Supplementary material

APPENDIX #1: HISTORICAL PERSPECTIVES: SLACKLINING AND AMI

1.1 SLACKLINING

Slacklining is a modern 'trend-sport' being an adaptation of the ancient performing art of traditional rope walking[30, 123]. It was initially started by climbers and outdoor enthusiasts in the 1960s and 1970s in the European Alps of Switzerland, France and Austria, as well as the National Parks in East and Central USA, where ropes and cables were replaced with adapted lightweight webbing and ratchet technology that incorporated an easier, safer and more elastic line[37]. Slacklining was originally termed 'Line-walking' or 'Funambulaire' in Latin European regions, and 'Jultagi' or 'Eoreum' in Korea. Slacklining presumably began from necessity in quite ancient times when rope-making originated (40,000-15,000BC)[124] for fastening and construction[125]; where movement along ropes enabled passage between elevated structures and fixation points, including dwellings, across gorges, and eventually on boat-sails[125]. It developed into a performing art, social activity and recreational past-time, with marked notoriety in Ancient Greece and Roman times (circa 15,000BC-500AD) and the late 19th century[37]. The graded incorporation into rehabilitation was possible at any time, though medical literary writings by Galen-of-Pergamon insinuate around 150AD being the most likely[126]. Though slacklining's first published scientific literature was not till around 2009[29, 123], modern formal pre-/re-habilitation started in the 1980s[29, 30] when slacklining's applications were almost entirely for musculoskeletal conditions.

1.2 ARTHROGENIC MUSCLE INHIBITION (AMI)

In contrast AMI was likely first detailed by Hippocrates-of-Kos around 450BC[18, p314, 61] as '*atrophy associated with luxation of the femur at the knee'*. However, a more considered understanding of its basis

took till around 160AD when the father of 'Exercise in Medicine', Greco-Roman physician Galen-of-Pergamon noted '*wasting in the absence of local muscle or nerve damage'*[126]. This muscle-wasting phenomena was also referred to by the distinguished 18th Century Scottish Surgeon-Scientist John Hunter as a '… *sympathetic relationship between the muscles and the joint'*[18]. In-depth scientific definition as '*reflex inhibition*' was provided by several 19th century scientists, most notably the French with Vulpian (1875)[58, p329] noting in his seminal publication that '*reflex atrophy… is the result of a modification caused, in … the gray matter of the cerebrospinal nerve center, by the irritation of the peripheral extremities of certain sensory nerves'*. This centrally applied reasoning was also supported by the English Surgeon-Pathologist Sir James Paget as a '… *reflex action influencing the trophic centers in the cord'*[18].

The progression to the terminology of AMI was initiated by Harding[18] in her 1925 publication entitled *'Arthritic Muscular Atrophy',* and, subsequently, modified to *Arthrogenic Muscular Atrophy',* which was retained[127]. Over the ensuing decades other researchers and scientists continued to use both this terminology and 'reflex muscle inhibition'. However, the 1984 seminal paper by Stokes and Young[19] introduced the term '*arthrogenous muscle weakness',* before the current terminology of *AMI* was formalized by Hurley[57] in 1994, which has been adopted consistently since. Each terminology, including the Greco-Romanic patient descriptions by Hippocrates and Galen, have referred to the protective inhibitory action, leading to muscular atrophy, predominantly around injured peripheral joints[18, 20, 23], particularly the knee[19, 21, 22, 58], but also the hip[78], ankle[74], elbow[57], and shoulder[18], and most recently an expanded recognition to include the lumbar spine[6, 16, 17].

APPENDIX #2: NEUROPHYSIOLOGICAL CONSIDERATIONS FOR AMI

The CNS neurophysiological aspects associated with AMI at the local, spinal and central level, as detailed in the main manuscript, can be supplemented.

1. LOCAL/PERIPHERAL PATHWAYS IMPLICATED IN AMI

Local level changes result from sustained tissue damage causing altered afferent discharge in both muscle resting motor thresholds and articular sensory receptors[6, 19, 20, 58, 64].

1.1 Articular Sensory Receptors

Articular sensory receptor firing can vary due to the opposing effects that different joint afferent populations have on motoneuron excitability. Joint structural trauma and local sensory nerve ending damaged *decreases* afferent output[59, 65], whilst joint distention and laxity *increases* output which reciprocates with AMI[21]. The articular sensory receptor processing roles, in relation to AMI, are confirmed by the effects of small levels of inflammation or fluid within a joint space, e.g., 20-60 ml in a knee reduces' maximum quadriceps peak torque 30-40%[23, 62]. However, local interventions dramatically affect AMI: being abolished through either a local anaesthetic (LA) injection or aspiration; or prevented, by use of a LA injection prior to joint effusion[68].

1.2 Joint Laxity

Joint laxity facilitates increased intra-articular movement which increases mechanoreceptor and nociceptor activation. This further alters sensory receptor activation and indicators of joint limitations leading to AMI.

1.3 Distention from Swelling

Any swelling distends the joint, which increases the IAP and the pressure-sensitive and stretch mechanoreceptors Group II afferents discharge. This inhibits periarticular muscles α-motoneurons

through spinal cord Group-I non-reciprocal (Ib) inhibitory interneurons, resulting in local and potentially contralateral AMI[23, 62] without need for local structural damage[58, 61] or inflammation[19, 68, 69].

1.4 Distention from Inflammation

Distention from inflammation causes AMI as with distention from trauma related swelling, however it is due to peripheral sensitization and subsequent nociceptive signaling, including reduced activation threshold in the articular free nerve endings supplied by Group III and IV joint afferents[67]. It has been shown that these Group-III (lightly myelinated) and Group IV (unmyelinated) nerve endings are high-threshold, constitute the majority of afferent fibers innervating joints, where they function predominantly as nociceptors and mechanoreceptors, that signal potential and real joint damage from structures that respond to strong chemical, mechanical, and thermal stimuli. However, they are also activated by local mechanical stimulation and passive movements both of which are non-painful[67]. Consequently, articular structure stimulation from normally non-noxious mechanical sources causes Group III and IV afferent discharge[67] which is compounded by inflammatory mediators' that sensitize free nerve endings innervated by Group III and IV afferents.

2. SPINAL PATHWAYS IMPLICATED IN AMI

2.1 The Group Ib non-reciprocal inhibitory pathway and Group Ia, II, III and IV Fibers adjuvant role

The Ib spinal interneurons, within the spinal cord grey matter column lamina VI-VII, integrate sensory-motor input through signal relay actions between sensory-afferent and motor-efferent neurons. Afferent signaling derives from the musculo-tendinous junction adjacent the Golgi tendon organs, supplemented by convergent input from joint afferents and peripheral sensory receptors, namely large, myelinated afferent Group II and thinly myelinated/ unmyelinated Group III and IV fibers[72]. The Group Ia sensory fibers, from the muscle spindle stretch receptor, and Group II afferents, terminate in corpuscular nerve endings being activated by mechanical based pressure and stretch stimuli. The Group

III-IV muscle afferents play a dual autonomic control role via the Ib interneurons to mediate both central and peripheral fatigue response during exercise. This occurs via: facilitating inhibition and reciprocal excitation, through polysynaptic-pathways, to affect ventilation, cardiac output and, consequently peripheral blood flow. The central projections limit exercise via negative output from spinal motoneurons that decrease voluntary muscle activation which enhances central fatigue[72], a documented phenomenon with slacklining, particularly with neurological patients[31, 34] and reported implications with NSLBP patients[17]. In addition, the Groups III/IV have two subtype afferents: one responding to general aerobic exercise via endogenous intramuscular metabolites; the other to noxious muscle metabolite levels from ischemic contractions.

2.2 The Flexion reflex

The flexion reflex polysynaptic pathway with agonist excitability and reciprocal extensor inhibition is demonstrated in the quadriceps[68]. A similar pathway response has the potential to influence the MF and NSLBP, though to a low degree as there is no link between the flexion reflex response threshold and AMI severity[68, 73].

2.3 The Gamma (γ)-loop

The *γ*-loop spinal reflex circuit enables automatic regulation of muscle tension levels that ensures full muscle activation during voluntary contractions. The *γ*-motoneuron pool controls muscle fiber contraction within the muscle spindle and transmits excitatory impulses to the *γ*-motoneuron pool via Ia afferent fibers; while the α-motoneuron pool controls skeletal muscle contraction[73].

2.4 Spinal cord neuroplasticity

Spinal cord neuroplasticity contributes to human motor learning as neural reorganization and adaptive changes to learning complex skill tasks are time dependent. Initially the changes are ‘acquired’ as a general adaptation but after approximately 24 hours these adaptations become ‘retained’ and

task-specific. Consequently skill training induces a time-dependent reorganization of the spinal networks modulation that reflects the process of feed-forward motor command as an optimization process that is time-dependent[71].

3. SUPRA-SPINAL/CENTRAL PATHWAYS IMPLICATED IN AMI

Supra-spinal/central changes related to AMI are hypothesized as predominantly being from the supraspinal projections from the joint afferents[21, 75]. The changes influence the four areas summarized within the main manuscript and are further detailed below: cortico-spinal excitability/activity [22]; brainstem descending pathways[76] and the flexion reflex[75]; individual voluntary effort[21]; and 'informed awareness' through 'flow-experience'[77]. These supra-spinal actions, subsequently, affect the capacity and presence of both neuroplasticity[78] and resultant homunculus motor smudging[42, 43], as well as the individual desire and intention to achieve movement-fluency as reflected in HRM[79], more-so than simply achieving the activity and exercise itself. Consequently, the normalization of peripheral muscular activation is the primary goal to facilitate global equilibrium control, as central sources override down regulatory inhibition that enables muscles, normally repressed by central inhibition from traumatized/distended joints negative afferent input, to be re-activated.

3.1 Changes in Corticospinal Excitability

Changes in efferent corticospinal excitability activity as a consequence of joint injury, distention, and local manual intervention, is well documented as being associated with local muscle inhibition at the knee[81] and spine[82]. This includes the presence of AMI, as the descending pathways extend widely into the spinal level interneurons and motoneurons[75], which provides the capacity for strong influence[21-23]. These excitability changes at the area of the primary motor cortex projecting to the muscle's motoneuron pool can be either: decreased, due to lower muscle activity levels and diminished spinal-reflexive

excitability; or paradoxically increased, due to increased cortico-spinal drive[81]. This latter circumstance occurs despite reduced local muscle activity levels and capacity, as excitability increase consequentially counteracts the α-motoneuron inhibition by the spinal reflex pathways as the brain attempts to unsuccessfully increase the AMI affected muscle activation. This local AMI muscle affect is present as a consequence of the down regulatory pathway inhibition preventing final signal distribution[6, 21, 73]. This preventative action accounts for how various voluntary, involuntary, direct, and indirect mechanisms, previously discussed, provide effective MF activation locally or centrally by overcoming the inhibition, with such cortical changes being quantified with motor cortex Transcranial Magnetic Stimulation (TMS)[23, 42, 63]. Despite this mechanistic understanding of corticospinal involvement in AMI, supporting evidence for its presence in experimental knee joint effusion has been questioned[23]. It has been determined that the neural activation deficit could be sustained simply by ongoing spinal reflex inhibition, however, this cannot be generalized to all regions, conditions, and patient groups[23]. Recent investigations in patients with rupture of the knee ACL, affirmed the generally agreed assumption that a link to central brain origin output and inhibition would explain the presence of quadriceps AMI[22], and by extrapolation, this would be indicative of the deficiency in the MF[5, 6]. Consequently, there is a difference in brain motor area activation between individuals with and without AMI, which can be visualized with brain functional magnetic resonance imaging (fMRI)[43, 63, 69].

Altered electro-cortical brain activity occurs in the somatosensory cortex, with relation to the control of working memory processes, and subsequent neurocognitive function variability[128, 129]. This is due to changes in afferent proprioceptive information from the affected joint via the neural supraspinal projections to the recipient centers during essential movement and joint position sense tasks[77, 129]. This results in intra-cortical inhibition and the need for: increased frontal cortex Theta power, which is related

to higher focused attention and measured by electroencephalogram (EEG); and decreased parietal Alpha-2 power, which is related to differences in sensory information processing[22].

3.2 Brainstem Modulation and the Flexion Reflex

Descending brainstem pathways normally provide tonic inhibitory control over the varied spinal neurons involved in pain processing[76], and the flexion reflex[75]. Consequently there is modulation of efferent commands due to afferent information. In the presence of injury and inflammation the descending brainstem pathways' input is notably affected with paradoxical negative and positive components. Consequently, local joint damage can cause either: reduced descending inhibition effectiveness[59]; and/or enhanced descending facilitation[75]. This results in excitability increases in the flexion reflex pathways, and subsequently increased AMI as demonstrated in the knee[21, 68], and postulated in the spine[5, 6].

3.3 Reduced Voluntary Effort

Voluntary effort directly determines the level of muscle activation as a reflection of individual motivation and exertion. It has been speculated that muscle strength and activation reductions may be partially a consequence of adjustment in subconscious voluntary effort due to fear avoidance of a pain response[76], or further damage to the injured joint[59, 69]. However, to date, the experimental models utilized have not substantiated this hypothesis, and observed reductions in quadriceps activation are deemed to be primarily reflex related due to articular afferents, and not related to individual voluntary conscious or subconscious volition changes. Similarly, this is supported by the statistically significant reduction in perceived and observed exertion with concurrent statistically significant increased quadriceps activation during slacklining for subjects with post trauma knee AMI[30, 31]. However, such altered voluntary effort cannot be completely excluded[21].

3.4 Informed Awareness and Flow

'*Informed awareness*' is information about oneself in relation to the surrounding environment, and plays a critical role in survival through active positioning of conscious and unconscious actions. By contrast, 'flow' is the harmonious psychological state, involving movement-fluency and task/activity-absorptivity[77]. It is proposed that '*informed awareness*' can be re-conceptualized as '*flow experience*' when exhibiting certain *behaviors*, or performance activities, that require highly complex movements, such as slacklining[77]. The *behavior-experience* coupling can be empirically measured: *behavior* by the 'Hurst (H) exponent' (which quantifies whether a time-series regresses toward the mean or exhibits directional clusters)[130], as determined by accelerometry time-series and CoM; and *experience* on the 10-item 'Flow Short-Scale', where the total score derives from sub-scales on movement-fluency and absorptivity[130].

As AMI is influenced by supra-spinal projections, cortical alterations influenced by '*Informed awareness',* due to the interactive behavior-experience coupling, could influence pre-synaptic inhibitions through either a reductive or heightened input-level and, subsequently, change the level of AMI. This would potentially be under the FFFS from the 'primordial imperative'[97, 98], where brainstem survival reactionary influences cause 'inhibition of action'[97] through the cumulative balance between the BIS (*behavioral inhibition system)* and BAS (*behavioral approach system),* as outlined in RST (*reinforcement sensitivity theory)*[98].

APPENDIX #3: AMI SPECIFIC INTERVENTIONS AND APPLICABILITY TO NSLBP

The therapeutic interventions with a demonstrated effect in countering AMI have been predominantly employed in the lower limb[23, 74], but the basis of their effect should equally apply to NSLBP. These applications are discussed in detail in this section and considered within two broad categories: 1) *modulation of joint afferent* discharge; and 2) *muscle stimulation*[20, 22].

1) JOINT AFFERENT MODULATION TO REDUCE DISCHARGE

These techniques intend to reduce the neural signaling such that the CNS receives a lower degree of neural information and, subsequently, lowers the muscles inhibitory levels. These include:

i) Joint Aspiration, particularly from acute effusion that damages articular receptors and changes capsular compliance[131]; however, for chronic situations the clinical value is limited due to local pathologically induced reoccurrence.

ii) Intra-Articular Corticosteroid Injection in the presence of strong inflammatory conditions, such as rheumatoid arthritis (RA), via the reduction in effusion afferent signaling; however, in osteoarthritis (OA), the effects were non-significant[68] .

iii) Nonsteroidal Anti-Inflammatory Drugs (NSAIDS) have conflicting evidence. They assist AMI reduction in the presence of acute joint damage via effusion afferent signaling reduction, particularly with strong inflammation from joint pathology. Negative effects relate to the analgesic action which results in increased joint load with gravity based actions such as gait or loaded standing, sitting, and manual handling[48, 83] .

iv) Local Anesthetic (LA) also has opposing actions as it reduces joint afferent impulses, which, subsequently, reduces AMI. However pain and AMI reduction are not directly related, as AMI can remain in pain free joints[18, 19, 21, 62]. Further LA is invasive, short-acting, and impractical in clinical settings due to the number of injections required and the infection risk.

v) Cryotherapy provides temporary AMI reduction of up to 30 min. The advantages are that it is non-invasive, counters the H-reflex amplitude decline normally associated with joint swelling, negates joint movement deficiencies, including peak torque and power found in the knee; and provides an opportunity for adjuvant therapy, of which slacklining is an example, that facilitates or assists muscle activation[69].

vi) Transcutaneous Electrical Nerve Stimulation (TENS) enables increased muscle activation during and maximal voluntary contractions that vary with the output frequency. High-frequency (120 Hz, pulse width, 0.1 sec) negates the H-reflex amplitude, while low-frequency (4 Hz, pulse width, 1 sec) increases muscular force output, indicating improved voluntary activity[6].

vii) Electro-Acupuncture has a recognised analgesic effect, particularly in the presence of peripheral joint OA, which in turn improves *health related quality of life* (HRQOL). However, MF's depth and overlying soft tissue make this low evidence therapy for MF-AMI NSLBP[6].

viii) Altering fluid distribution/capsular compliance induces a positive joint signaling effect, particularly though the use of non-weight-bearing, submaximal contractions, and consequent local joint movement, particularly at the knee. This enables dispersion of intra-articular effusion and improved capsular compliance. Specific knee studies found positive results that have potential implications for MF-AMI related NSLBP[6].

2) MUSCLE STIMULATION AND ACTIVATION

Therapy directed to target muscle stimulation and activation aims to restore stability, predominantly through the MF segmental stabilizing action[6, 54], either alone[12] or with other 'core' muscles, as noted previously[15]. This is achieved through four combinations of *direct/indirect* activation through *voluntary/involuntary* mechanisms.

i) Direct-Voluntary techniques use the various singular or hybrid approaches through traditional MCE. These can be furthered with biofeedback, though with variable results and evidence levels[6, 13].

ii) Direct-Involuntary techniques are based on stimulatory interventions for non-activating AMI quadriceps[23]. These include:

1. *NMES* via a surgically implanted electrical stimulation device. This episodically contracts the MF[5, 6] via the L2 dorsal ramus medial branch[16]. However, the activation timing is non-specific and unrelated to function. The reported findings are both negative and positive[5, 16].
2. *Transcutaneous NMES* is a superficial technique and unsuitable as activation is nonspecific due to soft-tissue interference from overlying muscles, MF being a deep position, and its diffuse nature[5].
3. *Transcranial magnetic stimulation* (TMS) affects the required muscle via motor cortex induced changes in the central activation ratio (CAR)[44]; however, the associated costs appears highly prohibitive for widespread clinical uptake.
4. *Peripheral/transcutaneous magnetic stimulation (PMS)* is a noninvasive, painless muscle stimulatory technique delivering a rapid and pulsed, high-intensity magnetic field to activate the muscle via its motor nerve without brain involvement[132].

iii) Indirect-Voluntary techniques activate the deficient MF by either: specifically targeting the core muscle group as a whole, while incorporating the MF[6]; or through an overflow to the core from a global or whole-body exercise approach, particularly using the aforementioned MMB (*Modern Mind Body*) regimes (see Table 1). These include but are not limited to Yoga, Tai Chi, Pilates and so forth[79], as well as sling/suspension therapy, and may also include McKenzie's manual diagnostic therapy (MDT)[111].

iv) Indirect-Involuntary techniques are not currently recognized but would involve a strategy enabling involuntary activation that recruits the MF muscle indirectly. This would occur via: a reflex reaction, or centrally mediated activation process[78, 100]; or one that counters existing down-regulatory influences, such as those causing AMI[30, 31], as with slacklining. Consequently the lack of local activation is overridden.

A major obstacle to such exercise programs is that voluntary MF muscle control can be difficult to teach and the MF itself is virtually impossible to activate with efficacy and effectiveness in the presence of AMI[6, 44]. Further, each mentioned exercise strategy, apart from indirect-involuntary techniques, focuses

primarily on trunk/peripheral-based activation processes without accounting for CNS mediated down regulatory actions.

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