Applying Current Concepts in Pain-Related Brain Science to Dance Rehabilitation

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Abstract

Dance involves exemplary sensory-motor control, which is subserved by sophisticated neural processing at the spinal cord and brain level. Such neural processing is altered in the presence of nociception and pain, and the adaptations within the central nervous system that are known to occur with persistent nociception or pain have clear implications for movement and, indeed, risk of further injury. Recent rapid advances in our understanding of the brain's representation of the body and the role of cortical representations, or "neurotags," in bodily protection and regulation have given rise to new strategies that are gaining traction in sports medicine. Those strategies are built on the principles that govern the operation of neurotags and focus on minimizing the impact of pain, injury, and immobilization on movement control and optimal performance. Here we apply empirical evidence from the chronic pain clinical neurosciences to introduce new opportunities for rehabilitation after dance injury.

Dance requires exemplary movement control. Pain, injury, immobilization, and rest have clear impacts on the neural mechanisms that subserve movement. One way to minimize those impacts may be to employ strategies that maintain the cortical maps that subserve bodily representations. Recent discoveries in the clinical pain sciences have revealed much about the tight connections between brain-grounded representations of the way our body feels and the way it, and the space around it, is protected and regulated at a physiological level. There are two examples of this phenomenon. First, when a painful arm is magnified to appear to its owner as though it is swollen, the swelling evoked by movement is increased, even though the actual movements are no different.[1] As a second example, inducing the rubber hand illusion in which the participant feels that one arm has been "replaced" by an artificial counterpart, produces limb-specific cooling[2] and enhanced histamine reactivity[3] of the arm, and noxious stimuli delivered during the illusion evoke pain in the artificial arm with no loss of intensity.[4]

The discovery of disrupted spatial processing in people with unilateral pain syndromes and its inter-relationship with thermoregulation and movement control,[5-8] and disrupted thermoregulation with transcranial magnetic stimulation over the posterior parietal cortex (a brain area thought to be critical in subserving spatial and proprioceptive data),[9] added weight to the idea that a network of thalamocortical loops subserves protection and regulation of the body, and the space around it, at both perceptual and physiological levels. This concept was captured in the idea of a "cortical body matrix."[10] Relevant to this idea is the recent development of treatments that use multimodal illusions to modulate the cortical body matrix in real time. Some illusions show real promise for clinical applications,[11] but more work is clearly required to understand which illusions might work for whom.[12]

These developments may be particularly relevant for dance because of the imperative to produce precise movements, often sharing one's peripersonal space with that of another, frequently performing with an ongoing painful condition, and sometimes returning from time off due to injury. Dancers are no different

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from other elite performers insofar as compromised performance represents a potentially major threat that extends well beyond participation—to identity, income, and career prospects.

A Framework for Making Sense of Pain and Altered Movement

The present review is based on a framework for understanding pain and movement dysfunction that emphasizes three issues: 1. that perceptual (e.g., pain) and physiological (e.g., movement) outputs are subserved by brain-grounded neuroimmune representations that are intimately linked; 2. that these outputs are influenced by a potentially vast array of other "second level" brain-grounded neuroimmune representations that subserve credible evidence that the physical self is in danger and needs protecting; and 3. these "second level'" representations can prevent optimal pain and movement outcomes of rehabilitation but can be targeted and normalized with appropriate intervention.

Inherent in this framework is the goal of minimizing the impact of pain and injury. It is appropriate that rehabilitation after acute dance-related injury involve initial rest and gradual re-introduction of movement, an approach that focuses on recovery of strength, range of motion, and endurance, so that the structures of the body can again withstand the full mechanical loads of performance. One might suggest that this kind of rehabilitation targets the body's periphery, the muscles, tendons, bones, and joints. This is all well and good, but adaptations are known to occur within the central nervous system when people are injured, at real or perceived risk of injury, or have marked changes in behavior, for example immobilization.[13-15] There is compelling evidence that these adaptations do not necessarily resolve when the injury does,[13,14,16-18] which raises the very real possibility that movement and interaction with peripersonal space will remain disrupted, and the pain production system will continue to be overly sensitive and responsive to situations that are in fact not dangerous. Moreover, common strategies to minimize performance loss during time-off from dance, for example facilitative imagery,[19,20] are unlikely to address some of these disruptions. It is these investigators' contention, then, that comprehensive management of the dancer must include appraisal of a range of neuroimmune representations, including psychosocial and contextual factors (although not simply as contributors to poor coping with pain or self-efficacy).[21] In order to understand the implications of this hypothesis, it is important first to visit current concepts in pain science and the relationship between pain and movement.

Pain, Movement, and Protection

Understanding the distinction between nociception and pain is critical in clinical practice and in research. Nociception can be considered "danger detection and transmission,"[22] whereas pain is a conscious experience that compels us to take protective action.[23] We have known for a long time that nociception is neither sufficient nor necessary for pain,[23-25] yet the presumption that they are one and the same is still common in clinical practice[26] (although, critically, not to the dominant extent it was just a decade ago).[27] Activation of primary (peripheral) nociceptors can certainly trigger reflex responses—most obviously the withdrawal reflex—and modulate spinal motor output, without necessarily generating a sensory experience,[28,29] yet pain triggers whole-organism moto-behavioral outputs—i.e., purposeful responses such as limping, staying still, moving away, or seeking professional care. Because pain is necessarily *felt,* it can be considered as occurring in consciousness rather than within biological structures, whereas nociception is never felt and theoretically occurs entirely within biological structures. Modern pain science requires us to conceptualize pain as an "output" of our biological substrate, most conveniently described as an output of the brain[30,31] (although see Thacker and Moseley[32] for important traps associated with that convenience). Pain can be conceived as a perceptual inference[33] rather than a message that is generated at the tissues and transmitted to the brain.[34] As such, pain is most obviously about protection of the physical self rather than a perceptual marker of the state of the body tissues.

Similar to pain, movement is a very effective protector of bodily tissues. Movement results from the integration of a potentially complex array of sensory and contextual information. Critically, both pain and movement can be seen as outputs that make things happen; the outputs themselves do not occur within the brain, but they are subserved by brain-grounded processes. This differentiates them from the infinite array of influences that occur within the brain and modulate "from the inside." Each of those influences, for example, contextual or proprioceptive data of which we are unaware, are represented or "held" within the brain and can be thought of as exerting their influence via the output of those representations. Insofar as their effects are exerted within the brain, we can conceptualize this potentially infinite array of influences that occur within the brain as modulatory outputs.

Inherent to this understanding of pain and movement is that they are not hierarchically differentiated. This is contrary to previous models of pain and motor control, which suggest that pain has sometimes subtle effects on motor commands.[35] The research underpinning those models clearly shows that nociception modulates motor commands,[36] and that those in pain move differently from those without pain.[36] However, we contend that concluding that an effect of pain when one delivers a noxious stimulus, suffers from conflation of nociception and pain, and concluding that pain causes a sufferer to move differently suffers from the causality assumption (an association does not imply causation).

Movement is potentially modified in many ways—proprioceptive input, competitive motor require-

ments (most obviously "perform or protect"), motor planning, the motor command, spinal modulation by incoming nociception, the executor "organs" themselves (i.e., muscles), or a combination of any of these (see Wolpert and Ghahramani[37]). That central nervous system changes in motor output can persist after symptoms resolve has led to new approaches to rehabilitation in people with chronic pain, most notably the assessment and re-training of the neural representations associated with the movement, protection of the body, and the space around it (the "peripersonal space").[38] The large number of discoveries related to these neural representations and how they interact have led to the proposal of a new theoretical model that can be used to guide our assessment and rehabilitation of people in pain or recovering from injury.

Theoretical Models: The Cortical Body Matrix, Action, and Modulation Neurotags

Digging deeper into the "cortical body matrix," a highly complex network of cortical representations that integrates information about the body, the self, and the surrounding space and regulates and protects us at a physiological (e.g., temperature regulation) and perceptual (e.g., "feeling" cold) level[10] (see also Bellan and colleagues in this issue), compels us to broaden the scope of our assessment and intervention for injured dancers or those in pain. Inputs from sensory organs, including visual, auditory, tactile, olfactory, proprioceptive, and vestibular, as well as from memory, emotion, and other cognitive factors, are all capable of modulating the outputs of the cortical body matrix.[10] The precise neuroanatomy and biological mechanisms that substantiate the cortical body matrix and its function are yet to be untangled (although broad functional neuroanatomy has been suggested[10]), but the prevailing theory relates to the concept of neural signatures,[39] or "neurotags"—i.e., discrete (but also dynamic) neuroimmune networks that both influence and compete against each other.[40]

One can think, then, of the cortical body matrix as consisting of an infinite number of neurotags that, together, form a kind of "protection-meter" that continually drives bodily preservation. The biological levers with which this protection-meter affects preservation include both consciousness (feelings of pain, hunger, fatigue, for example) and bodily responses.[31,41] The clinician or teacher can think of the cortical body matrix in terms of neurotags that cause either action or modulation. Action (or "primary"[40]) neurotags exert an influence beyond the brain, for example, into consciousness or the movement of joints, and are therefore the neurotags that represent the "observables"—i.e., what someone feels, states, or does. Modulatory (or "secondary"[40]) neurotags exert their influence only within the brain, and therefore represent the non-observables. Endorsement of this idea leaves the inquiring clinician considering how disruption of the observables, for example, pain, stress, or suboptimal movements, might reflect the influence of certain non-observables. That is, the clinician considers a much wider range of potential influences on pain and movement rather than focussing solely on the movement, or solely on the pain, as though it is generated in the tissues and transmitted to the brain. Although the erroneous nature of this idea of pain has been known for decades, clinicians of all persuasions are reluctant to give it up[26] (although things are changing—see Moseley[42,43] for a brief discussion). This distinction between old and current concepts as it is applied to movement rather than pain might be captured thus: when struck with a disruption of movement (the observable), rather than asking only "what muscles are weak and strong and what joints are stiff and loose?" one might ask "what are the influences on the movement neurotags?" In so doing, the clinician must look for any credible evidence of danger, a search that necessarily involves psychological and contextual domains.

Let us consider, then, the action neurotags that underlie movement sequences of dance in a dancer returning from injury: the desired movement outputs subserve the Newtonian and aesthetic requirements for the performance; however, a potentially broad array of modulatory neurotags that represent credible evidence of danger (for example, those neurotags that represent cues that the body part is broken, vulnerable, or weak) will influence motor outputs that subserve protection rather than performance. If these "protective neurotags" are sufficiently influential, then the motor output is affected and the performance is compromised. That is, the final movement reflects a "best guess"[33] on what is an optimal response under the competing demands of performance and protection (Fig. 1).

The Motor System

Ultimately, the highest priority of the motor system is protection of the organism—to use the movement generators to prevent body tissue from being (re)injured, allow it to heal if it is injured, and to accommodate fundamental survival needs (i.e., eating and drinking[44]). We have argued that nociception—activity in high threshold primary neurones that are distributed across most of the tissues of the body (with some exceptions, for example, the brain and the inner sections of knee menisci)—is best considered a danger detection system. Sudden or large changes in the mechanical, chemical, or thermal state of the tissues activates nociceptors that transform and transmit the signal to the central nervous system (CNS) and then, via spinal nociceptors, to the brain.[45] This mechanism is very effective at influencing brain activity in the presence of otherwise undetected threats. The most immediate, effective (or "least costly") response is usually a motor response.[33,46,47]

Earlier we touched on the abundant literature pairing pain with altered motor output (see Moseley and colleagues[48] for review). The prevailing theme throughout the literature is one

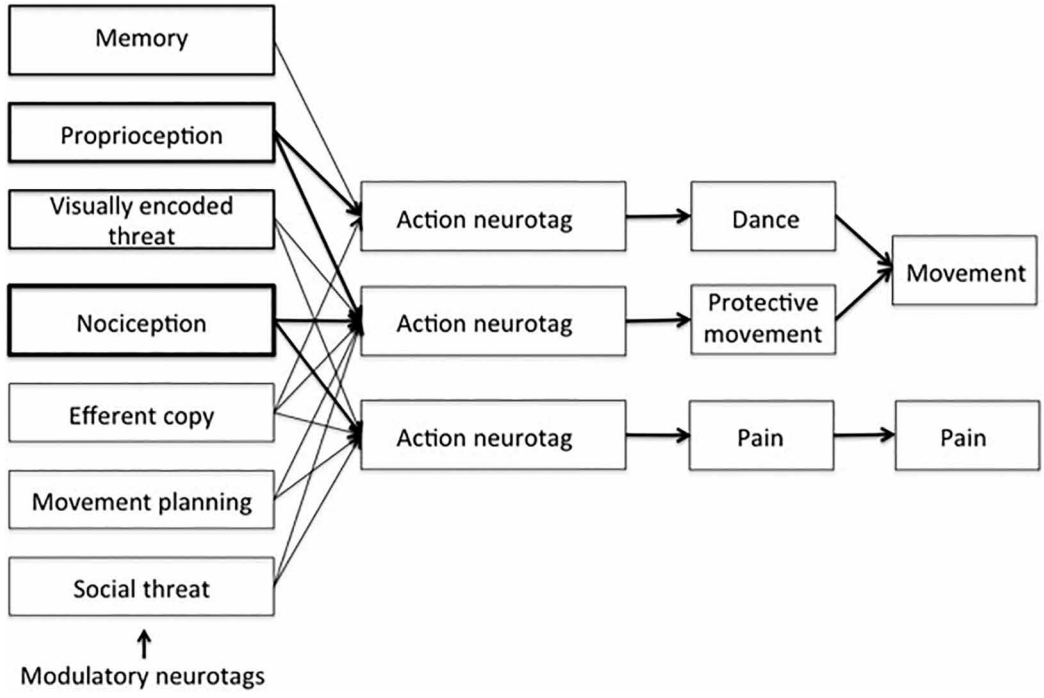

Figure 1 According to the present paradigm, the "observables" of movement and pain are the result of activation of brain-grounded neuroimmune representations, or "neurotags." These neurotags are themselves under the constant influence of other neurotags, which subserve anything that provides credible evidence that the physical self is in danger. Those neurotags that promote protection and regulation by affecting consciousness (e.g., pain or other feelings) or the levers that alter bodily function (e.g., movement or immune response) can be conceptualized as action neurotags. Those that influence the action neurotags can be considered "second level" or "modulatory neurotags." Here, the influence of a modulatory neurotag is denoted by the thickness of the lines that surround it and project to the action neurotag. One can see that the "observable" output—e.g., pain of 6/10 or a particular movement—can be the result of a potentially infinite mix of modulatory neurotags. Here one can also see that the optimal performance-related motor output will be compromised if there is any protection-related motor output. The astute clinician, then, on detecting suboptimal motor performance, will consider anything that provides credible evidence that the body is under threat.

of protection under threat. For example, clinical back pain, experimentally induced back pain, the expectation of experimentally induced back pain, and the conviction that one's back is inherently fragile are all associated with "protective" motor strategies for simple behaviors such as arm or leg movements.[15,37,49-51] Changes in muscle recruitment patterns associated with movement during a bout of acute experimental pain vary according to which muscles are injected with hypertonic saline solution[52] and seem to be dictated by the ability to redistribute load to the other limb or to other muscle groups.[53] In regard to postural control, the movement strategies adopted by people during experimental back pain appear to limit trunk motion but *usually* return to normal patterns when pain resolves.[54] Some healthy subjects lose variability in postural control during experimental back pain and then maintain that loss, which *may* be mediated, at least in part, by their beliefs about their back.[51] Here, the literature presents a shift from the exclusive sequential trio of nociception, pain, and motor control into the presentation of a multiple neurotag network whereby pain, movement, and protection are intimately linked, but where protection seems to be the prevailing and over-riding driver of output (Fig. 1).

One aspect of the framework described here that we contend to be important is that motor output is potentially modulated by anything that provides credible evidence of threat, including purely cognitive factors, such as the belief that one's foot is weak, or that one's teacher is unfair, or that one's well-being is at stake. As is always the case, the easiest methods to investigate such concepts involve the simplest (and in many ways least ecologically valid) experimental approaches. With regard to motor output, the degree to which purely cognitive stimuli can modulate motor control is nicely illustrated by models of reflexive behavior. Reflex paradigms provide an objective and robust platform with which to test automatic and implicit motor responses to impending threat. Reflexes are not vulnerable to reporter bias, modelling, or observer bias (presuming analysis is automated).

One reflex that is clearly protective is the somatosensorily evoked hand-blink reflex (HBR), which is elicited by electrical stimulation to the median nerve at the wrist. The HBR has until recently been considered to be an entirely subcortical reflex that is mediated through circuitry in the brainstem (i.e., not under descending control), an idea that has now been overturned on the basis of discoveries

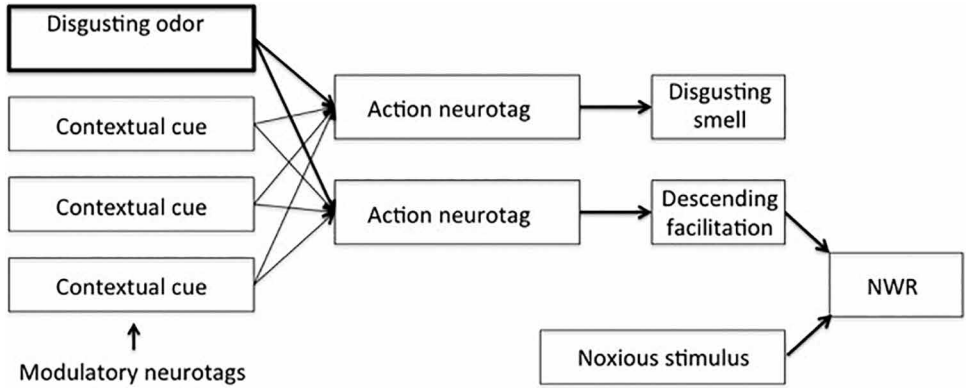

Figure 2 An explanatory model for an increase in the magnitude of the nociceptive withdrawal reflex induced by an unpleasant odor.[32] The disgusting odor activates a modulatory neurotag, which in turn activates two action neurotags—one that produces the disgusting smell and one that increases protection by facilitating the nociceptive withdrawal reflex. Contextual cues here might include the environment of an experimental laboratory, the presence of electrodes on the skin, or the social environment. The neurotag that causes descending facilitation can be conceptualized as an action neurotag because it exerts its effect outside of the brain. NWR = Nociceptive withdrawl reflex.

such as a larger blink response occurring when the stimulated wrist is close to the ipsilateral eye than when it is far from it.[55] Furthermore, the regulation of the HBR magnitude seems to be based on the predicted coordinates of the hand during stimulation. It is therefore under feedforward control.[56] Thus, even this supposedly "basal" motor reflex is under the influence of top-down protective modulation acting in realtime and in a predictive capacity.

The HBR is a poignant, but not isolated, example of cognitively mediated reflex modulation. The auditory startle (eye-blink) response is greater when people smell unpleasant odors than no odor at all[57] and when people view pictures to which they have an aversion.[58] The magnitude of the nociceptive withdrawal reflex is augmented by the presence of an unpleasant odor; these same odors also modulate pain ratings (Fig. 2).[59] Think of how remarkable this is: the presence of a disgusting smell increases the protective reflex response to a noxious stimulus. We can make sense of these intricate modulations according to the model of modulatory neurotags and their effect on action neurotags and competition among neurotags for influence; the odor activates a modulatory neurotag, which in turn influences action neurotags (Fig. 2). Indeed, this same explanatory approach can be replicated for the range of findings that show modulation of reflexes according to cues of threat.

There is much redundancy in the human protective system. This redundancy is evident at a danger detection level, whereby a range of receptors are responsive to stimuli in the noxious range,[22] at a spinal level, causing multiple pathways and modulatory interneurons to contribute to spinal nociceptor discharge,[60] and at a brain level, where multiple feelings and systems can drive protective behavior.[44] For example, fatigue initiates the urge to rest, hunger initiates the urge to eat, thirst initiates the urge to drink, and pain initiates the urge to protect the body part that is hurting.[44] Contemporary theories about how these outputs occur emphasize the importance of higher center modulation. For example, 1. pain (output) is clearly influenced by the nociceptor; however, nociception is modulated at the dorsal root ganglion,[61] dorsal horn of the spinal cord,[62] and the thalamus,[63] and still is not sufficient nor necessary for pain to occur; 2. joint stiffness is equally open to modulation within the nervous system, such that hearing a creaking noise during back mobilizations makes the back *feel* stiffer than it does with no noise or a control "whooshing" noise (Stanton and coworkers under review); and 3. exercise-induced fatigue is highly influenced by, for example, tissue lactate acidosis, but contemporary theories emphasize cortical mechanisms (see the so-called "central governor theory"[64]).

Proprioceptive Contributions to Motor Dysfunction

Movements depend on accurate models of the current position, alignment, and biomechanical characteristics of the body.[65,66] There is a growing accumulation of evidence to suggest that brain-held models of the body, broadly termed "cortical proprioceptive representations" or "proprioceptive neurotags," are inaccurate in those with persistent or recurrent pain. Some examples include people with chronic pain are less able to detect an incongruence between actual head movement and an artificial excursion of the visual field than healthy controls are[67]; tactile acuity at the site of pain is worse in people with low back pain,[68] arthritis-related knee pain,[69] or complex regional pain syndrome (CRPS)[70] than it is in healthy controls (see Catley and colleagues[71] for review and meta-analysis); the ability to return to a given position, or match a given position, is lower in those with a history of neck pain[72] or back pain[73] than it is in healthy controls; people with facial pain find it difficult to differentiate facial postures in oth-

ers.[74] These examples provide clear evidence of disrupted cortical proprioceptive representations. Many cannot be explained by disrupted motor execution or proprioceptive feedback; none can be fully explained by either; all involve disrupted cortical proprioceptive neurotags.

Adaptation

Biological adaptability, or plasticity, has profound implications for the integration of the modern science of pain, protection, and the cortical body matrix. Although the concept of neuroplasticity is not new,[75] it is enjoying a resurgence in popularity. There are many mechanisms by which adaptation occurs within the nervous system, and the broad and guiding principle is one of use-dependent modulation over time. Neurotags that are activated often become more influential and seldom become less influential. The implications of widespread neurotag plasticity in association with protection are potentially profound for dancers because of the fine line that exists between satisfactory and unsatisfactory performance. For example, neurotags that represent cues of impending or present threat will increase their influence over motor output in line with how frequently they are activated; when injury prevents execution of some movements, the neurotags that represent those movements will gradually lose their influence.

That the influence of protective neurotags increases over time, according to known changes in the neuroimmune mechanisms that subserve protection, underlies the need to have a truly biopsychosocial approach to identifying, and eliminating where possible, all cues that provide the dancer with evidence that he or she is in danger and needs protection. This requires the clinician and dancer to look beyond nociception for such cues and, indeed, to every modality of input, for example, cognitive, contextual, or systemic. The cortical body matrix is fundamental in this regard given its "protection meter" function and is captured by modern rehabilitation approaches, including the "Protectometer."[41]

The Protectometer guides the participant through an exploratory process, looking for evidence of danger in the things they say, do, hear, and see; in the people they are with or places they go; and in the variable state of their immune, endocrine, or autonomic systems. Alongside this search for evidence of danger can run a search for evidence of safety—the cues that can be utilized by the participant to activate "safety neurotags" that will compete against protective neurotags in their influence on movement, feeling, or other action neurotags. This approach has immediate relevance for dance, and the interested reader can refer to other accounts for more information (e.g., Moseley and Butler, 2015[31]; Moseley and Butler, 2015[41]).

Decreased influence of performance neurotags over time underlies the integration of "neurotag rehabilitation" after acute injury when movement is not possible, overly compromised by danger cues such as nociception or predicted injury, or considered deleterious for tissue healing. Dancers are well versed in the use of imagery to enhance their performance (i.e., "performance" neurotags), and we would predict that the same approach could be used to minimize the loss of precision and influence of performance neurotags during such times as actual dance is not possible. Our prediction is that if movement rehabilitation could be commenced *prior* to movement, the cortical influence of performance-related neurotags would be maintained, which may reduce risk of further injury or re-injury and hasten return to performance. Moreover, such "neurotag rehabilitation" would minimize isolation and time away from company activities. In an enterprise where injury can be debilitating socially, physically, and professionally, maximizing rehabilitation outcomes would seem vital. That imagery still plays a potential role in training even when a dancer is unable to execute movements is well recognized,[19] but one component that seems not to have been considered is that, by focussing only on action neurotags—or the outputs themselves—disruption of modulatory neurotags remains in place. That is, the dancer may be practicing, via imagery, problematic outputs. This is where the framework we are interested in here may add something new to dance rehabilitation by also considering modulatory neurotags.

Maintaining Modulatory Neurotags

Spatial and tactile neurotags are potentially very influential over the action neurotags of both movement and pain. Indeed, the maintenance of modulatory neurotags that represent, for example, the space around the body, the location of stimuli within or on the body, and movement or task preparation, is now gaining some interest in the rehabilitation field.[30,76] Furthermore, that disrupting some of these modulatory neurotags clearly impacts both movements and bodily feelings is now supported by many studies.[8,38,77-85] The most studied aspects of this new field are movement preparation and touch.

Assessment of Movement Preparation Neurotags

Recognition of a pictured body part as belonging to, or facing, one side of the body or the other requires the participant to prepare to move his or her own body to match the picture.[86,87] Importantly, once participants have gained some proficiency at this task (usually requiring about 40 trials), they stop activating action neurotags associated with movement execution.[88] Functional imaging data uphold this position; although movement execution and imagined movements involve activation of primary motor cortex, left and right judgments do not.[89,90] That is, the modulatory neurotags are activated but the action neurotag, or primary output of movement, is not. Critical here is the distinction between implicit motor imagery, most commonly involving left and right judgements of body parts in those who are practiced at the task, and explicit mo-

tor imagery—imagined movements or left and right judgments in those who are not yet proficient at the task.

Performance in implicit motor imagery can be measured in two ways: the time that it takes for someone to complete the task (the response time, RT) and the accuracy of their performance (what proportion of responses are correct, as a percentage). Side-specific deficits in RT are common in people with chronic or recurrent pain, for example, hand pain (specifically CRPS[91-93] and phantom limb pain,[94] neck pain,[72] back pain,[95,96] knee pain,[8] and facial pain[74]). Side-specific RT deficits can be interpreted as reflecting unequal influence of the modulatory neurotags that represent the affected and unaffected limbs or the affected and unaffected sides of space. That is, when an ambiguous image is presented, the initial implicit judgment is biased toward the healthy limb. In this instance, error is detected during the second stage of the task, preparing to adopt the position, which causes a delay while the initial judgment is remade.[88]

Deficits in accuracy are less well understood than deficits in RT. The prevailing theory is that decreased precision of modulatory neurotags that subserve proprioception underlie the deficit.[95] There are interesting findings that cast doubt over this interpretation; for example, regular yoga practitioners are no more accurate than healthy age and gender matched controls,[97] nor are children who regularly engage in sport and musical instruments,[98] and people who suffer from dizziness are no worse than matched controls who do not.[99] There is now a large pool of normative data for the most common motor imagery tools, and recommendations exist for identifying a deficit in RT or accuracy,[88] and research into underlying mechanisms is ongoing.

Assessment of Touch-Related Neurotags

Tactile acuity is the ability to locate accurately a stimulus on the skin's surface. The most commonly used assessment is the two point discrimination threshold, i.e., the minimal separation at which two stimuli will evoke the perception of two stimuli not one. Aβ neurones with specialized receptors transform mechanical deformation of the skin and transmit that signal along the spinothalamic tract to the thalamus and primary sensory cortex (see Gallace and Spence[100] for a review of tactile physiology). Many chronic pain disorders are associated with anatomically confined areas of poor tactile acuity, but the deficits can seldom be explained by poor detection of the stimulus or transmission of the signal.[101] The prevailing interpretation, then, is a loss of precision at a cortical level, i.e., reduced precision of a modulatory neurotag that represents the surface of the body. Changes in cortical maps established by tactile-evoked responses in the primary sensory cortex lend weight to this hypothesis.[102,103] There are also now normative data for tactile acuity at most bodily locations, and recommendations exist for identifying a deficit in it.[104] This is important for our consideration of dance rehabilitation, because the brain cells that subserve anatomical locations within the neurotags that produce a sense of touch are likely also to subserve those that produce other bodily feelings and upon which movement commands are based.[88]

Training Modulatory Neurotags in Rehabilitation; Implicit and Explicit Motor Imagery

As previously noted, use of imagery is common among dancers.[21] Most imagery strategies can be categorized as facilitative (for example, imagining oneself performing well) or debilitative (for example, imagining oneself falling over while performing).[21] This type of imagery can be considered according to the framework being presented here as analogous to explicit motor imagery. That is, these strategies involve imagining the output of action neurotags. We contend that imagery can extend to modulatory neurotags. One therapeutic strategy that is based on this contention is graded motor imagery (GMI).[105] GMI was developed for the treatment of chronic pathological arm pain in which even imagined movements can evoke pain and swelling.[106,107] GMI is now used widely and in a variety of contexts, including sport, although, critically, strong evidence for its effectiveness is limited to those with pathological arm or leg pain.[108] GMI aims to retrain movement neurotags in a graded fashion by first focussing on modulatory neurotags that influence the action neurotags of movement.[88] This approach is very useful when movement is not possible or compromised, for example, in states of heightened protection, as when explicit motor imagery (imagined movements) triggers protective neurotags. Such a situation has been clearly demonstrated in people with pathological arm pain who perform imagined movements of their painful arm. The task might be described as "facilitative imagery," yet it results in increased pain and swelling.[106,107]

Importantly, there are no data, to our knowledge, that extend this work to dance, but therein lies a key objective of this article: we hope to initiate discussion as to whether, should the clinician encounter a similar problem—that is explicit motor imagery apparently reinforcing protective outputs, including percepts—he or she use implicit motor imagery as a sensible intermediary step. This is relevant to the finding that athletes who normally engage in facilitative imagery do so less when they are injured[19,20]—perhaps because their imagery reflects the outcome of the same competing mix of modulatory neurotags that would be in place should they attempt to actually execute the behavior.

Training Modulatory Neurotags in Rehabilitation—Tactile Maps

Tactile acuity rehabilitation involves training one's ability to locate a tactile stimulus accurately on the body's surface. Such practice has long been commonplace in rehabilitation after peripheral nerve trauma or surgery and was introduced to pathological pain in an elegant study of amputees

over a decade ago.[109] Tactile acuity training has also been associated with pain reduction in people with pathological pain.[110,111] Such training can take many forms, all of which require the participant to rely on tactile input alone to differentiate one stimulus from another. Recommendations for protocols are provided elsewhere.[112] Similar to GMI, tactile acuity training can commence immediately after an injury as long as skin integrity is intact and peripheral sensitization is not present. However, again similar to GMI, there is a paucity of empirical studies applying these approaches to other populations, and although there is no evidence of side effects or risks, we urge the enthusiastic clinician not to over-interpret the data.

Conclusion

Here we have attempted to introduce the dance community to a framework for rehabilitation that incorporates essential elements of three prevailing pain-related concepts: the biopsychosocial model, the cortical body matrix model, and the concept of action and modulatory neurotags that compete for influence. This framework predicts that any credible evidence of danger, represented within the cortical body matrix by modulatory neurotags, has the potential to influence both movement commands and bodily feelings subserved by action neurotags. This framework predicts that neurotags that subserve the location of body parts, spatially and anatomically defined coordinates, contribute to and can disrupt action neurotags. Moreover, the principle of neuroplasticity suggests that when dance is not possible, both modulatory and action neurotags associated with dance performance can be maintained with specific strategies. There are assessments that can highlight a loss of precision in some modulatory neurotags, and there are established strategies that can reverse disruptions and imprecision. Finally, clinical tools, such as the Protectometer,[41] can be used to guide clinical assessment and rehabilitation. This approach is applicable to other high-performance areas,[40] but there are unique contextual considerations for dance. We hope that this review will stimulate fresh research into these ideas and generate vigorous discussion in the field.

References

1. Moseley GL, Parsons TJ, Spence C. Visual distortion of a limb modulates the pain and swelling evoked by movement. Curr Biol. 2008 Nov 25;18(22):R1047-8.
2. Moseley GL, Olthof N, Venema A, et al. Psychologically induced cooling of a specific body part caused by the illusory ownership of an artificial counterpart. Proc Natl Acad Sci U S A. 2008 Sep 2;105(35):13169-73.
3. Barnsley N, McAuley JH, Mohan R, et al. The rubber hand illusion increases histamine reactivity in the real arm. Curr Biol. 2011 Dec 6;21(23):R945-6.
4. Mohan R, Jensen KB, Petkova VI, et al. No pain relief with the rubber hand illusion. PLoS One. 2012;7(12):e52400.
5. Moseley GL, Gallace A, Spence C. Space-based, but not arm-based, shift in tactile processing in complex regional pain syndrome and its relationship to cooling of the affected limb. Brain. 2009 Nov;132(Pt 11):3142-51.
6. Moseley GL, Gallace A, Iannetti GD. Spatially defined modulation of skin temperature and hand ownership of both hands in patients with unilateral complex regional pain syndrome. Brain. 2012 Dec;135(Pt 12):3676-86.
7. Reid E, Wallwork SB, Harvie D, et al. A new kind of spatial inattention associated with chronic limb pain? Ann Neurol. 2016 Apr;79(4):701-4.
8. Stanton TR, Lin CW, Smeets RJ, et al. Spatially-defined disruption of motor imagery performance in people with osteoarthritis. Rheumatology (Oxford). 2012 Aug;51(8):1455-64.
9. Gallace A, Soravia G, Cattaneo Z, et al. Temporary interference over the posterior parietal cortices disrupts thermoregulatory control in humans. PLoS One. 2014 Mar 12;9(3):e88209.
10. Moseley GL, Gallace A, Spence C. Bodily illusions in health and disease: physiological and clinical perspectives and the concept of a cortical 'body matrix.' Neurosci Biobehav Rev. 2012 Jan;36(1):34-46.
11. Preston C, Newport R. Analgesic effects of multisensory illusions in osteoarthritis. Rheumatology (Oxford). 2011 Dec;50(12):2314-5.
12. Boesch E, Bellan V, Moseley GL, Stanton TR. The effect of bodily illusions on clinical pain: a systematic review and meta-analysis. Pain. 2016 Mar;157(3):516-29.
13. Woolf CJ. What to call the amplification of nociceptive signals in the central nervous system that contribute to widespread pain? Pain. 2014 Oct;155(10):1911-2.
14. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain. 2011 Mar;152(3 Suppl):S2-15.
15. Moseley GL, Hodges PW. Chronic pain and motor control. *In*: Jull G, Boyling J: *Grieves Modern Manual Therapy of the Vertebral Column* (3rd ed). Edinburgh: Churchill-Livingstone, 2004, pp. 215-222.
16. MacDonald D, Moseley GL, Hodges PW. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain. 2009 Apr;142(3):183-8.
17. Moseley GL, Hodges PW. Reduced variability of postural strategy prevents normalization of motor changes induced by back pain: a risk factor for chronic trouble? Behav Neurosci. 2006 Apr;120(2):474-6.
18. Moseley GL, Nicholas MK, Hodges PW. Does anticipation of back pain predispose to back trouble? Brain. 2004 Oct;127(Pt 10):2339-47.
19. Milne M, Hall C, Forwell L. Self-efficacy, imagery use, and adherence to rehabilitation by injured athletes. J Sport Rehabil. 2005 May;14(2):150-67.
20. Sordoni C, Hall C, Forwell L. The use of imagery in athletic injury rehabilitation and its relationship to self-efficacy. Physiother Canada. 2002 Summer;54:177-85.
21. Nordin-Bates SM, Walker IJ, Baker J, et al. Injury, imagery, and self-esteem in dance healthy minds in injured bodies? J Dance Med Sci. 2011 Jun;15(2):76-85.
22. Woolf CJ, Ma Q. Nociceptors—noxious stimulus detectors. Neuron. 2007 Aug 2;55(3):353-64.
23. Wall P, McMahon S. The relation-

ship of perceived pain to afferent nerve impulses. Trends Neurosci. 1986;9(6):254-5.
24. Butler D, Moseley GL. *Explain Pain* (2nd ed). Adelaide, Australia: Noigroup Publications, 2013.
25. Moseley GL. Reconceptualising pain according to its underlying biology. Phys Ther Rev. 2007 Sep;12(3):169-78.
26. Madden VJ, Moseley GL. Do clinicians think that pain can be a classically conditioned response to a non-noxious stimulus? Man Ther. 2016 Apr;22:165-73.
27. Moseley L. Unraveling the barriers to reconceptualization of the problem in chronic pain: the actual and perceived ability of patients and health professionals to understand the neurophysiology. J Pain. 2003 May;4(4):184-9.
28. Hagbarth KE. Spinal withdrawal reflexes in the human lower limbs. J Neurol Neurosurg Psychiatry. 1960 Aug; 23(3):222-7.
29. Le Bars D, Gozariu M, Cadden SW. Animal models of nociception. Pharmacol Rev. 2001 Dec;53(4):597-652.
30. Moseley GL, Flor H. Targeting cortical representations in the treatment of chronic pain: a review. Neurorehabil Neural Repair. 2012 Jul-Aug;26(6):646-52.
31. Moseley GL, Butler DS. Fifteen years of explaining pain—the past, present and future. J Pain. 2015 Sep;16(9):807-13.
32. Thacker MA, Moseley GL. First-person neuroscience and the understanding of pain. Med J Aust. 2012 Apr 2;196(6):410-1.
33. Tabor A, O'Daly O, Gregory RW, Jacobs C, et al. Perceptual inference in chronic pain: an investigation into the economy of action hypothesis. Clin J Pain. 2016 Jul;32(7):588-93.
34. Foster M. *Lectures on the History of Physiology During the Sixteenth, Seventeenth and EIghteenth Centuries.* Cambridge: Cambridge University Press, 1901.
35. Tucker K, Butler J, Graven-Nielsen T, et al. Motor unit recruitment strategies are altered during deep-tissue pain. J Neurosci. 2009 Sep 2;29(35):10820-6.
36. Hodges PW, Moseley GL, Gabrielsson A, Gandevia SC. Experimental muscle pain changes feedforward postural responses of the trunk muscles. Exp Brain Res. 2003 Jul;151(2):262-71.
37. Wolpert DM, Ghahramani Z. Computational principles of movement neuroscience. Nat Neurosci. 2000 Nov;3 Suppl:1212-7.
38. Lotze M, Moseley GL. Theoretical considerations for chronic pain rehabilitation. Phys Ther. 2015 Sep;95(9):1316-20.
39. Melzack R. Phantom limbs and the concept of a neuromatrix. Trends Neurosci. 1990 Mar;13(3):88-92.
40. Wallwork SB, Bellan V, Catley MJ, Moseley GL. Neural representations and the cortical body matrix: implications for sports medicine and future directions. Br J Sports Med, 2016;50(16):990-6.
41. Moseley G, Butler D. *The Explain Pain Handbook: Protectometer.* Adelaide, Australia: Noigroup Publications, 2015.
42. Moseley GL. Unravelling the barriers to reconceptualisation of the problem in chronic pain: the actual and perceived ability of patients and health professionals to understand the neurophysiology. J Pain. 2003 May;4(4):184-9.
43. Moseley GL. Teaching people about pain: why do we keep beating around the bush? Pain Manag. 2012 Jan;2(1):1-3.
44. Williams MT, Gerlach Y, Moseley L. The 'survival perceptions': time to put some Bacon on our plates? J Physiother. 2012;58(2):73-5.
45. Dubner R. Neurophysiology of pain. Dent Clin North Am. 1978 Jan;22(1):11-30.
46. Tabor A, Catley MJ, Gandevia S, et al. Perceptual bias in pain: a switch looks closer when it will relieve pain than when it won't. Pain. 2013 Oct;154(10):1961-5.
47. Tabor A, Catley MJ, Gandevia SC, et al. The close proximity of threat: altered distance perception in the anticipation of pain. Front Psychol. 2015 May 13;6:626.
48. Hodges PW, Moseley GL. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. J Electromyogr Kinesiol. 2003 Aug;13(4):361-70.
49. Moseley GL, Nicholas MK, Hodges PW. Pain differs from non-painful attention-demanding or stressful tasks in its effect on postural control patterns of trunk muscles. Exp Brain Res. 2004 May;156(1):64-71.
50. Moseley GL, Nicholas MK, Hodges PW. Does anticipation of back pain predispose to back trouble? Brain. 2004 Oct;127(Pt 10):2339-47.
51. Moseley GL, Hodges PW. Reduced variability of postural strategy prevents normalization of motor changes induced by back pain: a risk factor for chronic trouble? Behav Neurosci. 2006 Apr;120(2):474-6.
52. Hug F, Hodges PW, van den Hoorn W, Tucker K. Between-muscle differences in the adaptation to experimental pain. J Appl Physiol (1985). 2014 Nov 15;117(10):1132-40.
53. Hug F, Hodges PW, Tucker K. Task dependency of motor adaptations to an acute noxious stimulation. J Neurophysiol. 2014 Jun 1;111(11):2298-306.
54. Moseley GL, Hodges PW. Are the changes in postural control associated with low back pain caused by pain interference? Clin J Pain. 2005 Jul-Aug;21(4):323-9.
55. Sambo CF, Liang M, Cruccu G, Iannetti GD. Defensive peripersonal space: the blink reflex evoked by hand stimulation is increased when the hand is near the face. J Neurophysiol. 2012 Feb;107(3):880-9.
56. Wallwork SB, Talbot K, Camfferman D, et al. The blink reflex magnitude is continuously adjusted according to both current and predicted stimulus position with respect to the face. Cortex. 2016 Aug;81:168-75.
57. Ehrlichman H, Brown S, Zhu J, Warrenburg S. Startle reflex modulation during exposure to pleasant and unpleasant odors. Psychophysiology. 1995 Mar;32(2):150-4.
58. Bradley MM, Cuthbert BN, Lang PJ. Pictures as prepulse: attention and emotion in startle modification. Psychophysiology. 1993 Sep;30(5):541-5.
58. Bartolo M, Serrao M, Gamgebeli Z, et al. Modulation of the human nociceptive flexion reflex by pleasant and unpleasant odors. Pain. 2013 Oct;154(10):2054-9.
60. Moore KA, Baba H, Woolf CJ. Synaptic transmission and plasticity in the superficial dorsal horn. Prog Brain Res. 2000;129:63-80.
61. Kusano K, Gainer H. Modulation of voltage-activated Ca currents by pain-inducing agents in a dorsal root ganglion neuronal line, F-11. J Neurosci Res. 1993 Feb 1;34(2):158-69.
62. Woolf CJ, Salter M. Plasticity and

pain: the role of the dorsal horn. *In:* McMahon SB, Koltzenburg M: *Melzack and Wall's Textbook of Pain* (5th ed). London: Elsevier, 2006, pp. 91-107.
63. Duncan GH, Kupers RC, Marchand S, et al. Stimulation of human thalamus for pain relief: possible modulatory circuits revealed by positron emission tomography. J Neurophysiol. 1998 Dec;80(6):326-30.
64. Noakes TD. Fatigue is a brain-derived emotion that regulates the exercise behavior to ensure the protection of whole body homeostasis. Front Physiol. 2012 Apr 11;3:82.
65. Gandevia S, Kinesthesia: roles for afferent signals and motor commands. *In:* Rothwell L, Shepherd JT: *Handbook of Physiology, Section 12, Exercise: Regulation and Integration of Multiple Systems.* New York: Oxford University Press, 1996, pp. 128-172.
66. Gandevia SC, Refshauge KM, Collins DF. Proprioception: peripheral inputs and perceptual interactions. *In:* Gandevia SC, Proske U, Stuart DG: *Sensorimotor Control of Movement and Posture.* New York: Kluwer Academic/Plenum Publishers, 2002, pp. 61-68.
66. Harvie DS, Hillier S, Madden VJ, et al. Neck pain and proprioception revisited using the Proprioception Incongruence Detection Test. Phys Ther. 2016 May;96(5):671-8.
68. Luomajoki H, Moseley GL. Tactile acuity and lumbopelvic motor control in patients with back pain and healthy controls. Br J Sports Med. 2011 Apr;45(4):437-40.
69. Stanton TR, Lin CW, Bray H, et al. Tactile acuity is disrupted in osteoarthritis but is unrelated to disruptions in motor imagery performance. Rheumatology (Oxford). 2013 Aug;52(8):1509-19.
70. Maihöfner C, Neundörfer B, Birklein F, Handwerker HO. Mislocalization of tactile stimulation in patients with complex regional pain syndrome. J Neurol. 2006 Jun;253(6):772-9.
71. Catley MJ, O'Connell NE, Berryman C, et al. Is tactile acuity altered in people with chronic pain? A systematic review and meta-analysis. J Pain. 2014 Oct;15(10):985-1000.
72. Stanton TR, Leake HB, Chalmers KJ, Moseley GL. Evidence of impaired proprioception in chronic, idiopathic neck pain: systematic review and meta-analysis. Phys Ther. 2016 Jun;96(6):876-87.
73. O'Sullivan K, Verschueren S, Van Hoof W, et al. Lumbar repositioning error in sitting: healthy controls versus people with sitting-related non-specific chronic low back pain (flexion pattern). Man Ther. 2013 Dec;18(6):526-32.
74. von Piekartz H, Wallwork SB, Mohr G, et al. People with chronic facial pain perform worse than controls at a facial emotion recognition task, but it is not all about the emotion. J Oral Rehabil. 2015 Apr;42(4):243-50.
75. Lindley EH. A study of puzzles with special reference to the psychology of mental adaptation. Am J Psychol. 1897;8(4):431-93.
76. Wand BM, Parkitny L, O'Connell NE, et al. Cortical changes in chronic low back pain: current state of the art and implications for clinical practice. Man Ther. 2011 Feb;16(1):15-20.
77. McCormick K, Zalucki N, Hudson M, Moseley GL. Faulty proprioceptive information disrupts motor imagery: an experimental study. Aust J Physiother. 2007;53(1):41-5.
78. Wand BM, Abbaszadeh S, Smith AJ, et al. Acupuncture applied as a sensory discrimination training tool decreases movement-related pain in patients with chronic low back pain more than acupuncture alone: a randomised cross-over experiment. Br J Sports Med. 2013 Nov;47(17):1085-9.
79. Wand BM, Stephens SE, Mangharam EI, et al. Illusory touch temporarily improves sensation in areas of chronic numbness: a brief communication. Neurorehabil Neural Repair. 2014 Oct;28(8):797-9.
80. Wand BM, Szpak L, George PJ, et al. Moving in an environment of induced sensorimotor incongruence does not influence pain sensitivity in healthy volunteers: a randomised within-subject experiment. PLoS One. 2014 Apr;9(4):e93701.
81. Butler D, Moseley GL. *Explain Pain.* Adelaide, Australia: Noigroup Publications, 2003.
82. Gallace A, Torta DM, Moseley GL, Iannetti GD. The analgesic effect of crossing the arms. Pain. 2011 Jun;152(6):1418-23.
83. Gilpin HR, Moseley GL, Stanton TR, Newport R. Evidence for distorted mental representation of the hand in osteoarthritis. Rheumatology (Oxford). 2015 Apr;54(4):678-82.
84. Luomajoki H, Moseley GL. Tactile acuity and lumbopelvic motor control in patients with back pain and healthy controls. Br J Sports Med. 2011 Apr;45(5):437-40.
85. Reid E, Wallwork SB, Harvie D, et al. A new kind of spatial inattention associated with chronic limb pain? Ann Neurol. 2016 Apr;79(4):701-4.
86. Parsons LM, Fox PT. The neural basis of implicit movements used in recognising hand shape. Cogn Neuropsychol. 1998;15(6-8):583-615.
87. Parsons LM. Integrating cognitive psychology, neurology and neuroimaging. Acta Psychol (Amst). 2001 Apr;107(1-3):155-81.
88. Moseley GL, Butler DS, Beames TB, Giles TJ. *The Graded Motor Imagery Handbook.* Adelaide, Australia: Noigroup Publications, 2012.
89. Moseley GL, Schweinhardt P, Wise R, et al. Virtual, imagined and mirror movements - a novel approach to complex regional pain syndrome (CRPS1). European Federation of IASP Chapters Triennial Conference. Prague, Czech Republic: 2003, Abstract 686T, p. 422.
90. Walz AD, Usichenko T, Moseley GL, Lotze M. Graded motor imagery and the impact on pain processing in a case of CRPS. Clin J Pain. 2013 Mar;29(3):276-9.
91. Coslett HB, Saffran EM, Schwoebel J. Knowledge of the human body: a distinct semantic domain. Neurology. 2002 Aug;59(3):357-63.
92. Schwoebel J, Friedman R, Duda N, Coslett HB. Pain and the body schema: evidence for peripheral effects on mental representations of movement. Brain. 2001 Oct;124(Pt 10):2098-104.
93. Moseley GL. Why do people with complex regional pain syndrome take longer to recognize their affected hand? Neurology. 2004 Jun;62(12):2182-6.
94. Nico D, Daprati E, Rigal F, et al. Left and right hand recognition in upper limb amputees. Brain. 2004 Jan;127(Pt 1):120-32.
95. Bray H, Moseley GL. Disrupted working body schema of the trunk in people with back pain. Br J Sports Med. 2011 Mar;45(3):168-73.
96. Bowering KJ, Butler DS, Fulton IJ, Moseley GL. Motor imagery in people with a history of back pain, current back pain, both, or neither. Clin J Pain. 2014 Dec;30(12):1070-5.

97. Wallwork SB, Butler DS, Wilson DJ, Moseley GL. Are people who do yoga any better at a motor imagery task than those who do not? Br J Sports Med. 2015 Jan;49(2):123-7.
98. Dey A, Barnsley N, Mohan R, et al. Are children who play a sport or a musical instrument better at motor imagery than children who do not? Br J Sports Med. 2012 Oct;46(13):923-6.
99. Wallwork SB, Butler DS, Moseley GL. Dizzy people perform no worse at a motor imagery task requiring whole body mental rotation; a case-control comparison. Front Hum Neurosci. 2013 Jun 6;7:258.
100. Gallace A, Spence C. *In Touch with the Future: The Sense of Touch from Cognitive Neuroscience to Virtual Reality.* Oxford: Oxford University Press, 2014.
101. Catley MJ, O'Connell NE, Berryman C, et al. Is tactile acuity altered in people with chronic pain? a systematic review and meta-analysis. J Pain. 2014 Oct;15(10):985-1000.
102. Di Pietro F, McAuley JH, Parkitny L, et al. Primary somatosensory cortex function in complex regional pain syndrome: a systematic review and meta-analysis. J Pain. 2013 Oct;14(10):1001-18.
103. Di Pietro F, Stanton TR, Moseley GL, et al. Interhemispheric somatosensory differences in chronic pain reflect abnormality of the healthy side. Hum Brain Mapp. 2015 Feb;36(2):508-18.
104. Weinstein S. Tactile sensitivity of the phalanges. Percept Mot Skills. 1962 Jun;14:351-4.
105. Moseley GL. Graded motor imagery is effective for long-standing complex regional pain syndrome: a randomised controlled trial. Pain. 2004 Mar;108(1-2):192-8.
106. Moseley GL. Imagined movements cause pain and swelling in a patient with complex regional pain syndrome. Neurology. 2004 May 11;62(9):1644.
107. Moseley GL, Zalucki N, Birklein F, et al. Thinking about movement hurts: the effect of motor imagery on pain and swelling in people with chronic arm pain. Arthritis Rheum. 2008 May 15;59(5):623-31.
108. Bowering KJ, O'Connell NE, Tabor A, et al. The effects of graded motor imagery and its components on chronic pain: a systematic review and meta-analysis. J Pain. 2013 Jan;14(1):3-13.
109. Flor H, Denke C, Schaefer M, Grüsser S. Effect of sensory discrimination training on cortical reorganisation and phantom limb pain. Lancet. 2001 Jun 2;357(9270):1763-4.
110. Moseley GL, Wiech K. The effect of tactile discrimination training is enhanced when patients watch the reflected image of their unaffected limb during training. Pain. 2009 Aug;144(3):314-9.
111. Moseley GL, Zalucki NM, Wiech K. Tactile discrimination, but not tactile stimulation alone, reduces chronic limb pain. Pain. 2008 Jul 31;137(3):600-8.
112. Moseley G. Rehabilitation of complex regional pain syndrome. *In:* Mogil J: *World Congress on Pain.* Montreal, Canada: IASP Press, 2010, pp. 125-136.