

An examination of the muscle amnesia theory

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OVERVIEW

The theory of muscle amnesia advances the idea of insufficient neural activation as a common cause of muscle dysfunction. The theory has so far gone unaddressed within the literature. The author presents here a review of this theory, together with a review of the surrounding literature, in order to assess its claims. The conclusion is that, rather than insufficient neural activation, it is the mechanical properties of connective tissue and the interaction of this with the neuromuscular system that drives the observed problems. This interaction shapes function and adaptation, mediating the transfer of muscle-generated forces, and thus determines how the system can respond to imposed demands and the resultant adaptations.

Problems with the muscle amnesia theory

The theory focuses on insufficient muscle use as a cause of diminished neural activation. This insufficient use is attributed to large periods of sedentary behaviour,^{23,43,47,68} which can have an impact even if regular, periodic physical activity is undertaken.^{47,68} However, the research does not support this: some disuse models have found no change, or even increased neural activation.²⁷ Although other studies do report a reduction in maximal neural activation, this is tempered by an increase in submaximal activation.⁸⁶ The spaceflight model used in Edgerton²⁷ may be of more relevance in this discussion as it represents a reduction in load, albeit severe, and not a cessation of movement, as seen in the limb suspension model of Schulze.⁸⁶ Within the context of the muscle amnesia theory, this would seem to better apply.

Setting such considerations aside, yet further doubt is cast by Schulze et al.⁸⁶ They showed that, even with limb suspension, resistance training every third day maintained normal neural function. This preservation of function was further supported by Gordin.³⁵ Taken together, the findings show that peripheral neural function, at least, is well preserved during periods of reduced loading and large periods of disuse, provided that intermittent use and loading occurs.

When examining the claims derived from lower cross syndrome, support is found for static stretching of a

Introduction

Muscle amnesia is a theory that seeks to explain muscle dysfunction, attributing it to diminished neural activation.^{23,43,47,68} Despite a wealth of articles being published, the theory has yet to be critically examined, with no peer-reviewed literature addressing the claims. The articles written are inconsistent in how they describe the phenomenon, confusing what the involved mechanisms of the theory are proposed to be. However, common through all descriptions is the theory that neural activation of affected muscle diminishes, with reduced strength and, consequently, function.^{23,43,47,68} Commonly cited for this is the lower cross syndrome, although not always explicitly named.⁴³ The idea is that excessive tightness and/or shortness in one muscle leads to another being chronically lengthened, the increased length eventually eliciting depressed neural

signalling. The prominence of lower cross syndrome within muscle amnesia has led to it generally being discussed with regards to glutes, with terms such as gluteal amnesia and dead butt syndrome common.

The predominance of discussion around glutes and lower cross syndrome often focuses attention around disuse on sitting.^{43,68} The problems recorded include compromised function, poor movement and coordination, apparent difficulties in getting the muscle to effectively contribute, and even pain and pathology.^{23,43,47,68} However, without investigation, the efficacy of the claims made within the theory cannot be determined, and its implementation is therefore left to question. This paper seeks to provide an initial assessment of these claims: in order to do this effectively, the theory – as well as the observed problems it attempts to explain – are discussed.

muscle diminishing neural activation.³⁴ However, the general nature of the claim needs further examination. It is established that sarcomeres will adapt their length in series to optimise their contractile capabilities.^{11,36,42,110} Evidence also exists to show how the system can adapt to range of movement (ROM) demands, bringing it within tolerance and eliminating stretch stimulus at a given position.^{11,89} The evidence of diminished neural activation is found only acutely after stretching,³⁴ with values returning to normal over time. With the system's adaptive capabilities, any stretch induced by a habitual position will cease to elicit a stretch response over time. Muscle force production is also protected by adaptation of sarcomere length.

Although loss of neural activation is indicated in arthritic elderly⁹⁸ and complete disuse populations (a population who, either for a specific limb or in totality, have no movement at all – either for a set time, eg, where a bone break has been cast, or permanently, eg, those who are wheelchair-bound),^{16,35,86,98} in healthy populations activation is completely preserved with very minimal loading.^{27,35,86} This fact conflicts with the theory's triggering mechanism, and the universality with which it can be applied. Furthermore, there does not seem to be scope for chronic positions to apply continual stretch to a muscle, as the system will adapt to accommodate this. Without the chronic stretch, any compromising of neural function is a temporal event, and normal function will return. The literature therefore does not support the theory within healthy populations.

However, two points remain to be answered:

Are the observed problems of the theory of neural origin?

Is the uptake in the theory due to efficacy of the treatment?

Physiology and mechanics

This section details how mechanics and physiology interact: it also provides evidence for an inextricable intertwining of the two. The evidence provided here shows the influence of imposed mechanical demands on our

physiology, the system response, and the resulting internal mechanics. With the nervous system's resiliency to low usage in healthy populations, this relationship between physiology and mechanics holds possible answers to the remaining questions.

BIOTENSEGRITY: A MODEL FOR THE BODY

Biotensegrity is a model of construction for the human body, where the soft tissues are tensional elements and bones are intermittent compression resistant elements floating within this tensional frame. The biotensegrity structure being described has two elements: tension (which is continuous) and compression (which is intermittent). Bones are those elements that deal with compression.^{13,25,56,99} (See ref 81 for a more in-depth review.)

Apart from its general importance for examining forces and mechanics within the body, biotensegrity's place in this discussion comes from the anatomy of connective tissue and muscle. The general connective tissue is an extensive tissue interconnecting and wrapping all tissues and organs,^{1,109,113} and is crucial in creating tension. As bone floats within the tensional frame of the soft tissues, muscle floats within connective tissue.^{10,117,75,80,83,93,99,104,115} Images show that when muscle is removed from frame, connective tissue creates a honeycomb-like structure, surrounding and encapsulating the whole muscle fibre.⁷⁵ Muscle therefore is an isolated contractile cell engine, only linked to the body through its place within the tensional frame and the tissues that form it. Force transfer from muscle to the broader system wholly depends, therefore, on the surrounding connective tissue; this relationship forms the resultant movement.

MECHANICS AND ADAPTATION

The basics of mechanics centres on the stress/strain curve. Stress is the internal distribution of an applied force, calculated as applied load divided by cross-sectional area,⁶³ and strain is the degree of deformation.⁶³ Although inert materials such as steel have linear stress/strain curves,⁶⁷ biological soft-tissues possess slightly non-linear curves,⁵⁰ although often presented as linear.⁸¹ The angle of the slope on the curve is known as stiffness,⁵⁰ and describes the amount of stress required to elicit a given amount of strain.

The more the slope moves towards stress, the greater the stiffness and the more stress required to create a given level of strain.

Stiffness relates to biotensegrity through tension. Tension is a type of strain; what is shown by stress/strain curves is the greater the strain the more stress required to create further deformation. Within the biotensegrity model, the more tension under which the system is held, the greater the system stiffness. Therefore, in this discussion stiffness and tension can be viewed as intertwined properties.

Adaptation is heavily influenced by mechanics. When a mechanical demand is imposed it is transmitted through increasingly small scale structures until it reaches the extracellular matrix (ECM), from which it can begin to act upon the cell. The ECM is the anchor point for cells and the environment from which they receive their mechanical information. Through their adhesion points cells sense and are subject to the stress and strain of the applied stimuli.^{3,9,31,32,44,109} The process of mechanical forces acting on cells, described by mechanotransduction, has been demonstrated to be vital and fundamental to the behaviour and functions of cells, even determining cell lineage and fate.^{19,20,39,44,45,52,66,70,107} With cells the driving force behind all tissue adaptation,⁶⁹ mechanical stimuli are shown to be crucial in system health, form, and function.

MECHANICAL SAID

The idea of 'specific adaptations to imposed demands' (SAID) is the bedrock of strength and conditioning. Important within this discussion is how adaptations are mechanically specific. Although this is far from established, there is enough evidence between the known mechanisms of adaptation, responses to imposed demands, and the properties of various tissue materials to make educated guesses where direct evidence is not available.

Mechanical adaptations will favour one of two outcomes:

- high stiffness, creating a stress dominant stress/strain curve
- low stiffness, creating a strain dominant stress/strain curve

This 'either-or' dichotomy is heavily dependent upon the materials and architecture of the connective tissue, particularly collagen. Reviewed previously in a mechanical context,⁸⁹ of particular relevance here is the toe region,^{4,77} as it has a significant effect on the mechanical properties of the connective tissue. The smaller the toe region the greater the stiffness will be; the larger it is the greater the system strain will be. Of significance here is differentiating between tissue strain capacity and system strain capacity or ROM. As the toe region increases, the total ROM available will increase. But tissue strain capacity seems relatively fixed,⁵⁴ with the toe region only occurring before true fibre deformation, meaning the region beyond that is essentially homogenous. Because of this, increasing stiffness equates to an increased ultimate stress capacity.

Another important material in connective tissue ECM is elastic fibres.⁴⁹ It must be clarified that the term elastic here is not the mechanical definition, but speaks to a high strain capability. The core of these fibres is elastin: surrounding it are fibrillin rich microfilaments.^{37,44,49} Elastic fibres have astounding mechanical properties, with strain capacity of over 150%,⁴⁴ and a long half-life.^{26,76,90,92} They form a continuous network that aids cellular adhesion,¹¹³ and wraps and links the cells.^{37,71} Elastic fibres are the cell housing through which cells are connected with each other and their mechanical environment. With the importance of deformation in cellular sensing of imposed demands,^{61,87,88,105} the threshold of reaction is increased due to the high strain elastic fibres housing them.

The management of elastic fibre mechanical properties is not clear. There is very little evidence of meaningful elastin turnover after maturation,^{26,76,90,92} suggesting that material changes are unlikely to be the driving cause of any alterations to the stress/strain curve. That the stress/strain curve alters at all is suggested by the current understanding of crimp angle formation, with small crimp angles requiring a more homogenous stiffness value between collagen fibres and inter-fibrillar materials.⁴¹

Kiely⁴⁹ provides a potential mechanism for elastic fibre mechanical

changes: describing intramolecular folding of fibrillin. This study crucially shows that elastic fibres can extend or retract. Materials can also cross-link the elastin^{31,53,79} and the fibrillin^{46,100} of elastic fibres, with elastin and fibrillin also able to bind to each other.⁷⁸ Kiely shows that elastic fibres can alter their length. This, in combination with cross-linking and binding of fibrillin and elastin, allows the ultimate strain capacity to be varied by restricting how far fibres can extend and slide past each other, altering stiffness through its relationship to strain.

What emerges from this are highly variable mechanical properties to connective tissue. Its relation to muscle means that the transfer of force from muscle to the rest of the system and broader environment is also highly variable, as it is wholly dependent upon its surrounding tissue. Force production and resultant movement is, therefore, not only a product of the neuromuscular system, but the connective tissue it is housed within. Furthermore, the variable mechanical properties influence the sensitivity of cells to imposed mechanical demands, meaning the threshold of adaptation is dependent upon, and altered by the mechanical properties of connective tissue.

We are far from having a complete understanding of the signalling pathways at the heart of connective tissue's adaptive processes. However, simple pathways for the sensing of stress and strain are potentially understood. The relationship between stress and strain means any tissue damage is always an issue of applied stress. The fibroblast-dependent repair process of tissue may represent a stress sensing pathway.^{7,24,65,101,102,108} Additionally both cells^{61,87,88,105} and fibronectin³¹ possess cryptic binding sites, which under sufficient strain are exposed. In cells a signalling cascade is triggered, while fibronectin acts as a mechanosensor,³¹ feeding back to the cells.

From this it is apparent that the sensing of and therefore adaptive responses to mechanical stimuli are specific and local. Understanding this and applying it to the biotensegrity theory creates an integrated and singular biotensegrity system, but the local mechanics throughout it can alter and vary.

Because of this the system will sense and respond to imposed demands differently. This mechanical variability will ultimately mediate and alter the mechanical stimuli experienced at the cellular level, and dictate the adaptive processes that are triggered.

What is shown, then, is that at all scales the mechanical properties of connective tissue influences the system function and adaptation. The influence on muscle force transfer determines how the system applies force. What is revealed in this is that muscle function cannot be properly understood or discussed when confined solely to the neuromuscular system, but must include the mechanical properties of the connective tissue as well.

The model presented below provides insight into how biotensegrity, mechanics, and neuromuscular function all come together, and how movement is the sum of this. By detailing how these all contribute to, and influence each other, the questions posed, both by the muscle amnesia theory and the assessment of it here, are answered. Furthermore, the mediating role that the mechanical properties possess on cellular sensing of imposed demands heavily influences the manner in which the system adapts. As such, the properties of the neuromuscular system depend upon the mechanical properties for determining adaptation.

A MODEL FOR POOR FUNCTION: NOT OFF, BUT A SLACKER

The muscle amnesia theory suggests that muscle activation is severely diminished, and that this drives the dysfunction. The suggestion here is that muscle is unaffected, but its surrounding environment changes. Rather than an issue of neuromuscular inhibition, therefore, the system is mechanically inhibited. The implications of this are significant, and go beyond discussion of the muscle amnesia theory. They suggest that muscle function and, as a result, biomechanics and movement are dependent upon the mechanical properties of the encasing connective tissue. The evidence above sets out the importance of tissue mechanics. Outlined in this section is how the interaction of the different elements provides a different answer to the still remaining questions. The

mechanoneuromuscular system is put forward as a basis for argument.

Biotensegrity forms the starting point. It changes the discussion of posture from one dominated by the neuromuscular system to one dominated by connective tissue and the mechanical forces acting upon and governing them. These principles surrounding posture remain when examining dynamic movement. Biotensegrity arrives at its strength and equilibrium through tension against intermittent compression.^{5,13,25,74,93,99} The balance of tension determines position; change tension in one place and the equilibrium changes, and with it the structural shape.⁹³ The nature of biotensegrity and the mechanics governing it mean that the system will organise its shape and equilibrium around points of high tension/stiffness. Tissue stiffness is associated with a myriad of performance benefits, and crucially that includes time to force production.^{8,14,15,28,57,59,62,73} This means that tissues with higher stiffness have a higher ultimate capacity, as described earlier, and can generate force faster than low stiffness areas. This is particularly important in sport, where large peak forces typically occur in a fraction of a second.

When considering the effects of connective tissue on muscle, the toe region becomes vital. Low stiffness is characterised by a larger toe region, or larger region of high deformation and low tension. When a muscle contracts within its connective tissue housing, the force it generates cannot be transmitted until the connective tissue is under tension. The relationship between stress and strain means you cannot have one without the other. Therefore, if there is no strain at the distal connective tissue, there is no stress, and no force transfer. There must first be tension across the whole connective tissue. If you consider a bungee rope tied at one end and arranged in the same crimp pattern as collagen, if you begin to pull at the end not tied, force will not act upon the tied end until the crimp pattern is lost and the rope begins to tense. As tension increases, the force transmitted to the far end will increase, both in efficiency and magnitude. In this the effects of tissue mechanics on muscle function is shown.

Imagine two muscles of equal strength and contractile speed: one is the agonist and is within a high stiffness area, the other the antagonist within a low stiffness area. When both contract maximally, the agonist will transfer more force more quickly through its encasing connective tissue, more rapidly developing greater stiffness and tension. The principles of biotensegrity mean that the system will organise around and subsequently bias load at these points. This develops loading patterns and subsequently has a large contribution to movement patterns and ensuing adaptations.

Applying this to the remaining questions, and the claim of mechanical rather than neural inhibition, I suggest that the following is happening. Chronic positions alter the mechanical properties of various tissues, driving increased strain capacity in some and reducing it in others. Any periods of loading therefore result in preferential loading of the higher stiffness areas, especially so when time to force expression is a factor as in sports. Loading of muscle becomes imbalanced through this, as those in high stiffness areas deal with load first, and are therefore used as the system foundation, resulting in more load as the biotensegrity body organises around these points. In contrast the low stiffness areas are slower to transmit force as they have more slack to pull on before tension is sufficient. As a result they are placed at longer working lengths, increasing leverages, as the system orientates around the high stiffness areas. This may elicit some additional increases in ROM and so further reduce stiffness as stretching a contracting muscle has been shown to elicit ROM increases comparable to traditional stretching protocols at significantly shorter lengths.⁴⁸ But, as already discussed, this change in mobility will likely have limited scope as it is confined by the movement demand.

Furthermore, effects on adaptation will embed and amplify these mechanical differences, enshrining them in the neuromuscular system. Loading of the connective tissue at short ranges will increase stiffness,^{12,89} while the long ranges will increase tissue stiffness, but it will not be as great, due to reduced loading and maintenance

of the greater toe region.^{12,89} The difference in muscle loading will produce imbalanced strength and hypertrophy improvements, with high stiffness areas benefitting more. Neural adaptations will also show disparity. Chronically increased loading improves individual motor unit force production, synchronicity, and function,^{18,33,103} needing less motor neuron recruitment for a given task.¹⁸ This reduces the chance of neural interference such as antagonist activation,¹⁸ increasing efficiency and performance. Of further note is that when trained as prime movers, muscles were more active when subsequently used as antagonists,⁶ possibly indicating a suppression of reciprocal inhibition.^{2,21,60} This suggests that the imbalanced loading will elicit greater neural adaptations at points of high stiffness, creating higher force outputs with greater efficiency and a more dominant recruitment pattern and activation, even when acting as an antagonist. However, this bias does not indicate diminished neural function in low stiffness areas.

The idea of the mechanoneuromuscular system, therefore, is that mechanics serves as the foundation from which the neuromuscular system operates. Movement is therefore built upon mechanics and refined through the neuromuscular system, both completely reliant on the other.

Turning to the remaining questions from the muscle amnesia theory, the problems it seeks to deal with are more likely to be mechanical in origin, although the imbalance of neural adaptation does introduce a neural disparity. However, despite differences in proposed mechanisms, what the theory advances for resolving the problem is largely supported here, as it is targeted load application.^{23,43,68} Using this method, mechanical properties can begin to be balanced out.

Practical applications

Changing the focus from neural to mechanical properties, the proposed methods for resolving the issue can be further advanced. Although permanent resolution of the problem can only be found through proper management of applied load, this process can be augmented by acute, short-term

Three example interventions for the resolution of slackness around the glutes.**Intervention 1**

DESCRIPTION	EXERCISE	SETS	REPS	INFO	%1RM
Mobility (stiffness reduction)	Anterior hip foam roll	1	3-5 mins	e/l	-
Primer (stiffness augment)	Banded clams	4	8-12	e/l	-
Primary exercise	BB Hip thrust	4	6	5+1(8s Iso)	85%+

Intervention 2

DESCRIPTION	EXERCISE	SETS	REPS	INFO	%1RM
Mobility (stiffness reduction)	Adductor foam roll	1	3-5 mins	e/l	-
Primer (stiffness augment)	Banded X walks	4	8-12	e/l	-
Primary exercise	Bulgarian split squat	4	5	e/l	80%+

Intervention 3

DESCRIPTION	EXERCISE	SETS	REPS	INFO	%1RM
Mobility (stiffness reduction)	Anterior/lateral leg foam roll	1	3-5 mins	e/l	-
Primer (stiffness augment)	Single leg rack holds	4	5	5 sec e/l	90%+
Primary exercise	Back squat	4	3		85%+

changes to the tissues mechanical profile. Connective tissue's contractile capabilities offer one avenue for this. Although nowhere near the order of magnitude of muscle, the contractile fibroblast cell population allows connective tissue to contract, and is able to do so for many hours.^{9,40,82,84,85,111} Connective tissue can also have its stress/strain curve shunted towards the strain end, reducing stiffness for short durations through longer duration (~5 minutes) stretching and myofascial release methods.⁶⁴ Although this would seem short lived, maybe ~10 minutes,^{38,58} it helps to acutely balance the mechanical properties.

Interventions should aim to apply and bias load towards the deficient area, while at the same reducing load and/or increasing required range of movement on the dominant area. A combination of compound and isolation movements can be very effective, with targeted mobility work to supplement this. The intent is to create long-term balance in tissue mechanical properties.

Above are three example interventions for the resolution of slackness around the glutes. In them, each mobility exercise is done before beginning the resistance exercises. The primer and primary exercises are a superset, with short rests of ~30-60s between them. The concept is to benefit the mechanical

properties of the tissue, without adding fatigue that limits the working sets.

The purpose of these interventions is to use mobility to try and offset the dominant mechanical environment and to use load in order to improve the balance of tension both acutely (primer) and chronically (primary). This requires a maximisation of the mechanical work performed, indicating heavy loads over large displacements and/or held for prolonged periods of time.

Conclusion

With no peer-reviewed literature directly examining the theory, the claims of the muscle amnesia theory have so far gone untested within an academic context. Although this is not a thorough investigation directly testing the theory, it is hoped that this review goes some way to properly addressing the claims made, as well as the surrounding literature.

What has been advanced is that the dysfunction being suggested by the muscle amnesia theory is in fact a legacy of the variable mechanical properties of the connective tissue. It is also hypothesised that it is the properties of the encasing connective tissue and the subsequent repercussions on force transfer from the encased muscle to the broader system that is being observed.

This integrated system of mechanics and neuromuscular function was here termed the mechanoneuromuscular system, and details how mechanics can play a large governing role on both movement and adaptation. The greatest moderator of mechanical properties is load.

Therefore, by using the manipulation of load and how it is applied, we can alter the foundations of an athlete's movement. Load is crucial, and can be benevolent or tyrant. Within strength and conditioning we have the opportunity to create the former from the latter, and benefit from all the possibilities that arise therein. However, we must be cognizant of the principles of biotensegrity within this. If a fault, or mechanical imbalance, is seen in one place, the singular integrated whole of a biotensegrity system guarantees that a fault exists somewhere else. No problem exists in isolation.

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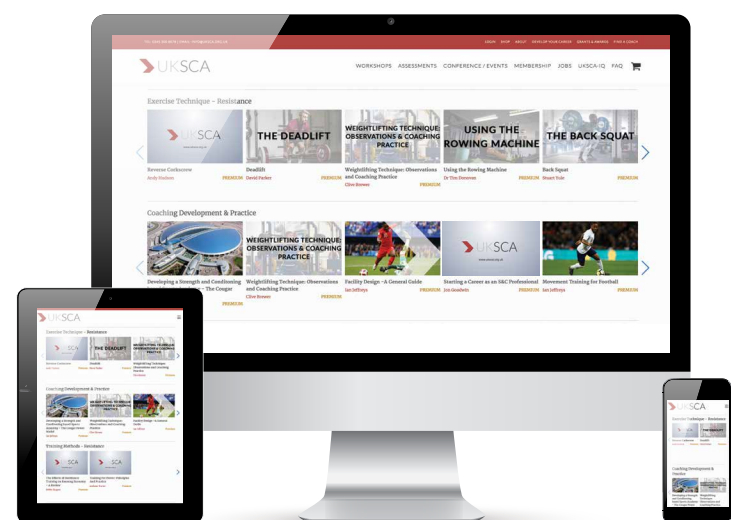
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