

# Anatomy and mechanics:

## Part 1 - Position and movement

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### OVERVIEW

The role of passive tissues and their mechanical profile has typically been viewed as secondary to neuromuscular functions in movement. In a four-part series of articles, I will discuss the foundations to mechanics and their interplay with anatomy. Part 1 details how this relationship combines to shape and direct both position and movement. Within this, the article addresses the biased view of soft tissues being responsible for locomotion and discusses the need for a reconceptualisation of these tissues.

### The importance of passive tissues on function

Investigation to date has typically dealt with passive tissues in isolation, either as a single tissue,<sup>13</sup> or within its muscle-tendon complex.<sup>16</sup> Although there is recognition of a far more extensive and significant network of passive tissues, principally fascia, this has received little attention within biomechanics, resulting in poor understanding of the network's role within movement.

### Introduction

The basic principles that frame our conceptions around biomechanics and human locomotion have their roots in research from the 1680s,<sup>9</sup> in which muscular force acts to move levers. These principles have been built upon, but the foundations have remained largely unchanged. Typically, the body is still viewed as a system of levers with spring-like elements that store and release energy.<sup>21,54,75</sup> More recent studies on the role of fascia have raised questions as to how complete these foundations are.<sup>26,68,69,73,96</sup>

The information that is accumulating provides compelling evidence that traditional biomechanics has missed important detail, with the transmission and return of force through passive tissues an important and far more complex and diversely affecting factor. However, this more recent branch of research commonly falls to the same problems of compartmentalising and dealing with tissues as distinct elements. This article argues that these delineations are limiting the literature

and constraining a full understanding of biomechanics. Further to this, although the mechanical profile of these passive tissues is frequently raised, the application of mechanics is too often simplistic and lacking nuance and detail. Values are often stated as an absolute (eg, <sup>48</sup>); however, the discussion of any soft tissue can only be a snapshot in time and velocity due to the adaptive nature of the tissues, the viscoelastic properties of connective tissues,<sup>52,91</sup> the force-velocity relationship in muscle,<sup>24</sup> and the type of force imposed. In short, biomechanics is too often dealt with in material absolutes where there are none, often ignores tissues crucial to function, and is too often lacking in mechanics.

This will be the first in a four-part series, with the aim to explore the anatomy of locomotion and how it interacts with mechanical demands. Part 1 will begin with a general overview of critical theories and principles, before applying these practically within sprinting development and hypertrophy training (Part 2) and understanding aspects of injury (Parts 3 and 4).

Fascia encapsulates muscle at every scale from fascicle to whole muscle<sup>59</sup> and is part of a continuous network of passive tissues that includes fascia, tendon, ligaments, and aponeurosis.<sup>2,35,72</sup> The housing of muscle within fascia is frequently acknowledged, but any discussion rarely expands beyond the anatomical existence of fascia and fails to capture its importance or the implications of passive tissue interconnectedness.

Muscular force must be transmitted through passive elements such as fascia and tendon before it can interact with external factors.<sup>47,59</sup> The efficiency and speed of force application are therefore dependent not only on the neuromuscular system, but the passive tissues that house it. This involvement of passive tissues in force expression brings mechanical principles more prominently into the discussion, as it is the mechanical profile of the tissues that determines their magnitude and speed of force transmission. When considering the qualities of efficiency, magnitude, and speed in isolation, the greater the stiffness of a tissue the greater the performance.<sup>7,19</sup>

## ‘When considering the qualities of efficiency, magnitude, and speed in isolation, the greater the stiffness of a tissue, the greater the performance’

Revealed in this is that the study of biomechanics and the understanding of physical performance cannot be complete without first acknowledging the importance of passive tissues within force expression. This goes beyond the return of passive force and shapes the contribution of the neuromuscular system, as the passive tissues are the messenger that relays the muscle output. This leads to several fundamental questions:

- What factors influence the adaptation and function of these passive tissues?
- What are the implications of mechanics mediating neuromuscular expression?
- How much of movement is determined by active coordination and production of force and how much is derived from passive return and transmission of force?

Discussed below are theories and established mechanisms that provide answers to these questions. This begins to frame a more complex and elegant system governing movement.

### **Biotensegrity as a foundation to passive tissue function**

Biotensegrity is the theory that biological systems find structural integrity through constant tension acting against floating intermittent compression resistant elements, meaning none of the compression resisting elements touch.<sup>5</sup> This has been theorised in humans from whole body to cellular levels.<sup>28,67</sup> Tensegrity structures always arrive at a balance between tension and compression, as any change in applied force spreads

along the tension elements and distorts the structure.<sup>70</sup> In short, nothing occurs in isolation as any change in load will radiate outwards, changing tension. The nature of these changes can be understood by viewing a biotensegrity system as a tug of war: if tension increases in an element, then it will pull harder, bringing any compression resistant elements closer to it. However, if tension is reduced, the compression-resistant element will be surrendered to the opposing tension element. When viewed within the human body, the passive tissues become the tension elements, while bone becomes the floating compression resistant elements.

The input on speed, efficiency, and magnitude of neuromuscular force held by passive tissues is informed by biotensegrity. The tension in the system allows for higher speed of force transfer,<sup>5</sup> as force travels less distance at greater velocity. The greater speed of transmission allows greater rate of loading in passive tissues which loses less force as heat (efficiency).<sup>57</sup> Because tension is derived by strain, to increase it requires operating closer to ultimate strain. As an increasing strain requires an ever greater stress, an increase in tension is equivalent to an increase in stiffness, and will also equate to greater resilience. The increasing resilience allows for a greater magnitude of force to be stored and returned. Each of these demonstrates the relationship between tension and speed, efficiency, and magnitude of force transmission.<sup>79</sup> The example of a basketball helps conceptualise this. When flat, there is no tension and the ball dissipates energy before it can be returned. As tension increases, the variables described above will improve; it will rebound faster for a

given impulse, it will rebound higher for a given impulse, and its ultimate rebound height will be greater than a lower tension ball.

Aside from improved performance through tension, the outcome when tension is low is important. As a structure is acted upon by a force it will strain, as strain increases the force trying to restore the structure to its original form increases. With the basketball this is seen in the impact of the bounce. A passive material cannot return to its original form unless the enacting force is removed. In the bounce this occurs because force is mass x acceleration. At the collision, acceleration reduces until it is zero. At this point there is no force acting on it and the ball can return to its original form, creating the bounce. If the tension is low, the resilience is low and so only a small amount of energy is stored. As tension diminishes, total energy stored reduces – as does efficiency – meaning increasingly small amounts of force are available to return the ball to its original shape. If these forces fall below the requirement to overcome gravity, there will be no bounce. From this we understand position is derived from internal force minus external force; a net balance and nothing happens, positive value and movement occurs, negative work and position is lost. The important thing is that this changes the perspective of coordination and movement from one dominated by the neuromuscular system,<sup>56</sup> to one whose first foundation is passive tension. Neuromuscular actions are a coordinated alteration in the balance of tension to create movement or to resist an imposed force. But this only builds upon the existing tension, it does not override it or replace it. Both aspects must be in view and addressed, as limitations in the foundation will diminish the function of anything building from it. The balance of tension becomes vital to optimising both tension and force expression. And so we must understand how to alter it.

The description above demonstrates how tension requires greater relative strain to increase. Tension can be altered, therefore, by applying a greater stress or by reducing the total strain possible. Changes to imposed stresses are largely transient (although they can produce adaptive responses – discussed later), meaning that chronic tension

is derived from changes to relative strain. These changes are mediated by architectural changes, such as fibre cross-linking, and material changes, such as crimp.

Discussion of cross-linking is quite straightforward; cross-links are found in collagen and extra cellular matrix (ECM) fibres, and inhibit the range of movement (ROM) by limiting how far fibres slide past each other.<sup>13</sup> Crimp requires more explanation. Crimp is found in collagen fibres, the predominant material in passive tissues;<sup>32,74</sup> it describes a wave-like pattern to the fibres at rest<sup>39</sup> and produces the characteristic toe region in passive tissues,<sup>36</sup> the initial phase of the stress/strain curve that has a high strain at low stress. Through the folding of collagen, crimp allows greater total length but a preservation of tension. But, as with a low-tension basketball and demonstrated by the high strain and low stress profile of the toe region, the force generating tension in crimp is low. Crimp therefore has a governing role in tension, as it modulates the length of collagen. However, crimp is not fixed: its formation is not fully understood, but appears to be a cell-governed process mediated by the difference in stiffness between ECM and collagen fibres.<sup>22,39</sup> The more homogenous the stiffness, the smaller crimp becomes. The material determining stiffness cannot be changed, which leaves architectural alterations. Fibre cross-linking allows total strain capacity to reduce, increasing stiffness for a given ROM. This suggests alteration of crimp becomes the primary target for adjusting tension. As loading increases fibre cross-linking and stiffness,<sup>13,32</sup> and high ROM is associated with greater crimp, it shows tension is a product of loading history. High loads with small ROM will develop high tension, whereas low loads and high ROM will develop low tension.

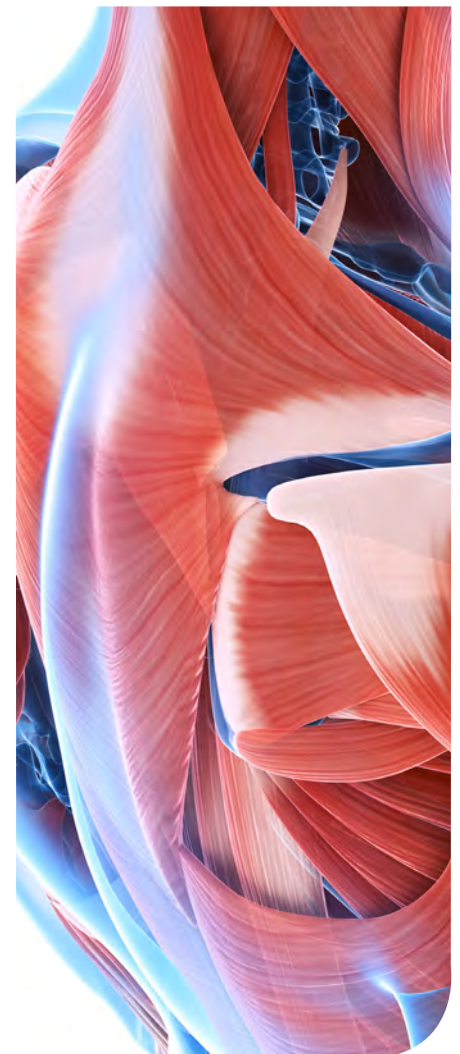
Biotensegrity, aside from introducing underlying mechanical principles at work provides a different perspective on how movement is achieved. Movement becomes the distortion of a whole,<sup>5</sup> rather than an isolated action. Within a tensegrity structure, forces radiate three-dimensionally in all directions. The efficiency of force transmission in tension has been set out, and means the body is incredibly sensitive to

mechanical forces and cues, not just locally but distally. The magnitude of force and degree of tension will then dictate how far that force is transmitted. Because forces act three-dimensionally, forces dissipate inwards to act on smaller scale structures such as cells (discussed in further detail in the mechanotransduction section), as well as laterally through tissues.

This view of global rather than specific lines of force transmission has growing evidence to support it, with lateral force transmission observed within muscle-tendon complexes,<sup>45</sup> as well as passive force transmitting to antagonist and agonist actions.<sup>27,44,61</sup> The theory of biotensegrity provides an immensely elegant model of movement, in which self-propelling tensegrity structures can generate their own internal forces to modulate tension and control the distortion of the system: movement. The transmission of these forces radiates outward through the whole system to produce not only propulsion, but stabilising forces from the same muscle action. And further to this, it creates a deeply integrated system that cannot be understood through isolation of elements, and in which interventions act globally as well as locally.

Applying the ideas of long-range three-dimensional force transmission to a biotensegrity system has interesting implications. A tensegrity system distorts around areas of highest tension, and tension is derived from strain. The application of a load therefore creates a radiating, yet reducing, increase in tension. This means the system will distort around it. How far the tension radiates will determine how much of the system will distort. It presents movement as the coordination of tension changes. And because of the three-dimensional nature of it, this becomes distinct from the traditional idea of muscles pulling levers along specific lines. The neuromuscular system is not diminished in this, but passive tissues are elevated. Passive tissues are the foundation to tension: an imbalance in loading will distort how the system holds at rest and has the potential to create tension deficits the neuromuscular system cannot overcome. The dependence of the neuromuscular system on the passive tissues to transmit forces means its modulation of tension will be inhibited

in lower tension tissues. Slower expression of force delays the increase in tension, meaning higher tension areas will load first and the system distorts to them. The need for load to increase tension means higher tension areas will have biased loading and so increase the imbalance in tension. This means movement patterns can be altered through passive mechanical forces, not just neuromuscular coordination. This details how the passive tension in the system underpins all of movement. It does not re-write biomechanics, but provides another layer for consideration by coaches. It allows movement error to be viewed in a different manner, as although it does not rule out coordination issues, it raises the possibility of imbalanced tension preventing effective coordination. And because the tensegrity system acts as a whole, it requires any intervention be viewed locally (eg, area of loading), and globally (eg, balance of whole body tension).



# 'The input on speed, efficiency, and magnitude of neuromuscular force held by passive tissues is informed by biotensegrity'

## Mechanical adaptation within passive tissues

So far, the role of mechanics has been touched upon – both in enacting forces and the profiles of the tissues – but not fully explored. In this section the full scope of mechanics is examined and ranges from tissue to cellular level. This begins with mechanotransduction, the foundation of mechanically derived adaptation. It reveals the significance and scope of mechanical cues on physiology. Important to carry forward is that discussion of tension in the biotensegrity section is equal to stiffness here.

### MECHANOTRANSDUCTION

Mechanotransduction is the process by which cells sense and respond to mechanical cues.<sup>58</sup> This process is significant in governing cell behaviour, including: migration,<sup>81,84</sup> proliferation,<sup>92</sup> apoptosis (programmed cell death),<sup>10</sup> and tissue deposition (tissue hypertrophy).<sup>29</sup> When a mechanical cue acts upon the cell surface it generates strain, which can reveal cryptic binding sites and trigger a chemical signal cascade.<sup>64,94</sup> Mechanical cues can also be transmitted to inside the cell, acting on its nucleus and therefore able to affect DNA transcription.<sup>3,83</sup> In this it becomes apparent that the importance of mechanical cues is profound, affecting the functioning and structure of the body in its entirety.

Mechanotransduction in the context of this paper can be defined as the means by which mechanical forces become biological actions. The importance of this is simple: mechanics shapes biology. What adds to the complexity of this is that biology shapes the mechanical cues received.<sup>85</sup> Within the biotensegrity section, stiffness was associated with greater force transmission. However, the process of mechanotransduction requires strain. The greater the stiffness, the greater

the force must be to achieve a given strain. This means that as tissues adapt, they create an increasingly insulated environment for the cell, reducing the mechanical cues it receives. Increasing stiffness makes it harder to stimulate adaptation. Therefore, from this we can understand that passive tissues housing the cell populations affect adaptation not just acutely through mediating force expression and resultant movement, but chronically by mediating cell strain.

The biological aspect of mechanotransduction is dominated by cells. Viewing these cells and their surrounding environment, the ECM, provides more clarity on the extent to which passive tissues shape adaptation, and by that, chronically shape movement.

The ECM is a network of predominantly fibrous proteins (such as collagen) alongside glycoproteins and other constituent materials.<sup>6,33</sup> It can be understood as a smaller scale passive tissue; where fascia encases muscle at different levels, the ECM encases individual cells and provides the frame for cells to function and interact with chemical and mechanical information.<sup>33,50</sup> The mechanical profile of the ECM is adaptive, and as with all passive tissues this can be achieved through alterations in both materials and architecture (both of these are discussed in the next section).<sup>13,23,37</sup> Crucially, the mechanical profile of the ECM itself can drive all cell behaviours listed above.<sup>86</sup> Cells don't just respond to acute loading, but the chronic mechanical environment of the ECM. This demonstrates that tissue development (eg, hypertrophy, cross-linking) is heavily mediated by the passive tissue ECM.<sup>15,18,86,87</sup> What we can understand from this is that the inherent properties of passive tissues direct and inform cell function and behaviour, and through this mediate chronic adaptation.

Not only do passive tissues serve as the meeting ground between imposed mechanical demands and cellular biological actions, but they are themselves a mechanical cue to cells and direct their function. In this their place in chronic adaptation is both direct and in-direct. Of particular interest is that cells will preferentially migrate to areas of higher stiffness,<sup>86</sup> raising the possibility that areas of greater stiffness hold a higher adaptation potential. The process of mechanotransduction is mediated by biotensegrity, as the system tension allows forces to be transmitted further and three-dimensionally. But the greater stiffness inherent to tension insulates the cells from mechanical cues, increasing the threshold of adaptation.

### FRAMING ADAPTATION TO LOAD

Thus far, the mediating role that passive tissues have on force transmission has been detailed; this occurs acutely by affecting the rate, efficiency, and magnitude of neuromuscular force transmitted. It also occurs chronically, as passive tissues mediate cell signalling and function and thereby influence adaptation. In this section, a general summary will follow of some key adaptations and the implications on training interventions.

The specialisation of different passive tissues provides a useful starting point as it can reveal more universal traits of adaptation as well as specialised markers to achieve specific outcomes. All passive tissues are dominated by collagen.<sup>30,32,51</sup> However, although the materials are typically similar across the tissues,<sup>32,51</sup> their architecture is far more variable. Evidence within the literature suggests these passive tissues originate from the same source, but the application of mechanical load leads to specialisation in some tissues.<sup>80</sup> A clear example of this can be found between fascia and tendons. Tendons are highly specialised passive tissues. Nearly all

tendon fibres and cells are arranged along the long axis of the tendon,<sup>30,65</sup> this specialised architecture<sup>62</sup> allowing enhanced resistance of the tensional demands.<sup>42,66</sup> In contrast, fascia is far more generalised in the demands placed on it, acting as a general linking tissue across tissues and scales, most clearly seen in muscle.<sup>59</sup> These generalised demands are mirrored by multiple directions of fibre alignment and cell population.<sup>74</sup> The architectural generalisation of fascia prevents it from attaining the mechanical peaks of tendons, but allows it to operate within mechanically diverse environments. Demonstrating the significance mechanical forces have on tissue material and architecture is the gradual progression of tendon into bone. As compressive forces increase nearer the insertion point, cartilaginous properties of the tendon increase concurrently.<sup>8</sup> Evidence suggests that these cartilaginous properties are adaptable, and can begin to form in the presence of compression.<sup>63</sup> This helps to frame both the importance of mechanotransduction, and the immense scope of mechanical force on tissue properties.

The description of cell actions in adaptation has so far explained the mechanism of sensing imposed demands and the scope of those demands to direct material and architecture, but not specific cell actions. Yet to properly contextualise the architectural specificity of tissues, the cell actions during adaptation needs to be understood. The process of tissue deposition is a complex balance of degradation and synthesis,<sup>12,14</sup> overseen by the

local cell population, predominantly fibroblasts within passive tissues.<sup>40</sup> In addition to orchestrating the complex chemical process of tissue adaptation, these cells are capable of contracting, and will contract areas of new tissue deposition,<sup>25,38,40,95</sup> creating a pre-tension to it. The magnitude of contraction is dependent upon the imposed demands and the stiffness of the existing tissue, meaning the greater the stiffness the greater the contraction strength.<sup>11</sup> The preference of cells for stiff environments<sup>86</sup> means that not only will a stiffer tissue more easily increase tension, but it will hold a greater cell population to drive adaptation and contract tissue. Alongside this, application of load leads to fibroblasts becoming more active.<sup>12,20,31</sup> This results in increased material deposition and architectural arrangement<sup>43,77</sup> and triggers contractile actions.<sup>55,71</sup> Taken together, this demonstrates that increasing tension/stiffness of a tissue creates a higher and more active cell population. The implications of this on training is that greater amounts of tissue can be deposited and set under higher contractile forces, further developing tension/stiffness and perpetuating the cycle.

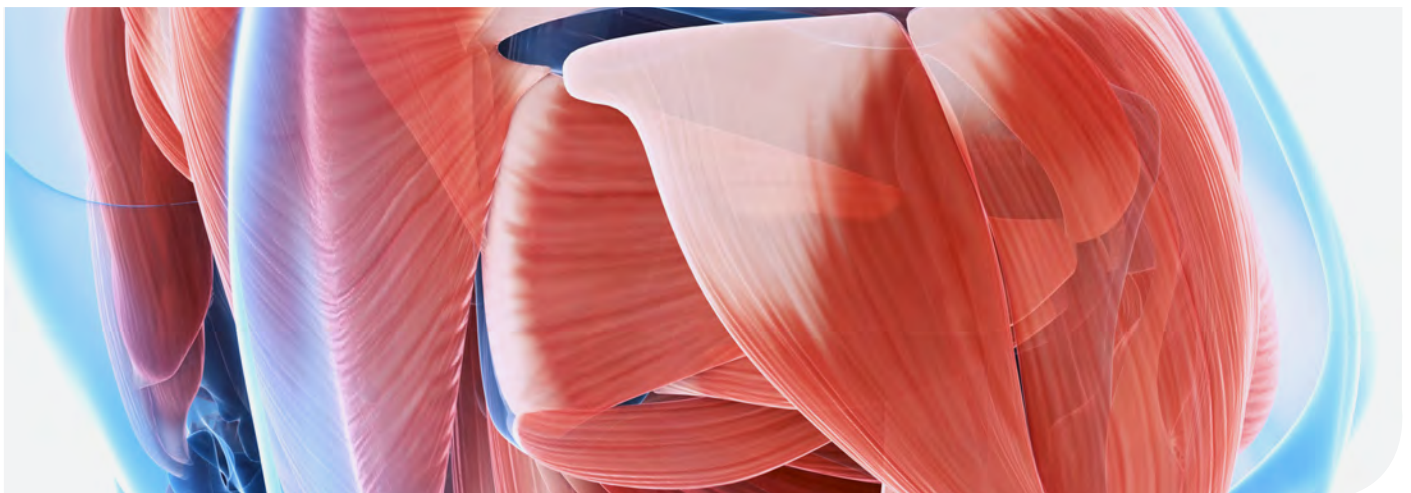
### **Can passive tissues and the neuromuscular system be viewed as discrete?**

The severe imbalance in research between neuromuscular and passive tissues has left the fact the neuromuscular system is functionally useless without the network of passive tissues largely ignored. But this functional dependence raises the

question of whether they are genuinely distinct tissues, or if this distinction is a direct result of dissection and over simplification of anatomy. The theory of biotensegrity sets huge importance on the passive tissue network, but this network is inert and also functionally useless without the neuromuscular system. If neither can operate without the other, at what point does it become counterproductive to view them as single and distinct tissues? Furthermore, there is blending of passive and active force production within the two tissues. Passive tissues possess the active force production fibroblast cells,<sup>40</sup> while the passive force production element titin is within muscle cells.<sup>41</sup> Even the general anatomy of passive and active tissues sees them interwoven with each other; fascia when viewed without muscle is a honeycomb-like structure,<sup>59</sup> showing active is continuous with passive and vice versa.

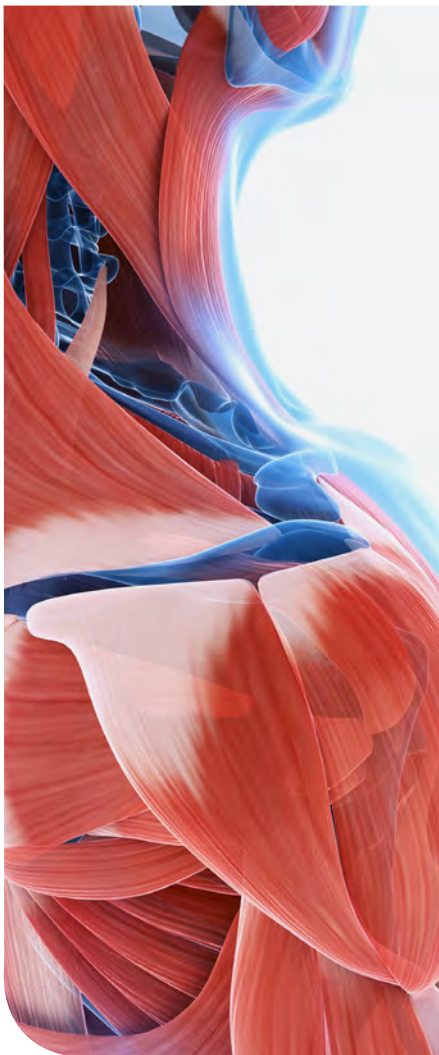
Although the role of velocity is to be examined in far greater depth in part two of this series it has huge importance here in providing a perspective on how these tissues operate and whether they can be seen as distinct from each other. When considering velocity it is important to understand the viscoelastic properties of passive tissue mean their mechanical profile is augmented by increasing velocity. This includes increased ultimate capacity, greater force return, reduced energy loss, and reduced speeds to perform these functions.<sup>57</sup>

Data shows that it takes between 0.2s,<sup>1</sup> and 0.3s,<sup>78</sup> to reach around 80% maximum voluntary contraction (MVC), with a significant slowing



beyond this point shown, with 0.1s only bringing 5% more of MVC.<sup>78</sup> Data focusing on the first 0.05s of muscle contraction shows only 16% of MVC produced.<sup>46</sup> Examination of elite sprinting demands shows an aerial time between contacts of 0.12s,<sup>88</sup> with time to peak force 0.028s,<sup>89</sup> giving a total time for active force production of 0.148s. This leaves it well short of even 80% of MVC in force output, with this possibly reduced further by the delay in motor unit activation.<sup>78</sup> Given the increasing peak force demands with greater movement velocities, this diminished force production capacity brings serious questions over how active force production contributes to propulsion in severely time constrained actions. If it is the primary source, it would appear prohibitively slow.

Related to active force production output is the energy demand of these actions. As shortening velocity of the muscle increases, the energy demand



increases. The literature points to several mechanisms involved with mitigating this rising energy cost. As velocity increased, it was found that muscles in the triceps surae altered their contraction type from concentric to isometric/near isometric.<sup>4,53</sup> Because of this, the distance required for the whole muscle to move drops to zero/near zero. This isometric action creates a very simple solution: it not only reduces the energy demands of the muscle by two to three times,<sup>17</sup> but also overcomes the force-velocity relationship observed within muscle<sup>24</sup> as there is no shortening velocity. However, this comes at a cost: isometric actions do not produce movement or store energy. Therefore, the passive tissues become vital as they are able to store imposed forces (the implications of this are explored fully in Part 2 of this series). Further to this, increased fascicle length has been linked with improved sprinting speed, theorised to be a product of enhanced shortening velocity and greater tendon strain.<sup>34</sup> But it has also been shown that greater fascicle length is more energy demanding.<sup>17</sup> This again demonstrates active and passive tissues work synchronously; the active tissue enhances its contribution (and also that of the passive tissue), while the increased metabolic cost can at least in part be offset by the improved passive contribution. The isometric action of the muscle also enhances its force contribution while augmenting the mechanical properties of the passive tissue. Equally, the passive tissue makes the isometric action of the muscle possible, as it can strain and store energy.

The above demonstrates, therefore, that mechanisms exist to diminish or completely overcome deficiencies in active force production when completing higher velocity movement. However, the mechanisms that allow this are dependent upon passive force production for offsetting energy cost or for generating work. And the presence of these mechanisms does not negate the established time course for active force production, meaning only fractions of total potential force will be available within high velocity-high peak force-short duration movements. Introducing velocity brings into view not just active and passive tissue interdependence, but fluidity in the means of force production and how this allows enhanced efficiency

and performance from active tissues.

This article also demonstrates how passive tissues don't just support the active tissues, but that the two are a singular composite tissue, functioning as one. The action of active tissues, both in expressing force and in mitigating the negative effects of shortening velocity cannot occur without the passive tissues. And the properties of the passive tissues are enhanced and coordinated by the active tissues. Neither works without the other; their anatomy is intertwined and active and passive elements are found in both tissues – their physiology is influenced and developed by each other, they enhance the function of each other, and they address the shortcomings of each other. Aside from the means of force production, there is no functional means of differentiating active and passive elements. The implication of velocity on force production provides the clearest example of this.

Suggested here is that the traditional perspective of discrete tissues is both reductionist and flawed: it cannot allow proper understanding of movement. What is proposed instead is a single composite material, where differentiation comes only from the means of force production. Although scientific investigation may still find value in isolated examination, without relaying this back into the whole composite environment the findings will always be limited.

### Summary and conclusion

Setting this composite tissue within the framework so far set out adds an ever-increasing elegance to the form and function of the body. Further to what has been discussed, two systems of governance emerge: electrical (nervous system) and mechanical (composite tissue and cellular population). The electrical system deals with acute functions, and through this shapes the system exposure and influences subsequent chronic adaptations. The mechanical system deals with both acute and chronic: acutely, by translating both expressed and imposed mechanical demands, and chronically through its translation of mechanical force to cells and the adaptations this elicits, in particular the altered tissue tension

determining the balance and properties of the system as a whole. The composite tissue responsible for this system-wide tension has self-contained engines (muscle) that through tensegrity each generate propulsive and stabilising forces. This process entails a less linear view of movement. Rather than a muscle holding a singular line of force through pulling of tendons, it instead has a dominant line but with force radiating three-dimensionally, creating propulsive and stabilising forces from a single source. It provides a far more complex and nuanced view but shows greater efficiency: force does not act solely along distinct lines, but globally through a tensioned web, yet this allows each force production to go somewhat towards stabilising itself. This tissue operates incredibly fluidly, allowing it to specialise according to the velocity demands placed upon it, utilising high energy cost internal force production at lower velocity, but increasingly utilising low energy cost external force through passive return as velocity rises, increasing efficiency. This describes a system of incredible mechanical fluidity, allowing for extremely varied force production to be performed with a great deal of specificity. Additionally, the extreme sensitivity to mechanical cues provided through the tensegrity structure means these imposed demands are spread three-dimensionally and distally, allowing the body to simultaneously shape itself to better meet the imposed demands acutely (eg, fibroblast/muscle fibre contraction of tissues) and chronically (tissue adaptation). Although load spreads through tensegrity structures, it does not do so homogeneously, meaning adaptation will still be localised and specific, with the effects gradually diminishing. This gives scope for shape change to occur, leading to changes in posture and default positions during movement as the system will defer to points of high stiffness and tension for those reasons already set out.

What emerges from this is the modality through which movement and loading history shape the way future demands are met. This demonstrates a great degree of plasticity in posture and how we move that is mediated firstly through passive tension and the adaptive processes governing it. Although this tension has neuromuscular control, its mechanical foundation will always

## **‘Setting this composite tissue within the framework so far set out adds an ever-increasing elegance to the form and function of the body’**

exert influence and control. This means that movement and injuries cannot simply be viewed as a process of neuromuscular training, but require effective loading in correct positions to develop a balance of tension that optimises the performance of the task, providing stability for the expression of force and mitigating the occurrence of acute and chronic injuries.

Additionally, the conceptualisation of a single composite tissue alters the view of force production and resultant movement, creating electrical and mechanical governance to force production. Through this, it can fluidly adjust the means of force production depending upon imposed demands, and within the scope of that demand maximise force production while mitigating energy demand. The practical implications of this necessitate deeper exploration of the topic within an applied setting: this will be described in Part 2 through examination of sprint performance. The final papers review the interplay of mechanics and physiology within injury.

### **AUTHOR'S BIO**



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of sports from recreational to national and international standard competition. With a particular focus on rehabilitation, this has led to a strong research interest in the physiological underpinnings of movement.

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