

Slacklining as therapy to address non-specific low back pain in the presence of multifidus arthrogenic muscle inhibition

Charles Philip Gabel, Hamid Reza Mokhtarinia, Markus Melloh, Sébastien Mateo

ORCID number: Charles Philip Gabel 0000-0001-8354-4545; Hamid Reza Mokhtarinia 0000-0002-5181-4894; Markus Melloh 0000-0002-8819-799X; Sébastien Mateo 0000-0003-4867-4146.

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Charles Philip Gabel, Department of Physiotherapy, Access Physiotherapy, Coolum Beach 4573, QLD, Australia

Hamid Reza Mokhtarinia, Department of Ergonomics, University of Social Welfare and Rehabilitation Sciences, Tehran 0001, Iran

Hamid Reza Mokhtarinia, Department of Physiotherapy, University of Social Welfare and Rehabilitation Sciences, Tehran 0001, Iran

Markus Melloh, School of Health Professions, Zurich University of Applied Sciences, Winterthur 8310, Switzerland

Sébastien Mateo, INSERM U1028, CNRS UMR5292, Lyon Neuroscience Research Center, Université de Lyon, Lyon 69000, France

Corresponding author: Charles Philip Gabel, BPhy, MSc, PhD, Director, Department of Physiotherapy, Access Physiotherapy, PO Box 760, Coolum Beach 4573, QLD, Australia. cp.gabel@bigpond.com

Abstract

Low back pain (LBP) represents the most prevalent, problematic and painful of musculoskeletal conditions that affects both the individual and society with health and economic concerns. LBP is a heterogeneous condition with multiple diagnoses and causes. In the absence of consensus definitions, partly because of terminology inconsistency, it is further referred to as non-specific LBP (NSLBP). In NSLBP patients, the lumbar multifidus (MF), a key stabilizing muscle, has a depleted role due to recognized myocellular lipid infiltration and wasting, with the potential primary cause hypothesized as arthrogenic muscle inhibition (AMI). This link between AMI and NSLBP continues to gain increasing recognition. To date there is no 'gold standard' or consensus treatment to alleviate symptoms and disability due to NSLBP, though the advocated interventions are numerous, with marked variations in costs and levels of supportive evidence. However, there is consensus that NSLBP management be cost-effective, self-administered, educational, exercise-based, and use multi-modal and multi-disciplinary approaches. An adjuvant therapy fulfilling these consensus criteria is 'slacklining', within an overall rehabilitation program. Slacklining, the neuromechanical action of balance retention on a tightened band, induces strategic indirect-involuntary therapeutic muscle activation exercise incorporating spinal motor control. Though several models have been proposed, understanding slacklining's neuro-motor

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mechanism of action remains incomplete. Slacklining has demonstrated clinical effects to overcome AMI in peripheral joints, particularly the knee, and is reported in clinical case-studies as showing promising results in reducing NSLBP related to MF deficiency induced through AMI (MF-AMI). Therefore, this paper aims to: rationalize why and how adjuvant, slacklining therapeutic exercise may positively affect patients with NSLBP, due to MF-AMI induced depletion of spinal stabilization; considers current understandings and interventions for NSLBP, including the contributing role of MF-AMI; and details the reasons why slacklining could be considered as a potential adjuvant intervention for NSLBP through its indirect-involuntary action. This action is hypothesized to occur through an over-ride or inhibition of central down-regulatory induced muscle insufficiency, present due to AMI. This subsequently allows neuroplasticity, normal neuro-motor sequencing and muscle re-activation, which facilitates innate advantageous spinal stabilization. This in-turn addresses and reduces NSLBP, its concurrent symptoms and functional disability. This process is hypothesized to occur through four neuro-physiological processing pathways: finite neural delay; movement-control phenotypes; inhibition of action and the innate primordial imperative; and accentuated corticospinal drive. Further research is recommended to investigate these hypotheses and the effect of slacklining as an adjuvant therapy in cohort and control studies of NSLBP populations.

Key Words: Slacklining; Arthrogenic muscle inhibition; Low back pain; Therapy-intervention; Multifidus; Hypothesis

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Core Tip: Non-specific low back pain (NSLBP) is the leading problematic musculoskeletal condition for individuals and society. With no consensus definition, depleted lumbar multifidus stabilization is recognized with fatty infiltration and wasting, where arthrogenic muscle inhibition is a probable cause. With no gold-standard therapy, management consensus recommends cost-effective, self-administered, exercise-based multi-modal approaches. 'Slacklining' addresses these criteria as an adjuvant therapeutic rehabilitation exercise, rationalized by a hypothesized over-ride of central down-regulatory induced muscle insufficiency. This allows neuroplasticity, normalized neuro-motor sequencing and muscle re-activation for stabilization. Four neuro-physiological pathways are proposed with further research required into the hypotheses and slacklining's potential NSLBP rehabilitation role.

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INTRODUCTION

Low back pain (LBP) is a major global health and economic concern due to its prevalence, heterogeneous classification and multiple causes and diagnoses. It affects more individuals than any other musculoskeletal condition^[1], but is mostly 'non-specific' (approximately 85%), as the source of abnormality and symptoms is undefined^[2]. Non-specific LBP (NSLBP) is characterized by factors that cross biophysical, psychological and social domains, and presents on a spectrum from neuropathic to nociceptive pain^[3]. The neuropathic component is predominantly from nerve root compression within or adjacent the spinal canal from several potential causes, whereas nociceptive is associated with mechanical stress^[4] and non-neural tissue damage^[5,6]. This leads to broad problems for the individual's function, ability, activities of daily living (ADL), societal participation, and economic situation. There are also societal demands on health-care and support networks, that vary considerably

within and between countries, due to local approaches, social attitudes, and legislative influences^[1,2]. The most widely used categorization of NSLBP is acute (< 6 wk), subacute (6-12 wk), or chronic (> 12 wk), causing symptoms in > 50% of days^[3,7,8].

However, there is no 'gold standard' management that demonstrates a strong, cost efficient, evidence-based effect^[2]. This is partly because consensus is divided on the terminology used and the approaches advocated. Different terms such as mechanical or axial LBP refer to the same condition, causing inconsistency in nomenclature due to varied interpretations by clinicians, researchers, and society in general; while the many diverse and conflicting approaches and interventions recommended perpetuate the perplexity^[6]. Systematic reviews and meta-analyses indicate the different interventions show markedly varied levels of cost efficiency and supportive evidence, both between and within approaches^[2]. One area with general agreement is that spinal stability^[9-11] is critically important, particularly at the segmental level^[12,13] where the abdominal and lumbar muscles^[6] contribute significantly^[14,15], with the multifidus (MF) muscle having a strategic functional role^[12]. Accordingly, any condition that disrupts and compromises this lumbo-pelvic stabilization capacity, such as myocellular lipid (MCL) infiltration and wasting leading to depletion of the MF stabilizing role, should be investigated and addressed^[6,16,17].

One such causative condition is arthrogenic muscle inhibition (AMI)^[17-22], which is summarily defined as "... a neural activation deficit [of the muscles]"^[23] (see [Supplementary material, Appendices 1-3](#)). Consequently, AMI has gained increasing recognition and research interest over recent decades as a primary contributor to NSLBP^[5,6,17]. Therefore, intervention techniques that address MF deficiency induced through AMI (MF-AMI), use direct/indirect muscle activation approaches that target the MF through voluntary/involuntary activation, and are clinically important^[24], particularly if they are self-directed, and cost effective^[13,15].

Within this clinical framework of self-directed, cost effective interventions addressing MF deficiency, any new techniques are encouraged^[15]; with a very recent paradigm being indirect-involuntary muscle activation, where slacklining is a specific example. Slacklining, 'the neuromechanical action of balance retention while standing on a tightened webbing band'^[25-28], has been introduced over recent decades as a strategic musculoskeletal and neurological therapeutic pre- and re-habilitation exercise^[29,30]. However, published slacklining research is recent and available evidence remains limited^[31-33], particularly for clinical rehabilitation implications^[30,34-36], and formulated explanatory models^[28,31], despite slacklining's ancient origins^[37]. Slacklining invokes a moderated indirect muscle activation that occurs involuntarily and facilitates achieving optimum balance according to the needs of the motor task through muscle, joint, and subsequent limb and body control^[31,38]. It has been clinically demonstrated to overcome AMI during rehabilitation in peripheral regions and joints, particularly the knee and ankle^[23,30,39], and is recently reported as potentially showing promising clinical results in reducing MF-AMI induced NSLBP^[17].

Consequently, this paper aims to rationalize why and how slacklining, as an adjuvant therapeutic exercise, may positively affect patients with NSLBP that is due to MF-AMI induced depletion of spinal stabilization. We consider slacklining's centrally mediated pre-synaptic actions that provide either an over-ride or inhibition of the down-regulatory induced MF insufficiency that occurs as a consequence of AMI. This action will, consequently, allow neuroplasticity, the restoration of normal neuro-motor sequencing that enables MF re-activation, subsequent spinal stabilization, and the resultant improvement in symptoms and function. This in-turn addresses and reduces NSLBP and its concurrent symptoms and functional disability.

This action is hypothesized to occur through one or a combination of four neuro-physiological processes that occur within the following pathways: (1) Finite neural delay accompanying 'time available' processing; (2) Movement control phenotypes affected by an exercise reasoning hypothesis; (3) The inhibition of action and the innate primordial imperative; and (4) Accentuated corticospinal drive. To comprehend these actions and understand their implications requires a step-wise rationalization that involves a process of: presenting a summary review of NSLBP and spinal stability; the MF stabilizing role; how AMI depletes this stabilization; recognized NSLBP rehabilitation methods; slacklining as an adjuvant therapeutic exercise; and, subsequently, slacklining's neuro-physiological effects of centrally mediated pre-synaptic actions that provide either an over-ride or inhibition of the down-regulatory induced MF, that counter the local stabilization inadequacies that result in NSLBP.

NSLBP AND SPINAL STABILITY

The classic Panjabi spine-stability model^[9,10] hypothesizes that NSLBP originates from the embedded mechanoreceptors of passive spinal support structures (ligaments, disc-annulus, and facet-capsules), which are subject to large-singular or cumulative-micro trauma causing sub-failure injuries. This model was initially amended to include the thoracolumbar fascia^[40], then further adapted to integrate mobility^[11]. The injury-affected structures generate corrupted transducer signals that alter the neuromuscular control units response patterns, which disrupts muscular onset-shutoff coordination and force magnitude characteristics^[6,9,10]. Lumbar spine vertebral joints are overloaded^[4] from 'functional instability' due to 'motor-control system anomalies'^[9,11,16]. These are both a cause and effect of neuroplastic changes in sensorimotor control^[41-43] and impaired lumbar MF corticomotor control^[6,44]. Addressing these factors involves muscle activation, such as 'motor control exercises' (MCE) at the local and or global level, which aim to restore coordinated and efficient use of the muscles that control and support the spine^[13,45]. These target the 'core' stabilizing muscles, transversus abdominus (TrA), erector spinae (ES), pelvic floor (PF), and MF^[6,46]; however, only MF provides segmental stability^[12,13].

The use of MCE therapy aims to restore muscle activation to the disrupted MF, either alone or in combination with the other core muscles^[6,46], though with conflicting available evidence^[47,48]. Systematic reviews of characteristic macroscopic structural MF changes in NSLBP patients indicate "... a loss of muscle size ... [especially] in the lower lumbar levels ..." ^[49]; and distinct differences in muscle size and levels of fat infiltration compared to age and gender related norms^[12,50,51]. However, correlation of these changes with clinical outcome improvement varies^[48], as supportive evidence is low-moderate in higher quality studies^[16,47], while lower quality studies overestimate MCE effectiveness^[45]. Consequently, MCE can be considered as supported, but without a unified consensus approach^[13].

LUMBAR MF ROLE IN SPINAL STABILIZATION

Researching trunk muscles structure, particularly MF, and their contributions to spinal stability and the clinical association with NSLBP, has continued for over 50 years^[45]. The MF has a specific segmental stability role^[10-12], and is the muscle with the strongest influence on lumbar stability against applied forces, especially in flexion and extension^[12,13], the two predominant motor control impairment clinical directional subgroup classifications^[52,53]. A lack of adequate segmental stability results in abnormal stress on ligaments, muscles, and mechanoreceptors; which leads to excessive facet joint loading^[10,13]. Since spinal ligaments have inherently poor healing, disc and facet joint degeneration accelerates. Persistence of such abnormal conditions results in NSLBP from: neural tissue inflammation, changes to biochemistry, nutrition, stem cells immune factors, endplate structure and composition, discs, and neural tissues with ingrowth into the intervertebral discs^[10]. In this situation, the MF's stabilizing role is depleted and hypothesized as due to AMI^[5,6,17]; with supportive evidence from MCE therapy^[13,14,24,45,54], and the effects of localized neuromuscular electrical stimulation (NMES) which directly induces involuntary episodic MF-contraction^[5,6,16,55].

The links between NSLBP, lumbar muscle wasting, and fatty infiltration by macroscopic MCLs was recognized over 25 years ago^[12,14], though there has been both subsequent support for the model, and questioning of whether these links have consistent correlations^[49]. The attribution of this lumbar MF wasting to AMI was first speculated in 2002^[56], while acceptance of AMIs contribution to NSLBP is only quite recent^[5,6,16]. Recognizing MF-AMI is a clinical diagnosis, but supported by physical measures of wasting on ultrasound (US) imaging^[44], and MCL infiltrates on dual/multi-echo magnetic resonance imaging (MRI) and MR-spectroscopy^[51]. The latter also provides muscular-MCL composition and distinguishes between extra-MCLs (EMCLs), associated with age-related change, and intra-MCLs (IMCLs) associated with NSLBP^[17,51]. Consequently, IMCLs and changes in their percentage presence may enable: the described diagnostic techniques to function as a proxy for MF-AMI induced NSLBP; and act as prognostic markers for the efficacy of NSLBP rehabilitation management techniques directed at MF activation/stimulation for spinal stabilization^[6,17].

ARTHROGENIC MUSCLES INHIBITION — CURRENT CONCEPTS

The introductions summary definition of AMI as “... a neural activation deficit [of the muscles]”^[23], can be further expanded and detailed through the current descriptive consensus of: Periarticular muscle inhibition, often with associated atrophy, from modulation of motor neuron pools within the uninjured muscle/s, that prevents voluntary recruitment about a damaged or distended joint^[21,57], and may include the contralateral unaffected side^[23]; where motor insufficiency and activation failure is a consequence of a presynaptic reflex reaction through neural inhibition and ongoing neural activation deficit of the muscles, due to compromised efferent drive in the local, spinal and supra-spinal pathways, resulting from aberrant afferent discharge from joint mechano-sensory receptors, which serves as a natural response to protect from further damage^[18-23,57-60].

The distinction of AMI from ‘disuse atrophy’, which has no central nervous system (CNS) or reflex basis^[58], has been made since Hippocrates around 400BC^[61]. However, the specific mechanism eliciting and controlling AMI remains unclear in all recent publications^[22,62], and has predominantly been postulated from the recognized actions and effects following knee joint injury^[6,23]. Consequently, though the physiological neuro-motor basis of AMI remains poorly understood, significant investigation continues, particularly research into the cerebral-based mechanisms of AMI^[22,63].

Clinically AMI results in an inability to completely activate the periarticular muscles as the available volume of motor units is reduced^[20,21]. This leads to a ‘vicious circle’^[19] of atrophy, weakness, damage, and dysfunction^[59]. Consequently, AMI is consistent across different joints and joint pathologies throughout the body, and often most severe in the acute stage of joint damage^[21]. The level of afferent discharge from changes in the articular sensory receptors is determined by several factors. Consequently, the mechanisms that drive the severity of AMI presentation are: the severity of the damage to the joint, distention due to inflammation and effusion, joint laxity^[6,20,23], the angular position of the affected joint/s, and time-duration since the injury^[21]. These factors, subsequently, initiate changes at all levels of the CNS^[21], and is expanded on below and in [Supplementary material, Appendix 2](#).

AMI — neurophysiology of AMI actions within the CNS

The mechanisms of action for AMI initiated by neural pathways at the local/peripheral, spinal, and supra-spinal/central levels of the CNS are summarized as follows.

Local/peripheral level changes: These are a consequence of sustained tissue damage that alters muscle resting motor thresholds, and articular sensory receptor afferent discharge^[6,19,20,58,64]. These predominantly include loss or irregularity of the affected articular sensory receptors themselves, joint laxity, and joint damage from distention due to swelling and/or inflammation^[21].

For articular sensory receptors, the degree of joint structural trauma affects the level of damaged local nerve endings, with decreases in afferent output when the sensory endings are damaged^[59,65], but increases with distention and joint laxity, which accounts for the strong association with AMI^[21]. With joint laxity there is altered sensory receptor activation. The presence of structural damage, and/or degenerative change, enables increased intra-articular movement and joint surface translation, which increases mechanoreceptor and nociceptor activation. This in-turn signals the joints range of movement limits. Consequently, anomalous sensory receptor firing during joint movement will be present.

With joint distention from swelling, including clinically undetectable effusions, intra-articular pressure (IAP) is raised. This increases Group II afferent discharge from the joint through stimulation of pressure sensitive and stretch mechanoreceptors. This strongly inhibits the periarticular muscles, as the Group-I non-reciprocal (Ib) inhibitory interneurons in the spinal cord in-turn inhibit localized muscle α -motoneurons, which prevents full muscle activation. This results in significant AMI, as shown directly in the quadriceps^[23,62], and can be implied for the local MF from studies on disc and nerve root trauma^[66]. With joint distention from inflammation, the presence of AMI is distinguished by peripheral sensitization and resultant nociceptive signaling. Prolonged local tissue and joint sensitivity changes occur as a result of a reduced activation threshold in the articular free nerve endings supplied by Group III and IV joint afferents^[67]. Consequently, normal non-noxious mechanical movement and activity causes articular structure stimulation which results in notable Group III and IV afferent discharge^[67]. The release of inflammatory mediators increases this joint afferent discharge by sensitizing the free nerve endings innervated by Group III and

IV afferents. This is independent of nociceptive signals, as reducing pain does not necessarily reduce AMI's severity^[21,62]. This action and the resultant reflex inhibition indicative of AMI, is also demonstrated for the lumbar MF at adjacent joints as well as the specific neural innovation levels, and in the presence of disc prolapse^[66]. Both distention scenarios support the historical and recent recognition that AMI can occur regardless of the presence of structural damage^[23,58,61,62] or inflammation^[19,68,69].

Spinal pathways implicated in AMI: The spinal pathways are particularly associated with abnormal afferent discharge from the affected joint/s that can alter the excitability of the reflex pathways within the cord. This particularly affects the Group Ib inhibitory pathway and the associated Group Ia, II, III, and IV, the flexion reflex, and the gamma (γ)-loop^[21]. However the neural connectivity at this level is able to show local neuroplastic change. A marker of observable neural change at the spinal level, the Hoffmann reflex (H-reflex), is measurable at the soleus through amplitude variations^[70]. The H-reflex can be used to compare training effects in relation to the learning of a new skilled task, such as slacklining, with measures pre-activity (baseline), immediate post-activity (acquisition), and 24 h post-activity (retention). After 'acquisition' of a new skilled task, changes at the spinal level are general, but by 24 h these become task-specific. Consequently neural reorganization and generalization of spinal adaptations appears to be time dependent with the task specific adaption occurring after one day^[71].

The Group Ib non-reciprocal inhibitory pathway utilizes signal relay actions to integrate sensory-motor information. Afferent musculo-tendinous junction neuronal signals are supplemented by Group Ia sensory fibers from the muscle-spindle stretch receptor, joint and peripheral sensory receptors through Group II, and unmyelinated/thinly myelinated Group III and IV fibers^[72]. In turn motor-efferent neuron actions are influenced. Consequently, these dual capacities influence the Ib fibers signaling where effusion increases Group II output, which increases AMI through facilitated muscle motoneuron pool inhibition^[21,72].

The flexion reflex in contrast is a polysynaptic pathway with facilitated agonist excitability and concurrent reciprocal extensor inhibition that may cause AMI^[68]. The distinction of the γ -loop spinal reflex circuit is that it enables automatic muscle tension regulation that ensures full activation during voluntary muscle contractions^[21,73]. Consequently, γ -loop dysfunction will contribute to AMI^[21] as joint injury reduces excitatory afferent output to the muscle γ -motoneuron pool which reduces discharge. This results in enhanced presynaptic inhibition of the muscle α -motoneuron pool *via* Ia afferent fibers^[74]. The spinal inhibitory interneurons projecting into the Ia afferent fibers synaptic terminals adjust the neurotransmitter release levels in response to the afferent input, which, subsequently, modulates the synaptic efficacy^[74].

Supra-spinal/ Central pathways implicated in AMI: These pathways are hypothesized as supra-spinal projections from the joint afferents that influence AMI^[21,75]. The changes include four main areas: (1) Cortico-spinal excitability/activity affecting the somatosensory cortex^[22]; (2) Brainstem descending pathways^[76] and the flexion reflex^[75], with efferent commands modulated by afferent input; (3) Individual voluntary effort^[21]; and (4) 'Informed awareness' that amalgamates behavior and 'flow-experience'^[77]. These supra-spinal actions affect both neuroplasticity^[42,43,78] and movement-fluency^[79] to facilitate global equilibrium control. Consequently, centrally mediated sources override existing down regulatory inhibition, through concurrent control of active spinal reflexes. This enables muscular re-activation that is repressed by central inhibition in response to negative afferent input from traumatized and/or distended joints.

AMI knowledge: Based on peripheral findings, particularly at the knee

Despite AMI's specific mechanism remaining unclear and the majority of knowledge being derived from recognized attributes post-trauma about the knee^[22,62], ongoing research into AMI's physiological neuro-motor basis continues^[5,6,17,22,23,63]. It is already well accepted that, in acute knee trauma maximizing available quadriceps activation through isometric exercises in the lowest IAP range is effective^[80], however this is not always recognized as occurring when addressing AMI^[23,68]. Consequently the focus in new research is on the CNS, particularly modification of the connectivity within the sensory-motor network^[63]. This includes efferent corticospinal excitability alterations post joint injury that facilitate local muscle inhibition at the knee^[81], and investigations in ACL injury related to therapeutic effectiveness in reducing AMI^[22]. The verification of the consensus assumption that central brain origin output and inhibition is the primary explanation for quadriceps AMI^[22,23,63], is demonstrated by brain motor area

activation variations, found under MRI, between individuals with and without quadriceps AMI^[69]. As these understandings improve, become recognized and accepted, the ability to progress and extrapolate such findings to the spine and MF-AMI depleted stabilisation will increase; as will the potential for management of NSLBP though AMI directed intervention to similarly evolve^[5,6,17].

AMI knowledge: NSLBP implications and hypothesis evolved from the peripheral context

Using the knowledge and precedent understanding of AMI at the peripheral joints, particularly the knee^[22,23,68], along with recent work from MF NMES^[5,6,16], and understanding MF MCLs^[17,51], the underlying basis of spinal MF-AMI can not only be implied but also understood through quantitative research.

This is supported by examples within the CNS neurophysiology, detailed above. This includes the analogous comparison of MF and quadriceps in the polysynaptic pathway of the flexion reflex with agonist flexion reflex excitability and concurrent reciprocal extensor inhibition^[68]. Also the neuroplastic change, as a response to neuro-motor sequencing and muscle re-activation, that advantageously facilitates innate spinal stabilization^[42,78]. Similarly the altered efferent corticospinal excitability following both joint injury and local manual intervention associated with local spinal muscle inhibition^[82] supports this premise.

It seems then a logical understanding to extrapolate the knowledge of peripheral AMI, particularly the knee, to the spine, as a recognized mechanism where central brain origin output and inhibition is a primary explanation. Consequently this would be, at least in part, an indicative explanation of the MF deficiency^[6], and by consequence that interventions directed at MF-AMI would facilitate NSLBP management^[5,17].

NSLBP: RECOGNIZED THERAPEUTIC INTERVENTIONS

Medical, general, non-surgical, and surgical interventions

Patients with LBP seek and are recommended in clinical practice guidelines to use symptom relief in a stepped approach stratified by duration^[3,7,8,48]. This approach is initially simple and non-interventional with moderate-quality evidence for advice, reassurance, and self-management. If improvement is insufficient, then more complex interventional techniques are considered including heat, massage, spinal manipulation, and acupuncture in the acute-subacute phase, which have low-quality evidence^[3,7,48]. Progression to medications, such as non-steroidal anti-inflammatory drugs (NSAIDs), but not Paracetamol or muscle relaxants, has moderate-quality evidence, being initiated only if non-pharmacological therapies are unsuccessful. This is advised to be under medical guidance and with caution, particularly if further medication progression is made to antidepressants, opioids, or for any prolonged medication use, which have moderate-quality evidence, and anticonvulsants or other (new) drugs, which have insufficient-quality evidence^[7,48,83]. At week 12, persistent pain is recognized and the recommended progression is stratified using a biopsychosocial (BPS) approach that employs patient reported questionnaires that screen for the risk of persistent problems and pain^[3,7,84,85]: where low risk indicates simpler less intense support with continued therapy and group exercises^[7]; while higher risk^[84] indicates structured exercise regimes^[86], psychological therapies including cognitive behavioral therapy^[82], and a multimodal^[7,85] and multidisciplinary approach, though the 'dosage' of input has no quality of evidence for a determined recommendation^[3,7,47,48]. With suspicion of radiculopathy or specific pathology, or non-improvement following four weeks of additional therapy, a specialist referral is recommended^[83].

This overall approach has gained consensus to achieve best practice^[8] in NSLBP and reflects general musculoskeletal problems. The approach is supported by recent systematic reviews with 11 'musculoskeletal pain care' summarized recommendations^[87] that parallel the stated NSLBP management^[6,7,85]. However, only three recommendations were specific to interventions, (education/information, physical activity/exercise, and adjuvant manual therapy treatment), but they were supplemented with a BPS approach (activity through work)^[87].

Other non-invasive, non-pharmacological interventions have mixed evidence in their effect. There is no evidence for therapeutic traction, heat, and US^[5,48]; insufficient^[48] or limited evidence for massage, non-structured exercise^[86], and transcutaneous electrical nerve stimulation (TENS)^[6]; and low to moderate evidence for photo-biomodulation (or laser therapy) and information/education alone^[48]; but

information/education improves significantly when combined with other therapies, particularly manual therapies^[16,48] and therapeutic exercise^[82], of which slacklining would be an example.

Invasive interventions have similar mixed findings in the evidence of their effect. These include: radiofrequency neurotomy, facet and nerve blocks, which have similarly varied findings with most recent meta-analyses questioning their use^[88]. Similarly spinal cord stimulation (SCS) is questioned, though high frequency multi-column-SCS added to optimal medical management has higher evidence for gained effects, particularly individuals with multi-level non-specific changes and central sensitization^[89]. Consequently, there remains a patient subgroup with significant symptoms and/or disability that fail with conventional management therapies and potentially require additional medical and invasive interventions^[6].

AMI specific interventions for NSLBP

Several therapeutic interventions are demonstrated as effective in countering the presence of MF-AMI, though evidence is predominantly derived from effectiveness determined in musculoskeletal settings, particularly the lower limb and the quadriceps post knee trauma^[23]. These techniques are generally achieved through a focus on local processes within two broad categories: (1) Modulation of joint afferent discharge; or (2) Muscle stimulation^[20,22] as detailed below (in [Supplementary material, Appendix 3](#)), and summarized in [Table 1](#). Consideration of management strategies for the lumbar MF-AMI that parallel those used for other AMI affected muscles, particularly quadriceps AMI at the knee, has occurred only recently through: involuntary-direct therapy *via* surgically introduced NEMS interventions^[5,16]; while conservative approaches look to explain and utilize existing voluntary-direct and voluntary-indirect MCE therapy^[6], and potentially involuntary-indirect therapeutic approaches, such as slacklining^[17].

Joint afferent modulation to reduce discharge: The joint afferent modulation techniques reduce the neural signaling such that the CNS receives a lower degree of neural information and, subsequently, lowers the muscles inhibitory levels. Some of these therapies appear transferable to other body regions, such as the lumbar MF, and include: very low evidence for US^[60] and vibration; low evidence for TENS^[6], disputed evidence for NEMS^[5,16] with reported findings both negative and positive^[5,16], and moderate to strong evidence for cryotherapy^[90].

Muscle stimulation and activation: The muscle stimulation techniques are achieved through four combinations of direct/indirect activation through voluntary/involuntary mechanisms. These target the restoring of stability, predominantly through the MF segmental stabilizing action^[6,54], either alone^[12] or with other 'core' muscles as previously noted^[14,15]. This includes open/closed/composite kinetic-chain resistance^[91] with fatiguing of the antagonist. However, many peripherally directed interventions for AMI may not be transferable to MF-AMI due to the MF diffuse fibers network, depth, additional overlying muscles, and that MF-AMI is virtually impossible to voluntarily activate^[6].

Consequently, there is no definitive consensus on which specific therapeutic intervention/s demonstrate the most effective management for NSLBP, including that due to MF-AMI^[6,13,85]. Ongoing disputes remain in the systematic reviews, meta-analyses, and RCTs, with discrepancies in guidelines^[7,8,48], even within the same author group^[83]. However, one area of consensus is management goals and aims, with recognition that for all NSLBP, including AMI affected joints and regions^[5,6], low cost, self-administered interventions, such as adjuvant therapeutic exercise with the potential to alleviate symptoms and disability, should be considered and investigated^[22,68].

Using published and current AMI cerebral-based research on peripheral joints, particularly the knee and the lower limb kinetic chain generally, the known positive effects from dis-inhibitory therapies that alter motor excitability, as cited in mild to moderate definitive scoping and systematic reviews^[22], can be used and extrapolated to provide relevant applications to NSLBP. This leaves slacklining as a unique rehabilitation exercise, in that it addresses the entire affected lower limb kinetic chain coupled with the lumbo-pelvic region as a single unit^[31,92], with the potential to overcome the presence of MF-AMI. This potential rehabilitation relationship between slacklining and MF-AMI for NSLBP management is significant, as slacklining presents an indirect-involuntary exercise therapy to address MF activation through a centrally mediated neural mechanism.

Table 1 Interventions that counter the effects of arthrogenic muscle inhibition

Modulation of joint afferent discharge		
Joint aspiration; intra-articular corticosteroid injection; nonsteroidal anti-inflammatory drugs; local anesthetic; cryotherapy; transcutaneous electrical nerve stimulation-TENS; electro-acupuncture; altering fluid distribution/capsular compliance		
Muscle stimulation		
	Voluntary activation	Involuntary activation
Direct	Therapeutic exercise; motor control exercise therapies: (1) Biofeedback/ultrasound guided; and (2) Individualized tailored hybrid convergence and divergence exercise-based approach of specific treatment of problems of the spine including: ‘movement system impairment’; ‘mechanical diagnosis and therapy’ (MDT); ‘integrated systems model’ incorporates ‘regional interdependence model’	(1) Neuromuscular electrical stimulation: (a) surgically implanted-effective; and (b) transcutaneous-ineffective; (2) Transcranial magnetic stimulation; and (3) Peripheral magnetic stimulation
Indirect	Therapeutic exercise: (1) Global/non-specific ‘core stabilization exercise’; and (2) Specific ‘core stabilization exercises’ including: ‘modern mind body’ incorporating: Yoga, Tai Chi, Qigong, Pilates, Alexander, Feldenkrais, Bounce-Back, Calisthenics, Gyrokinesis, Gaga, Core-Align and Human Harmony; and MDT	Slacklining – possibly, <i>via</i> reducing down regulatory inhibition

TENS: Transcutaneous electrical nerve stimulation.

SLACKLINING AND ADJUVANT THERAPEUTIC EXERCISE

What is slacklining

A complete understanding of the full neuro-motor mechanism of slacklining remains incomplete, though several hypothetical models are proposed^[28,92-95]. Most recently these models have been updated in an in-depth review with a revised paradigm proposed^[31]. The summary slacklining definition presented in this paper’s introduction can be expanded to include: Neuromechanical balance retention on a tightened webbing-band, fixed at each end, moveable in three dimensions during achievement of functional independence and dynamic stability from the interactions of the individual, where whole-body internal dynamics drive innate and learned responses to external environmental changes, that require adopting strategies that seek a compromise between maximum stability and minimal energy expenditure^[26,28,31].

Consequently as a complex task^[28,92], the individual learns, adapts, and adopts techniques that are self-developed neural strategies in response to the challenged balance and equilibrium^[30], and can be quantified using self-organizing maps, a class of vector learning algorithms with the capacity to explain visualization techniques, topological retention and high-dimensional data-sets^[94,96]. This results in lower-limb and core muscle activation as a primordial response^[97,98] for balance retention to ensure postural stability^[25,31] as a critical physical function^[99]. This is achieved through a combination of: neurological system controls from centrally derived dampening of the down-regulation that causes reflex inhibition at both the central^[78,100] and spinal segmental level^[71]; and learned motor skills, from muscle recruitment through higher demand^[78,101], that, subsequently improves coordination of movement and control^[11,31]. However, there is also the consideration of mindfulness and flow-experience^[77], the mind-body interaction that enables the movement-fluency^[79] required to achieve and pursue slacklining as a tool for social, pleasure, pre- or re-habilitation purposes^[29,37].

Efficacy of slacklining on AMI in NSLBP therapy

Slacklining has shown therapeutic promise in the clinical setting, particularly for the quadriceps AMI-inhibited post-trauma knee^[25,26,30]. Slacklining may induce over twice the activation of four standardized quadriceps exercises with less than half the effort^[30]. In employing this hypothesized basis, as previously detailed (presynaptic inhibitory actions on the CNS to over-ride central down-regulation induced muscle insufficiency^[31,78,100]), for AMI-MF NSLBP case studies^[17], the same neurophysiological, therapeutic-based reasoning of slacklining’s action applies to the lumbo-pelvic musculature. The AMI-MF is targeted, in unison with other core stabilizing muscles^[31], through an indirect-involuntary activation as a part of a whole body therapeutic exercise approach^[79]. Though the therapeutic practice and research is still in its infancy and definitive evidence are lacking, the concept appears viable^[17].

The consideration and inclusion of slacklining with other prevention and rehabilitation themes into NSLBP management derives from the sensory system’s contributing triad of, proprioception, vision, and vestibular somatosensory inputs^[102]. This is a consequence of slacklining’s unique properties^[30,31]: being a composite-chain

activity, *i.e.*, there is a weak link in the kinetic chain resulting in abnormal motor synergy patterns due to the contact surface of the loaded limb/s having free, partially supported, but unstable three-dimensional movement on a recoil resistance surface^[31,91]; and actions being distinctly different from other conventional balance activities^[27] and apparatus^[25,33]. The synergy of these qualities coupled with CNS contributions results in four integrated qualities^[30,31]: balance – equilibrium control of dynamic movement and center-of-mass within the base of support^[25]; postural control – positional control in space^[25]; muscle strength- muscular generated forces^[33]; and neuromechanical demand – integration of neurobiology and biomechanics^[28].

Evidence of the efficacy of slacklining in therapy and rehabilitation

The therapeutic direction of slacklining has progressively evolved. In particular the past decade has shown increased recognition of its adjuvant role in both the prevention and management of injury. In prehabilitation this is demonstrated in falls prevention in the elderly^[26], and specific sports including judo^[103], basketball^[33], badminton^[104], handball^[105], and football/soccer^[106]. In rehabilitation this is demonstrated in orthopedics^[17,30], neurology^[31,34,107] including systematic review support for reduction of falls and freezing of gait for Parkinson's disease^[36], sports training^[95], general physical training^[106], physical performance^[33,103], and recreation^[37].

For all forms of LBP the current recommendations for therapeutic exercise interventions suggest an individually tailored hybrid approach^[108] that accounts for individual preferences and abilities, and considers aligning areas of convergence and divergence that incorporate MCE^[14]. These approaches utilize a 'specific treatment of problems of the spine' (STOPS) approach^[109], where the key application principles are derived from four diverse areas. These are established evidence based/informed clinical physical therapy approaches where motor control is either a central or adjuvant feature^[14,108]: the kinesiopathologic mechanical LBP model of 'movement system impairment'^[110]; McKenzie's model of 'mechanical diagnosis and therapy'^[111]; individually tailored MCE for areas with assessed suboptimal features^[12]; and the 'integrated systems model'^[14] which is compatible with the musculoskeletal 'regional interdependence model'^[14]. This STOPS approach serves to guide clinicians and provide the platform for the proposed hybrid model^[14,108]. Consequently, slacklining can be seen to fit comfortably within a hybrid model^[14,108] as part of the STOPS approach^[109].

The inter-related effects of slacklining and AMI on NSLBP

To understand the hypothesized paradigm for how slacklining can address MF-AMI related NSLBP from the neuro-physiological perspective, it is critical to understand that AMI's defined CNS actions are present at each of the three levels^[21,23] as discussed previously. The presence of AMI, as a centrally derived presynaptic inhibitory action^[18,19,23], modifies existing down-regulation control with consequential local MF-AMI deficiency^[78,100]. This paradigm is particularly significant for exercise based NSLBP rehabilitation therapies that aim to facilitate normal neuro-motor sequencing^[93,94] for neuromuscular and proprioceptive impairments^[11,31,79], as similarly targeted in other musculoskeletal problems with cortical excitability deficits that alter function. Slacklining's action as an adjuvant therapy within rehabilitation simultaneously addresses this neural compromise by providing an override of central inhibition that enables indirect activation of the local neuro-motor inhibited muscles, where the presence of the lowest available IAP joint range inhibition initiates AMI through central mediated processes. This occurs immediately and directly at all CNS levels with significant implications for the MF-AMI related NSLBP, and the use of therapeutic slacklining. This therapeutic slacklining can be achieved with minimal required learning or therapist input^[30] to induce normalized function^[11,79] of the descending inhibition alterations^[21,31,59] and facilitate neuroplastic change^[41,94]. Consequently, in the presence of AMI there is "... the need to address cortical function early in rehabilitation ..."^[62].

This leaves an open and potential role for slacklining to be considered within 2-4 wk of any LBP rehabilitation initiation; but particularly NSLBP, when natural response progression falters, not simply as an alternative therapy and option of potential last resort. This therapeutic use could prevent the descent into persistent pain^[5,6], particularly in circumstances such as soft tissue trauma and operative interventions, where AMI is likely to ensue^[21,62]. The proposed model and paradigm shift on potential muscle specific activation, that this paper presents, can assist in closing what appears to be a 'knowledge gap', but may simply be a matter of interpretation, and consequently an 'implementation gap'^[112].

A practical implementation of therapeutic slacklining exercises can be derived from

the operational definition of 20-steps over 5-stages provided in previous publications^[30]. These progressive stages enable the patient and clinician to both document and sequentially progress the slackline exercise capacity as an adjuvant to other incorporated and multi-modal and multi-practitioner approaches in a manner that is both self-taught and self-progressed. The sequence of steps and stages act as a guide and can be followed or altered for the individual (Table 2).

SLACKLINING'S NEURO-PHYSIOLOGICAL ACTIONS THAT POTENTIALLY COUNTER MF AMI INDUCED NON-SPECIFIC LBP

As presented above and per the aims, to rationalize why and how slacklining should be effective in reducing NSLBP symptoms, the ameliorating capacity is a consequence of slacklining's neuro-motor action that addresses the basis of MF-AMI^[6,18-21,59], facilitating a reduction in down-regulated inhibition^[31,78,100]. Consequently, this reductive capacity causes signal redundancy in the cerebral pre-synaptic inhibition section of the neuro-motor closed loop feedback system^[102]. The action/s enable/s the brain to recourse to the normal or primordial signaling action^[97,98,113] of neural flow through the spinal cord to the peripheral nerves and the local musculature, subsequently reducing the effect of AMI. This mechanism is supported by studies suggesting that central sensitization is influenced by impaired descending inhibition signaling^[41]. This causes alterations in the individual's conditioned pain modulation (CPM) which is found in NSLBP patients. This slacklining action is hypothesized to occur through one or several of the four established neuro-physiological pathways detailed below^[41,114].

Finite neural delay

The finite neural delay system occurs within the brain's limits for 'time available processing'^[113,115]. To facilitate optimal use of available processing time during complex integrated neuro-motor control, such as that present with slacklining, the CNS combines and integrates: incoming/efferent signals through 'sensory weights', to ensure the required representation of all comparative contributions of each sensory system; and simplifies subsequent motor control through 'muscle synergies', where groups of muscles are combined as a common neural signal to control a range of movements, which simplifies motor-control^[102,116]. Further, these neuromuscular strategies can be adapted and modulated by the individual to the required capacity necessary to facilitate stability^[116]. However, despite these efficiency strategies, a significant neural load remains^[117]. Consequently, the total information is interpreted as a 'package'^[102], and as such requires processing time that exceeds the available finite neural delay; this consequently allows an override of existing down regulatory inhibition. Slacklining, being one such complex neuro-motor action, could induce such sequence override^[31].

This is further supported by the understanding that nervous system processing is a principle consideration in relation to neural delay. This is due to the variations in different tissue-specific neural conductive pathways^[118] and subsequent delays that result in varied reaction times^[119] under different stressful situations^[120]. These systems detect changes in desired positional orientation and react in an integrated manner. This maintains functional balance-control through a closed-loop feedback system with varied constraints on the sensory integration process, including that of the 'sensory weighting' discussed above^[102,116]. Consequently, changes in motor activity or movement occur after nervous system processing^[121], and are predominantly primordially protective responses designated to prevent injury^[97,98,113].

NSLBP movement-control phenotypes, exercise reasoning hypothesis

The presence of two trunk movement control phenotypes^[54] has been proposed as a model to facilitate the understanding of the inter-relation within trunk motor control between muscle activity and kinematics in LBP individuals^[122]. In such a model there is a proposed spectrum continuity anchored at each end by control that is 'tight' and 'loose'. This may also be influenced by CPM response strategies that have a bias toward a pro-nociceptive phenotype in individuals presenting with NSLBP. Both the 'tight' and 'loose' control models are proposed to have beneficial effects, where 'tight' control protects against large tissue strains from uncontrolled movement, whereas 'loose' control protects against high muscle forces and resultant spinal compression^[54]. This concept is consistent with Panjabi^[9]'s modified model of spine stability that was

Table 2 Slacklining progressive competency phases—5 stages and 20 steps^[30]

Stage and steps	Description of position
1 – Beginner: Stand	Each description of stages 1-4 is for the slackliner standing on a slackline of 3 m length at strong tension anchored at each end 25 cm above soft terrain such as sand or grass
1	Single leg stand – on the dominant leg
2	Single leg stand – on the non-dominant leg
3	Single leg stand – on dominant leg, other foot touching the side of the line 1 foot length in front of the weight-bearing foot
4	Single leg stand – on dominant leg, other foot touching the side of the line 1 foot length behind of the weight-bearing foot
5	Single leg on non-dominant leg, other foot touching the side of the line 1 foot length in front the weight-bearing foot
6	Single leg on non-dominant leg, other foot touching the side of the line 1 foot length behind of the weight-bearing foot
2 – Moderate: Walk	
1	Walk forward along the line with minimal to no pause between steps
2	Walk backward along the line with minimal to no pause between steps
3	Tandem stance with the dominant leg back or closest to the anchor point
4	Tandem stance with the dominant leg forward or furthest from the anchor point
3 – Intermediate: Tandem	
1	Tandem stance with the dominant leg behind, then turn or pivot 180° on both feet to the natural side so that the dominant then becomes forward
2	Tandem stance with the dominant leg forward then turn or pivot on both feet to the non-natural side so that the dominant leg is behind
3	Tandem stance with the dominant leg behind, then turn or pivot 180° on the dominant foot to the non-natural side so that the non-dominant foot crosses over and returns to the forward position
4	Tandem stance with the dominant leg in front, then turn or pivot 180° on the non-dominant foot to the non-natural side so that the dominant foot crosses over and returns to the forward position
5	Side stand ‘surf posture’ – feet perpendicular to slackline and balance
4 – Advanced: Squats	
1	‘Surfer’ position and squat down feet perpendicular to the line approaching buttocks to the line
2	Squat in tandem, dominant leg behind – feet along the line approaching buttocks to the line
3	Squat in tandem dominant leg in front – feet along the line approaching buttocks to the line
4	Single leg squat all weight on the dominant leg – approaching buttocks to the line
5	Single leg squat all weight on the non-dominant leg – approaching buttocks to the line
5 – Extreme	Without using arms, without sight, bouncing
Other – tricks: Performance	Heel raises, walking on toes, jumps, spins, somersaults on line or as dismounts
	External focus (<i>e.g.</i> , throwing ball, juggling ball)
	Surfing (on very slack line) with oscillations or swinging perpendicular to the line

Slackline length and tension can be changed to modify the difficulty level.

evolved to include motor control^[11]. Should these proposed differential movement control phenotypes exist, then interventions using different motor-control exercises would be required, or a single exercise that provides the ability to address both forms^[54,122], particularly if they truly are parts of the same spectrum^[14]. Slacklining as a unique complex neuromechanical action could potentially be such an exercise with this dual capacity^[30,31].

The inhibition of action and the innate primordial imperative

A primary brain function is behavioral organization to determine ‘action’ or ‘inhibition of action’^[198]. This involves activation of the behavioral inhibition system (BIS)^[97], which occurs in the presence of threats and endangered health, to enable choice of the least

detrimental option, *e.g.*, injury is preferred to death. It also, additionally, occurs in three circumstances when: no previous response pattern exists; danger cannot be predicted; or the instinctive fight/flight/freeze response (FFFR) is impossible^[97]. The FFFR is instigated by fear and relates to individual underlying differences and experiences that, consequently, affect the level of adrenaline production. All three additional BIS circumstances would essentially be present in an initial exposure to slacklining^[30,31]. Consequently, this BIS activation pathway may explain the pre-synaptic AMI override, but such an assumption must be taken within the context of reinforcement sensitivity theory (RST). This RST predicts that the BIS role in coping-motivated problem solving through its central role in anxiety is moderated by the behavioral approach system^[97]. Further, it is important to recognize that BIS activation causes neuroendocrine responses; these involve multi-adrenocorticotrophic hormone production by the hypothalamus, pituitary and adrenal axis, as part of the stress response. This in turn leads to glucocorticoid secretion and elevated cortisol levels, which if prolonged or chronically elevated have detrimental consequences including reductions in hippocampal volume which, subsequently, effect spatial and hippocampal-dependent learning and memory task capacity^[15].

Preservation of existence is every organism's primordial imperative^[97] as the brain's purpose is to ensure survival, through this aforementioned action, or inhibition of action^[97]; not simply to think, but to act. This is because humans exist to maintain the structures that sustain life^[98] and to reproduce for the optimal potential of natural selection through self-organization. The nervous system, consequently, enables species to act both within and upon their environment with the intended purpose of survival^[99]. As such, the actions of slacklining through pre-synaptic inhibition could result from the consequential role of the inhibition of action and the innate primordial imperative^[97,98].

Accentuated corticospinal drive

Positive influence on corticospinal excitability occurs in the presence of individual muscular feedback and joint pathology^[68]. These cause an accentuated modulation in the CNS corticospinal drive^[21]. The consequential effect is an override of pre-synaptic inhibition that enables a counteraction to the α -motoneuron inhibition by the spinal reflex pathways^[23]. This positive influence on local AMI influenced muscles^[69], particularly as demonstrated in the knee^[22,23,30,31], could account for the capacity to initiate MF activation through the various direct and indirect, voluntary and involuntary mechanisms previously discussed.

CONCLUSION

Slacklining is a strategic indirect-involuntary therapeutic exercise that can facilitate the activation of MF-AMI deficient muscles; however, a thorough understanding of its neuro-motor mechanism of action remains incomplete. Slacklining has demonstrated clinical effects that overcome AMI in peripheral joints and recent case-studies indicate slacklining's potential in reducing MF-AMI related NSLBP. Slacklining's actions of effect are proposed to be provided by an: over-ride and inhibition of CNS down-regulatory induced MF-AMI, which normalizes the MF neuro-motor sequencing and muscle re-activation. These actions in-turn suggest slacklining as a potential adjuvant therapy that may address and reduce NSLBP symptoms and disability. Slacklining is simple to administer, use, and progress. Existing slacklining research on the neurological basis of effect, particularly in relation to AMI, has adequate acceptance to facilitate its introduction into NSLBP rehabilitation. However, what appears as a 'knowledge gap' could simply be interpretive, and merely an 'implementation gap' where the rehabilitation clinicians prescribing protocols are yet to recognize the full potential of this ancient excise and take it back to the future. Further investigation is required in research, cohort, and clinical populations to determine slacklining's efficacy, dose-response, and optimal time of implementation during pre-rehabilitation in managing MF-AMI deficient NSLBP.

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