Masterclass

# A neuroscience approach to managing athletes with low back pain

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A R T I C L E   I N F O

A B S T R A C T

Low back pain (LBP) is a common complaint within the athletic population and is commonly managed through a biomedical approach. The injured or damaged structure causing the LBP is identified and treated, and complete recovery from the episode is expected. Clinical experience shows us that often, athletes with LBP will not recover from their episode and may continue their sports participation despite persistent pain, or they may limit participation. Recent neuroscience research into the biology of pain suggests that clinicians involved in the management of athletes with LBP should embrace a biopsychosocial approach by engaging the brain and nervous system. This manuscript provides an overview of such a biopsychosocial approach, and presents information on the neurobiology of the athlete's pain experience.

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## 1. Introduction

Much has been written within the sports medicine literature on the prevalence and management of low back pain (LBP) in the athletic population. The typical research report will indicate that athletes have high rates of LBP (Bono, 2004; Kraft, 2002; Trainor & Wiesel, 2002); discussions will tend to focus on pathology involving anatomy and biomechanics (such as instability/spondylolisthesis) as the most probable source of pain and disability (Hides, Stanton, McMahon, Sims, & Richardson, 2008; Lundin, Hellstrom, Nilsson, & Sward, 2001; Standaert, Herring, & Pratt, 2004; Takemitsu, El Rassi, Woratanarat, & Shah, 2006) and treatment approaches will inevitably focus on correcting the pathoanatomy and biomechanics through spinal stabilization, either surgical or therapeutic (d'Hemecourt, Gerbino, & Micheli, 2000; George & Delitto, 2002; Hides et al., 2008; Nadler, Malanga, Bartoli, et al., 2002; Richardson, Hodges, & Hides, 2004). Such descriptions of LBP affecting athletes are a classic example of the biomedical model, which focuses heavily on anatomy, pathoanatomy and biomechanics.

The traditional biomedical model of sports medicine suggests that every disease process (dysfunction) can be explained in terms of an underlying deviation from normal function such as

a pathogen or injury. The model suggests that pathology and symptoms are correlated such that a greater expression of symptoms in the athlete would indicate greater underlying pathology (Fig. 1A). This model further proposes that a simple correction of the underlying pathology with a treatment (for example injection, surgery, manipulation or exercise) will result in elimination of the symptoms and subsequent restoration of normal function in the athlete (Fig. 1B). Clinical experience and epidemiological data on LBP often tells us otherwise, with many athletes demonstrating physical and diagnostic signs that they have recovered from injury and yet they will continue to experience symptoms/pain (Fig. 2C) (Iwamoto, Takeda, & Wakano, 2004). Additionally, it has been well demonstrated that many people, including athletes often have significant tissue pathology (arthritis of the spine, bulging discs, bone spurs, etc.), yet experience little to no pain (Fig. 2D) (Alyas, Turner, & Connell, 2007; Waris, Eskelin, Hermunen, Kiviluoto, & Paajanen, 2007)

The time has come for therapists who work with athletes and LBP to take on the more comprehensive biopsychosocial model (Foster & Delitto, 2011; Linton & Shaw, 2011). The biopsychosocial model encompasses more than just the biological factors (anatomy, physiology and pathoanatomy) in human functioning, by addressing the psychological (thoughts, emotions and behaviors), and social (work and playing status, culture and religion) factors which are known to play a significant role in athletic functioning in the context of injury or illness. A true biopsychosocial model includes a greater understanding of how the nervous system processes injury, disease, pain, threat and emotions.

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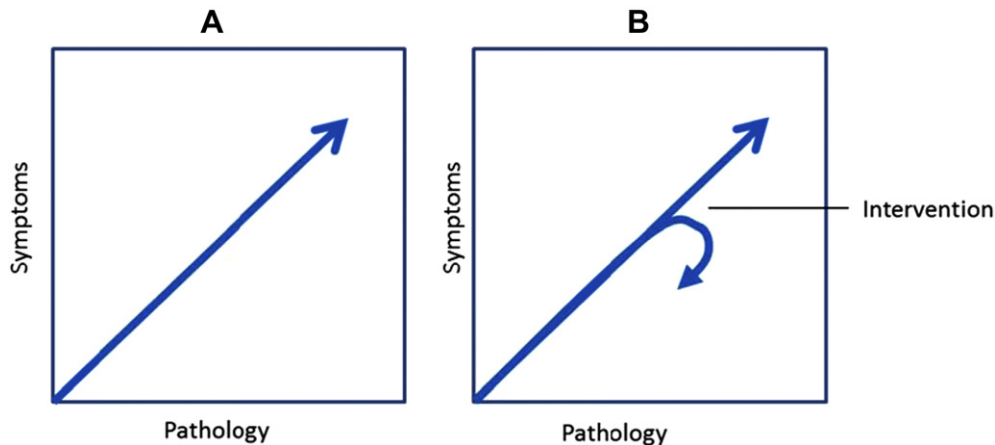   E-mail address: louie.puentedura@unlv.edu (E.J. Puentedura).

**Fig. 1.** The prevailing biomedical model of low back pain (LBP). A. Proposed correlation between symptoms and pathology. B. Proposed consequence of treatment intervention in the biomedical model. Adapted from Haldeman (1990).

## 2. A biopsychosocial approach

Opinions vary as to what constitutes a true biopsychosocial approach (Jull & Sterling, 2009; Weiner, 2008) and it could be argued that the list would vary, depending on each athlete and his/her specific injury. It is however proposed that a biopsychosocial approach include aspects of anatomy, pathoanatomy, biomechanics, brain representation of injury, the nervous system's processing of information, psychological issues associated with pain, evolutionary biology and fear avoidance (Fig. 3).

A clinician aiming to practice in a true biopsychosocial approach would need to be familiar with each of the proposed components of this approach and be able to incorporate this into clinical practice. The biopsychosocial approach includes knowledge of:

### 2.1. Anatomy

Knowledge of anatomy is essential for physical therapy, orthopedics and sports medicine. Anatomy has a significant value in developing a grounded knowledge of the human body and also has a potential for explaining pain to athletes in the acute stages of an injury. Clinicians will often use anatomy to explain pain, using a plastic spine model to show the delicate anatomical structures forming the intervertebral foramen, indicating a potential lack of space around a nerve root. In acute pain states, knowledge of anatomy may help an athlete understand why he/she may be experiencing pain (i.e., encroachment of the intervertebral nerve), but this model has a limited ability to explain persistent pain, widespread pain or pain driven by fear and emotion (Jull & Sterling, 2009; Weiner, 2008).

### 2.2. Biomechanics

Orthopedic and sports medicine therapists often excel in this area (Childs et al., 2007, 2005). The ability to analyze movement and determine normal movement patterns versus abnormal patterns are essential to therapy, especially in fine-tuned athletes (Louw, Manilall, & Grimmer, 2008; Standaert et al., 2004). It could be argued that minor biomechanical alterations will have profound implications for high level athletes, compared to activities of daily living (Nadler, Malanga, Bartoli, et al., 2002; Nadler, Malanga, Feinberg, et al., 2002). A therapist examining low back/hip pain in a marathon runner would require the ability to analyze the runner's gait, with a chance of finding slight biomechanical abnormalities in the kinetic chain, which may or may not be associated with the development of LBP (Bischof, Abbey, Chuckpaiwong, Nunley, & Queen, 2010; Cibulka, 1999; Geraci & Brown, 2005; Harrison, Harrison, & Troyanovich, 1997). A shortcoming of the biomechanical model is, once again, that it has limitations in explaining persistent and widespread pain, especially if the biomechanical abnormality has been corrected.
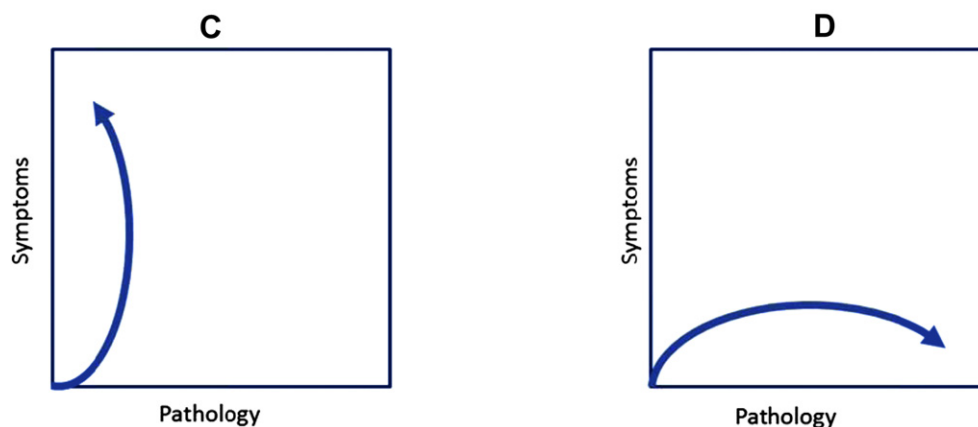


**Fig. 2.** The differing clinical expressions of low back pain (LBP). C. Patients may present with many symptoms, yet have little to no demonstrable injury/pathology. D. Patients may present with many observable pathologies, yet experience little to no symptoms/pain. Adapted from Haldeman (1990).
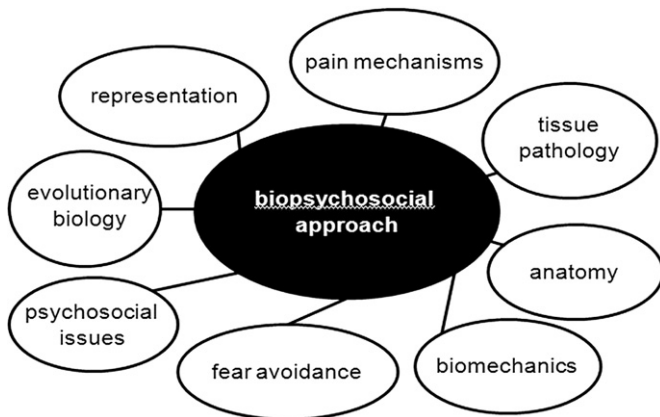
**Fig. 3.** Conceptual model of a comprehensive biopsychosocial model. From Butler (2011) − personal communication.

### 2.3. Tissue pathology

The tissue pathology model is an extension of the anatomy model, comparing "normal/healthy" tissue to "injured" tissue. The tissue pathology model is valuable in explaining acute pain states and is closely linked to the predicted stages of healing − injury, inflammation, regeneration and remodeling. The tissue pathology model his associated with specific timed intervals based on the current knowledge of tissue healing (Cook, Khan, & Purdam, 2002; Gross, Fetto, & Rosen, 1996; Vernon-Roberts, Moore, & Fraser, 2007). For example, a grade 1 muscle strain would most likely take 2–3 weeks to heal, while magnetic resonance imaging (MRI) studies show bulging discs reabsorb and clear over time (Autio et al., 2006; Masui et al., 2005). Tissues heal, and if pain is seen to persist beyond the predicted stages of healing, clinicians utilizing only this model may struggle to explain pain to the athlete.

### 2.4. Pain mechanisms

The pain mechanism model may be a good first step out of a traditional biomedical model by not only acknowledging the aforementioned three models of anatomy, biomechanics and tissue injury, but utilizing a larger view of the pain process (Butler, 2000; Moseley, 2007) (Fig. 4). The pain mechanism model, proposed by Gifford in 1998, provides an increased understanding of the nervous system's processing of the athlete and his/her LBP (Gifford, 1998). The pain mechanism model can be divided into three overlapping processes of input, processing and output.

#### 2.4.1. Input dominant mechanisms

Injuries in athletes are common (Bono, 2004; Louw et al., 2008) and athletes may experience pain from tissue injuries. Based on traditional training, tissue injuries and their healing stages are well understood and predictable. As previously stated, this is a dominant model in orthopedics and sports medicine and needs no further discussion. Tissue injuries however, occur in various environments, which may alter the perception of the injury or threat the injury represents. Environmental issues are known to alter pain (Bayer, Baer, & Early, 1991; Moseley & Arntz, 2007). The study by Moseley and Arntz showed that manipulation of visual input altered pain responses. Patients presented with red rods contacting the skin, which is associated with heat and potential increased tissue damage evoked more pain versus blue colored rods associated with cold, non-damaging input, even though both colored rods were the same temperature (Moseley & Arntz, 2007). Similarly, Bayer showed that patients attached to a sham stimulator reported higher pain ratings when the stimulator was turned higher, even
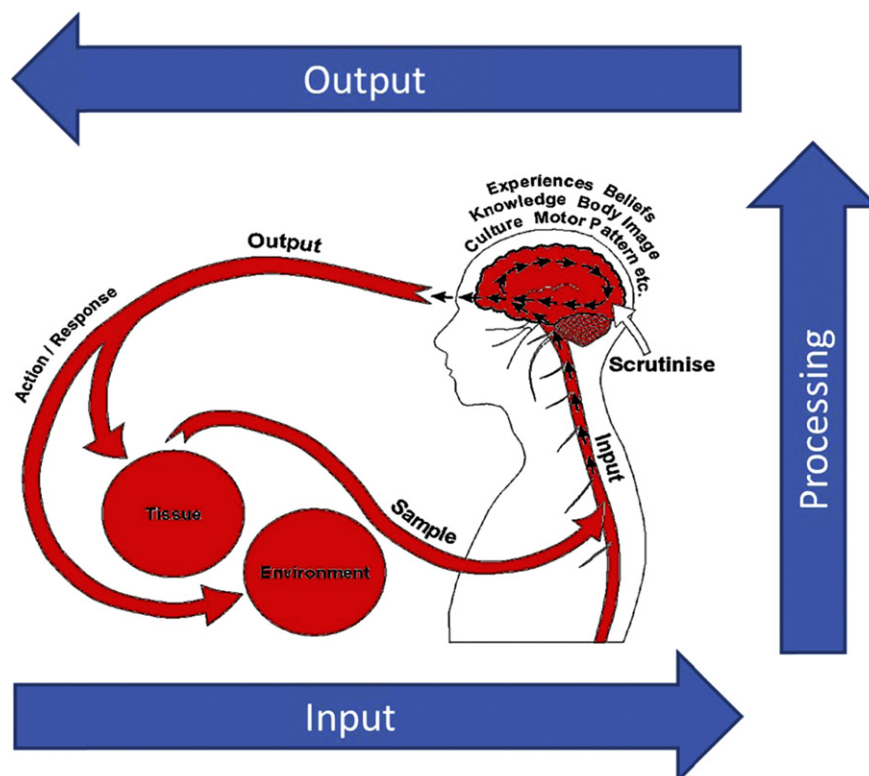


**Fig. 4.** A pain mechanism model. From Gifford (1998).

when the patient was not connected to the stimulator rods (Bayer et al., 1991). Various studies have shown that injury in stressful environments is linked to poorer outcomes (Holm, Carroll, Cassidy, Skillgate, & Ahlbom, 2007; Marras, Ferguson, Burr, Schabo, & Maronitis, 2007; Simotas & Shen, 2005). Given the competitive nature of sports, it is important that therapists realize that environmental issues may modulate pain. A skilled clinician should not only evaluate the injury, but have a broader understanding and appreciation of the environment the LBP was acquired in, including playing status, importance of a game, place on the team roster, etc.

Following tissue injury and environmental issues, a third process related to input is the delivery of the information from the tissue to the spinal cord and brain via electrochemical communication. The peripheral nervous system and spinal cord are instrumental in delivering the message of impending threat to the brain. Nociceptive input, mainly via C-fibers and A-delta fibers from the affected area (low back) are sent via the dorsal horn of the spinal cord to the brain for further processing (Woolf & Salter, 2005). With injury, the nervous system in and around the affected area becomes hyper excitable to relay the impending threat to the central nervous system (CNS). This process is referred to as peripheral nerve sensitization (Butler, 2000; Gifford & Butler, 1997; Malick & Burstein, 2000; Merskey & Bogduk, 1994). As time goes by and the athlete heals, the peripheral nervous system in and around the affected area should respond accordingly, by decreasing its sensitivity. The longer pain persists, however, the nervous system is less likely to decrease its sensitivity, and may even increase its sensitivity (Cook, Woolf, Wall, & McMahon, 1987; Woolf, 1994; Woolf & Doubell, 1994). Clinically, these patients will have heightened responses to stimuli, including palpation of the peripheral nervous system (Walsh & Hall, 2009b) and active and passive neurodynamic tests such as straight leg raise (SLR) and slump (Boyd, Wanek, Gray, & Topp, 2009; Coppieters, Alshami, Babri, et al., 2006; Coppieters, Alshami, & Hodges, 2006; Walsh & Hall, 2009a). A good example of heightened response to neurodynamic testing and tying it into environmental cues (Section 2.4.1) is study by Coppieters et al. In this study, subjects with upper extremity pain were either told the ensuing neurodynamic test was a test of nerves (associated with pain and sensitivity) versus muscle (less threatening), and showed a heightened response to the neurodynamic tests thought to be "nerve tests" versus "muscle tests." (Coppieters, Alshami, & Hodges, 2006)

### 2.4.2. Processing dominant mechanisms

Information from the tissues and the peripheral nervous system is received via the dorsal horn, mediated via descending inhibition from the brain and segmental inhibition via the interneuron (Woolf, 2007; Woolf & Mannion, 1999; Woolf & Salter, 2005). Information is passed to the brain via second order neurons for further analyses. In processing dominant systems (central sensitization), the spinal cord, brain stem and cerebral hemispheres become the source of dysfunction, with or without peripheral input (Nijs, Van Houdenhove, & Oostendorp, 2010; Woolf, 2007). The CNS, due to persistent input (particularly via C-fibers) increases its sensitivity over time. In many cases the original injury may have healed. The athlete may complain of LBP, but it has been present for 5 years. Similar to peripheral nerve sensitivity, the patient reports increased sensitivity to physical tests and movements, but it becomes more widespread and affects areas other than the original area of the injury. Pain is also now heavily affected by thoughts, feelings and emotions. (See representational model).

### 2.4.3. Output dominant mechanisms

In the presence of persistent pain, failed treatments, multiple diagnoses and opinions, decreased coping skills and increased fear,

homeostatic systems will engage to protect the athlete. Various systems, such as the endocrine, immune, motor, respiratory, sympathetic and parasympathetic systems will alter their function to protect the athlete in pain (Butler & Moseley, 2003; Moseley, 2007). These systems, although designed to deal with acute, immediate threat, are not designed to be overactive for prolonged periods. Changes associated with these output mechanisms include decreased blood flow to muscles (George, Dover, & Fillingim, 2007; Larsson, Cai, Zhang, Larsson, & Oberg, 1995), endocrine changes such as altered cortisol production (Janig, Chapman, & Green, 2006), muscle fiber representation in stabilizing muscles of the spine including atrophy and altered recruitment patterns(MacDonald, Moseley, & Hodges, 2006; Moseley, Hodges, & Gandevia, 2002), immune system changes with increased cytokine production (Watkins & Maier, 2002; Watkins, Milligan, & Maier, 2003), sympathetic nervous system changes associated with increased nerve sensitivity (Baron & Janig, 2004), changes in pain modulation with increased sensitivity (George & Delitto, 2002), and changes in breathing, mood and possibly performance.

A quick view of the pain mechanism model (Fig. 4) should underscore the statement that therapists who incorporate such a model will already enhance their biopsychosocial approach since the model not only includes biomedical concepts, but presents a more elaborate model dealing with various systems; especially the nervous system's processing of the injury.

### 2.5. Representation

The representation model of pain takes on the brain and its processing of pain. Pain is complex (Moseley, 2003b, 2007) and athletes will often perform/practice while having LBP (Bono, 2004; George & Delitto, 2002; Hangai et al., 2009). It is important for therapists and athletes alike to understand that 'nociception' is not synonymous with 'pain'. Nociception refers to the neural processes of encoding and processing of noxious stimuli (Loeser & Treede, 2008). Nociception is therefore merely input into the nervous system which has the potential to trigger a variety of responses and may or may not result in the experience of pain (Moseley, 2007). Nociception is neither sufficient nor necessary for the experience of pain (Acerra & Moseley, 2005; Bayer, Coverdale, Chiang, & Bangs, 1998; Melzack, 2001; Moseley, Brhyn, Ilowiecki, Solstad, & Hodges, 2003). Therapists who work with athletes may recall many anecdotal examples of tissue damage (nociception) not resulting in pain. An example might be a college football player who shrugs a fierce tackle and manages to sprint to the end zone for a touchdown only to discover some time later, that the tackle injured his acromioclavicular joint. In such a scenario, nociception (injury to the acromioclavicular joint caused by the tackle) did not result in pain. Research has also demonstrated that pain can be experienced in the absence of nociception (Acerra & Moseley, 2005). Pain is therefore more accurately defined as conscious decision by the brain to defend the athlete in lieu of the perceived threat of the injury (Moseley, 2003b, 2007)

New functional MRI (fMRI) and positron emission tomography (PET) scans have allowed scientists to show that when the brain processes information from the tissues, numerous areas are activated to deal with the threat of an injury, disease or situation (Flor, 2003; Moseley, 2003b, 2005; Peyron, Laurent, & Garcia-Larrea, 2000). It has long been thought that pain is processed within a certain area of the brain, commonly associated with sensation. The fact that a single area of the brain is associated with processing pain has been disputed for several decades and the use of the new imaging devices have allowed scientists to show this is not the case, but rather, various brain areas are active in processing pain (Flor, 2000; Flor, 2003). Numerous studies investigating various types

of patients, including those with LBP, have shown that common areas of the brain are frequently "ignited" in various pain states (Flor, 2000; Flor, 2003). These areas however have functions other than processing threat and pain (Fig. 5). These commonly ignited areas, via connections, recursive and backfiring neurons generate, in essence, a "pain map", which is referred to as a neural signature, neuromatrix or neurotag (Butler & Moseley, 2003; Melzack, 1999; Melzack, 2001; Moseley, 2003b). Adding further complexity, the neural signature is not dependent on any specific tissue (i.e., disc, facet or nerve), but rather the impending theat. "Emotional pain" uses similar areas to "physical pain." It is important to realize that even though there are some common pathways and areas activated in all people, each person's neuromatrix is individualized, which further underscores the reason why pain education sessions utilizing one-on-one treatments may have better outcomes than programs designed for groups of patients (Moseley, 2003a). The individualism of the neuromatrix can be understood considering the map's modulation with perception, memories and social context which will be different for each individual (Butler & Moseley, 2003).

Nociceptive information via the peripheral nervous system and spinal cord is thus processed by various areas of the brain (Melzack, 1999; Melzack, 2001; Moseley, 2003b). Pain is an output and ultimately a conscious decision by the brain, based on the sum of all the information it receives from the tissues and surrounding environment (Moseley, 2003a). If the sum result of the brain's processing of the information concludes that tissues are in danger, it is logical for the brain to produce pain as a means of protection. Any time the neural signature of LBP is activated, for example via nociceptive input from the back via exercise, bending or a tackle, the map activates, "runs" and *may* produce pain. Additional constituent maps form as well (Fig. 6). For example, maps related to beliefs, knowledge/logic, other sensory cues, social issues, anticipated consequences, healthcare provider and more (Moseley, 2003a, 2007).

The primary LBP map will form synapses with the "beliefs" map and therefore, any issues related to beliefs may activate the LBP map (Moseley, 2003b). For example, the athlete may believe that any LBP is potentially career-ending and thus activates the LBP map whenever he/she engages in thoughts related to these beliefs. The "knowledge" map will also synapse into the LBP map, and thus, any knowledge associated with LBP will activate the map. The athlete may have poor or faulty knowledge of LBP and what it means. As an example, a college football player may know nothing about LBP, except that the quarterback of an opposing team developed it 2 years ago and is no longer able to play. The primary LBP map can therefore receive increasing input from various other maps and will continually grow allowing LBP to potentially be influenced by fear, anxiety and memories (Moseley, 2003b). Therapists treating athletes with LBP should realize that by addressing the tissue issues (e.g. joint strain, instability) with typical therapeutic interventions (e.g. spinal stabilization exercises) they are only addressing one of perhaps many issues associated with the development of that LBP. The athlete may have such an innate fear of LBP that any activation of the amygdala may activate the LBP map, even though "the tissues may have healed." If medical care continues on the path of "seeking the injured joint or tissue" and results in more medical tests, more opinions, more failed treatment, then fear itself may increase and LBP may persist. Pain is a multiple system output, driven by the neuromatrix, which is activated by perceived threat (Butler & Moseley, 2003; Moseley, 2003b). Athletes with LBP often deal with injury (Bono, 2004; Hangai et al., 2009; Hides et al., 2008), disease (Hind, Truscott, & Evans, 2006; Ong, Anderson, & Roche, 2003), pain (Bono, 2004; George & Delitto, 2002; Kraft, 2002; Lundin et al., 2001), stress (Nadler, Malanga, Feinberg, et al., 2002; Nadler, Moley, et al., 2002), competition and fear (Bono, 2004; Standaert et al., 2004; Trainor & Wiesel, 2002), all of which can be implicated in driving the neuromatrix.

### 2.6. Evolutionary biology

It has also been proposed that a true biopsychosocial approach incorporate a viewpoint of pain and survival via an evolutionary model (Ness & Young, 2000). Pain is defined as a sensory and/or emotional experience associated with potential and actual tissue damage and described in such terms by the International Association on the Study of Pain (IASP) (Wall & Melzack, 2005). Pain, although unpleasant, is normal and part of survival. Evolutionary models help us create a better understanding of why certain physiological processes occur in the nervous system that seem
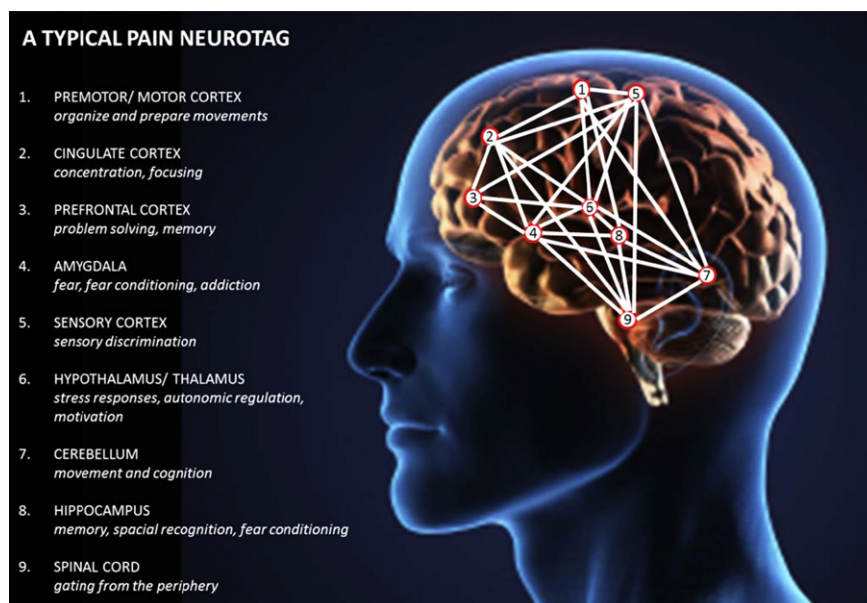


**Fig. 5.** Various areas of the brain commonly associated with the development of a pain neural signature or 'neurotag'. Re-drawn from Butler & Moseley, 2011.
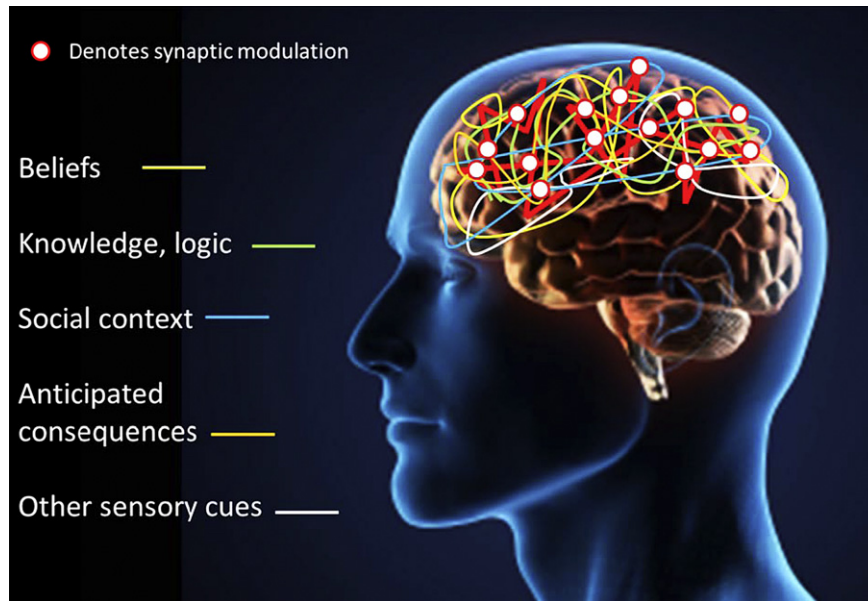
**Fig. 6.** Constituent maps alongside the pain neuromatrix or 'neurotag'. From Moseley, personal communication.

detrimental to the athlete. Processes such as neuronal death, neuroplasticity and receptor field changes and expansion (spreading pain) can be seen as processes aimed at survival (C. J. Woolf, 2007). Unfortunately, processes such as these may contribute to increased pain and unpleasant, though a logical survival strategy of the brain to deal with impending threat. Evolutionary biology models include aspects of pain as a learned behavior (nurture) as well as genetic issues associated with the development of pain (nature).

### 2.7. Psychosocial issues

It is well established that pain is not purely due to nociception and is heavily influenced by several other factors. The onion skins model (Fig. 7) (Loeser, 1999; Waddell, 2004) depicts the multi-faceted issues associated with pain. An athlete may have nociception (tissue injury), yet it may be modulated by issues such as attitudes and beliefs, suffering, pain escape behaviors and more.

### 2.8. Fear avoidance

It has been stated that "the fear of pain may be worse than pain itself." (Arntz & Peters, 1995). This statement is underscored by the fact that numerous studies evaluating LBP include the use of scales addressing fear, such as the fear avoidance beliefs questionnaire (FABQ) (Fritz & George, 2002; Fritz, George, & Delitto, 2001; George, Bialosky, & Fritz, 2004; George, Fritz, Bialosky, & Donald, 2003; George, Fritz, & Erhard, 2001; George, Fritz, & McNeil, 2006). Fear within the general population is often associated with the belief that increased activity, movement or exercise with not only increase pain, but further damage tissues. Athletes dealing with LBP deal with the unknown, including the time injury takes to heal, return to sport, diagnosis, how the back pain may/may not influence income, etc. The clinical manifestation of these unknowns may present itself as increased fear.

It is clear from the description above that athletes and LBP should be viewed from a more complex biopsychosocial approach, rather than a simple biomedical model. It could be argued that athletes, due to their increased demand from each and every bodily

system need a biopsychosocial model more than patients presenting in therapy from the general population due to the higher demands placed on their tissues, cognitions and brain.

### 3. The neuromatrix, athletes and performance

The fact that the neuromatrix engages various areas of the brain during the processing of LBP, leads us to consider how this might affect the "normal" functioning of these brain areas. Although there has been very little research (most focusing on concussion) (Bailey, Echemendia, & Arnett, 2006; Echemendia, Putukian, Mackin, Julian, & Shoss, 2001; Nielsen & Cohen, 2008); a good starting point would be to realize that sports performance is an output of the brain.
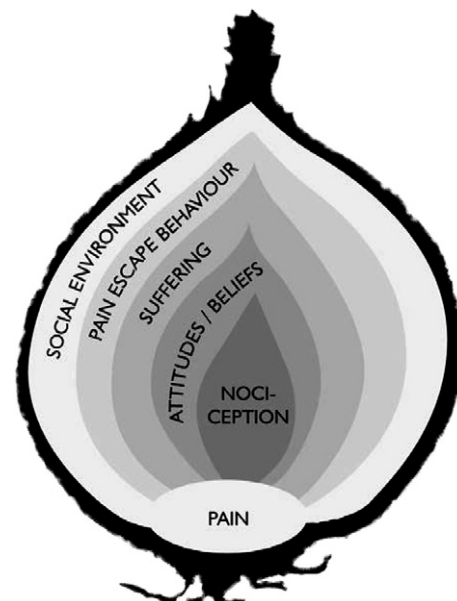


**Fig. 7.** Onion skins model. From Butler & Moseley, 2003 − With permission.

Numerous athletes, by honing their skill through seemingly endless repetition, may in fact strengthen neural pathways and, in essence, develop powerful "sports skills" maps in their brain. By repetitively practicing a task, the map of that task will become enhanced (Moseley, 2003b; Nielsen & Cohen, 2008). Synaptic activation is optimized. Dopamine, an excitatory neurotransmitter, is thought to help in solidifying these connections (Girault & Greengard, 2004). This neural function is extremely useful for the athlete, and it can essentially explain the neurobiology underpinning an athlete's smooth and refined golf swing or baseball pitch. This same process occurs in patients with pain, but in a negative way (Moseley, 2003b). Patients who continue to "live their pain" are essentially sealing the pain pathways via the repeated activation of neuro-transmitters such as dopamine (Girault & Greengard, 2004). This is why changing pain (or any other addictive behavior) is so difficult. If we return to our athlete with LBP, and consider that the LBP map can be affected by adjacent maps (fear, anxiety, etc.) (Moseley, 2003b) it may provide a possible explanation as to why an athlete may have a problem performing their athletic tasks with their customary smoothness and precision. With the increased reach of the LBP map, many different influences may activate the pain map, which may in turn influence optimal movement and performance.

Additionally, we should consider the "normal" function/activity of the brain areas activated during a typical pain neuromatrix. Numerous studies examining fMRI and PET scans of patients with various forms of pain have helped scientists identify key areas of the brain which are frequently activated during a painful experience (Fig. 5) (Flor, Braun, Elbert, & Birbaumer, 1997; Moseley, 2003b, 2005). These areas include the pre-motor area (organize, plan and prepare movement); motor area (motor control); cingulate cortex (focus/concentration); amygdala (fear); hypothalamus/thalamus (stress responses/autonomic regulation/motivation); and cerebellum (movement/balance/proprioception) (Flor, 2000; Flor, 2003; George, Wittmer, Fillingim, & Robinson, 2006; Melzack, 2001; Moseley, 2003b). A key question for athletes with LBP is now noted. If these areas, normally used to perform athletic skills, are now used to process nociception as part of the pain experience, how effective can they be at performing their primary tasks? It could be argued that for optimal performance, all areas of the brain should function at optimal capacity associated with performing a specific task to help the athlete perform at his/her highest level (Nielsen & Cohen, 2008). If the motor cortex is engaged in processing nociceptive input as part of the pain experience, it may not be able to provide for an optimal output dedicated to, for instance, motor control – a vital component in athletes and LBP. Injury to the low back as well as experimentally induced nociception have been associated with pain as well as altered motor control (Hides, Stokes, Saide, Jull, & Cooper, 1994; Richardson & Jull, 1995; Richardson, Snijders, Hides, 2002).

Pre-motor and motor areas are significantly activated in patients with LBP (Peyron et al., 2000). Motor control is significantly affected by fear of pain (Hodges & Moseley, 2003; Moseley et al., 2003); anticipation of pain (Hodges & Moseley, 2003; Moseley et al., 2003; Moseley, Nicholas, & Hodges, 2004a); catastrophization (Moseley, 2004); past history of LBP (Hodges & Moseley, 2003); and by thoughts and emotions. All of these observed changes are considered to be normal. Clinical observation validates this concept as pain is seen to change/affect motor control (Hodges & Moseley, 2003; Sterling, Jull, & Wright, 2001). Even the pre-motor area, often activated by thoughts, vision or sound is active in the neuromatrix processing pain. Athletes also spend significant time preparing mentally and rehearsing techniques, and the pre-motor area may be limited in this regard if it is actively contributing to a pain neuromatrix.

A final consideration related to the neuromatrix involves the body's ability and innate desire to protect itself. With activation of the pain neuromatrix, the brain produces pain (Moseley, 2003b, 2005). The body will then react by engaging multiple systems designed to protect the athlete (muscle guarding, limping, etc.) (Butler & Moseley, 2003).As previously stated, pain can be more precisely defined as a multiple system output activated by the neuromatrix in the face of impending threat (Moseley, 2003b) The brain, based on all the information available to it, activates the pain neuromatrix and engages systems to protect the athlete. These are homeostatic systems which include the sympathetic nervous system, motor system, immune system, parasympathetic nervous system, pain system, respiration, mood and even language (Butler & Moseley, 2003; Johnson, Kamilaris, Chrousos, & Gold, 1992) and are there to help athletes cope with immediate (acute) danger, but if left in a heightened state over time (weeks, months or even years) may cause changes which can be clinically observed.

The sympathetic nervous system changes adrenaline levels and prolonged activation is associated with fatigue, sleep disturbance, and increased sensitivity of the nervous system (Segal, Hindmarsh, & Viner, 2005; Van Houdenhove, Van Den Eede, & Luyten, 2009). Although the exact correlation and mechanism concerning the parasympathetic nervous system (PNS) and altered sleep is unknown, altered sleeping patterns have been associated with possible abnormal PNS function which may lead to fatigue and irritability (Zhong et al., 2005). The systems associated with pain decrease thresholds and lead to increased pain perception (primary and secondary hyperalgesia). Changes in the neuroendocrine system lead to changes in circulating cortisol. Altered cortisol levels have been linked to changes in the immune system, depression, mood changes, sleep disturbance, appetite changes and fatigue (Ben Ounis et al., 2011; Crewther, Heke, & Keogh, 2011; Tanskanen et al., 2011). Cortisol further alters cytokine levels, which in turn alters the immune system (the athlete may be more susceptible to infections) (Vukelic et al., 2011) and increase nerve sensitivity. Respiration changes to a more superficial pattern, activating accessory muscles, thus diminishing diaphragmatic breathing and thus leading to poor oxygenation of blood. A long list of such deleterious changes may be seen to occur. The key issue is that persistent pain has a widespread effect and limiting our view of an athlete's LBP to a local joint issue to be treated with local techniques may not be adequate, especially in a high-performing athletic population. The longer the pain lasts and the more ineffective local treatments are, the more these systems, activated by the pain neuromatrix will be engaged to defend the athlete.

## 4. Treating athletes with a neuroscience approach

It is clear that LBP in athletes cannot be reduced to tissue injury, pain and treatment aimed at reducing nociception, and that a more complex biopsychosocial view is warranted. Such an approach embraces the typical treatments aimed at treating mechanical acute LBP such as spinal mobilization and manipulation (Childs et al., 2004; Flynn et al., 2002), exercise (Critchley, Ratcliffe, Noonan, Jones, & Hurley, 2007; Goldby, Moore, Doust, & Trew, 2006; Puentedura, Brooksby, Wallmann, & Landers, 2010) and modalities (Wong, Schumann, Townsend, & Phelps, 2007) but also urges the therapists to address issues far more complex, such as fear, anxiety, goals and perception.

One strategy which aims to address a true biopsychosocial approach is pain neuroscience education. Pain neuroscience education aims to explain to athletes with LBP (or any other pain problem) the biology of their pain (Moseley, 2004; Moseley, Nicholas, & Hodges, 2004b; Moseley, 2002). It is hypothesized

**Table 1**
Summary of the content and delivery methods of neuroscience education for musculoskeletal pain and dysfunction (Louw et al., 2011).

Content of neuroscience education:
- Neurophysiology of pain
- No reference to anatomical or pathoanatomical models
- No discussion of emotional or behavioral aspects to pain
- Nociception and nociceptive pathways
- Neurones
- Synapses
- Action potential
- Spinal inhibition and facilitation
- Peripheral sensitization
- Central sensitization
- Plasticity of the nervous system

Professionals performing neuroscience education:
- Neuroscience education in the reviewed studies was performed by physiotherapists.

Duration and frequency of neuroscience education:
- The duration and frequency of the neuroscience education sessions were quite varied.
- Educational sessions lasted as long as 4 h, while more recent studies reported sessions lasting 30 min.
- Educational sessions were also varied between single educational sessions and multiple sessions.
- The most common frequency between multiple educational sessions was one week apart.
- Considering studies varied between single educational interventions and multiple interventions, total education time was also determined.

Educational format:
- The format in which the neuroscience education was delivered was primarily by means of one-on-one verbal communication.
- Only 2 studies utilized group sessions.

Educational tools:
- Prepared pictures
- Examples
- Metaphors
- Hand drawings
- Workbook with reading/question-answer assignments
- Neurophysiology Pain Questionnaire

Adjunct treatment to the neuroscience education: Neuroscience education was thus preceded, combined with, or followed by, various therapeutic activities.
- Manual therapy including spinal mobilization and manipulation
- Soft tissue treatment/massage
- Neural tissue mobilization
- Spinal stabilization exercises
- Home exercises
- Circuit training
- Aerobic exercise
- None (neuroscience education only)

that this approach disengages parts of the "pain neuromatrix" (Moseley, 2005; Moseley et al., 2004b). Pain neuroscience education has primarily been used with patients experiencing chronic LBP, especially widespread pain (Moseley et al., 2004b; Moseley, 2002) Studies which utilize neuroscience education have been shown to decrease fear and change a patient's perception of his/her pain (Moseley, 2003b; Oliveira, Gevirtz, & Hubbard, 2006). Additionally, neuroscience education has been shown to have an immediate effect on improvements in patients' attitudes about and relation to pain (Moseley, 2003b); improvements in pain cognition and physical performance (Moseley, 2004); increased pain thresholds during physical tasks (Moseley et al., 2004b); improved outcomes of therapeutic exercises (Moseley, 2002); and significant reduction in widespread brain activity characteristic of areas involved in processing pain during abdominal draw in tasks in spinal stabilization (Moseley, 2005). Furthermore, these neuroscience studies have shown results to extend beyond the short term

and to be maintained at one-year follow-up (Moseley, 2003b; Moseley, 2002; Oliveira et al., 2006). A recent systematic review on neuroscience education summarized the content and education delivery methods used in neuroscience education addressing pain, anxiety and stress in musculoskeletal pain (Table 1) (Louw, Diener, Butler, & Puentedura, 2011). The findings from this review concur with recent articles regarding the practical application of explaining neuroscience education to patients and can be used as a guideline for clinicians (Clarke, Ryan, & Martin, 2011; Louw, Puentedura, & Mintken, 2012; Nijs, Paul van Wilgen, Van Oosterwijck, van Ittersum, & Meeus, 2011).

## 5. Conclusion

Therapists should certainly continue to utilize their manual therapy and therapeutic exercise skills in rehabilitating the athlete with LBP. It is well documented that pain affects motor control (Hodges & Moseley, 2003; Sterling et al., 2001) and that manual therapy elicits an immediate change in motor control and pain (Fernandez-de-las-Penas, Perez-de-Heredia, Brea-Rivero, & Miangolarra-Page, 2007; George, Bishop, Bialosky, Zeppieri, & Robinson, 2006; Raney, Teyhen, & Childs, 2007). However, the complexity of the neuromatrix demands that clinicians also spend time (during other treatments and by itself) addressing the psychosocial aspects of pain in athletes; especially fear, anxiety and faulty knowledge regarding their LBP. The idea behind such a biopsychosocial approach is to systematically determine factors associated with the persistent pain state and work on strategies to disengage those adjacent maps. For instance, spending time addressing fear and helping an athlete to better understand their pain and thus decrease fear, may help to disengage the connection of the fear map to the LBP map. Systematically, as the clinician addresses other issues (imaging results, diagnoses, failed treatments, etc.), the influence of these adjacent maps should diminish and the LBP map may not only become activated less often (LBP becomes less frequent), but it will take a stronger input from the tissues to activate the primary LBP map (the tolerance to exercise, movement and activity will increase).

Physical therapists are ideally positioned to treat athletes with LBP, especially if they incorporate the neuromatrix model of pain. Physical therapists have the ability to affect athletes on so many levels, all at the same time. Skillful delivery of manual therapy, including spinal manipulation, and segmental spinal stabilization exercises are part of daily physical therapy practice and should form a key part of the management of an athlete with LBP. Physical therapists should utilize adjunct treatments aimed at reducing nociceptive input to the central nervous system from the periphery, through the use of manual therapy and modalities. However, a 'top-down approach' is also needed.

Therapists are also able to (and should) educate the athlete about the neurobiology of their pain. They should explain how pain works and how it is processed. Therapists should avoid anatomical models that may induce fear and anxiety, and avoid using words such as "instability" and "ruptured" or "herniated" disc. Every therapy session should be aimed at calming down the nervous system, mainly by addressing fears, expectations, anxiety and goals. Aerobic exercise is also important. Aerobic exercise has been shown to have good efficacy in managing chronic patients, who clearly have very active and widespread pain neuromatrices. Aerobic exercise helps increase oxygen and blood to various tissues and has been shown to help decrease nerve pain, help patients sleep better, improve mood, help depression and more. All of the treatments described above are part of physical therapy. Physical therapists should embrace the biopsychosocial approach to athletes with LBP by engaging the brain and nervous system.

Athletes do experience LBP. Some of those episodes of LBP may well be due to structural issues and "instabilities"; however, treatment should focus on much more than just manual therapy and stabilization exercises.

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