprioceptive deficits in CRPS, dystonia, fibromyalgia, and whiplash associated disorder, although for some musculoskeletal conditions this evidence is mixed. Proprioceptive deficits could compromise motor predictions and/or provide distorted sensory feedback.

2.2.2. Evidence that sensory changes relate to altered cortical processing

Some patterns of tactile and proprioceptive deficits indirectly suggest that changes in sensory processing are at least partly cortically-driven, such as bilateral proprioceptive deficits in unilateral CRPS (Lewis et al., 2010), and experiencing less illusory movement in dystonia (Avanzino et al., 2018a; Conte et al., 2019). Complementing these clinical patterns, studies using brain imaging and stimulation techniques have directly linked changes to touch and proprioception in people with painful conditions to altered cortical activity and/or organisation. Cortical activation during tactile stimulation is altered in people with CRPS (Defina et al., 2021; Kuttikat et al., 2018). Two-point discrimination threshold is associated with structural changes in chronic low back pain (Kim et al., 2020), and altered cortical activation patterns in CRPS (Di Pietro et al., 2020; Pleger et al., 2006), but not with GABA or glutamate concentrations (Lee et al., 2020). There is also evidence that cortical processing of proprioceptive information is altered in dystonia (for review see Avanzino and Fiorio, 2014), and low back pain (Goossens et al., 2019). In summary, there is some evidence that sensory deficits relate to changes in CRPS, dystonia, and musculoskeletal pain.

2.2.3. Evidence that sensory changes relate to pain and other clinical characteristics

The sensorimotor theory of pain (Harris, 1999) posits that cortically-driven (non-painful) sensory changes lead to pain. Most available evidence supports the conclusion that tactile deficits are associated with pain, although none of these studies provide insights into whether one causes the other. Pain intensity correlated with two-point discrimination threshold for people with musculoskeletal pain conditions, such as low back pain (Wang et al., 2020; Zamorano et al., 2015), neck pain (Harvie et al., 2018), and shoulder pain (Barbosa et al., 2021), but not for people with knee osteoarthritis (Stanton et al., 2013).

Pain intensity also correlated with somatosensory temporal discrimination threshold in people with fibromyalgia (Gunendi et al., 2019), and migraine (Vuralli et al., 2016). Mislocalization of tactile stimulation delivered to the painful body part correlated with mechanical hyperalgesia in people with CRPS (Maihöfner et al., 2006). However, tactile mislocalization errors were not related to pain intensity in people with low back pain (Wand et al., 2013).

Proprioceptive impairments have been related to motor deficits in CRPS (Bank et al., 2013), and disability in low back pain, although its relationship with pain is not clear (for reviews see Ghamkhar and Kahlæe, 2019; Lin et al., 2019). Nonetheless, a prospective study of people with low back pain found that the extent to which participants reweighted proprioceptive sensitivity whilst standing, from trunk-focused to more ankle-focused control, predicted pain two years later (Claeys et al., 2015). This finding suggests that the way that proprioceptive information is processed may be important for progression of low back pain, and it is evidence that sensory deficits can precede pain. Overall, there is evidence that changes to touch and (to a lesser extent) proprioception are related to pain.

2.2.4. Interim summary

In summary, there is evidence of several sensory changes CRPS, dystonia, fibromyalgia, headache/orofacial, musculoskeletal pain, neuropathic pain, and whiplash associated disorder, which could contribute to a sensorimotor incongruence. Impaired tactile acuity, greater somatosensory temporal discrimination threshold, tactile mislocalization, and referred sensations could compromise the quality of the sensory information available to the nervous system, which would contribute to sensorimotor conflict (Azañón et al., 2016; Wolpert and Flanagan, 2001). There is also evidence of impaired proprioception in CRPS, dystonia, fibromyalgia, musculoskeletal pain, and whiplash associated disorder. In musculoskeletal pain, proprioceptive deficits have been found to prospectively relate to pain. However, as the relationship between pain and proprioception can be bidirectional, and influenced by fatigue and muscle trauma (Röijezon et al., 2015), there might be multiple factors leading to the development of such deficits. Therefore, sensory changes in several pathological pain conditions offer at least partial support for the sensorimotor theory of pain (Harris, 1999).

2.3. Body representation

Internal models of the body are used to generate predictions about the sensory outcomes of a movement (Shadmehr et al., 2010). Cognitive representations of the body are closely related to cortical representations, whilst also incorporating perceptual features and somatic input (Longo et al., 2010). Central to Harris (1999) proposal was the idea that distortions in the cortical representation of the affected limb give rise to errors in predicting the sensory consequences of movement, and thereby sensorimotor incongruence. If this is the case, there should also be evidence for distorted body representations in people with pathological pain (2.3.1). Temporary distortions are often seen following injury without leading to pathological pain. Therefore, it appears that distorted body representations are not themselves sufficient to explain pathological pain, but it is also possible that some of the processes that are involved in maintaining or updating body representations are altered (2.3.2). Furthermore, changes in body representations should be related to altered cortical processing (2.3.3), and pain (2.3.4).

2.3.1. Evidence for distorted body representations in people with pathological pain

Distorted body representations are widespread in CRPS, fibromyalgia, musculoskeletal pain, and neuropathic pain. Often people report that their painful body part feels different in its shape and/or size to its physical dimensions, or that it feels foreign, as described in interviews with people with CRPS (Lewis et al., 2007; Tajadura-Jiménez et al.,

2017), fibromyalgia (Valenzuela-Moguillansky, 2013), low back pain (Moseley, 2008), orofacial pain (Dagsdóttir et al., 2016), phantom limbs (Carlen et al., 1978; Giummarra et al., 2010; Jensen et al., 1983), and shoulder pain (Alaiti et al., 2019). Body representation distortions have also been demonstrated objectively (for reviews see Fuchs et al., 2018; Giummarra and Moseley, 2011; Haggard et al., 2013; Makin and Flor, 2020; Senkowski and Heinz, 2016; Tsay et al., 2015; Viceconti et al., 2020). For instance, people with CRPS (Moseley, 2005a; Peltz et al., 2011), hand osteoarthritis (Gilpin et al., 2015), and scoliosis (Szczygiel et al., 2021) perceived their painful areas as larger than the physical sizes, as indicated by visual estimation tasks. In contrast, people with low back pain showed no such distortion (Meier et al., 2021). These findings show that when misperceptions of the size of the affected body part do occur, the error is typically towards perceiving the body as larger than its physical size.

The problems shown by people with pathological pain when identifying the laterality of pictures corresponding to the painful body part have been taken as evidence that body representations are degraded and/or harder to access, and/or how difficult it would be for them to perform the same movement (for meta-analyses see Breckenridge et al., 2019; Ravat et al., 2019). Such impaired performance (i.e. reduced speed and/or accuracy) has been reported in CRPS, fibromyalgia, headache/orofacial pain, musculoskeletal pain, neuropathic pain, and whiplash associated disorder (see Table 2):

Some studies find that performance on hand laterality judgement tasks is only impaired for pictures corresponding to the painful body part, as seen in carpal tunnel syndrome (Schmid and Coppeters, 2012), CRPS, (Lee et al., 2021; Reid et al., 2016; Schwoebel et al., 2001), and knee osteoarthritis (Stanton et al., 2012), or when these are presented in the visual field corresponding to the painful side of the body for people with CRPS (Reid et al., 2016). Overall, most studies of limb laterality recognition show deficits in people with CRPS, fibromyalgia, headache/orofacial pain, musculoskeletal pain, neuropathic pain, and whiplash associated disorder. There is inconsistency concerning whether these deficits are for identifying both the affected and unaffected limb, or limb-specific problems.

2.3.2. Evidence for difficulties with updating body representations in people with pathological pain

Body representations flexibly adapt according to our experiences to support sensorimotor processes (for review see Martel et al., 2016), such as motor predictions (Haggard and Wolpert, 2005). If body representation disturbance underlies sensorimotor incongruence, then there should be a reason why body representations do not normalise/update.

Amputees and people with CRPS have been found to update their body representations. Upper limb amputees scale the representation of their limb depending on whether they were wearing a prosthesis or not (for review see Niedernhuber et al., 2018). Similarly, people with CRPS modified their gait consistent with changes in perceived body weight (Tajadura-Jiménez et al., 2017), and to accommodate tools (Vittersø et al., 2020). When using tools, however, we found a difference in the way that body representations of the non-affected and of the affected hand were updated. This finding suggests that people with CRPS update their body representations differently to pain-free individuals, and that their body representations may be less stable.

2.3.3. Evidence that distorted body representations relate to altered cortical processing

Body representations are underpinned by several cortical areas, including the primary somatosensory cortices, parietal lobes, anterior temporal cortices, inferior frontal cortices, and the anterior insula (Longo et al., 2010). If distorted body representations can influence sensorimotor processing, then we would also expect them to relate to altered cortical processing. The evidence from phantom limb pain is unclear (for review see Makin and Flor, 2020). In contrast, evidence from people with CRPS (Kohler et al., 2019; Strauss et al., 2021), and

Table 2

Laterality Recognition. Summary of studies examining body part laterality recognition in people with pathological pain, organized by population.

	Population	Rotated body part	Results
CRPS			
Bultitude et al. (2017)	CRPS; controls	Hands	Longer RTs for CRPS than controls.
Kohler et al. (2019)	CRPS; controls		Longer RTs for CRPS than controls.
Moseley (2004b)	CRPS; controls	Hands	Longer RTs for CRPS than controls.
Schwoebel et al. (2001)	CRPS; controls	Hands	Longer RTs for affected hand than non-affected hand.
Reinersmann et al. (2010)	CRPS; controls	Hands	Longer RTs for CRPS than controls.
Reinersmann et al. (2012)	CRPS; UL pain; controls	Hands	No sig. differences in RTs
Lee et al. (2021)	CRPS LL; CRPS LL	Hands, feet	Longer RTs for feet than hands for LL CRPS. Longer RTs for hands than feet for UL CRPS. No group differences for accuracy.
Fibromyalgia			
Martínez et al. (2019)	FMS; controls	Hands	Longer RTs and lower accuracy for FMS than controls.
Headache/orofacial pain			
Luedtke and Edlhaime (2021)	Migraine; controls	Neck	Lower accuracy for people with migraine, no group-difference in RTs. RTs varied with the migraine cycle.
Musculoskeletal pain			
Tompra et al. (2021)	Achilles tendinopathy; controls	Hands, feet	No difference in RTs or accuracy.
Coslett et al. (2010c)	Arm/shoulder pain; other pain; controls	Hands	Longer RTs for arm/shoulder pain group than controls. Correlated with pain severity for arm and shoulder pain group
Bray and Moseley (2011)	Back pain; controls	Hands, trunks	RT similar. Accuracy lower for trunks in back pain group than controls
Wallwork et al. (2020)	Chronic neck pain; acute neck pain; controls	Hands, necks	Lower accuracy for chronic neck pain than controls when judging neck rotation. No difference in RTs.
Richter et al. (2010)	Chronic neck pain; controls	Hands	Shorter RTs for chronic neck pain than controls.
Breckenridge et al. (2020a)	Frozen shoulder	Shoulders	Longer RTs for images corresponding to the affected side than the healthy side. No difference in accuracy.
Mena-del Horno et al. (2020)	Frozen Shoulder; controls	Shoulders	Longer RTs and poorer accuracy for images corresponding to the affected side, but not relative to controls.
Magni et al. (2018)	Hand OA; controls	Hands	Longer RTs and lower accuracy for hand OA than controls.
Conboy et al. (2021)	Irreparable rotator cuff tear; controls	Hands	Longer RTs for people with irreparable rotator cuff tear than controls.
Stanton et al. (2012)	Knee OA; arm pain; controls	Hands, feet	Lower accuracy for knee OA than controls.
Wiebusch et al. (2021)	Lateral elbow tendinopathy; controls	Hands	No difference in RTs or accuracy.

(continued on next page)

Table 2 (continued)

	Population	Rotated body part	Results
Coslett et al. (2010a)	Leg or foot pain; other pain; controls	Feet, legs	Longer RTs and lower accuracy for leg or foot pain group than other pain, and controls.
Bowering et al. (2014)	Low back pain; controls	Trunks	Lower accuracy for people with low back pain than controls.
Summers et al. (2021)	Persistent posterior thigh pain; controls	Hands, feet	Longer RTs for hands and feet relative to controls.
Breckenridge et al. (2020b)	Shoulder pain; controls	Shoulders	No difference in RT* or accuracy.
Caseiro et al. (2021)	Shoulder pain	Shoulders, feet	No difference in RT or accuracy between the affected and the non-affected side.
Pelletier et al. (2018)	Wrist/hand pain	Hands, feet	Accuracy predicted by self-reported motor imagery ability.
Neuropathic pain			
Schmid and Coppieters (2012)	CTS; controls	Hands, feet, necks, SM	RT similar. Accuracy lower for CTS hands and necks, not for feet or SM.
Lustenhout et al. (2020)	Neuralgic amyotrophy; controls	Hands	Lower accuracy for people with neuralgic amyotrophy relative to controls. No difference in RTs.
Reinersmann et al. (2010)	PLP; controls	Hands	Longer RTs for PLP than controls.
Osinski et al. (2020)	Spinal cord injury (with pain, without pain); controls	Hands, feet	No difference in RT or accuracy.
Whiplash associated disorder			
Richter et al. (2010)	WAD; neck pain; controls	Hands	Shorter RTs for WAD than controls.

* After controlling for age differences between the groups. Controls = pain-free controls; CTS = Carpal tunnel syndrome; FMS = fibromyalgia syndrome; NA = not applicable; NT = not tested; OA = osteoarthritis; RT = reaction time; sig = significant; SM = Shepard Metzler figures; UL = upper limb; WAD = whiplash-associated disorder

low back pain (Goossens et al., 2019) shows an association between behavioural measures of body representations and an altered pattern of cortical activity, as would be predicted by the sensorimotor theory of pain (Harris, 1999).

2.3.4. Evidence that distorted body representations relate to pain and other clinical characteristics

According to the sensorimotor theory of pain (Harris, 1999), distorted body representations lead to inaccurate motor predictions, sensorimotor incongruence, and thereby pain. Associations between self-reported body representation distortion and pain severity have been reported by several studies in CRPS (e.g. Halicka et al., 2020a; Lewis and Schweinhardt, 2012; Moseley, 2005a; Schulte-Goecking et al., 2020; Vittersø et al., 2021b), although these associations are not always found (Lewis et al., 2019; Ten Brink et al., 2021). Evidence for an association between body perception and pain has also been reported for people with knee osteoarthritis (Tanaka et al., 2021), and orofacial pain (Dagsdóttir et al., 2016). Overall, these studies suggest an association between pain and self-reported body representation.

In studies using limb laterality recognition, pain severity was associated with reaction times and/or accuracy for people with arm or shoulder pain (Coslett et al., 2010b), CRPS (Moseley, 2004b), and spinal cord injury (Osinski et al., 2020). No such associations were found for people with musculoskeletal back or shoulder pain (Bray and Moseley, 2011; Mena-del Horno et al., 2020), or carpal tunnel syndrome (Schmid and Coppieters, 2012). Overall, there is evidence that self-reported, and

to a lesser extent objectively measured, body representation distortion is related to pain and/or other clinical characteristics in CRPS, headache/orofacial pain, musculoskeletal pain, and neuropathic pain.

2.3.5. Interim summary

Distorted body representations are common in CRPS, fibromyalgia, musculoskeletal pain (i.e. shoulder pain, osteoarthritis, low back pain), neuropathic pain (i.e. phantom limb pain), and orofacial pain. The evidence is particularly consistent for self-reported body representation distortions, although findings of worse performance on limb laterality recognition also suggest problems with body and/or motor representations. There is also substantial evidence that distorted body representations are related to cortical changes, pain, and other clinical characteristics. Many of these pathological pain conditions are associated with heightened inflammatory states (e.g. Littlejohn, 2015), and there is preliminary evidence to suggest an association between inflammation and altered multisensory processing related to body representations from people with allergies (Finotti et al., 2018), coeliac disease (Finotti and Costantini, 2016), and obesity (Scarpina et al., 2016). Irrespective of the underlying mechanism, both motor representations (Jeannerod and Decety, 1995) and body representations (Haggard and Wolpert, 2005) are involved in motor predictions. Thus, distorted representations of the body could impair motor predictions, and thereby result in sensorimotor incongruence and pain.

2.4. Peripersonal spatial representations

The representations of space near our body (i.e. peripersonal) are thought to facilitate defensive and goal-directed action (De Vignemont and Iannetti, 2015). Therefore, one possible source of sensorimotor conflict is distorted peripersonal spatial representations (2.4.1). Spatial representations are malleable, and will update as we interact with the external world (e.g. Serino, 2019). Thus, unusual distortions in peripersonal space should, in theory, dissipate if normal interaction is possible thereby prompting the representations to update/return to a more “normal” state (2.4.2). To support the sensorimotor theory of pain, changes to peripersonal space should relate to cortical processing (2.4.3) and pain (2.4.4).

2.4.1. Evidence for distorted peripersonal space representations in people with pathological pain

Distortions in peripersonal spatial representations can occur in several pathological limb pain conditions. For instance, people with CRPS have been reported to have spatially defined motor deficits (Reid et al., 2017), although these biases are not always found (Christophe et al., 2016b; Halicka et al., 2020a). Similarly, there was no evidence that people with shoulder pain had distorted peripersonal spatial representations based on reachability judgements (Alaiti et al., 2019).

Defensive peripersonal space can be altered for people with pathological pain. The hand blink reflex (Crucchi et al., 2006) is more pronounced when the hand is near the face, providing a means to map the dimensions of defensive peripersonal space (Sambo et al., 2011). For people with trigeminal neuralgia, the hand blink reflex indicated an enlarged representation of defensive peripersonal space for the painful side (Bufacchi et al., 2017). People with migraine demonstrated a greater overall hand blink reflex than controls, especially if they were experiencing a headache at the time of testing (Ayas et al., 2020), although the typical spatial modulation was not found. People with cervical dystonia also showed a greater hand blink response than controls and an inversion of the normal spatial modulation (Öztürk et al., 2018). Taken together, these findings suggest that there is abnormal higher-order cortical modulation of the brainstem reflex in dystonia, headache/orofacial pain, and neuropathic pain.

2.4.2. Evidence for difficulties with updating peripersonal space representations in people with pathological pain

Interacting with the external world typically causes peripersonal space representations to update (e.g. [Serino, 2019](#)). For instance, a period spent interacting with tools can lead peripersonal space to enlarge to encompass the space surrounding the tools ([Maravita and Iriki, 2004](#); [Martel et al., 2016](#); [Rizzolatti et al., 1997](#)). Using this approach, we showed that people with CRPS updated their representations of peripersonal space ([Vittersø et al., 2020](#)), although the representations were less stable for the affected limb. Furthermore, despite not being able to move the limb themselves, people with spinal cord injuries updated peripersonal space representations around their limb following passive movement ([Scandola et al., 2016, 2020](#)). Therefore, there is no evidence that CRPS, or neuropathic pain leads to difficulties updating peripersonal space.

2.4.3. Evidence that distorted peripersonal space representations relate to altered cortical processing

To the best of our knowledge, the relationship between peripersonal space representations and cortical processing has yet to be directly studied in the context of pathological pain.

2.4.4. Evidence that distorted peripersonal space representations relate to pain and other clinical characteristics

There is some evidence that pain is related to changes in peripersonal space representations, although only few studies have investigated this topic. For people with CRPS, placing the affected limb in the non-affected side of space, was found to reduce hand temperature asymmetries ([Moseley et al., 2012](#)). This effect depends on the perceived location of the limb ([Moseley et al., 2013](#)), although it may not replicate ([Vittersø et al., 2020](#)). For people with shoulder pain, the extent of peripersonal space was associated with the intensity of movement-related pain ([Alaiti et al., 2019](#)). People with trigeminal neuralgia showed greater spatial modulation of the hand blink response for the painful side compared to the non-painful side ([Bufacchi et al., 2017](#)). Overall, there are several studies supporting a relationship between distorted spatial representations and pain and other symptoms in CRPS, musculoskeletal pain, and neuropathic (orofacial) pain.

2.4.5. Interim summary

The evidence suggests that representations of peripersonal space can have altered dimensions or stability in CRPS, dystonia, headache/orofacial pain, and neuropathic pain. So far, there does not appear to be a consistent pattern in whether peripersonal space is enlarged or diminished across different conditions. These representations can be modulated by immobilisation ([Bassolino et al., 2015](#); [Toussaint et al., 2018](#)), fear ([de Haan et al., 2016](#)), and attention ([Cléry et al., 2015](#)), all of which are plausible mechanisms to explain how distortions can arise in pathological pain. Peripersonal space representations are needed for sensorimotor processing ([De Vignemont, 2010](#); [De Vignemont and Ianetti, 2015](#); [Di Vita et al., 2016](#); [Serino, 2019](#); [Soechting and Flanders, 1989](#)), as they facilitate multisensory processing for action. The uncertainty of peripersonal space representation should increase the likelihood of sensorimotor incongruence by impairing multisensory processing. Therefore, observations suggesting that peripersonal space representations can be altered in pathological pain are consistent with the sensorimotor theory of pain ([Harris, 1999](#)).

2.5. Multisensory processing

The brain receives sensory feedback from multiple modalities during movement. Under normal circumstances, the nervous system weights this information by its reliability ([Wolpert et al., 2011](#)) and can thereby deal with conflicting sensory information by giving priority to the most reliable signal(s). For sensorimotor conflict to lead to pain, we would hypothesise that the processes governing multisensory perception might

be altered in people with pathological pain (e.g. preventing down-regulation of noisy/compromised sources of sensory information). We therefore review the evidence that people with pathological pain have altered multisensory perception (2.5.1) and that these changes relate to altered cortical processing (2.5.2), and to pain (2.5.3).

2.5.1. Evidence for altered multisensory processing in people with pathological pain

There is evidence that people with CRPS, dystonia, fibromyalgia, and neuropathic pain are capable of multisensory processing, for instance from studies using the rubber hand illusion ([Botvinick and Cohen, 1998](#)). This illusion relies on multisensory processing of visual, tactile and proprioceptive information ([Costantini and Haggard, 2007](#)), and has been induced in people with CRPS ([Reinersmann et al., 2013](#)), fibromyalgia ([Martínez et al., 2018](#)), focal hand dystonia ([Fiorio et al., 2011](#)), and neuropathic pain ([Pozeg et al., 2017](#)), including phantom limb pain ([Ehrsson et al., 2008](#)). The successful induction of the rubber hand illusion suggests that multisensory processing is not completely disrupted in pathological pain. Evidence of increased (e.g. in dystonia, neuropathic pain) or decreased (e.g. in fibromyalgia) outcomes of the illusion in some conditions could suggest altered weighting of different types of sensory information.

For amputees and people with CRPS, watching the mirror image of their non-affected limb being touched can lead to pain and/or sensations such as tingling and prickling in the phantom/affected limb ([Acerra and Moseley, 2005](#); [Cohen et al., 2013](#); [Ramachandran et al., 1995](#)). This phenomenon, referred to as “dysynchiria”, implies that (problems with) multisensory processing can give rise to illusory sensations. Dysynchiria has not been found for people with neuropathic pain ([Krämer et al., 2008](#)). More broadly, we speculate that anomalous sensations reported by people with pathological pain ([Echalier et al., 2020](#); [Ten Brink et al., 2020](#)) could occur because of problems with the multisensory processes that normally down-regulate less important signals.

Multisensory processing has also been studied in the context of interoception. Interoceptive accuracy was reduced for people with CRPS ([Solcà et al., 2020](#)), and in a mixed group of pathological pain conditions ([Di Lernia et al., 2020](#)). These findings suggest that people with pathological pain conditions might make use of multisensory bodily information differently to pain-free individuals.

2.5.2. Evidence that altered multisensory processing relates to altered cortical processing

There is limited evidence of an association between multisensory processing and altered cortical processing. Women with fibromyalgia reported greater unpleasantness from multisensory stimulation, which correlated with higher activation in areas associated with later stages of sensory processing ([Harte et al., 2016](#); [López-Solà et al., 2014](#)). Furthermore, the cortical response to nociceptive and multisensory stimuli can distinguish people with fibromyalgia from pain-free individuals ([López-Solà et al., 2017](#)). People with CRPS showed a reduced cortical response during a heartbeat counting task ([Solcà et al., 2020](#)). These findings provide evidence that altered cortical processing is related to changes in the experience of multisensory stimuli in people with fibromyalgia, and for interoceptive processing in CRPS.

2.5.3. Evidence that altered multisensory processing relates to pain and other clinical characteristics

Women with fibromyalgia reported increased pain in response to multisensory stimulation ([López-Solà et al., 2017](#)). Reduced interoceptive accuracy was also related to pain intensity in a mixed group of pathological pain conditions ([Di Lernia et al., 2020](#)), and to pain duration and motor impairments in people with CRPS ([Solcà et al., 2020](#)). Thus, although the causal link is not clear, the few studies on this subject provide support for the idea that problems with multisensory processing are associated with pain.

2.5.4. Interim summary

There is evidence to suggest that multisensory processing can be altered in CRPS, dystonia, fibromyalgia, and neuropathic pain. Multisensory perception was found to relate to altered cortical processing and pain for people with fibromyalgia, and to pain for people with CRPS. These findings suggest that for some pathological pain conditions, errors in sensorimotor processing might arise when combining and weighting information from multiple modalities. It is possible that such errors contribute to anomalous sensations in people with pathological pain. Within the framework of the sensorimotor theory of pain (Harris, 1999), it has been speculated that such anomalous sensations are warning signals or pre-cursors to pain (McCabe and Blake, 2008).

2.6. Sensorimotor integration

By combining and comparing sensory information to motor predictions, the nervous system is able to evaluate the success of a movement, make adjustments, learn, and adapt to incongruent information (Kording and Wolpert, 2004). Sensorimotor adaptation demonstrates that the sensorimotor system normally has mechanisms for dealing with sensorimotor conflict. For sensorimotor conflict to lead to pain, therefore, the sensorimotor systems of people with pathological pain must not fully correct for and/or adapt to conflicting information (else there would be no conflict). However, we would not expect a complete disruption to sensorimotor integration, as it is required for sensorimotor incongruence to be detected. We therefore evaluate evidence that people with pathological pain conditions have altered sensorimotor integration (2.6.1), which is related to altered cortical processing (2.6.2), and pain (2.6.3).

2.6.1. Evidence for altered sensorimotor integration in people with pathological pain

Sensorimotor integration can be altered in CRPS, dystonia, fibromyalgia, musculoskeletal pain, and whiplash associated disorder. Studies using unimanual tasks have shown that changes in sensorimotor integration are common in dystonia (for reviews see Avanzino et al., 2015; Conte et al., 2019). People with CRPS showed impairments in their control and estimation of grip force (Bank et al., 2014), and people with CRPS and fixed dystonia did not weight force feedback and position information optimally (Mugge et al., 2013). People with CRPS also have less stable bimanual coordination (Bank et al., 2015). In contrast, we found that people with CRPS showed normal sensorimotor integration, although they weight sensory information differently (Bultitude and Petrini, 2020). People with CRPS were able to integrate auditory and motor information during walking (Tajadura-Jiménez et al., 2017). Standing balance can be impaired in fibromyalgia (for meta-analysis see Núñez-Fuentes et al., 2021), musculoskeletal pain: chronic low back pain (for review see Berenshteyn et al., 2019), Ehlers-Danlos Syndrome (Rombaut et al., 2011), and whiplash associated disorder (for meta-analysis see Mazaheri et al., 2021), but was normal for people with idiopathic neck pain (de Zoete et al., 2020). Taken together, these studies suggest that changes in sensorimotor integration can be seen for people with pathological pain.

Sensorimotor adaptation has been studied in dystonia, and CRPS. People with cervical dystonia with tremor showed reduced adaptation to catching balls of different weights (Avanzino et al., 2018b). In contrast, normal adaptation has been found for people with CRPS (Vittersø et al., 2021a), and Ehlers-Danlos Syndrome (Clayton et al., 2013). Therefore, research into sensorimotor adaptation largely shows that performance is unimpaired in pathological pain, with some exceptions.

2.6.2. Evidence that altered sensorimotor integration relates to altered cortical processing

No research has looked at whether altered sensorimotor integration in pathological pain is related to altered cortical processing.

2.6.3. Evidence that altered sensorimotor integration relates to pain and other clinical characteristics

In upper limb amputees, greater phantom limb pain was associated with a smaller bimanual coupling effect when they were asked to imagine drawing circles with their phantom limb whilst drawing straight lines with their intact limb (Osuni et al., 2015). Similarly, performance on a bimanual coupling task was related to pain in people with CRPS (Bank et al., 2015). Both studies suggest that sensorimotor integration may relate to pain.

2.6.4. Interim summary

Sensorimotor integration can correct for errors in the sensorimotor system (Wolpert et al., 2011). The evidence reviewed suggests that sensorimotor integration can be altered in conditions such as CRPS, dystonia, fibromyalgia, low back pain, and phantom limb pain. Altered sensorimotor integration could disrupt the way that the sensorimotor system compensates for sensorimotor conflict.

2.7. Causal links between sensorimotor incongruence and pain

Harris (1999) specifically proposed that sensorimotor incongruence leads to the affective experience of pain. Therefore, to evaluate evidence for causal links between sensorimotor incongruence and pain, we review evidence that incongruent sensorimotor information leads to pain (2.7.1) and that treatments that reduce such conflict can relieve pain (2.7.2).

2.7.1. Evidence that incongruent sensory and motor information causes pain

Many studies have used mirror visual feedback to examine the effects of induced sensorimotor conflict (Table 3). Incongruent mirror visual feedback creates a conflict between visual, proprioceptive, and motor information. Compelling evidence for the sensorimotor theory of pain (Harris, 1999) would be if pain arose in otherwise pain-free individuals who experienced incongruent sensory and motor information. Although such pain has been reported in three studies (Brun et al., 2018; Foell et al., 2013; McCabe et al., 2005), this is usually only in a small fraction of participants, and most studies find that pain is not reported (for review, see Don et al., 2017b). There is, however, consistent evidence that incongruent mirror visual feedback can cause anomalous sensations (e.g. tingling; for review, see Don et al., 2017b). These sensory disturbances are greater when individuals are subjected to experimental pain induction (Brun et al., 2017), which suggests that pain amplifies anomalous sensations.

People with CRPS, fibromyalgia, and whiplash associated disorders reported an increase in pain due to incongruent mirror visual feedback. It could be that participants have developed a heightened sensitivity to sensorimotor incongruence due to prolonged exposure. Other studies, however, showed no increase in pain for those with musculoskeletal pain, or whiplash associated disorder (for review, see Don et al., 2017b), and/or that these effects are not specific to incongruent mirror visual feedback. Many studies have shown an increase in the intensity of sensory disturbances in response to incongruent mirror visual feedback for people with CRPS, fibromyalgia, musculoskeletal pain, and whiplash associated disorder. When the difference between mirror visual feedback and control conditions (e.g. replacing the mirror with a whiteboard) are compared, however, there are often no differences in sensory disturbances (Table 3; for review see Don et al., 2017b). The latter suggests that the increase in sensory disturbances may not be specific to sensorimotor incongruence.

Sensitivity to sensorimotor incongruence has also been studied using optokinetic stimulation, whereby visual cues signalling self-motion conflict with vestibular cues. For people with CRPS, optokinetic stimulation caused an increase in limb pain (Knudsen and Drummond, 2015). Migraine sufferers experienced greater nausea, more persistent headache, photophobia, and greater fingertip pain after optokinetic

Table 3

Mirror visual feedback. Summary of studies examining the responses of people with pathological pain to incongruent sensorimotor information, for instance, using mirror visual feedback (MVF) with anti-phase limb movements, presented in chronological order. Studies that found evidence that is consistent with the sensorimotor theory of pain (Harris, 1999) are indicated (✓ or ~, see table note for definitions).

	Population	Method	Finding		
			Pain		Sensory disturbances
CRPS					
Brun et al. (2019)	CRPS, controls	MVF	Not reported*		Greater intensity of sensory disturbances for CRPS than controls following I-MVF. C-MVF analysis not reported. ✓
Brun et al. (2021)	CRPS, controls	MVF-K	Greater intensity of pain and discomfort for CRPS (affected limb) than controls (non-dominant limb), for I-MVF relative to C-MVF.	✓	Greater intensity of report feelings of peculiarity for controls (non-dominant limb) than CRPS (affected limb), for I-MVF relative to C-MVF.
Fibromyalgia					
McCabe et al. (2007)	Fibromyalgia, controls	MVF	Numerically larger number of people with fibromyalgia reported pain during I-MVF than C-MVF, but not I-Ctrl* *	~	Numerically smaller percentage of people with fibromyalgia reported sensory disturbances during I-MVF than C-MVF, and I-Ctrl* *
Martínez et al. (2019)	Fibromyalgia, controls	MVF	Greater pain intensity for fibromyalgia than controls following I-MVF. Data not reported for baseline or C-MVF.	✓	Greater frequency of sensory disturbances for fibromyalgia than controls following I-MVF. Data not reported for baseline or C-MVF.
Brun et al. (2019)	Fibromyalgia, controls	MVF	Not reported*		Greater intensity of sensory disturbances for FMS, relative to controls following I-MVF. C-MVF analysis not reported. ✓
Brun et al. (2020)	Fibromyalgia, controls	MVF-K	Not reported*		Greater intensity of sensory disturbances for fibromyalgia than controls, for I-MVF relative to C-MVF. ✓
Musculoskeletal pain					
Brun et al. (2019)	Arthritis, controls	MVF	Not reported*		No differences between arthritis, and controls following I-MVF. C-MVF analysis not reported.
Roussel et al. (2015)	Dancers with/without MSK	MVF	Not reported*		No difference in sensory disturbances between those with pain and those without pain.
Don et al. (2019)	Low back pain, controls	Video feedback	No influence on pain.		Greater frequency of sensory changes for low back pain relative to controls during I-VF and C-VF. ~
Daenen et al. (2010)	Violinists with/without pain	MVF	No influence on pain.		Greater frequency of sensory changes for those with pain for I-MVF and C-MVF. ~
Whiplash associated disorder					
Daenen et al. (2012)	WAD, controls	MVF	Not reported*		Greater intensity of sensory disturbances for WAD than controls. No difference between I-MVF and I-Ctrl for WAD. ~
Don et al. (2017a)	WAD, controls	MVF	Numerical increase in the frequency of pain, but not intensity for I-MVF relative to C-MVF, but not I-Ctrl, for WAD. No reports of pain for control participants.	~	WAD had an increase in frequency but not intensity of sensory disturbances for I-MVF, C-MVF, I-Ctrl, C-Ctrl relative to moving with vision of both arms. No differences between I-MVF, C-MVF, and I-Ctrl reported for WAD. No between-group comparisons reported.
Pain-free controls only					
McCabe et al. (2005)	Controls	MVF	Painful sensations reported by 7/41 participants, most commonly during the I-MVF condition* *	✓	Numerically greater frequency of sensory disturbances in the I-MVF condition relative to C-MVF and I-Ctrl conditions* *
Foell et al. (2013)	Controls	MVF	Painful sensations reported by 2/113 participants, to similar extent across conditions.		Greater frequency of sensory disturbances for I-MVF relative to C-MVF and I-Ctrl. No sig. difference between conditions in intensity of other sensory disturbances. ✓
Wand et al. (2014)	Controls	MVF	No change in pressure pain thresholds or pressure-evoked pain.		Not measured.
Bultitude et al. (2016)	Controls	MVF	Not measured.		Lower sensitivity to gaps in tactile stimulation during C-MVF relative to other conditions, with no difference in sensitivity during I-MVF relative to I-Ctrl.
Brun et al. (2018)	Controls	MVF-K	Painful sensations reported by 1/20 participant. Experimental condition not specified.		Greater frequency of sensory changes during incongruent than congruent condition. In the incongruent condition, sensory changes were more frequent if the movements were active compared to passive. ✓

*Reported with sensory changes, but no data and/or analysis of pain on its own. *No inferential statistics reported. ✓ Evidence is consistent with theory. ~ Evidence is consistent with theory, but not specific to incongruent mirror visual feedback condition compared to one or more other conditions. C-MVF = congruent mirror visual feedback; C-VF = congruent visual feedback; I-Ctrl = incongruent control condition (e.g. incongruent movement with a whiteboard); I-MVF = Incongruent mirror visual feedback; I-VF = Incongruent visual feedback; MSK = musculoskeletal pain; MVF = mirror visual feedback; MVF-K = mirror visual feedback using a KINARM exoskeleton (Kingston ON, Canada); WAD = chronic whiplash associated disorder.

stimulation (Drummond, 2002). Therefore, the evidence supports the idea that sensorimotor incongruence can influence pain. It does not, however, provide strong causal evidence that such incongruence directly leads to pain.

2.7.2. Evidence that targeting sensorimotor processing reduces pain

Demonstrating that reducing incongruence between sensory and motor information reduces pain supports the conclusion that the two are causally linked, consistent with the sensorimotor theory of pain (Harris, 1999). Here we review evidence for the efficacy treatment methods that were either designed to or could be interpreted as addressing sensorimotor conflict: sensory discrimination (2.7.2.1), bodily illusions

(2.7.2.2), visual feedback (2.7.2.3), mirror therapy (2.7.2.4), graded motor imagery (2.7.2.5), and prism adaptation (2.7.2.6).

2.7.2.1. Sensory discrimination. Sensory discrimination training involves stimulating a body part and making judgements about a given aspect of the stimulus, such as recognising the type of stimulation and/or its location (e.g. Flor et al., 2001). There is very low-quality evidence that sensory discrimination training reduces pain for people with fibromyalgia and phantom limb pain, whereas the evidence is mixed for low back pain (Graham et al., 2020; Kälin et al., 2016). Two small uncontrolled studies also showed that people with CRPS had reduced pain and improved tactile acuity after sensory discrimination training

(Moseley et al., 2008c; Schmid et al., 2017).

2.7.2.2. Bodily illusions. Several illusions have been trialled to create the experience of stretching, shrinking, disappearing, magnification, colour change, and/or the illusory experience of movement in the painful limb (Table 4). Such illusions have the potential to alter sensorimotor processing by altering the visual appearance of the limb to match the (distorted) body representation, and/or to match a normal

Table 4

Bodily illusions. Summary of studies examining the effects of bodily illusions on pain, for people with pathological pain, organised by population.

	Illusion type	Population	Finding
CRPS			
Matamala-Gomez et al. (2019b)	Transparency, magnification, minification (VR)	CRPS	Increasing transparency reduced pain. Magnification increased pain. Reduced pain relative to control condition that was still present 2-weeks later
Lewis et al. (2021)	Virtual limb adjusted to patient's desired appearance	CRPS	Reduced pain relative to control condition that was still present 2-weeks later
Headache/orofacial pain			
Phoon Nguyen et al. (2020)	Colour and size of tongue	Burning mouth syndrome	No effect of size on pain. An illusory blue tongue reduced burning pain relative to baseline
Musculoskeletal pain			
Preston et al. (2020)	Stretching, shrinking, disappearing	Hand osteoarthritis	Stretching, and shrinking led to pain reduction. Disappearing did not influence pain
Preston and Newport (2011)	Stretching, shrinking	Hand osteoarthritis	Stretching, and shrinking led to pain reduction
Themelis and Newport (2018)	Stretching	Hand osteoarthritis	Stretching led to pain reduction
Stanton et al. (2018)	Stretching, shrinking	Knee osteoarthritis	Resizing led to pain reduction
MacIntyre et al. (2019)	Stretching, shrinking	Knee osteoarthritis*	Stretching had potential to reduce pain and swelling
Moseley et al. (2008a)	Magnification, minification	Hand pain	Magnifying increased movement-induced pain and swelling, minifying decreased movement-induced pain and swelling
Nishigami et al. (2019)	Muscular, reshaped, neutral	Low back pain*	Embodying a muscular back showed potential for pain reduction
Neuropathic pain			
Matamala-Gomez et al. (2019b)	Transparency, magnification, minification (VR)	PNI	Increasing transparency increased pain. Magnification reduced pain.
Cole et al. (2009)	Virtual limb (VR)	Phantom limb pain	Reduced phantom limb pain
Mercier and Sirigu (2009)	Virtual limb (VR)	Phantom limb pain	Reduced phantom limb pain
Sano et al. (2016)	Virtual limb (VR) with/without tactile feedback	Phantom limb pain	Reduced phantom limb pain was greater when VR was combined with tactile feedback
Osumi et al. (2017a)	Virtual limb (VR)	Phantom limb pain	Reduced phantom limb pain

*Case study. PNI = peripheral nerve injury; VR = virtual reality.

limb, and thus to correct any sensorimotor incongruence that might be present. Findings from these studies suggest that pain can be modified by bodily illusions in CRPS, osteoarthritis, peripheral nerve injury, and phantom limb pain (for reviews see Boesch et al., 2016; Dunn et al., 2017; Matamala-Gomez et al., 2019a; Senkowski and Heinz, 2016). However, the type of illusions that are effective for pain relief can vary both between and within conditions. For instance, magnification of a limb reduced pain for people with peripheral nerve injury, but increased pain for people with CRPS (Matamala-Gomez et al., 2019b). People with knee osteoarthritis (Stanton et al., 2018) or CRPS (Lewis et al., 2021) required different illusions (e.g. stretching versus shrinking) to attain pain relief, despite the fact that participants generally perceived their affected limbs to be larger than in reality (see 2.3.1).

2.7.2.3. Visual feedback. A greater availability of sensory information (e.g. visual information) will increase the precision of sensorimotor processing, and allow more reliable sources of information to be given priority (Kording and Wolpert, 2004). For people with low back pain (Diers et al., 2016, 2013; Wand et al., 2012), and neck pain (Beinert et al., 2019) visual feedback during a movement can reduce pain (for reviews see Daffada et al., 2015; Heinrich et al., 2019). Virtual feedback reduced pain in people with spinal cord injury (for review see Roosink and Mercier, 2014).

2.7.2.4. Mirror therapy. Mirror therapy is thought to reduce sensorimotor conflict by providing visual feedback of normally appearing and moving limb in place of a painful (phantom) limb, thereby restoring congruence between motor intentions and sensations. The evidence is not strong enough to recommend mirror therapy for phantom limb pain (for meta-analyses see Thieme et al., 2018; Thieme et al., 2016; Wittkopf et al., 2018), although it appears to be effective for CRPS pain, and post-stroke pain. The quality of evidence for the use of mirror therapy in both CRPS and phantom limb is low (Rothgangel et al., 2011).

2.7.2.5. Graded motor imagery. Graded motor imagery is a phased approach aiming to normalise central representations of the affected limb through sequential training in limb laterality recognition, imagined movements, and mirror therapy (Moseley, 2004a, 2005b). The combination of exercises is thought to reduce sensorimotor conflict by normalising body representations and motor imagery, and by providing visual feedback of a normally moving limb. Mentally rehearsing a movement without executing it can by itself lead to reduced musculoskeletal pain (Suso-Martí et al., 2020), and phantom limb pain (MacIver et al., 2008), whereas the evidence is mixed for people with spinal cord injury (Opsommer et al., 2020). When considered across a range of conditions, graded motor imagery is more effective than physiotherapy for pain reduction (Bowering et al., 2013), and pain relief can be maintained up to six-months (Limakatso et al., 2019). Graded motor imagery appears to be particularly effective for CRPS (for meta-analysis see Thieme et al., 2016). Furthermore, people with distal radial fractures who underwent graded motor imagery in addition to traditional rehabilitation showed a greater improvement in pain and function (Dilek et al., 2018). This finding suggests that targeting body representation and motor imagery in the early stages after injury can prevent pain from persisting.

2.7.2.6. Prism adaptation. During prism adaptation, people point to visual targets viewed through lenses that shift vision to one side. This procedure causes a conflict between visual, proprioceptive, and motor information, which is quickly resolved as the sensorimotor system recalibrates (Rossetti et al., 1998). Prism adaptation could therefore work by improving participants' sensorimotor integration (Bastian, 2008). Four small-scale uncontrolled studies suggested pain and other symptoms of CRPS reduced after prism adaptation (Bultitude and Rafal, 2010; Christophe et al., 2016a; Foncelle et al., 2021; Sumitani et al.,

2007). However, in a recent double-blind randomized controlled trial, we did not observe any benefit of prism adaptation over sham treatment (Halicka et al., 2020b, 2020c).

2.7.3. Interim summary

Central to Harris (1999) theory was the proposition that sensorimotor incongruence can lead to the affective experience of pain. Studies that have induced sensorimotor incongruence to directly examine this proposal provide very limited evidence of pain arising in previously pain-free individuals. Somewhat more consistent is the evidence that these procedures can both exacerbate pathological pain and cause anomalous sensations, neither of which clearly support a causal link between sensorimotor incongruence and pain. It is also possible that this link is indirect, whereby sensorimotor changes cause peripheral changes (e.g. spasms, or inflammatory responses) that cause pain, and that a more mechanistic approach is needed to understand the relationship between sensorimotor incongruence and pain. Nonetheless, the analgesic effects of treatments that aim to target sensorimotor processing could be interpreted as showing that sensorimotor incongruence leads to pain. This evidence is strongest for graded motor imagery, and to a lesser extent sensory discrimination, visual feedback, and mirror therapy. As there is limited evidence that sensorimotor conflict causes pain, yet somewhat greater evidence that treating sensorimotor processing can reduce pain, we suggest that the sensorimotor theory of pain (Harris, 1999) is more appropriate for explaining why pain persists rather than how it arises.

3. Discussion

3.1. Sensorimotor processing is altered in many pathological pain conditions

Regardless of the theoretical lens through which one views the evidence, it is apparent that there are changes in the components of sensorimotor processing in many pathological pain conditions. Substantial research has investigated motor deficits, sensory changes, and body representation distortions in pathological pain. Although the conclusions of these studies are by no means unanimous, the evidence generally supports changes to these functions across a broad range of conditions, including CRPS, dystonia, fibromyalgia, headache/orofacial pain, neuropathic pain, and musculoskeletal pain. Consistent with the sensorimotor theory of pain (Harris, 1999), the specific nature of some of these motor and sensory deficits imply altered cortical processing. For motor deficits, these include difficulties with imagined movements reported in CRPS (Moseley et al., 2008b), low back pain (La Touche et al., 2019), phantom limb pain (Kikkert et al., 2017), and spinal cord injury (Scandola et al., 2017); and bradykinesia, hypokinesia, and spatially defined motor deficits in CRPS (Galer et al., 1995; Reid et al., 2017). For sensory deficits, these include mislocalization of touch across a range of conditions, and impaired tactile sensitivity in people with CRPS in other areas of the side of the body ipsilateral to the affected limb (Rommel et al., 2001). There are also many imaging studies that relate motor and sensory function of people with pathological pain to changes in the structure and function of brain areas implicated in motor control, sensory processing, and sensorimotor integration. There is little evidence concerning relationships between body representation and cortical processing in people with pathological pain. However, body representation distortions have been frequently related to pain and other clinical symptoms, as have changes in motor and sensory functions. Overall, there is good evidence that 1) motor functions, sensory functions, and body representations are altered in many pathological pain conditions, and 2) some of these changes are related to changes in cortical processing, pain, and other clinical symptoms.

Our review also found evidence that sensorimotor integration and multisensory processing can be altered in people with CRPS, dystonia, fibromyalgia, musculoskeletal pain, neuropathic pain, and whiplash

associated disorder. In general, studies found that people with pathological pain can integrate sensorimotor information, but that the integration may be altered (Bultitude and Petrini, 2020) and not optimal (e.g. Mugge et al., 2013). Abnormal weighting of different sensory signals might explain anomalous sensations reported spontaneously and under experimentally induced sensorimotor conflict, although this idea has yet to be examined. No studies have examined how sensorimotor processing is related to cortical processing in pathological pain, however, there is evidence that it is related to pain for people with CRPS (Bank et al., 2015), and phantom limb pain (Osumi et al., 2015). Somewhat fewer studies have examined multisensory perception in pathological pain, which was associated with altered cortical processing and pain for people with fibromyalgia (Harte et al., 2016; López-Solà et al., 2014, 2017), and with pain for people with CRPS (Solcà et al., 2020). Overall, there is evidence that people with pathological pain can show both multisensory processing and sensorimotor integration, but that these are altered.

3.2. Evidence for changes in sensorimotor processing is stronger for some conditions than others

There is good evidence that actual or imagined movements, tactile and proprioceptive processing, and body representations are impaired or altered in people with CRPS, fibromyalgia, and low back pain. Another condition for which there is relatively strong evidence for altered sensorimotor processing is phantom limb pain. Compared to other conditions considered in this review, we found the strongest evidence for causal links between sensorimotor incongruence and pain in CRPS, and fibromyalgia, and to some extent in low back pain, and phantom limb pain. The only two studies that provide evidence that incongruent mirror visual feedback can increase pathological pain are of people with CRPS (Brun et al., 2021), and fibromyalgia (Martínez et al., 2019). Regarding efforts to alleviate pain, sensory discrimination, visual feedback, mirror therapy, and graded motor imagery are all therapies that could be argued to address one or more problem with the components of sensorimotor processing. There is good evidence that one or more of these therapies can reduce pain for people with CRPS, fibromyalgia, low back pain, or phantom limb pain. Overall, we found the most consistent evidence in support of the sensorimotor theory of pain (Harris, 1999) for CRPS, fibromyalgia, low back pain, and phantom limb pain. As the theory was intended to describe how pain can arise in the absence of clear tissue pathology, it could be that sensorimotor changes play a greater role in the pathology of these conditions than in other painful conditions.

3.3. The evidence that problems with sensorimotor processing cause pain is very limited

Establishing that sensorimotor incongruence actually leads to pathological pain is methodologically challenging. Once pain is established, it is impossible to determine whether any deficits in sensorimotor processing preceded or followed pain. Nonetheless, our review has revealed some limited evidence that problems with the components of sensorimotor processing can precede, and possibly lead to, pain. Observations that motor (Sadler, Spink, Ho, De Jonge, and Chuter, 2017) and proprioceptive function (Claeys et al., 2015) predict later low back pain suggest that movement and proprioceptive deficits lead (directly or indirectly) to pain. Somewhat stronger support that sensorimotor incongruence is relevant for the emergence of pain is provided by evidence that early implementation of graded motor imagery leads to better pain outcomes after acute fracture (Dilek et al., 2018). Thus, there are a small number of isolated findings that support the conclusion that pain can arise as a result of sensorimotor incongruence, although our review also highlighted findings that contradict this idea. Taken together, evidence that links sensorimotor incongruence and pain is lacking. Given the somewhat greater evidence that sensorimotor processing plays a role

in the maintenance of pathological pain (e.g. 2.7.2) rather than in its development, it could be that the sensorimotor theory of pain (Harris, 1999) is more relevant for understanding why pain persists rather than how it arises.

4. Conclusions

The sensorimotor theory of pain described how pain could arise in the absence of clear tissue pathology (Harris, 1999). We have reviewed evidence of sensorimotor changes in pathological pain, and that such changes relate to cortical processing and/or pain. Our review highlights that a broad range of changes in sensorimotor processing can be seen in people with CRPS, dystonia, fibromyalgia, headache/orofacial pain, musculoskeletal pain, neuropathic pain, and whiplash associated disorder, and be present in conditions with distinct pathologies. Most consistent were motor deficits, sensory changes, and body representation distortions, and they were most evident in CRPS, fibromyalgia, and low back pain. Although it is unclear what causes these changes, targeting them for treatment has proven to alleviate pain, which is one of the strongest arguments in favour of the theory. However, there are also examples where the theory has been contradicted. We therefore propose that the sensorimotor theory of pain (Harris, 1999) is more appropriate for understanding why pain persists rather than how it arises. Irrespective of their theoretical basis, this review demonstrates that sensorimotor changes are evident across a broad range of pathological pain conditions. Furthering our understanding of sensorimotor processing in pathological pain is a key step towards developing new and improving existing treatments.

Conflict of interest

The authors have no conflicts of interest to declare.

Data Availability

No data was used for the research described in the article.

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