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From muscle fibres to gears: How fibre rotation and shape change impact muscle function

Running title: Muscle's gear and dynamic changes

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Abstract

Skeletal muscle architectural design strongly affects force-generating capacity and excursion range, and its functional importance to animal and human movement is well-founded. Traditionally, this 'structure–function relation' has been inferred when assessing a muscle's architecture at rest, often using muscle images or geometrical measurements obtained from anatomical dissections or medical imaging techniques such as ultrasonography or magnetic resonance imaging. However, a more contemporary view recognises that muscles display a remarkable capacity to dynamically change shape–and therefore architecture–during contraction, quite literally allowing them to change gears as contractile conditions change. Muscle gearing refers to the velocity and force advantages obtained through the effects of fibre rotation and shape changes on muscle and fibre velocities, which significantly influence the length and velocity operations of both the fibres and whole muscle during contraction. This phenomenon can be characterised by the ratio of the velocities or displacement of the muscle to the fascicle. Given its function importance, muscle gearing should be considered an integral part of a comprehensive framework for understanding dynamic muscle function, as both fibre length and architecture, along with muscle shape changes, respond to varying mechanical demands and influence functionality in both normal and pathological conditions. The present review aims to (i) present a historical overview of our understanding of the muscle structure–function relation from measurements obtained in resting muscle as well as during dynamic, active contractions and (ii) define 'muscle gear', explain how muscle gear impacts muscle function under various movement conditions, describe intrinsic and extrinsic factors that determine or influence gearing, and overview the literature reporting gearing in animal and human studies while raising hypotheses in an attempt to explain the gearing differences identified across the literature. Given the current challenges in experimentally quantifying the unique influence of each identified factor, we employ a computational modelling approach using a previously validated three-dimensional finite element muscle model to gain a better understanding of the mechanical phenomena underlying muscle gearing. Finally, we briefly discuss the potential role of gearing in the observed changes in muscle function with ageing, injury, and exercise training. Overall, muscle gearing is a distinctive feature of skeletal muscles, with the modelling demonstrating that it is an emergent property of the physics of muscle contraction. A greater understanding of this phenomenon may provide significant insight into human muscle function and movement performance that cannot be predicted from examination of the architectural features of resting (non-contracting) muscles.

Keywords: biomechanics; muscle; mechanics; physiology; contractile properties; belly gear; AGR; ageing; neuromuscular disorders; muscle imaging

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I. Background

Skeletal muscles exhibit a wide range of contractile behaviours to meet mechanical demands across diverse movement tasks. A muscle can actively shorten to generate positive mechanical work and act as a motor, it can contract isometrically to brace a body part or to allow it to act as a strut, or it can actively lengthen to dissipate energy as a brake during eccentric (lengthening) contractions (Dickinson *et al.*, 2000). This remarkable versatility is partly determined by the structural arrangement of its fibres and fascicles because a muscle's structure determines both its force-generating capacity and its functional excursion range (Burkholder *et al.*, 1994). Consequently, a muscle's structure significantly influences its function, and this relationship has been a central focus in the study of animal and human movement for centuries (Lieber & Ward, 2011; Narici, Franchi & Maganaris, 2016).

Despite longstanding investigations on how a muscle structure impacts its function, much of our understanding of muscle structure in humans stems from observations made with the muscle at rest, typically obtained through anatomical dissections or imaging techniques (e.g., ultrasonography or magnetic resonance imaging), and with its function tested whilst working isometrically or as a motor during concentric (shortening) contractions. This is despite many years of research showing an extraordinary capacity of muscles to dynamically change shape and structure during contraction and the fact that many motor activities require muscles to function eccentrically. Dynamic changes in muscle shape fundamentally impact muscle mechanical performance as it directly influences both fascicles and muscle velocity in a process known as *muscle gearing*. Muscle gearing refers to how alterations in fibre or fascicle orientations allow muscles to optimise their contractile performance under specific mechanical conditions (Azizi, Brainerd & Roberts, 2008; Wakeling *et al.*, 2011). Throughout this review, we will use 'fibres' and 'fascicles' interchangeably as it is often assumed that fascicles represent fibre behaviours, even though they are anatomically distinct.

The purpose of this review is to describe our current understanding of muscle mechanics and function, focussing on the concept of 'muscle gearing' (also referred to as belly or architectural gear ratio; see ref. Pinto *et al.*, 2023 for a discussion of methodological and nomenclature differences) and to discuss some potential applications in the biological and health sciences. First, we present a historical overview of the evolution of our understanding of the relation between muscle architecture (structure) measured at rest and its contractile function. Second, we define 'muscle gearing', explain how it affects function, and explore how its dynamic change during contraction can impact function under various contractile conditions. We then review our current understanding of the factors underpinning muscle gearing and its changes during contraction and employ a computational modelling approach to address the limitations introduced by different experimental designs that can influence muscle gearing and complicate between-study comparisons. In this computational framework, we conduct a series of simulations using a previously published three-dimensional (3D) muscle model (Wakeling *et al.*, 2020; Almonacid *et al.*, 2024b) to investigate the mechanical phenomena underlying muscle gearing. We then review potential differences in gearing in both animal and human studies and discuss factors that might influence gearing in muscles operating *in vivo* that may not be captured in isolated muscle preparations. Finally, we briefly review the potential role of muscle gearing in the changes in contractile function observed in response to ageing, muscle and ligamentous injury, and physical exercise training.

II. The muscle structure–function relationship

(1) Importance of muscle architecture: the 'classic' view

An appreciation of the relationship between skeletal muscle structure and function dates back to the Renaissance period (for detailed historical perspective, see refs. Narici *et al.*, 2016; Kardel, 1990, 1994) with early anatomical drawings already distinguishing architectural

features. The classic modern review papers and texts typically classify a muscle's architectural design into one of two major categories: *parallel-fibred muscles*, with fibres oriented along the muscle's line of action, and *pennate muscles*, with fibres oriented at an angle to it. These structural differences have important functional implications because they directly impact a muscle's contractile capacity, including its force–length and force–velocity characteristics (Charles *et al.*, 2022; Sacks & Roy, 1982). As such, a muscle's architectural design is an excellent predictor of its force-generating capacity and functional excursion range – the distance over which a muscle can contract – and thus strongly impacts overall muscle function (Lieber & Ward, 2011; Lieber & Fridén, 2000).

In most cases, parallel-fibred muscles have longer fibres than pennate muscles so they contain more sarcomeres in series, are capable of larger excursion ranges, attain faster dynamic contraction speeds, and have a greater opportunity to work near the peak of their (usually wider) force–length relationship (Lieber & Ward, 2011; Gans & Bock, 1965; Woittiez, Huijing & Rozendal, 1983). Such design is thus thought to be useful for the execution of high-speed, dynamic movements (Blazevich & Sharp, 2005; Lieber & Fridén, 2000). Conversely, pennate muscles are thought to be better suited to higher force production because, although their fibres are typically shorter relative to the muscle's length and run at an angle to the muscle's line of action (decreasing the component of force in this line of action; Blazevich & Sharp, 2005; Gans & Bock, 1965; Zajac, 1992), pennate fibre arrangement allows for more contractile tissue to pack onto a given area of tendon or aponeurosis, thus increasing the number of parallel sarcomeres and the physiological cross-sectional area (Blazevich & Sharp, 2005; Gans & Bock, 1965). However, their muscle excursion range may be somewhat compromised due to their shorter fibres having fewer sarcomeres in series and shorter excursion ranges (Muhl, 1982; Lieber & Fridén, 2000; Alexander, 1983). These characteristics form the foundations of the

137 structure–function relationship and highlight the architectural trade-off between muscle force
138 versus shortening range and velocity capacities.

139 Historically, insights into how architectural design affects muscle force production was
140 primarily obtained from geometrical (mathematical) models or anatomical dissections in
141 cadaver specimens under non-contractile (fixed) conditions. Technological advances, such as
142 sonomicrometry and ultrasound imaging (Blemker, 2023; Roberts & Dick, 2023), have more
143 recently allowed for muscle architecture to be measured *in vivo* and then for muscle function
144 measurements to be made whilst imaging the muscle during contractions in animals *in vitro*
145 and *in vivo* (Blemker, 2023; Roberts & Dick, 2023). This has led to a more 'contemporary
146 view' of muscle design that considers the muscle's ability to dynamically change shape (and
147 its architecture) during contraction and how these changes affect function across a range of
148 contractile conditions (e.g. different forces or velocities). While fibre and fascicle behaviours
149 during contraction are influenced by a muscle's overall architectural design, other factors such
150 as connective tissues, intramuscular fat and internal and external pressures (Kelp *et al.*, 2023;
151 Eng, Azizi & Roberts, 2018; Roberts *et al.*, 2019) are thought to influence muscle shape
152 changes during contraction. Changes in muscle shape, in turn, affect fascicle behaviours,
153 implying that fascicle behaviour and shape changes cannot be accurately predicted solely from
154 the architectural features of relaxed muscles. In the following section, we present evidence for
155 and clarify our current understanding of how dynamic muscle shape changes impact fascicle
156 behaviour and thereby influence overall muscle function during dynamic, active conditions.

157

158 **(2) Muscle shape change and fibre behaviour during active muscle contraction: a**
159 **'contemporary' view**

160 It is now widely accepted that muscle contraction involves fibres shortening and
161 transverse expansion to conserve volume, causing both muscles and their fibres thicken (bulge)

as they shorten. Two early researchers, Jan Swammerdam and Niels Stensen, were instrumental in advancing our understanding of this phenomenon. In the 1660s, Jan Swammerdam demonstrated that touching the exposed nerve caused the isolated frog gastrocnemius muscle to contract and swell while maintaining constant volume (retrieved from Kardel, 1996). Around the same time, the Danish anatomist Niels Stensen (also known as Nicolaus Steno) proposed the geometric theory of muscle contraction, later published in *De Musculis* and *Elementorum Myologiae Specimen*, which included numerous geometrical diagrams of muscles (Castel-Branco & Kardel, 2022).

Stensen's theory emerged from his innovative anatomical practice of making longitudinal cuts along muscle fibres rather than the more common transverse anatomical sections of his time. This approach allowed him to visualise the internal organisation of muscle fibres and likely led to his proposition that muscles could be described using geometrical principles and Euclidean geometry (Castel-Branco & Kardel, 2022). Stensen described both parallel and pennate muscle contraction using parallelepiped geometrical models, a concept now widely adopted in biomechanics and known as the "unipennate actuator model" (reviewed in Kardel, 1994, 1990).

Like Swammerdam, Stensen theorised that muscle volume remained unchanged during contraction, but he further proposed that this process could be explained by geometry (Figure 1B) without recourse to the notion of animal spirits, a widely held belief at the time (Cobb, 2002). Using Euclid's *Elements*, Stensen demonstrated that variation in the oblique angles of muscle fibres during contraction increases muscle thickness while preserving volume. In doing so, he anticipated modern concepts of pennate muscle contraction mechanics, using novel anatomical observations with mathematical and physical reasoning.

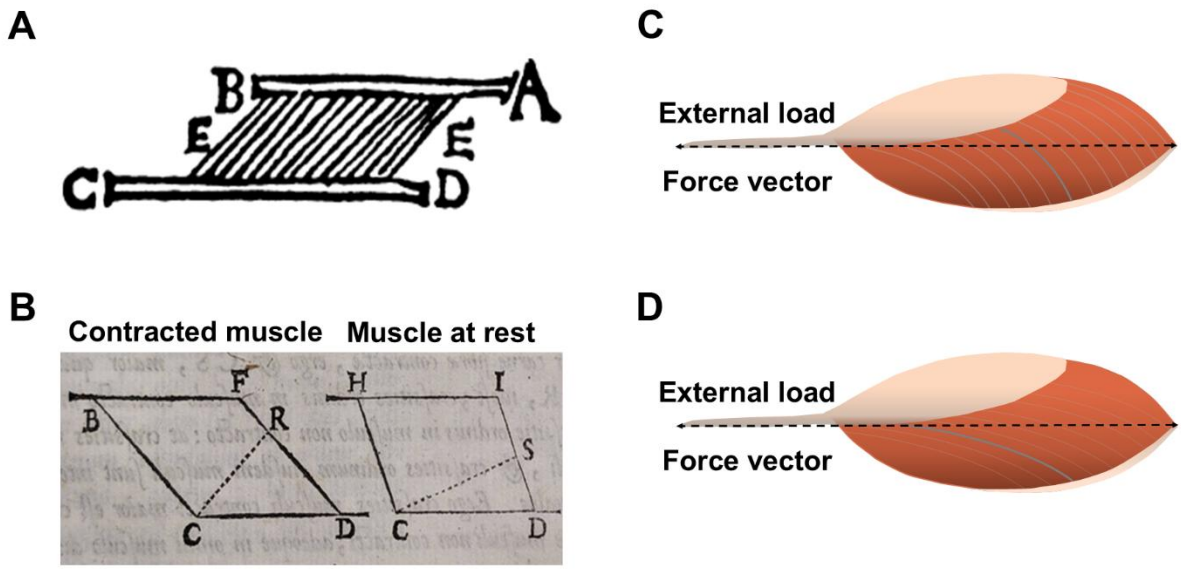

Figure 1. (A) Geometric representation of a muscle sketched by Niels Stensen on April 30, 1663. This sketch was proposed in a letter from Stensen to Thomas Bartholin and was used to represent the structures within a muscle using geometry (information and Figure retrieved from ref. Kardel, 1994; reprinted with permission). This is now known as the 'unipennate actuator model' and is commonly used in biomechanical and physiological studies of skeletal muscles. **(B)** Parallelogram representation from *Elementorum Myologiae Specimen* (1667) illustrating a muscle at rest and in the contracted state (adapted from Castel-Branco & Kardel, 2022; licensed under CC BY 4.0). Stensen applied Euclidean geometry to demonstrate that muscle volume remains constant during contraction. Shortening and rotation of the obliquely oriented fibres (from CS to CR) increases muscle thickness, defined here as the shortest (perpendicular) distance from point R to the intersection of the lower aponeurosis (line CD) without altering the area of the parallelogram. This geometric interpretation anticipated modern concepts of pennate muscle contracting, in which fascicle rotation during contraction causes the muscle to bulge, conserving volume despite visible bulging. **(C)** Representation of a muscle with large and **(D)** small pennation angles (relative to the muscle's line of action). Notice that, during muscle contraction, the change in segment length is expected to be greater than the fascicle length change in B, however there is a cost to peak force production because the component of fibre force produced along the muscle's line of action (external load/force vector) is reduced when compared to a muscle with lesser pennation, i.e., force along tendon = fibre force × cos(pennation angle). This illustrates a fundamental trade-off between shortening capacity and transmission effectiveness.

Although Swammerdam's and Stensen's contributions were foundational, Stensen's geometrical theories faced significant resistance for over 300 years. It was not until the 1920s that further work provided more detailed description of the geometric changes occurring during contraction of pennate muscles, incorporating mechanical principles to explain these changes more effectively. In 1925, Beritoff (1925) demonstrated that pennate muscle fibres shortened

less than the whole muscle and its associated tendon due to increases in pennation angle during contraction. Beritoff (1925) further contended that whole muscle shortening distance would depend on muscle thickening (bulging) as it allows for greater fibre rotation. This marked a critical transition from our understanding of muscle behaviour *during* contraction.

Subsequent studies have confirmed and expanded on Beritoff's findings. For instance, Feneis recorded fibre angle changes during locomotion of transparent crabs ((Feneis (1935); reviewed in ref. Benninghoff & Rollhäuser, 1952)), and Benninghoff & Rollhäuser (1952) experimentally demonstrated increased muscle shortening distance in pennate muscles. These findings were also later reviewed in the theoretical work of Gans & Bock (1965). However, experimental data confirming Beritoff's results were not published in English until the 1980s. In a key study published in 1982, Muhl exposed the digastric muscle of rabbits and captured photographs of the fascicles and whole muscle belly both at rest and during active contractions at several muscle lengths while recording forces at the tendon. Consistent with earlier predictions, Muhl (1982) observed changes in both fascicle length and angle during contraction, concluding that "for a given length of fibre, muscle excursion is enhanced by pinnation, relative to muscles in which the fibres lie in parallel to the line of muscle action." Other experimental and modelling results confirmed greater whole muscle than fibre shortening distance and velocity in unipennate muscles, which was not observed in parallel-fibred muscles of equal fibre length and physiological cross-sectional area (Huijing & Woittiez, 1983; Woittiez *et al.*, 1984; Woittiez & Huijing, 1984). Mathematical models confirmed that this dissociation between fibre and whole-muscle velocities resulted from changes in fibre angle during shortening (Woittiez *et al.*, 1984; Huijing & Woittiez, 1983; Otten, 1988). While these studies have collectively shown that fascicle rotation could impact muscle function through its effect on force–velocity and force–length relations, many researchers subsequently either neglected its contribution to force production (Bobbert, Huijing

& van Ingen Schenau, 1986; Bobbert & van Ingen Schenau, 1990; Scott & Winter, 1991) or assumed fibre angles remained fixed during contraction (Sacks & Roy, 1982; Spector *et al.*, 1980; Scott & Winter, 1991).

The functional consequences of incorrectly assuming fixed fibre angles during contraction, or instead correctly assuming that angles change in proportion to fibre length change, were tested in the models developed by Scott & Winter (1991). In the early 1990's, the authors demonstrated that fibres would contract faster than the whole muscle if they contracted at a fixed pennation angle (i.e., no fibre rotation). This implies reduced *fibre* force-generating capacity. However, when fibres were allowed to rotate during shortening, *fibre* speed was slower than *muscle* shortening speed. This would provide significant benefit to *fibre* force generating capacity; specifically, it would allow the fibres to work more optimally in relation to their force–length and force–velocity relations and for the muscle to operate over a broader range of lengths (widening the *muscle's* force–length relationship and its power–velocity relationship; as in Figures 2 and 3). Thus, fibre rotation was found to enhance muscle length change and velocity capacities (Scott & Winter, 1991), with profound implications for muscle function. Similar findings were confirmed experimentally in animal species by Zuurbier & Huijing (1992, 1993).

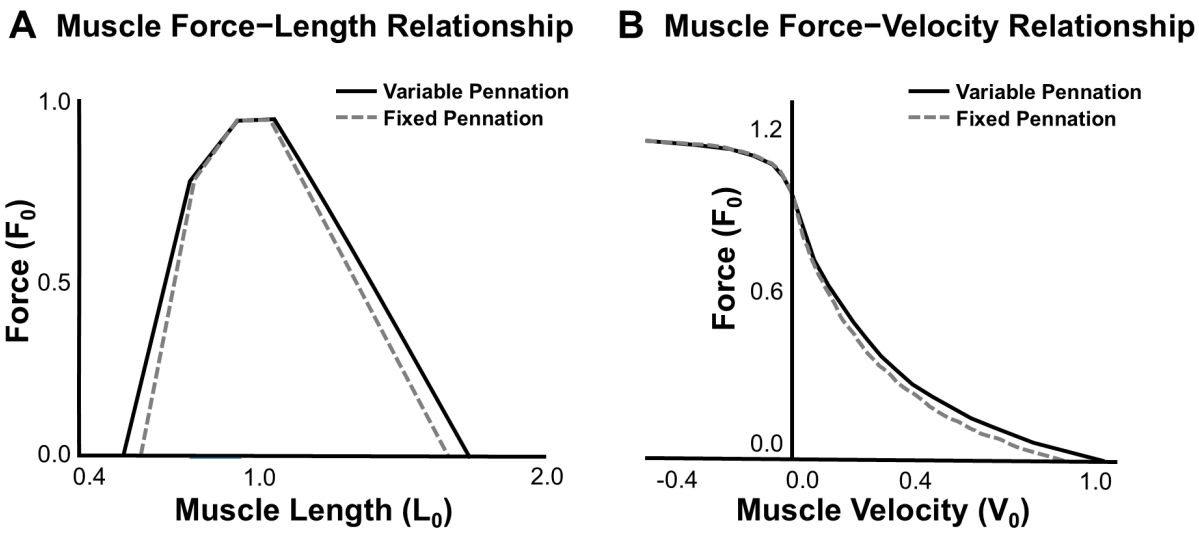

Figure 2. Effects of variable and fixed pennation (solid black and broken grey lines, respectively) on muscle (A) force−length and (B) force−velocity relationships. Figures adapted from Scott & Winter (1991). Reprinted with permission.

Nonetheless, while there was some appreciation of the effects of fibre rotation on muscle function, the question remained as to which intrinsic and extrinsic factors impacted the magnitude of rotation. Earlier studies of contracting muscles had already shown that pennate muscles increased in thickness during shortening and decreased during elongation, which, if not accounted for in the models, would significantly affect the muscle and tendon length changes for a given fibre shortening distance because changes in thickness significantly impact fibre rotation (Huijing & Woittiez, 1983; Woittiez *et al.*, 1984). However, such models did not consider that the muscle does work on surrounding tissues such as the aponeurosis and skin (not to mention neighbouring muscles and bones) as it expands (thickens) during shortening (Reinhardt *et al.*, 2016; Wakeling *et al.*, 2020) – thickness expansion was often described as small to negligible so that it has been neglected for many decades. These tissues have elastic properties that can resist deformation when work is done, only stretching to store a limited amount of energy. In 1969, Alexander's theoretical framework suggested that the angled collagen fibres in fish myosepta could impact the fish's lateral body movement and muscle bulging during shortening, although he (Alexander, 1969) and others (Otten, 1988; Gans & Bock, 1965) equivocally assumed that, because muscle maintains constant volume during contraction (Baskin & Paolini, 1967), the distance between the tendon plates (aponeuroses) must remain constant. Overall, these studies and others published in the 1920's (Beritoff, 1925), 1970's and 1980's provided evidence that (i) muscles could change in thickness (bulge) during contraction and that this would impact the magnitude of fascicle rotation and consequently muscle and tendon excursions, and (ii) non-contractile tissues surrounding muscles could potentially impact the amount of muscle deformation, but it was unclear to what extent these assumptions affected overall muscle function.

In the early 2000's, researchers provided preliminary evidence for the effect of connective tissues' material and structural properties on changes in muscle thickness and fibre rotation, and thus the extent to which these complex muscle-connective tissue structures influenced skeletal muscle function. Using the salamander's segmented musculature as a study model, Azizi, Gillis & Brainerd (2002) demonstrated that the extent of decoupling between muscle segment and fibre strains was potentially affected by the magnitude of the muscle segment bulging in the height or width directions – note that muscle bulging in height is synonymous with 'muscle thickness' in the literature, which is normally the shortest or vertical distance between the superficial and deep aponeuroses, while bulging in the width direction is equivalent to the change in distance orthogonal to the thickness direction in the transverse plane. They showed that segment strains exceeded fibre strains when shortening was accompanied by bulging in the thickness direction but not when thickness remained constant (a common assumption in earlier models; Alexander, 1969; Otten, 1988). Muscle volume was assumed to remain constant and connective tissues were considered to contribute to limiting bulging in one direction and thus prompting bulging in the orthogonal direction to maintain a constant muscle volume (Azizi *et al.*, 2002). Morphological analysis of the salamander's myoseptum (the long, intra-muscular connective tissue or aponeurosis) revealed that collagen fibre orientation was angled to help resist deformation in the width (mediolateral) direction, redirecting the bulging to the thickness (dorsoventral) direction. This 'bulge control hypothesis' was proposed to explain muscle bulging direction and magnitude in pennate muscles, subsequently influencing the magnitude of fibre rotation and consequent increases in the muscle-to-fibre strain ratio.

In subsequent studies, Brainerd & Azizi (2005) proposed that the effect of fascicle rotation could be characterised by determining the ratio between muscle segment and fibre shortening, which was referred as the 'architectural gear ratio' (AGR). Using mathematical

308 simulations, they demonstrated that muscle segments shortened more for a given fibre
309 shortening distance as fascicle rotation increased, resulting in a higher AGR, and that AGR
310 was significantly influenced by the initial fibre angle and bulging condition. Larger initial fibre
311 angles resulted in greater fibre rotation and AGR, which were further increased when the
312 muscle bulged in thickness. In 2008, Azizi *et al.* (2008) demonstrated that AGR varied with
313 the mechanical demands of the task, with a higher AGR enhancing muscle-to-fibre shortening
314 velocity in low-force contractions but a low AGR improving force output during high-force
315 contractions. Overall, these findings provided a novel perspective for understanding muscle
316 function by highlighting the importance of dynamic muscle architectural and whole-muscle
317 shape changes during contraction, which had been previously under-appreciated.
318

319 **III. Muscle gearing: definition and functional importance**

320 Muscle gearing refers to a mechanism that affects muscle length and velocity during
321 contraction through fibre rotation and its relation to muscle shape change (Azizi *et al.*, 2008;
322 Wakeling *et al.*, 2011). Fibre rotation partly decouples fascicle velocity from the whole muscle
323 velocity, and this gearing effect can be characterised by the ratio of whole muscle (or muscle
324 segment) to fibre (or fascicle) length change or velocity (for nomenclatures and methods, see
325 ref. Pinto *et al.*, 2023) (Azizi *et al.*, 2008; Brainerd & Azizi, 2005). Gear is >1.0 when muscle
326 velocity is greater than fibre velocity, is 1.0 when the muscle shortens or lengthens at the same
327 velocity as its fibres and is <1.0 when muscle fibres shorten or lengthen faster than the whole
328 muscle. In pennate muscles, the muscle gear ratio is generally ≥1.0 (Eng *et al.*, 2018), although
329 this has not been systematically described and is within the purpose of the current review (see
330 Table 2).

331 Changes in muscle shape during contraction significantly affect the magnitude of
332 fascicle rotation and therefore the decoupling of fibre/fascicle velocity from whole muscle

333 velocity. Given that shape changes and rotation are governed by the force exerted by a muscle
334 and the load imposed on it (Eng *et al.*, 2018; Azizi *et al.*, 2008) the extent of this decoupling
335 changes in response to the mechanical demands of the contraction. Therefore, the gear in which
336 a muscle operates depends on the force produced during contraction (Azizi *et al.*, 2008; Eng *et*
337 *al.*, 2018). Muscles have been shown to operate in high gear when contracting against light
338 loads or when generating lower force and possibly higher velocity (Azizi *et al.*, 2008). These
339 contraction conditions are characterised by substantial fibre rotation and changes in muscle
340 thickness, resulting in large muscle length changes (Azizi *et al.*, 2008). Conversely, muscles
341 shift to low gear when contracting against heavier loads and producing high forces with lower
342 velocity (Azizi *et al.*, 2008). Under these conditions, fibres rotate less and muscle shape
343 changes more predominantly in width, resulting in less muscle length change (Azizi *et al.*,
344 2008) since fibres are more aligned to the tendon (Figures 1C and D). Whilst it remains unclear
345 as to whether gearing is indeed affected by force or rather velocity (Wakeling *et al.*, 2011;
346 Azizi *et al.*, 2008; Dick & Wakeling, 2017), changes in gear, i.e., *variable gearing*, allow
347 muscle force output to better match the demands of a task by changing fibre behaviours such
348 that, like in other geared systems, gear ratio will adjust to favour force production under high
349 loads and movement speed under low loads (Eng *et al.*, 2018; Roberts *et al.*, 2019; Dick &
350 Wakeling, 2017).

351 Load-dependent fascicle behaviour and gearing have important functional implications.
352 During *concentric* contractions, variable gearing broadens the muscle's functional range
353 (Figure 3), shifting and perhaps more importantly broadening (widening) the power−velocity
354 relation (although it has no effect on the peak power). Whilst shifts in gear may allow muscles
355 and their fibres and fascicles to partially overcome the force−velocity constraints associated
356 with cross-bridge kinetics (time-dependent cross-bridge detachment and re-attachment; Rome
357 *et al.*, 1999; Podolsky, Nolan & Zaveler, 1969) by enabling them to operate more optimally

within their force–length and force–velocity relations, most of the benefit of variable gearing appears to manifest at the whole muscle level, as depicted in Figure 3. A *muscle* that maintains constant gear in response to variations in load (and thus muscle force) displays a narrower power–velocity relation that is largely dictated by and closely mirrors the *fibre* power–velocity relations. In contrast, a muscle that appropriately changes gear displays a broader whole-muscle power–velocity relation that extends beyond the limits of the fibres (e.g., Figures 3 and 11). During *eccentric* contractions, variable gearing allows muscles to lengthen more than the fascicles at any given external load and is therefore a feature of pennate muscles that reduces active fibre strains and potentially minimises muscle damage and the risk of strain-related injury during eccentric (lengthening) contractions (Pinto *et al.*, 2023; Azizi & Roberts, 2014). Given that variable gearing allows the muscle to broaden its performance range, understanding the mechanisms of gearing may have a range of clinical and health applications.

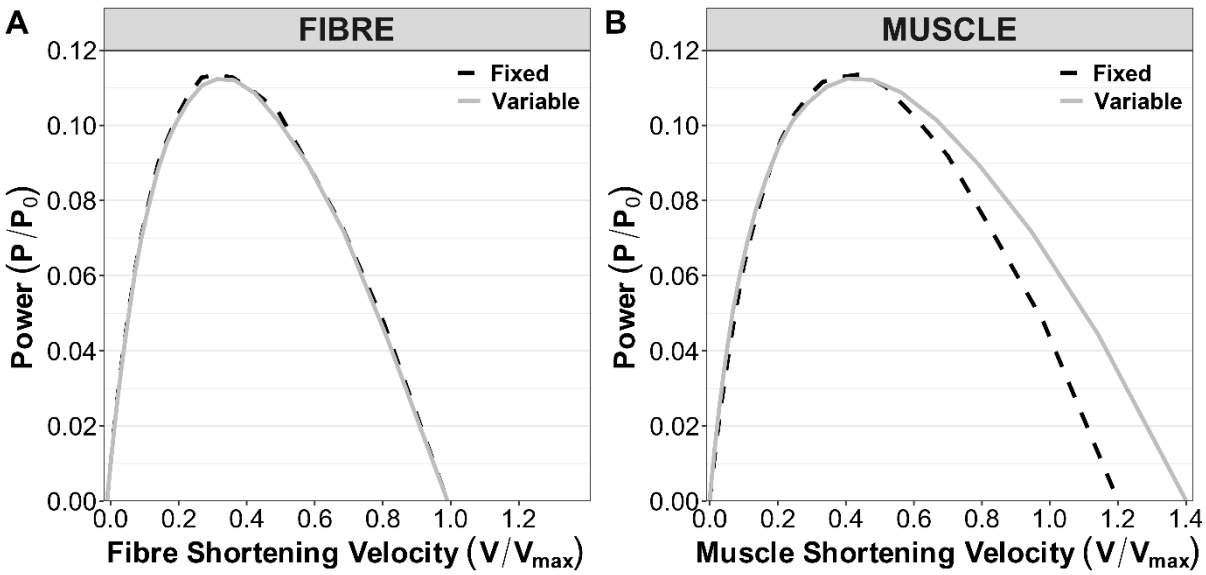

Figure 3. Effect of fixed (gear = 1.2; black dashed line) and variable (continuous grey line) gearing on fibre and muscle power–velocity relationships. Variable gearing broadens the range of muscle power production over a fibre velocity range. This figure is adapted from Fig. 6 in Azizi *et al.* (2008), where both force and shortening velocity are expressed relative to the maximal force and velocity, respectively. Power was calculated from the available shortening velocity and force data using values estimated using open available graph digitiser (WebPlotDigitiser, https://apps.automeris.io/wpd/).

However, despite the functional importance of variable gearing, our understanding of the mechanisms and structures influencing fascicle rotation, muscle shape changes, and gear shifts remains an evolving area of research. A range of studies have investigated different aspects of the mechanisms of variable gearing, including the role of pennation, three-dimensional expansion, tissue properties, intramuscular pressure, external pressure and external work on the muscle surface. In the following section, we present evidence for the inferred and deduced mechanisms for these different aspects.

(1) Mechanisms of variable gearing: what have we learnt from studies of isolated muscles of animals and physical models?

Initial evidence supporting variable gearing predominantly comes from studies of isolated muscle preparations, where supramaximal and short-lived stimulations are delivered to muscles to evoke supramaximal and non-transient activations, in addition to bioinspired muscles. These studies overall suggest that this feature is not imposed by the nervous system (Azizi *et al.*, 2008; Azizi & Roberts, 2013; Sleboda, Roberts & Azizi, 2024; Wang *et al.*, 2022). Instead, a growing body of evidence indicates that variable gearing and muscle shape changes during contraction result from the incompressible nature of skeletal muscle tissues and their composite structure (Eng *et al.*, 2018). Variable gearing appears to be mediated by how the fibre (vectorial) forces and elastic connective tissue elements affect, and therefore interact with, one another (Roberts *et al.*, 2019; Eng *et al.*, 2018). These elastic structures are found at the levels of the fibre, fascicle, and whole muscle level, and their arrangement in addition to their quantity appear to contribute to gearing. These structures are tightly packed within a volume constrained structure (the whole muscle belly). Fibre orientation, i.e., pennation angle, is also an important factor influencing muscle gearing and, as such, it influences load-dependent gear

change (Azizi *et al.*, 2008; Brainerd & Azizi, 2005). Experimental and theoretical evidence for the influence of each of these factors on the dynamics of muscle gearing is discussed below.

(a) Pennation angle

Resting pennation angle strongly influences muscle gear partly because muscles with a higher pennation at (rest and) the start of muscle contraction display greater fibre or fascicle rotation. This greater rotation more effectively contributes to muscle length change for a given amount of fascicle shortening (Eng *et al.*, 2018; Brainerd & Azizi, 2005) (as illustrated in condition A, Figure 4, Table 1). However, high gear should also be observed in muscles with greater initial pennation, even when undergoing *equal* fascicle rotation compared to those with less initial pennation. This is because a given change in fascicle angle results in a larger change in muscle (segment) length when initial pennation is higher. Specifically, the end points of a given fascicle would move more in the *longitudinal direction* when initial pennation is high but more *orthogonally* (in the thickness direction) when pennation is lower. This behaviour can be explained by the geometrical principles of sine and cosine laws. For a given change in angle, there will be more significant increases in the height (i.e. muscle thickness) and less reduction in the adjacent (muscle) segment length when initial angle is low (although this will vary according to fascicle length and its changes). Conversely, when initial angle is high, the same change in angle will contribute more to the reduction of the segment length and less to height increase. As muscles often increase in thickness during dynamic muscle contraction, this is an expected behaviour, although in some cases muscles may also exhibit little to no change in thickness during contraction *in vivo* (Maganaris, Baltzopoulos & Sargeant, 1998; Narici *et al.*, 1996; Wakeling *et al.*, 2011). However, this behaviour should occur even if muscle thickness remains constant during contraction, as muscles with greater starting pennation will work in

higher gear. Thus, the initial pennation angle is an important factor influencing gear (Azizi *et al.*, 2008; Wang *et al.*, 2022; Brainerd & Azizi, 2005).

Despite evidence indicating that higher pennation is associated with greater rotation and larger gear ratios (Tijs, Konow & Biewener, 2021; Brainerd & Azizi, 2005), it is important to note that muscles with higher pennation do not always exhibit a higher gear (Azizi & Brainerd, 2007). This inconsistency illustrates muscle gearing as a multifactorial phenomenon (as further discussed below).

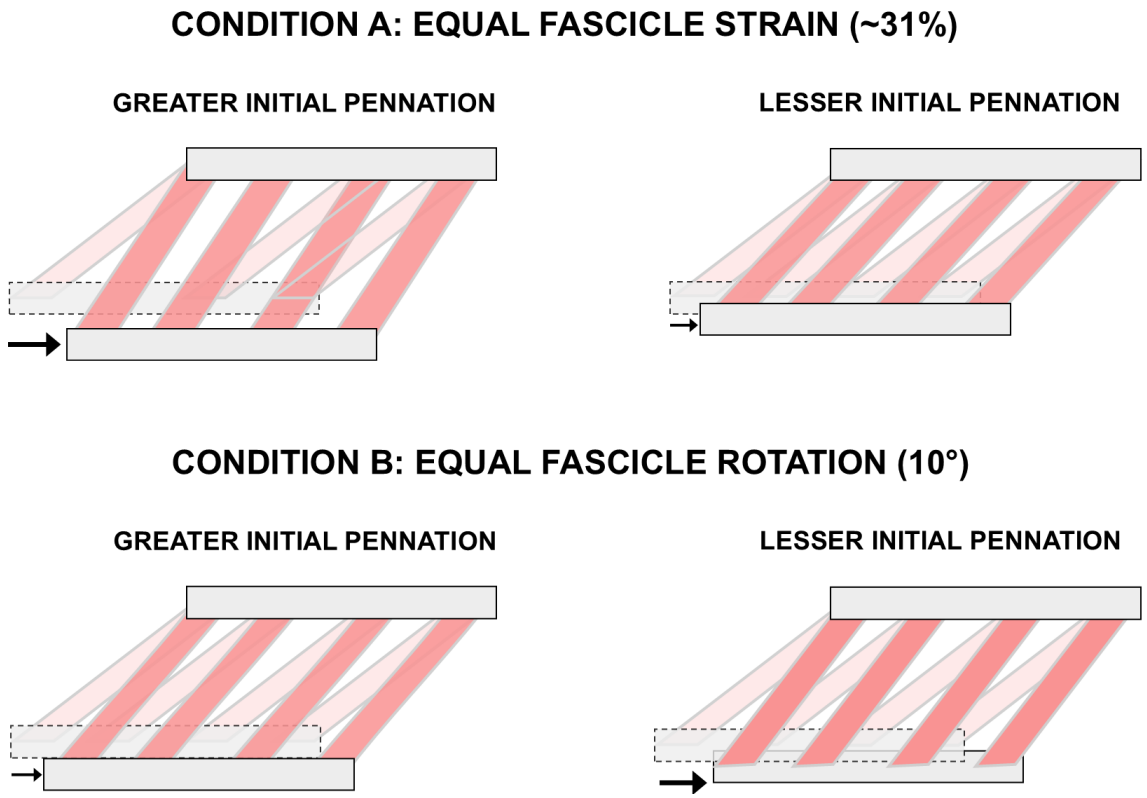

Figure 4. The effect of initial pennation angle on fascicle rotation, muscle segment length change, and muscle gear during contraction with equal fascicle shortening (condition A) and equal fascicle rotation (condition B). The architectural characteristics of each example are given in Table 1. Black arrows indicate the magnitude of muscle segment shortening. Note: figures are not drawn to scale; they are representations of the phenomena.

Table 1. Architectural parameters for the biplanar sketch in conditions A and B in Figure 4.

CONDITION A - Equal fascicle shortening, 20% thickness increase

	Higher initial pennation					Lower initial pennation				
Before shortening	L_f	L_t	β	L_{seg}	Gear ratio	L_f	L_t	β	L_{seg}	Gear ratio
	6.55	3.40	31.3	5.60		6.55	2.16	19.3	6.18	
After shortening										

	4.50	4.10	64.6	1.94	**1.80**	4.50	2.60	35.1	3.70	**1.22**
Change (Δ)	2.03	0.68	33.3	3.66		2.03	0.43	15.8	2.48	

CONDITION B - Equal fascicle rotation, 20% thickness increase

	Higher initial pennation					Lower initial pennation				
Before shortening	L_f	L_t	β	L_{seg}	Gear ratio	L_f	L_t	β	L_{seg}	Gear ratio
	6.55	3.40	31.3	5.60		6.55	2.16	19.3	6.18	
After shortening										
	6.19	4.10	41.3	4.65	**2.61**	4.41	2.16	29.3	4.63	**1.25**
Change (Δ)	0.36	0.68	10.0	0.95		1.24	0.43	10.0	1.55	

L_f = fascicle length (cm); L_t = muscle thickness (cm); β = pennation angle (degrees); L_{seg} = muscle segment length (cm); Change (Δ) = absolute difference between parameter before and after shortening.

(b) Fibre radial expansion vs. thickness forces

When pennate muscles shorten to generate active force, the fibres and fascicles undergo transverse deformations to maintain (approximately) constant volume (Baskin & Paolini, 1967). As fibres are packed within a volume-constrained muscle space, running adjacent to each other and with tight packing density, the radial expansion of adjacent fibres pushes neighbouring fibres away from each other, and this occurs as fibre rotates to greater pennation angles (Dick & Wakeling, 2017; Figure 5, blue arrows). Decreased radial expansion of the fibres concurs with reduced rotation of the fibres (Ryan *et al.*, 2019). Similarly, decreasing the spacing between neighbouring McKibben actuators reduces actuators rotation and eliminates variable gearing likely due to decreased transverse forces acting on adjacent actuators (Sleboda *et al.*, 2024).

Fibre rotation creates a force that acts to separate the aponeuroses and increases their distance in the thickness direction (Figure 5, green double headed arrow). This force is simultaneously counteracted by the component of the fibre shortening force that acts to reduce muscle thickness and bring the aponeuroses closer together (thickness compression; refer to Figure 5, grey arrows). The change in overall muscle belly thickness is therefore influenced by the balance of these two forces. A visual representation of this process is provided in Figure 5A-C.

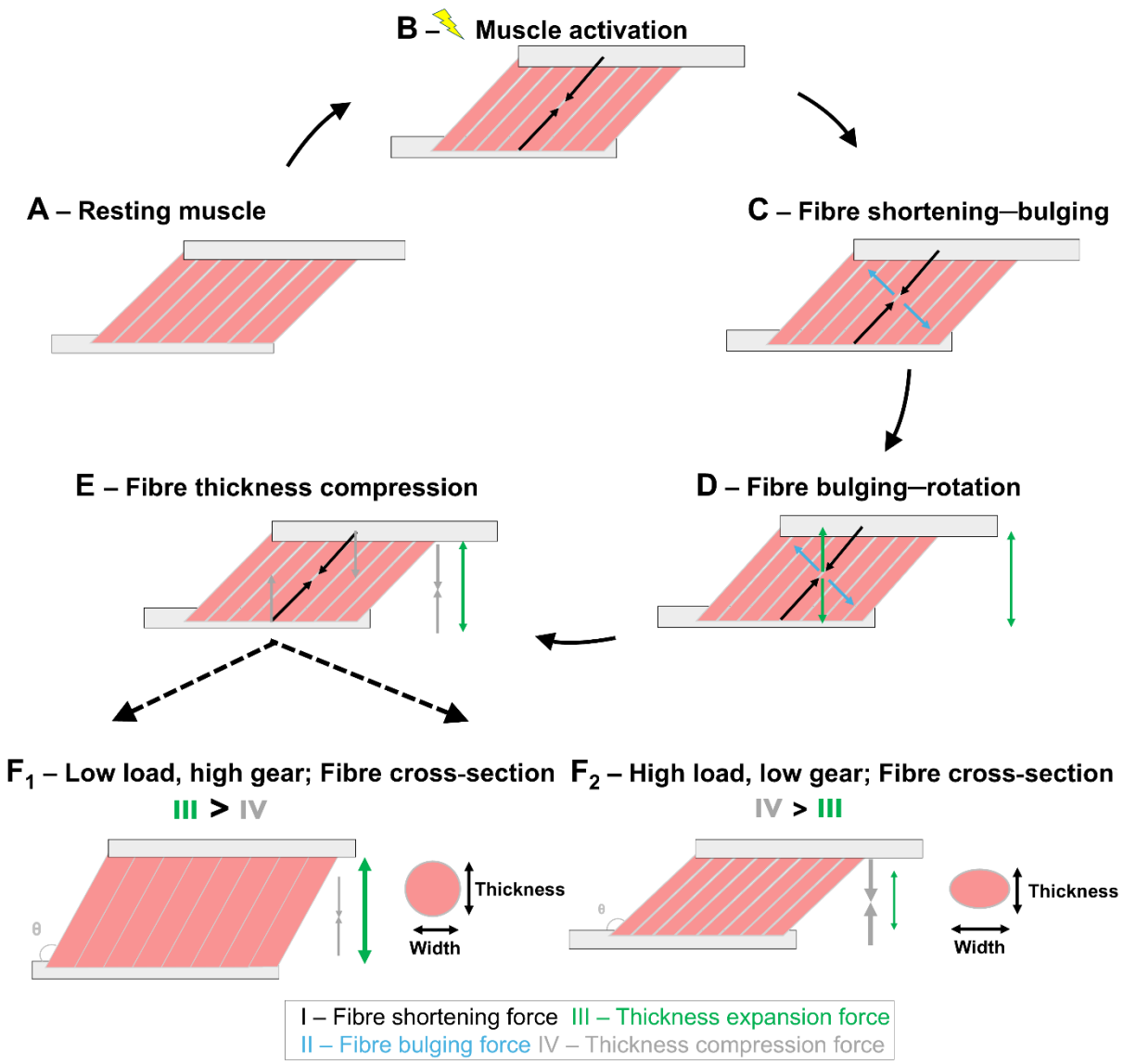

Figure 5. Architectural, muscle shape, and gear changes during muscle contraction. (A) Illustration of pennate muscle architecture in a non-activated, relaxed state, with fibres packed within a volume-constrained muscle space. (B) Upon activation and contraction, the contractile components within the fibres generate forces that shorten the fibres along their line of action (black arrows). (C) Since fibres remain nearly constant volume, fibre shortening is accompanied by radial expansion, which produces forces orthogonal to the fibres' line of action (blue arrows). Orthogonal fibre forces push neighbouring fibres away and pennation increases i.e., fibres rotate. (D) Fiber rotation creates a force that separates the aponeuroses apart, increasing the muscle belly thickness (green arrow). (E) This force is counteracted by a component of the fibre shortening force that acts to reduce muscle thickness and bring the aponeuroses closer together illustrated in grey. The change in muscle thickness is partly mediated by the balance between fibre bulging forces that promote thickness expansion and orthogonal fibre shortening forces that promote thickness compression. This balance is dictated by the amount of force fibre need to produce in response to the external load. (F) When the load is low, the thickness forces are smaller than the forces imposed by fibre rotation, resulting in greater fibre rotation and higher muscle velocity relative to fibre velocity, as illustrated in (low load, high gear). (G) When the external load is high, the fibres generate greater force,

compressing the muscle thickness and resulting in less fibre rotation and slower, more similar whole muscle velocity to that of the fibres. The muscle operates at low gear in this scenario.

Variable gearing occurs because fibre radial expansion and the fibre thickness compressive force are determined by the force fibres produce in response to the external load (Eng *et al.*, 2018; Roberts *et al.*, 2019; Azizi *et al.*, 2008). When the load is low and fibres generate low forces, fibre radial expansion occurs approximately equally in both thickness and width (assuming isotropy). This equally distributed radial expansion promotes fascicle rotation, with rotation being facilitated because the fibre thickness compressive force is smaller than the force produced by fibre radial expansion that induces fibre rotation and increases thickness (Figure 5D). However, under high loads in which fibres generate greater force, muscle radial expansion is constrained in the thickness direction because the component of fibre shortening force pulling the aponeuroses together is stronger, limiting thickness expansion but not resisting fibre and belly width-wise expansion that occurs to maintain a constant volume, as in Figure 5E. With less opportunity for belly thickness expansion and thus fascicle rotation, muscle length change is closer to the fascicle length change, and the muscle gear is low (~1.0; Figure 5). Importantly, observations that asymmetrical transverse deformations of fascicles (Randhawa & Wakeling, 2018; Kinugasa *et al.*, 2012) and muscles (Hodson-Tole & Lai, 2019) occur have been documented human muscles *in vivo*.

(c) Connective tissue material properties and architecture

Fibre forces compressing the muscle in thickness can be opposed by structural properties or alternatively by their own material properties during contraction (Eng *et al.*, 2018). The structural arrangements and material properties of the extracellular matrix (ECM) at various scales have the potential to resist such deformation (Eng & Roberts, 2018; Azizi *et al.*, 2017; Azizi *et al.*, 2002; Roberts *et al.*, 2019; Holt *et al.*, 2016; Alexander, 1969).

503 Connective tissues envelop single fibres (endomysium), fascicles (perimysium), and the whole
504 muscle (epimysium, often including the aponeurosis), and may potentially influence changes
505 in muscle shape during contraction by limiting fibre and fascicle radial expansion (Azizi *et al.*,
506 2017; Purslow, 2020) and resisting fascicle compressive forces that reduce the muscle in
507 thickness. These should ultimately influence fascicle and muscle behaviour.

508 The endomysium consists of an organised honeycomb-like network of collagen fibres
509 that envelope and connect individual adjacent muscle fibres (Purslow, 2020). Each shell forms
510 tubular structures along their longitudinal direction that run parallel to muscle fibres (Sleboda,
511 Stover & Roberts, 2020; Gillies & Lieber, 2011), although in a quasi-randomly oriented, wavy
512 pattern (Purslow & Trotter, 1994; Rowe, 1974). These structures are connected adjacently
513 (Purslow, 2020) and wrapped by an outer layer of mesh-like connective tissue sheaths known
514 as the perimysium (Gillies & Lieber, 2011; Purslow, 2020). The perimysium is also a
515 continuous network that links muscle fascicles adjacently and is predominantly composed of
516 collagen (Purslow, 2020). It exhibits a helically wound cable structure consisting of collagen
517 fibril bundles (Sleboda *et al.*, 2020), sometimes called perimysium collagen cables (Gillies &
518 Lieber, 2011), crossing each other at angles within multiple layers and in a crimp pattern
519 (Gillies & Lieber, 2011). These ECM structures provide passive resistance to fibre and fascicle
520 radial deformations through longitudinal and shear resistances, and the structural differences
521 between the endomysium and perimysium have been hypothesised to serve distinct functions
522 (Purslow, 2020). Purslow (2020) argues that the shear-compliant perimysium potentially
523 facilitates shape changes and rotation of fascicles by allowing them to slide past each other
524 while the shear-stiff endomysium serves to maintain tight fibre alignment and thus facilitate
525 force transfer. Current evidence indicates that collagen fibre length and orientation changes as
526 the muscle fibres and fascicles shorten and bulge radially (Purslow, 1989; Trotter & Purslow,
527 1992), thus doing mechanical work on the ECM collagenous network to maintain constant

volume (Purslow, 1989; Baskin & Paolini, 1967). Azizi *et al.* (2017) constrained radial expansion by placing the frog (parallel-fibred) palmaris longus muscle within a rigid tube and showed a notable reduction in longitudinal mechanical muscle shortening work. Mathematical models using a fibre-wound cylinder revealed that fibre collagen angle and area substantially influenced radial expansion and longitudinal strain, consistent with the empirical data (Azizi *et al.*, 2017). As illustrated in Figure 6, collagen fibres parallel to the longitudinal axis of fibre and fascicles limit fibre shortening in the longitudinal direction and fibre circumferential shape. Thus, ECM material and structural properties may influence the magnitude of fibre rotation and thus the gear in which a muscle operates, potentially impacting skeletal muscle function.

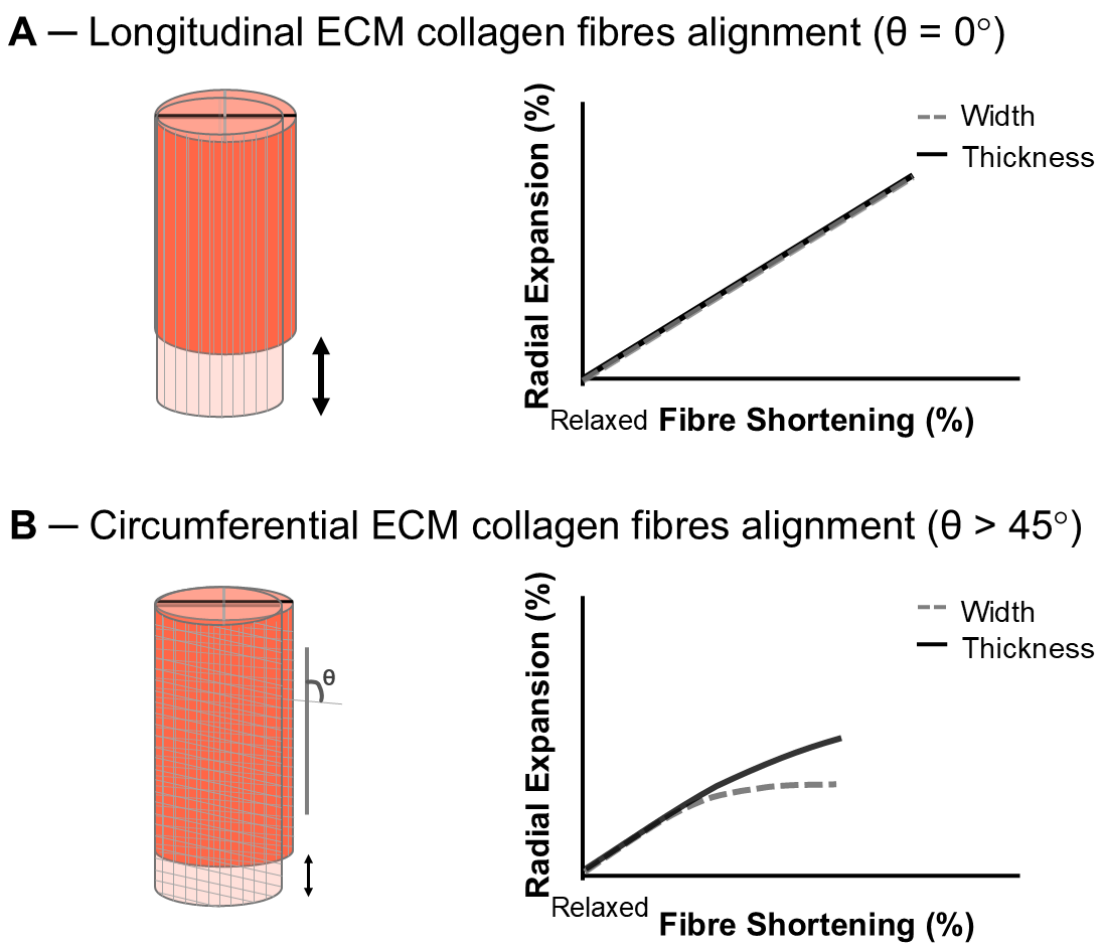

Figure 6. Hypothetical example illustrating the effect of extracellular matrix (ECM) collagen fibre orientation on fibre or fascicle strain and shape changes in width (grey) and thickness (black), assuming constant fibre or fascicle volume. Relaxed fibres (light pink) are overlaid on active fibres (orange) to illustrate deformation and longitudinal fibre strain (double-headed arrow). In panel A, the ECM collagen fibres align with the fibre's longitudinal axis at a negligible angle (θ = 0°), primarily resisting longitudinal fibre shortening (Azizi *et al.*, 2017).

If the ECM behaves isotropically, providing uniform resistance in all directions, width and thickness strains remain equal, preserving circular cross-section. However, ECM anisotropy may lead to slight deviation from perfect circularity. In panel B, ECM collagen fibres are more circumferentially orientated at a high pitch angle ($\theta > 45°$), restricting both longitudinal fibre strain and radial expansion. In this condition, ECM anisotropy is assumed, so width and thickness strains may differ and result in asymmetric deformation. If circumferential constraints (high hoop stiffness) preferentially limit width expansion, then width changes will be less than thickness changes, altering the fibre's cross-sectional shape. The grey line represents width strain, and the black line represents thickness strain.

The outermost ECM layer, the epimysium, contains thick collagen bundles arranged in a crimp-like pattern (Sleboda *et al.*, 2020; Rowe, 1974). It functions to connect and envelop the muscle fascicles, ultimately terminating by attaching to the aponeurosis or external tendon (Sleboda *et al.*, 2020). In pennate muscles, this robust sheet-like aponeurosis acts as a base for muscle fascicle attachment, into which they insert at an angle to its longitudinal direction. This collagen-predominant connective tissue is highly effective in resisting tensile stress but less so in resisting compressive loads (Morrow *et al.*, 2010; Purslow, 2020), like those exerted from fascicle forces acting to compress the muscle belly in thickness. However, during contractions in which the muscle is compressed in thickness and expands in width, these structures are stretched to resist tensile stress (Morrow *et al.*, 2010; Eng *et al.*, 2018). This resistance to width expansion is thought to promote thickness expansion to maintain constant muscle volume, ultimately influencing the change in muscle shape during contraction (Eng *et al.*, 2018; Eng & Roberts, 2018).

There is emerging evidence that the ECM influences muscle shape changes and gearing. Holt *et al.* (2016) observed that older rats worked in a near-constant (intermediate) gear regardless of the force produced, whereas young rats shifted from high to low gear as force changed from low to high. The older rats had significantly stiffer aponeuroses in the transverse direction and stiffer muscle fibre bundles in the longitudinal direction than the young rats. The use of higher gear during high-force contractions in older rats was attributed to an increased resistance to thickness compression due to the stiffer aponeurosis, resulting in less thickness

574 compression and width expansion. Consequently, the fibres of old rats rotated more, and the
575 muscle worked in high gear despite the high force, although the opposite occurred during
576 low-force contractions. Eng & Roberts (2018) demonstrated that small, longitudinal incisions
577 in the aponeurotic sheet, presumably reducing transverse stiffness and allowing width-wise
578 bulging, reduced gear during high force contractions but did not abolish variable gearing in
579 adult birds. The use of low gear at high forces was hypothesised to have resulted from increased
580 thickness compression and width expansion, indicating an inability to resist deformation and
581 prevent muscle flattening during contraction. The findings from these studies are consistent
582 with results from finite element models demonstrating that changes in aponeurosis stiffness
583 affect fibre geometry and behaviour (Rahemi, Nigam & Wakeling, 2014), and suggest overall
584 that ECM alterations can influence muscle shape changes and load-dependent muscle gear.
585

586 **(d) Mechanical pressure: internal and external sources**

587 Muscles generate internal pressure during contraction (Hill, 1948), which can influence
588 muscle performance by modifying stress distribution and resisting sarcomere shortening forces
589 (Daggfeldt, 2006). This internal pressure has been empirically measured and modelled (Van
590 Leeuwen & Spoor, 1993; Sejersted *et al.*, 1984; Heukelom, van der Stelt & Diegenbach, 1979),
591 and is thought to arise from a combination of forces, including those produced by fibre bulging
592 and internal fluids as well as structural interactions between closely packed fibres and fascicles.

593 Beyond internal sources, intramuscular pressure is further modulated by external
594 factors such as the forces exerted by adjacent muscles (Reinhardt *et al.*, 2016), bones,
595 intermuscular fat, and the ECM, which either passively resist transverse expansion or, in the
596 case of adjacent muscles contracting to general lateral pressures, actively compress the muscle.
597 External compressions or loads provide further resistance to fibres bulging and muscle shape
598 changes (Ross *et al.*, 2020; Wakeling, Jackman & Namburete, 2013; Siebert *et al.*, 2016). These

599 pressures, in turn, can influence overall muscle shape changes, velocity, and gearing, thus
600 having significant implications for muscle function.
601
602 **(i) Internal: the potential role of fluid and structural pressures**
603 Skeletal muscles contain a substantial amount of fluid, distributed within muscle fibres
604 and in interstitial spaces and blood vessels surrounding the fibres (Bhave & Neilson, 2011).
605 These fluids can influence internal pressure and thus fascicle behaviour in complex ways.
606 While fluid pressure is often cited as a factor influencing intramuscular mechanics, its precise
607 role in muscle shape changes and force transmission remains debated. Some interpretations
608 consider muscles as a hydrostatic system, where fluid movement plays an active role in
609 redistributing stress and mechanically interacts with elastic elements surrounding muscles,
610 ultimately influencing muscle shape changes and fascicle behaviour during contraction (Eng
611 *et al.*, 2018; Kier, 2012). An alternative view is that muscles behave and can be modelled as a
612 poroelastic material (Wheatley *et al.*, 2017, 2018), where internal pressures contribute to
613 structural deformation without requiring bulk fluid displacement.
614 During contraction, fibres and fascicles shorten and expand in girth (Figure 5), which
615 is thought to generate internal pressures that influence stress distribution. If fibres are implicitly
616 considered as fluid-filled hydrostats, then the hydraulic forces would redistribute contractile
617 stress between longitudinal and radial directions (Eng *et al.*, 2018). These hydraulic stresses
618 have been suggested to redirect compressive fascicle forces, causing transverse expansion that
619 stretches the surrounding connective tissues. Connective tissues resist this stretch, resulting in
620 fluid redirection (i.e., hydraulic stress redistribution) that counters thickness compression (Eng
621 *et al.*, 2018). This dynamic interplay between fibre forces, connective tissues, and fluid has
622 been suggested to involve substantial flow of energy across the fascicular and whole-muscle
623 levels and likely influence their behaviours (Roberts *et al.*, 2019; Eng *et al.*, 2018). However,

624 it should be noted that the flow of energy linking longitudinal to transverse deformations can
625 also be explained by simple structural models (Otten, 1988; Siebert, Till & Blickhan, 2014a)
626 and more complex continuum models of muscle in which fluid is absent (Wakeling *et al.*, 2020;
627 Ryan *et al.*, 2020a; Blemker, Pinsky & Delp, 2005). Poroelastic models, which account for
628 both solid and fluid interactions (Wheatley *et al.*, 2018; Böl *et al.*, 2022), may be an additional
629 framework to understand these energy dynamics.

630 Evidence from experimental studies suggests that increasing muscle fluid volume (via
631 hypotonic solutions) increases a muscle's resistance to stretch (Sleboda, Wold & Roberts,
632 2019; Sleboda & Roberts, 2017) and produces a passive shortening forces along the muscle's
633 line of action even in the absence of electrical input (Wold, Sleboda & Roberts, 2021). These
634 effects are thought to result from pressure-induced changes in ECM collagen fibre (angle)
635 orientation in a way that resists longitudinal stretch (Sleboda *et al.*, 2019; Sleboda & Roberts,
636 2017) and facilitates muscle shortening (Wold *et al.*, 2021). This behaviour resembles that of
637 'hydrostatic skeletons', like worms, where internal pressures modulate stiffness and connective
638 tissue structural arrangement to influence movement mechanics (Kier, 2012).

639 Further evidence supporting fluid pressure-driven shape changes comes from variable
640 gearing observed in McKibben pneumatic actuators arranged similarly to pennate muscles
641 (Azizi & Roberts, 2013; Sleboda *et al.*, 2024; Wang *et al.*, 2022). Azizi & Roberts (2013)
642 designed an array with actuators arranged side by side, mimicking the configuration of pennate
643 muscles. These actuators are made with elastic tubes covered by braided sleeve wires, which
644 expand radially when filled with air, causing reorientation of the braided sleeve wires and the
645 actuator to shorten (see Fig. 3 in ref. Azizi & Roberts, 2013). Actuator radial expansion and
646 shortening resulted in actuator rotation that increased array thickness, promoting transverse
647 bulging and variable gearing. However, unlike skeletal muscles, which are quasi-constant
648 volume structures (Baskin & Paolini, 1967), McKibben actuators increase in volume to

649 shorten; thus, McKibben actuators lack the complex fluid-pressure constraints imposed in
650 biological (muscle) tissues.

651 Further research is needed to examine how fluid, or the absence of it (as seen in
652 dehydrated muscle preparations), affects muscle gearing and function. Notably, dehydration
653 may induce cell stiffening and shape deformation (Guo *et al.*, 2017), raising questions as to
654 whether these effects would stem from fluid dynamics or material properties changes. The
655 mechanical properties of the solid phase alone may be sufficient to drive shape changes, as
656 anisotropic deformations can emerge through internal stress redistribution and energy transfer
657 in structured materials. Alternatively, poroelastic models offer an emerging theoretical
658 framework for understanding internal fluid pressures and stress distributions in muscle
659 (Wheatley *et al.*, 2017, 2018) as they are able to capture the biphasic nature of muscle.
660 However, ECM material properties and structural constraints likely play a more dominant role
661 in muscle shape changes and gearing than bulk fluid movement, particularly in complex muscle
662 groups that experience mechanical interactions and compressions as they contract (see below),
663 and a model without poroelastic effects should be sufficient to explain muscle shape changes
664 and variable gear.

665

666 **(ii) External pressure: surrounding structures and the effect of mass**

667 Neighbouring muscles, organs, intermuscular fat, and bones can influence transverse
668 muscle expansion and shape changes even in the absence of connective tissue alterations.
669 Indeed, intermuscular pressures between 10 and 70 kPa have been measured at the surface of
670 contracting muscles operating within their muscle package (Reinhardt *et al.*, 2016). Since many
671 vertebrates have muscles grouped tightly operating within compartments *in vivo* (Reinhardt *et*
672 *al.*, 2016; Bernabei, van Dieën & Maas, 2017), both adjacent muscles and other passive

673 structures should exert external mechanical forces on contracting muscles and thus limit
674 transverse expansions and influence shape changes.

675 Earlier evidence supporting the above was published by Siebert and colleagues. In a
676 sequence of studies, they applied transverse external compression to the surface of the rat
677 medial gastrocnemius during contraction and demonstrated reductions in the rate of force
678 development and maximal force output (Siebert, Günther & Blickhan, 2012; Siebert *et al.*,
679 2014b; Siebert *et al.*, 2016; Siebert *et al.*, 2014a). The muscle lifted the load, thus performing
680 external mechanical work to displace the load, but at the cost of reducing muscle force along
681 its line of action. This is thought to occur because forces acting at the muscle's surface must
682 be counteracted by the redirection of muscle work to the surface, consequently reducing
683 internal force generation capacity along the line of action (Wakeling *et al.*, 2020; Siebert *et al.*,
684 2014a). Further to this, Wakeling *et al.* (2013) demonstrated that external compression via
685 elastic bandage reduces belly gear and fascicle angle and muscle thickness changes during
686 contraction. These findings indicate that fascicle shortening velocity increases when transverse
687 expansion is constrained, which may reduce fascicle force-generating potential.

688 More recently, research has shown that imposing transverse pressures via external loads
689 and compressive bandages significantly impacts muscle geometry (Ryan *et al.*, 2019; Stutzig
690 *et al.*, 2019; Monte, 2021), force production (Ryan *et al.*, 2019; Stutzig *et al.*, 2019; Siebert *et*
691 *al.*, 2018), and energetic cost (Monte & Zamparo, 2023), and that such effects predominantly
692 occur when the load is placed along the muscle thickness axis but not width axis (Ryan *et al.*,
693 2020b). Further evidence from empirical data and finite element modelling indicates that
694 compression-related force reduction is exacerbated when a muscle is at shorter length (Sleboda
695 & Roberts, 2020) or has lower initial (resting) pennation (Ryan *et al.*, 2020a).

696 Further, muscles seem to show compensatory behaviours when operating within their
697 synergistic group *in vivo*. For example, Randhawa, Jackman & Wakeling (2013) showed that

698 medial gastrocnemius (MG) reduced in thickness during both isovelocity and isotonic
699 concentric plantar flexion contractions whereas the lateral gastrocnemius (LG) increased in
700 thickness; these observations have been consistently replicated in subsequent studies
701 (Randhawa & Wakeling, 2018; Wakeling *et al.*, 2011; Kelp *et al.*, 2021; Kelp *et al.*, 2023) and
702 suggest that their distinct architectural configurations influence their behaviours such that while
703 one muscle reduces in thickness the others may increase to stabilise the compartment, i.e., to
704 'fill the space'. Nonetheless, the mechanisms underlying variations in gear and shape changes,
705 and potential moderators, require further investigation.
706

707 **(2) Determinants of muscle gearing: computational modelling shows that muscle gearing**
708 **is an emergent property of pennate muscles**

709 The evidence reviewed above supports that variable gearing is a multifactorial
710 phenomenon that appears to result from the complex non-linear interactions of (i) pennation
711 angle, (ii) contractile element vectorial forces, (iii) connective tissue architectural and material
712 properties, and (iv) internal and external pressures. Given this complexity, we introduce a
713 theoretical framework that allows these effects to be considered in synergy. Such an approach
714 allows interrogation of knowledge gaps that have not yet been experimentally mapped, and to
715 address more general mechanistic questions about gearing.

716 To understand the fundamental mechanisms underlying muscle gearing, it is ideal to
717 quantify the independent influence of each factor reviewed herein. Alternatively, one could
718 remove some or all structural or biomechanical features to determine whether gearing still
719 occurs. These approaches are challenging to implement experimentally; however,
720 computational (*in silico*) modelling offers a framework for understanding such complexities
721 and examining the specific factors underlying muscle gearing. It enables 'what-if' experiments
722 (DiSalvo & Blemker, 2024), allowing specific factors to be manipulated independently or

instead explored using a simplified base model to reduce computational time. This approach also facilitates testing unresolved questions, such as whether dynamic changes in muscle shape and gearing are influenced by force, velocity, activation, or a combination of these factors. Unlike animal studies, which typically involve supra-maximal muscle activation, computational models allow exploration of a range of activation conditions that reflect *in vivo* contractions.

Therefore, to probe deeper into the mechanisms underlying muscle gearing and changes in muscle shape during contraction, we ran a series of dynamic (rather than quasi-static) simulations using a 3D finite element muscle model (Wakeling *et al.*, 2020; Ryan *et al.*, 2020a; Almonacid *et al.*, 2024b, 2024a). Unlike previous quasi-static models, this model explicitly accounts for the internal effects of muscle mass and fibre shortening velocity. The model geometry was styled from the average (young) rat medial gastrocnemius muscle reported by Holt *et al.* (2016), resulting in a block of muscle tissue of 18.6 × 6.58 × 6.95 mm with an initial pennation angle of 26.1° and a mass of 0.9 g. For each simulation we stretched the block to 110% of its initial (resting) length then ramped the activation to a predetermined level and finally shortened the block in an isovelocity manner while it was still active. When the activated block shortened back to its initial length (L_0), the mean muscle velocity, mean fibre velocity, fibre pennation angle, and the muscle block thickness and width through its midsection were measured. The gear ratio was computed at this timepoint, and the force at the end of the muscle was determined in the line-of-action of the muscle block. Figure 7 displays the simulation time-series and the muscle block and fibre-level visualisations at key events of the protocol.

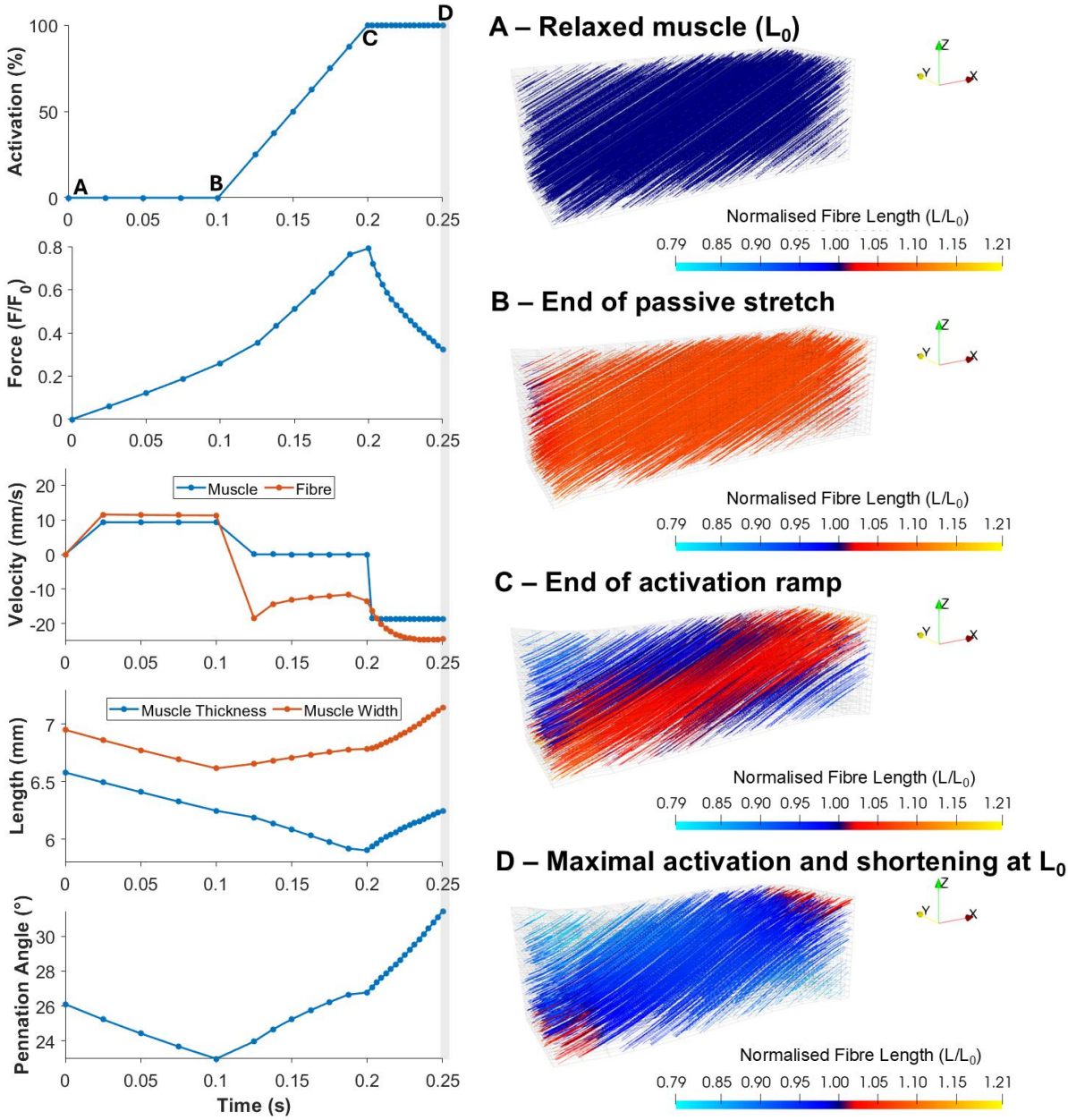

Figure 7. Dynamic simulation of muscle gearing during active shortening. **Left panel:** Time-series plot showing the sequence of events from resting (A) to, passive stretch (B), activation ramp whilst isometric (C), and active shortening at constant activation (D) for activation (% of maximum), muscle force (normalised to optimal length), fibre and muscle velocities, muscle shape (muscle thickness and width at the midsection), and fibres pennation angle. The grey region denotes the time in which variables were measured for analyses reported below. The muscle block was first passively stretched to 110% of its initial length (L_0), followed by a ramped activation phase and then actively shortened back to L_0 under constant velocity. **Right panel:** Visualisations of the muscle block shape and fibres displacement distribution (coloured-coded) at the four time points illustrating both fibre and muscle shape changes during the key events. Arrows in each panel indicate the orientation of the coordinate system (x: anterior-posterior/ muscle block length; y: medial-lateral/ muscle block width; z: superior-inferior/ muscle block thickness).

759 We formulated the muscle model in the same manner as previously described
760 (Wakeling *et al.*, 2020; Almonacid *et al.*, 2024b), as a fibre-reinforced composite biomaterial.
761 The one-dimensional fibres encoded the contractile elements of the muscle and had active and
762 passive force–length and force–velocity properties typical of a Hill-type muscle model (with a
763 maximum unloaded strain rate of 5 s^{-1}, and a maximum isometric stress of 200 kPa). These
764 fibres were encased in a three-dimensional volume representing the tissue properties of the
765 muscle. Tissue properties included the energetic cost of the muscle changing shape, small
766 volume changes (<1%), and the kinetic energy of the tissue. Simulations were run with a time
767 interval between steps such that 16 time-steps of simulation were computed during active
768 shortening (i.e., after the muscle reached 110% of its initial length and the predetermined
769 activation level). At each time step, the total strain energy of the system was minimised (i.e.
770 the energetically optimal solution was found for that time).

771 When the model was run, the fibres shortened and also rotated to increase in pennation
772 as the muscle block shortened (Fig. 7). The rate of fibre rotation increased with both muscle
773 shortening velocity and increased activation (Fig. 8). The fibre shortening velocity increased
774 with muscle velocity but decreased at higher activations (Fig. 8). The muscle gear (mean
775 muscle block velocity/mean fibre velocity) decreased with muscle force (Figs. 9 and 10),
776 increased with muscle velocity (Figs. 9 and 10), and increased with activation (Fig. 9). There
777 was a velocity-dependence to muscle gearing that was independent of force (Fig. 9). This effect
778 is consistent with previous reports on human muscle (Wakeling *et al.*, 2011), and emerged
779 because the simulations tested a range of activation levels that have not been previously
780 possible in *in-situ* or *in-vitro* animal preparations.

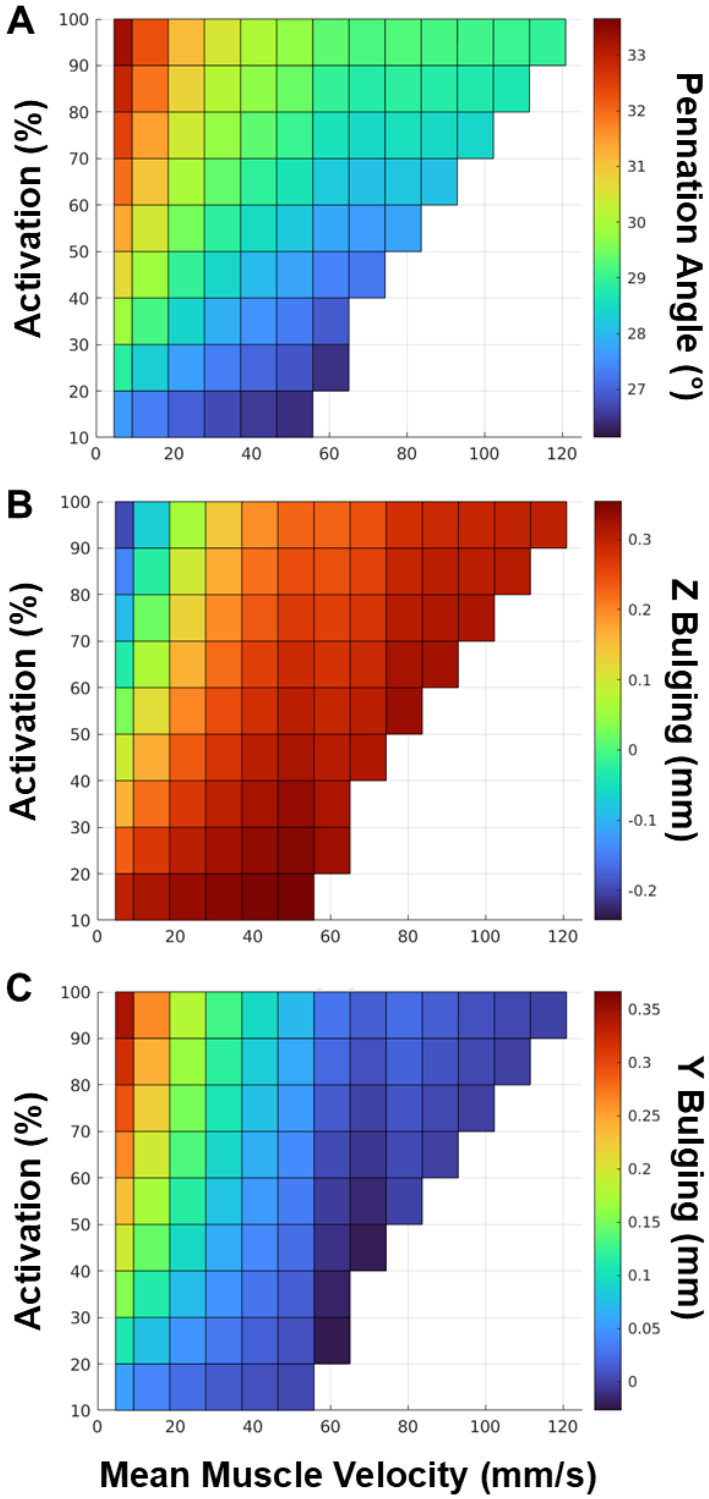

Figure 8. Activation- and velocity-dependent changes in muscle pennation angle and transverse shape during active shortening. **(A)** Pennation angle increases with greater activations and lower shortening velocity. **(B)** Muscle bulging along the thickness direction (Z-axis, orthogonal to the line of action) decreases with activation, suggesting compression at high activation levels and expansion at low activation levels, with a small influence of velocity. **(C)** Muscle bulging along the width direction (Y-axis, orthogonal to thickness) increases with both activation and shortening velocity. The schematic below/above illustrates the modelled muscle block before and after shortening, depicting both architectural and dimensional changes corresponding to panels A-C.

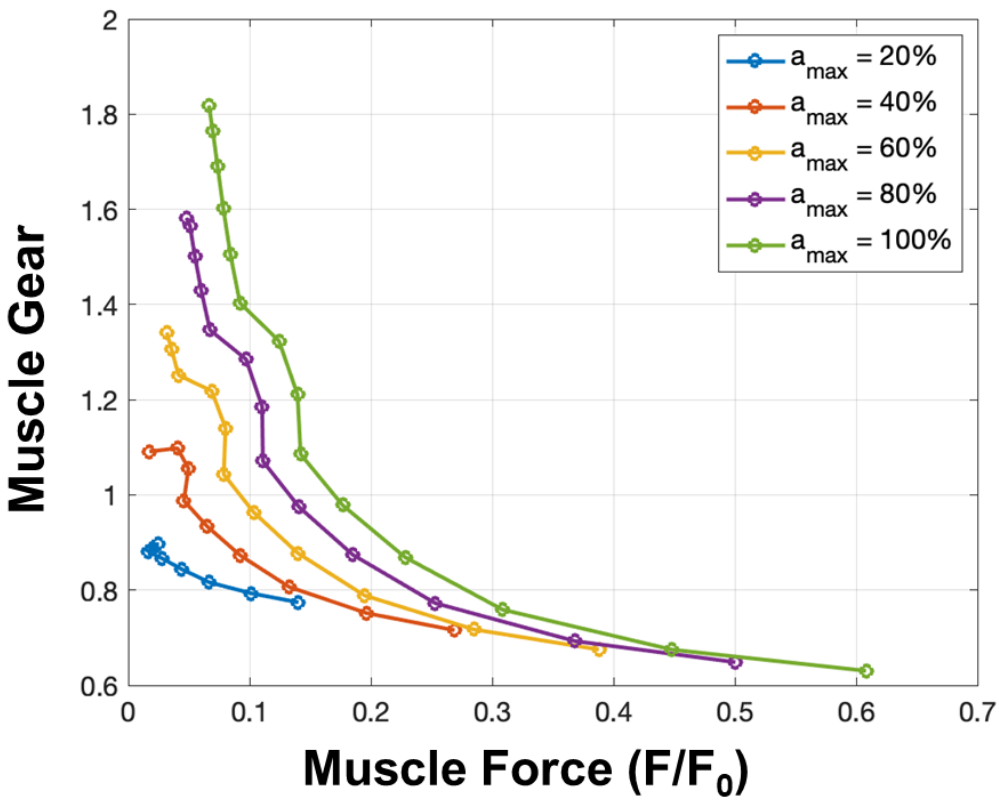

Figure 9. Effect of activation level on the muscle gear–force relationship. Muscle gear ratio decreases non-linearly with increasing normalised muscle force (F/F_0), with higher levels of maximal activation (a_{max}) enabling higher gear at low force outputs. This dynamic interaction between activation and the mechanical gearing of muscle fibres during force generation suggests that muscle gear is activation-dependent.

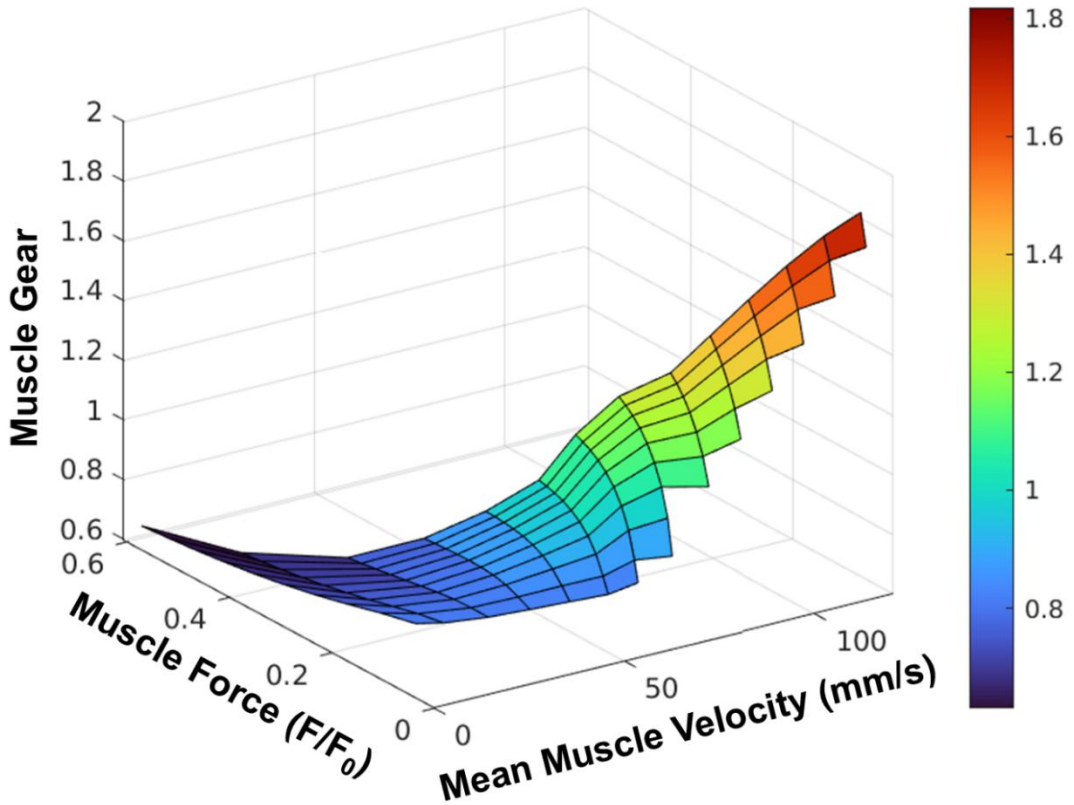

Figure 10. Relationship between muscle gear, muscle force, and mean muscle velocity during active muscle shortening. Muscle gear ratio decreases with increasing force but increases with mean fibre velocity, largely independent of force level. Gear values are particularly high during high shortening velocities, when gearing would be beneficial to limit fascicle length changes (i.e., both velocity and length operations) during rapid contractions.

As the muscle block shortened, it bulged in its midsection in an attempt to conserve its volume. At low velocity and high activation, the thickness of the muscle (in the fibre plane) decreased a small amount, but at higher velocities the thickness increased (Fig. 7). The width of the muscle block was maintained or increased in all conditions (Figure 7). Thus, the highest belly gearing occurred with a large increase in thickness but minimal change in width, whereas the lowest gearing coincided with a decrease in thickness by an increase in width. These findings are consistent with the model proposed by Azizi *et al.* (2008).

Of note from these simulations is that all the general features of muscle gearing emerge from the physics of the contractile elements and the muscle tissue properties alone, and do not

813 need effects from external tissues to occur. Importantly, additional effects from tissues such as
814 aponeurosis (Rahemi *et al.*, 2014; Rahemi, Nigam & Wakeling, 2015; Wakeling *et al.*, 2020),
815 ECM (Konno, Nigam & Wakeling, 2021a) and intramuscular fat (Rahemi *et al.*, 2015) can be
816 incorporated into this modelling framework but were not necessary to address the gearing
817 mechanisms described herein. Further, the simulations predict similar gearing effects that have
818 been reported for maximally activated muscles in *in situ* animal studies but also now predict
819 the more nuanced effects that occur at submaximal activations, as is the case during human
820 movement. This simulation framework shows how force, velocity, and activation
821 independently affect muscle gear. The exact state of the tissue when the measures were probed
822 depends on the energy balance at each condition, which in turn depends on the complex
823 interaction between tissue stiffness, compressibility, and mass as well as the activation state
824 and fibre contractile properties.
825

826 **IV. Variable gearing in human muscle preparations *in vivo*: what have we learnt so far?**

827 In the preceding sections, we presented evidence for the existence of muscle gearing
828 and its underlying mechanisms predominantly from animal studies and computational models.
829 A common question in the biological sciences is whether the physiological and mechanical
830 behaviours observed in animal studies can be extrapolated to living animals (including humans)
831 operating with muscles functioning *in vivo* under normal physiological conditions. This section
832 synthesises current *in vivo* research on muscle gearing and highlights potential commonalities
833 and discrepancies between animal and human studies.

834 A summary of studies investigating muscle gearing is presented in Table 2 and includes
835 *in situ*, *in vitro, ex vivo*, and *in vivo* investigations across both non-mammalian and mammalian
836 pennate muscles. Reported gear values, operation ranges, and responses to mechanical
837 demands vary across experimental models. In isolated muscle preparations from animals, gear

838 ratios typically range between ~1.0 and 2.0 during singular concentric contractions and from
839 1.5 and 4.5 during singular eccentric contractions, i.e., when concentric and eccentric
840 contractions are not performed consecutively like in a stretch-shorten cycle. In contrast, *in vivo*
841 human studies suggest gear ratios between 1.0 to 1.5 for concentric contractions and
842 approximately 1.0 to 3.1 for eccentric contractions.

843 Both animal and human studies have reported torque- or force-dependent shifts in gear
844 ratios, although only studies in humans have reported gear to vary with velocity. Furthermore,
845 differences in variable gearing exist, with most *in vivo* human studies reporting gear ratio to
846 increase with force or torque (or to increase with velocity) (Son & Rymer, 2020; Kelp *et al.*,
847 2023; Randhawa *et al.*, 2013; Coenning *et al.*, 2024) whereas animal studies report gear to
848 decrease with force increase. To our knowledge, no studies on animals muscles have
849 demonstrated an increase in gear ratios with force, and only two studies in humans have
850 reported a gear-force (or torque) relationship similar to that of animal experiments (Dick &
851 Wakeling, 2017; Monte *et al.*, 2021).

852 The reasons for these discrepancies remain unclear and may arise from variations in
853 experimental approaches—*in situ*, *in vitro*, or *ex vivo* versus *in vivo* human studies—or they
854 may reflect fundamental differences in muscle function between human and non-mammalian
855 animals.

Table 2. Summary of studies reporting muscle gearing during concentric, isometric, and eccentric contractions as well as passive lengthening.

References		Population	Muscle	Task	Gear ratio*		Variable gear
Concentric (predominant)							
Brainerd & Azizi (2005)	Animal	Salamander *Siren lacertina*	EO	Steady swimming	>1.0		–
Azizi & Brainerd (2007)	Animal	Salamander *Siren lacertina*	EO IO	Steady swimming	EO IO	1.3-1.6 1.3-2.7	– –
Azizi *et al.* (2008)	Animal	Wild turkey *Meleagris gallopavo*	LG	Isotonic contractions at 10%, 30%, 50%, 70%, 90% of MVIC	1.0-1.4		↓ with ↑F
Wakeling *et al.* (2011)	Human	Y M	MG LG Sol	Cycling at different cadences and crank torque	MG LG Sol	1.4-1.7 1.4-1.6 1.3-1.5	↑ with ↑V ↑ with ↑V ↑ with ↑V
Wakeling *et al.* (2013)	Human	Y M	MG	Cyclic isotonic contractions at different torques	1.05-1.08***		–
Randhawa *et al.* (2013)	Human	Y M	MG LG	Isokinetic and isotonic contractions at different velocities	MG LG	Isok 1.07-1.10 Isot 1.09-1.16 Isok 1.07-1.09 Isot 1.06-1.1	↓ with ↑V ↑ with ↑F & ↑V No V effect No V or F effects
Randhawa & Wakeling (2013)	Human	Y, O M	MG LG	Isokinetic contractions at different velocities	MG LG	Y 1.07-1.13 O 1.06-1.08 Y 1.07-1.08 O 1.05-1.06	↓ with ↑V ↓ with ↑V ↓ with ↑V Unclear
Azizi & Roberts (2014)	Animal	Bullfrog *Rana catesbeiana*	PL	Isotonic contractions at 10%, 30%, 50%, 70%, 90% of maximal MVIC	1.2-2.0		↓ with ↑F
Holt *et al.* (2016)	Animal	Y and O rats *Fischer 344-Brown Norway*	MG	After-loaded isotonic contractions at 10%, 30%, 50%, 70%, 90% of MVIC	Y: O:	0.9-1.5 1.3-1.4	↓ with ↑F = with ↑F

Dick & Wakeling (2017)	Human	Y M, F	MG	Cycling at different cadences and crank torque		1.0-1.5	↓ with ↑F
Eng & Roberts (2018)	Animal	Wild turkey *Meleagris gallopavo*	LG	Supra- and sub-maximal isotonic contractions at 10%, 20%, 60%, 80%, 100% of MVIC		1.1-1.4	↓ with ↑F
Bohm *et al.* (2019)	Human	Y M, F	Sol	4-min running		1.1****	–
Aeles & Vanwanseele (2019)	Human	Y M, F	MG	Maximal effort single-leg forward jump (muscle shortening phase)		1.2-1.3	–
Laird *et al.* (2020)	Animal	Tufted capuchin *Spajus apella*	SAT	Chewing food of different material properties		1.0-2.0	–
Monte *et al.* (2021)	Human	Y M	VL	Explosive dynamic contractions of different accelerations		1.1-1.23	↓ with ↑T
Tijs *et al.* (2021)	Animal	Rat *Sprague-Dawley*	MG	Isotonic contractions at 10%, 30%, 50%, 70%, 90% of MVC	Proximal	1.8-2.1	↓ with ↑F
					Distal	1.3-1.6	↓ with ↑F
Bohm *et al.* (2021)	Human	Y M, F	Sol VL	4-min running	Sol	0.016$******	–
					VL	0.003$******	–
Monte *et al.* (2022)	Human	Y M, F	MG VL	Level walking at incremental speeds	MG	1.0-1.14	↑ with ↑V up to 5km/h
					VL	1.06-1.1	↓ with ↑V for velocities >5
Brusco, Pinto & Blazevich (2023)	Human	Y M, F	BF	Isokinetic contractions at 55% and 100% of MVIC		1.1-1.8	= with ↑T
Laird *et al.* (2023)	Animal	Tufted capuchin *Spajus apella*	SAT SMT SPT	Chewing food of different material properties	SAT	-0.5-1.0	–
					SMT	-1.0-1.54	–
					SPT	-1.5-1.9	–

Mornas *et al.* (2024)	Human	Y M, F	MG	40-min running at10 km/h (fascicle shortening during stance and swing phases)	1.0 - 1.1		–
Coenning *et al.* (2024)	Human	M	MG LG Sol	Contraction on a sledge with external load of 125kg (~30% of body mass) through 40° of ankle rotation	MG LG Sol	1.03-1.26 1.0-1.1 1.08-1.21	↑ with ↑T = with ↑T ↑ with ↑T
Isometric (Fixed-end)							
Son & Rymer (2020)	Human SS	Y-O M	MG	0%, 20%, 40%, 60% and 100% of MVIC at multiple ankle angles	P NP	1.0 1.1-1.2	= with ↑T ↓ with ↑T
Monte & Zignoli (2021)	Human	Y M	MG	Explosive MVIC	1.1		–
Monte (2021)	Human	Y M	MG	Explosive MVIC and short-range stretching	1.0-1.2***		–
Werkhausen *et al.* (2022)	Human	Y M, F	VL	Explosive MVIC	1.0-1.1		–
Monte *et al.* (2023)	Human CP and C	O M, F	VL	Explosive MVIC. Gear analysed at multiple time-windows as torque increases	CP C	1.0-1.1 1.1-1.1	↓ with ↑T ↓ with ↑T
Kelp *et al.* (2023)	Human	Y, O M, F	MG LG	10%, 30%, 50%, and 100% of MVIC	MG LG	Y:1.0-1.1 O:1.0-1.1 Y:1.0-1.1 O:1.0-1.0	↑ with ↑T ↑ with ↑T ↑ with ↑T ↑ with ↑T
Monte & Zamparo (2023)	Human	Y M	MG	Cyclic submaximal ramp contractions	1.03-1.13***		–
	Human	M	MG		MG	1.04-1.34	↑ with ↑T

Coenning *et al.* (2024)			LG Sol	Ramp contraction from resting up to 90% MVIC	LG Sol	1.05-1.1 1.06-1.21	= with ↑T ↑ with ↑T
Eccentric							
Shin *et al.* (2009)	Human	Y M	MG	Cyclic (hydraulic induced) isotonic contractions at 40% MVC		1.0-1.2**	–
Azizi & Roberts (2014)	Animal	Bullfrog *Rana catesbeiana*	PL	Isotonic contractions at 110%, 130%, 150%, 170%, 190% of MVIC		1.8-4.3	↑
Guilhem *et al.* (2016)	Human	Y M, F	MG	Maximal isokinetic		~1.1	–
Hollville *et al.* (2019)	Human	Y M	MG VL	Drop landing	MG VL	1.1-1.2 1.0-1.1	–
Aeles & Vanwanseele (2019)		Y M,F	MG	Maximal effort single-leg forward jump (muscle lengthening phase)		0.8-1.2	–
Pinto *et al.* (2023)	Human	Y M	MG	Maximal isokinetic at slow velocity		1.2–3.1	↑ with ↑T
Brusco *et al.* (2023)	Human	Y M, F	BF	Isokinetic contractions at 55% and 100% of MVIC		0.9-1.1	= with ↑T
Pinto, Nosaka & Blazevich (2024)	Human	Y M	MG	Isokinetic contractions at 40% and 60% of MVEC at slow velocity		1.2 - 1.7	↑ with ↑T
Mornas *et al.* (2024)	Human	Y M, F	MG	40-min running at10 km/h (fascicle lengthening during swing phases)		1.05	–
Passive							
Shin *et al.* (2009)	Human	Y M	MG	Cyclic (hydraulic induced) isotonic contractions		1.0–1.3**	–

Pinto *et al.* (2023)	Human	Y M	MG	Isokinetic passive rotations	1.1-1.2	–
Takahashi *et al.* (2023)	Human	Y M, F	MG	Ankle rotated and measured stationary at multiple angles	1.4	–

*For simplicity, we ignore whether (i) the study reported the change in muscle-to-fascicle length or velocity, (ii) the change was measured in a muscle segment or the whole muscle, (iii) the study assumed linear vs. curved fascicles, fascicles were measured manually or using semi-automated tracking, and (iv) the mean joint excursion or muscle length was used to calculate gear, despite these variables being shown to affect gear (Pinto *et al.*, 2023). Additionally, studies reporting a muscle gear across different contraction intensities or joint ranges of motion are reported as a range of values unless otherwise indicated. Studies reporting a mean gear during a particular task or at a single contraction intensity are reported as a single mean gear value, unless otherwise indicated.

**Gear range represents different measurements within the muscle (proximal, middle, distal belly)

***Gear range represents measurements of muscle gear obtained during different conditions. In this case, the conditions were varying external loads on the muscle.

****There is an eccentric phase during the stance phase of running but values were not reported for both phases; thus, it was considered to be a predominantly concentric task.

$The velocity decoupling coefficient is an adaptation of the belly gear ratio used during functional tasks in which fascicle velocities may be close to zero (isometric behaviour).

Additional notes: Average gear is presented when reported in the manuscript's text but values were estimated using an open-source graph digitiser (WebPlotDigitiser, https://apps.automeris.io/wpd/) and data expressed as mean, median, or minimum and maximum values, according to the metric displayed in the figure (they are therefore an approximation) when gear was only presented in figures. Muscle gear is reported for baseline (pre-intervention) tests for studies that included an experimental intervention (e.g., muscle fatigue, aponeurosis incision).

Abbreviations:
Y = young adults; O = older adults; M = male; F = female; CP = clinical population; C = control, SS = chronic hemispheric stroke survivors; P = paretic side; NP = non-paretic side;
BF = biceps femoris; EO = external oblique; IO = internal oblique; MG = medial gastrocnemius; PL = plantaris; SAT = superficial anterior temporalis; SMT = superficial medialis temporalis; SPT = superficial posterior temporalis; VL = vastus lateralis.
MVIC = maximal voluntary isometric contraction; MVEC = maximal voluntary eccentric contraction; Isok = Isokinetic; Isot = Isotonic.

857 **(1) Differences in testing paradigms**

858 Experimental approaches for testing muscle gearing differ substantially between animal
859 and human studies, resulting in substantial variations in the mechanical conditions tested.
860 Methodological discrepancies may influence observed outcomes and interpretations of how
861 force and/or velocity modulate muscle gearing.

862 In animal studies, muscle gearing is examined under highly controlled laboratory
863 conditions using servomotor-driven experiments that enable force and length measurements.
864 The muscle-tendon unit is surgically exposed and freed from surrounding muscles, with one
865 tendon and its attached bone fixed to a rigid apparatus while the other tendon is calcified and
866 clamped to a servomotor lever arm (Azizi & Roberts, 2014; Azizi *et al.*, 2008). Muscle fibre
867 lengths and velocities are estimated using sonomicrometry (surgically implanted piezoelectric
868 crystals) while whole-muscle length and velocities are measured via the servomotor. Electrical
869 stimulation via implanted cuff electrodes to branching nerves induces tetanic contractions
870 against predefined forces or muscle lengths.

871 A common protocol used for gearing assessment begins with an isometric ramp
872 contraction, during which force is gradually increased to a predefined percentage of the
873 maximal isometric force. This is followed by an isotonic phase, where muscle force is held
874 constant as the muscle (and its fascicles) shortens or lengthens at approximately constant
875 velocities. Gear ratio is measured during this constant-force phase to minimise the influence of
876 series elastic element compliance (Azizi *et al.*, 2008), as tendon elasticity affects muscle length
877 changes (discussed below). A key advantage in these experiments is the ability to control initial
878 fibre length and pennation angle, which are kept the same at the onset of contractions across
879 force levels, including during the initial plateau of the isometric phase (see Figure 3 in Azizi *et*
880 *al.*, 2008). Furthermore, gear ratios are determined during the constant-force phase occurring
881 at or near the plateau of the force−length relationship at the muscle's optimal fascicle (L_0)

during all force conditions (Azizi *et al.*, 2008; Holt *et al.*, 2016). These controlled testing conditions ensure that gearing is not confounded by differences in fibre strain, force−length properties, or initial pennation angle (Brainerd & Azizi, 2005), while also minimising variations in fibres or fascicle radial expansion due to passive elastic properties. This effectively isolates muscle shape changes as the primary driver of gearing and enables comparisons between muscles to determine whether differences in gear arises from variations in muscle shape changes or in initial pennation angles.

In contrast, human *in vivo* studies rely on non-invasive imaging techniques, such as ultrasound, to estimate fascicle length and muscle-tendon junction displacement (Roberts & Dick, 2023). When ultrasonography is combined with motion analysis systems, accurate estimations of whole-muscle length changes are possible. While conceptually similar to the animal preparation experiments that use sonomicrometry and servomotors, human experiments lack the precise control over muscle forces, velocities, and activation timing. Such limitations make direct comparisons with *in vitro* and *in situ* animal studies challenging.

A variety of experimental paradigms have been employed in human studies (see Table 2), including isometric, isotonic, and isokinetic contractions, as well as more complex tasks involving time-varying force and velocity (Monte *et al.*, 2022). Muscle gearing has been investigated during isometric contractions under both time-varying (explosive) (Monte *et al.*, 2023; Monte & Zignoli, 2021) and approximately constant force or torque conditions (Kelp *et al.*, 2023; Monte, 2021). Dynamic contractions have been tested, such as isotonic (Randhawa & Wakeling, 2013; Randhawa *et al.*, 2013), explosive (Monte *et al.*, 2021), and isokinetic (Randhawa & Wakeling, 2013; Randhawa *et al.*, 2013; Pinto *et al.*, 2024; Pinto *et al.*, 2023) contractions. In these studies, external loads, cycle frequencies, and set velocities have been systematically manipulated to assess their effects on muscle gearing (Randhawa & Wakeling, 2013; Randhawa *et al.*, 2013). Some studies have used cycling-based paradigms, where bicycle

907 crank torque and velocity can be manipulated via bicycle gear ratios and cadences to partly
908 decoupling of force- and velocity-effects on muscle gearing (Wakeling *et al.*, 2011; Dick &
909 Wakeling, 2017). However, unlike in constant-force paradigms in animal models, many of
910 these human studies determine gear ratios during force-changing conditions and estimating
911 muscle segment velocities (Wakeling *et al.*, 2011; Monte *et al.*, 2021), which can alter
912 interpretation of findings. Other methodological differences also exist. For example, gear ratios
913 have been calculated during transient (Dick & Wakeling, 2017) or time-averaged periods (Pinto
914 *et al.*, 2024; Pinto *et al.*, 2023; Monte *et al.*, 2023; Monte *et al.*, 2022). Gear ratios have been
915 assessed at specific time points during the contraction cycle (Monte *et al.*, 2022), at different
916 parts of the contractile cycle during peak muscle and fibres velocities (Randhawa & Wakeling,
917 2013; Randhawa *et al.*, 2013; Dick & Wakeling, 2017), or at the muscle's optimal fascicle
918 length (Monte *et al.*, 2021). While findings mostly show variability in gearing, results suggest
919 that gear ratios depend on force or velocity, with force-dependent gearing often—though not
920 always—similar to that observed in animal studies where gear decreases with force (see Table
921 2).

922 The significant methodological differences and testing paradigms between animal and
923 human complicate direct comparisons of how force and velocity affect muscle gearing. The
924 precise control over force, velocity, and starting fascicle angles and lengths in isolated animal
925 muscle experiments is difficult to replicate *in vivo*. However, studies examining rat medial
926 gastrocnemius shape changes *in vivo* under time-varying force conditions, such as during gait
927 and slope conditions like decline walking or incline galloping, have shown force-dependent
928 shape changes similar to those observed in animal models (Konow, Collias & Biewener, 2020).
929 This raises the question of whether observed differences in muscle gearing primarily stem from
930 experimental design and muscle preparation or reflect inherent functional differences between
931 humans and non-mammalian animals

The muscle model presented herein addresses some of these challenges by incorporating testing paradigm similar to those used in animal studies while also simulating activation levels comparable to human experiments. The model demonstrates that gearing is independently affected by force, activation, and velocity, with high gear ratios occurring at high velocities independent of muscle force. Of note, this model does not include an in-series tendon, meaning that tendon compliance effects could not be a contributing factor to observed gearing behaviours in the simulation. Thus, differences in testing paradigms should be carefully considered when interpreting muscle gearing results and extrapolating findings from animal models to human movement. Nonetheless, potential intrinsic species differences should not be overlooked (see below).

(2) Factors within the muscle-tendon unit (MTU)

In animal experiments, muscle gearing is measured during conditions where force is constant and the influence of the tendon is minimised (Azizi & Roberts, 2014; Holt *et al.*, 2016; Eng & Roberts, 2018; Azizi *et al.*, 2008). To achieve this, the in-series tendon is either calcified (Azizi *et al.*, 2008) or otherwise made short and stiff by clamping it close to the muscle's end (Tijs *et al.*, 2021; Holt *et al.*, 2016). These methodological constraints, combined with the constant-force condition, limit the contribution of the in-series tendon to force-dependent muscle length changes. As a result, muscle shape changes and gearing occur with minimal interference from tendon elasticity. However, it is nearly impossible to remove the influence of tendon compliance in human muscles with long tendon, such as the gastrocnemii. Tendon elasticity significantly affects muscle force-length and force−velocity relationships as well as overall mechanics of the muscle-tendon unit. During contraction, muscles fibres shorten and increase in pennation, while the in in-series tendon stretches to accommodate these changes. This tendon compliance influences initial pennation angles, which can in turn influence gearing

957 (Brainerd & Azizi, 2005). Unlike in animal experiments, human studies cannot precisely
958 control for these effects. Consequently, tendon elasticity may cause muscle gearing to increase
959 rather than decrease with force, leading to different outcomes than those observed in isolated
960 muscle preparations.

961 The mechanical properties of a muscle's ECM may impact shape changes during
962 contraction and therefore gearing (Eng *et al.*, 2018; Roberts *et al.*, 2019). Whilst our
963 model show variable gearing despite absence of an aponeurosis or specific connective
964 tissues around the muscle, ECM components may still contribute to muscle shape changes
965 and gearing. Muscle fibres and fascicles are encapsuled by endomysial and perimysial
966 connective sheets, and research has shown that their quantity and morphology vary among
967 vertebrates (Sleboda *et al.*, 2020) and even among muscles within the same species
968 (Purslow, 2020). Given that ECM properties appear to influence shape changes at the
969 fibre and fascicle levels, one might expect gearing differences to exist between
970 functionally similar muscles across species, as well as between different muscles within
971 the same species. However, there is limited evidence to determine whether, or the extent
972 to which, between-species ECM structural and mechanical property variations can
973 explain differences in gearing between animals and human muscles. This is an area that
974 would benefit from computational modelling studies where the effect of tissue
975 manipulations can be addressed directly.

976 Spatial variations in motor unit activations also occur during tasks with different force
977 or velocity characteristics (Wakeling, 2009) and influence muscle metabolic cost (Lai,
978 Biewener & Wakeling, 2018) and muscle force (Wakeling *et al.*, 2012) possibly through
979 alterations in fibre behaviour and gearing. Rahemi *et al.* (2014) developed a 3-D finite element
980 model based on human lateral gastrocnemius to explore the effect of submaximal isometric
981 contractions with uniform and non-uniform (regionalised) activation on fibre and muscle

982 behaviours. All models had 10% of the fibres activated. Regionalised activation also caused
983 greater regionalisation of thickness changes, and thus larger fascicle strains and rotations.
984 These findings suggest that regionalised activation can influence shape changes and thus
985 variable gearing (Rahemi *et al.*, 2014). Differences in spatial fibre activation should affect
986 muscle shape, particularly during submaximal activation, as regions of inactive fibres do not
987 contribute to force production but still add mass, increasing non-active tissue inertia (Holt,
988 Wakeling & Biewener, 2014; Ross *et al.*, 2020) and potentially limiting radial bulging of active
989 fibres and fascicles. These may in turn reduce contraction efficiency (Ross & Wakeling, 2021;
990 Ross *et al.*, 2020) through its impact on gearing.

991 Spatial activation variation is an inherent physiological phenomenon also reported in
992 animal muscles *in vivo* (Higham & Biewener, 2011; Higham, Biewener & Wakeling, 2008).
993 However, studies examining variable gearing in isolated muscle preparations are conducted
994 under supra-maximal activation conditions in which the whole muscle is assumed to be
995 uniformly activated (Eng & Roberts, 2018). An exception to this was shown in the isolated rat
996 medial gastrocnemius (Tijs *et al.*, 2021) in which lower gear ratios were observed during
997 contractions triggered by submaximal rather than maximal stimulation intensities, although it
998 did not reduce fibre shortening velocity or alter the gear−force relationship (Tijs *et al.*, 2021).
999 It remains to be determined whether these findings are applicable to muscles functioning *in*
1000 *vivo* due to differences between asynchronous voluntary (as in *in vivo*) vs. synchronous
1001 electrically-evoked motor unit stimulation patterns.

1002 As with some animal muscles, regional muscle architectural variation is also observed
1003 in human muscles (Takahashi *et al.*, 2022; Shin *et al.*, 2009) and has the potential to affect
1004 gearing (Tijs *et al.*, 2021; Pinto *et al.*, 2023; Azizi & Deslauriers, 2014; Takahashi *et al.*, 2023;
1005 Shin *et al.*, 2009). Regional pennation variation can result in asymmetric transverse muscle
1006 belly strains, regional fascicle rotations, fascicle velocities, and muscle segment length

1007 changes (Blazevich, Gill & Zhou, 2006). Most gearing studies are conducted in small animals
1008 possessing relatively small muscles and relatively homogenous architecture (Azizi & Roberts,
1009 2014; Eng & Roberts, 2018), although this is not always the case (Tijs *et al.*, 2021; Holt *et al.*,
1010 2016). Recently, Tijs *et al.* (2021) demonstrated region-specific gear ratios in the rat medial
1011 gastrocnemius, with lower gear in muscle compartments exhibiting lower pennation regardless
1012 of activation status. However, gear changes with contractile force were similar to those reported
1013 in previous small animal data sets.

1014 Finally, intramuscular adipose tissue (fat) may be a potential factor influencing variable
1015 gearing (Rahemi *et al.*, 2015; Kelp *et al.*, 2023). Fat accumulates within skeletal muscle tissue
1016 (e.g. with ageing and disuse) and can be stored around myofibrils (intramyocellular fat or lipids
1017 droplets) and surrounding muscle fibres and fascicles (extramyocellular fat) (Altajar & Baffy,
1018 2020). Muscle must use internal work to deform the fatty inclusions, and this affects the way
1019 in which the contractile tissue deforms (Wakeling et al. 2020). Fat may increase the stiffness
1020 of the muscle and this increase would resist fibre shortening and transverse bulging of fibres
1021 and the muscle belly, ultimately reducing fascicle rotation and gear (Rahemi *et al.*, 2015; Kelp
1022 *et al.*, 2023). Determining fat quantities in animal studies would help pinpointing whether fat
1023 could be a contributing factor to the gearing differences previously observed between humans
1024 and other, non-mammalian animals.

1025

1026 **(3) Factors residing outside the muscle**

1027 In many vertebrates, joint movement is produced by the activation and subsequent
1028 length change of multiple muscles tightly packed in muscle groups (compartments). Unlike
1029 studies using animal preparations that typically isolate muscles from others within their group
1030 to measure gearing, human muscles normally operate within their synergistic group *in vivo*
1031 (Reinhardt *et al.*, 2016; Bernabei *et al.*, 2017). Thus, gearing can be influenced by external

factors constraining radial expansion during muscle shortening, including compressive loads exerted onto a muscle by bones, inter-muscular fat, or neighbouring muscles within the group.

Several factors can influence lateral muscle pressures and deformations in synergistic muscle groups *in vivo*. First, synergistic muscles may lack common neural drive (Hug *et al.*, 2021), resulting in varying activation levels among muscles of a group. Second, synergistic muscles may possess different shapes and architectures (Aeles *et al.*, 2022; Blazevich *et al.*, 2006) and deform asymmetrically as they shorten and expand radially. Asymmetric radial expansion in a muscle within the group may promote lateral pressures differences along adjacent muscles (Bernabei *et al.*, 2017). Third, individual muscles within the group may attach proximally and distally through distinct tendons or (intermuscular) aponeuroses that differ in their mechanical properties (Bernabei *et al.*, 2017; Ando *et al.*, 2018), so muscle shortening (or lengthening) during contraction between muscles may differ even if they exert similar forces. Fourth, the presence of non-contractile materials such as inter-muscular fat and connective tissues may influence muscle deformations. And finally, differences in architectural features imply distinct muscle force contributions to the total synergistic force or joint torque (Wakeling *et al.*, 2011; Higham & Biewener, 2011). While these factors may be inter-related, their independent or combined effects may impact muscle length change and radial pressures even when they are subjected to the same external loads, and their effects on fibre rotation and gearing might not be easily or accurately estimated using current muscle models.

The first evidence demonstrating an effect of external force constraining radial bulging of human muscle and fascicle behaviours *in vivo* came from studies in which external compression was applied using elastic bandage. Wakeling *et al.* (2013) found reductions in belly gear, fascicle angle, and muscle thickness changes during contraction without significant effect on concentric joint torque production. Under these conditions, belly gear reductions imply an increased fascicle shortening velocity that would decrease fascicle force-generating

potential. Other studies have since imposed transverse pressures using external loads or compressive garments to compress the muscle and observed a significant impact on muscle geometry (Ryan *et al.*, 2019; Stutzig *et al.*, 2019; Monte, 2021), force production (Ryan *et al.*, 2019; Stutzig *et al.*, 2019; Siebert *et al.*, 2018), and energetic cost (Monte & Zamparo, 2023), with both empirical data from animal studies and finite element modelling indicating that compression-related force reduction is exacerbated when a muscle is at shorter length (Sleboda & Roberts, 2020) or has lower pennation (Ryan *et al.*, 2020a). These effects appear to be direction dependent, with loads placed in the in the thickness direction increasing longitudinal muscle forces at muscle lengths shorter than resting and decreasing for shorter lengths, whereas loads compressing in the width direction promote reductions in longitudinal forces without length-dependency effects (Ryan *et al.*, 2020a). When muscle is compressed from all directions and at shorter muscle lengths, no changes in force or fascicle behaviour are detected (Ryan *et al.*, 2020b)

Evidence indicating that architectural differences between synergistic muscles can contribute to relative lateral pressure variations has been partially presented. Randhawa *et al.* (2013) showed that MG reduced in thickness during both isovelocity and isotonic concentric plantar flexion whereas the LG increased thickness. These observations were subsequently replicated in other studies (Randhawa & Wakeling, 2018; Wakeling *et al.*, 2011; Kelp *et al.*, 2021; Kelp *et al.*, 2023) and demonstrated that between-muscle synergism occurs such that as one muscle reduces in thickness the other may increase in response to the pressure reduction in that direction (to 'fill the space'). This interaction, in which adjacent muscles exert pressure and perform work on each other, likely influences muscle shape changes. Such effects are not accounted for in animal experiments and may contribute to differences in muscle gearing between human and animal preparations.

V. Other functional benefits of fibre arrangement and muscle shape changes during contraction

Changes in muscle shape during contraction may confer additional benefits beyond their effects on fibre rotation and muscle gearing. Because muscles operate within tightly packed anatomical compartments, transverse expansion during contraction can lead to compression of adjacent structures and reduce mechanical efficiency (Wakeling *et al.*, 2020). Muscle architecture and shape changes may be finely tuned not only for mechanical efficiency but also for spatial accommodation. Features such as fibre orientation, muscle belly shape and curvature, and the direction of force transmission can influence how a muscle deforms during contraction, enabling it to avoid impinging surroundings. Dynamic changes in muscle shape offer an elegant anatomical solution for managing spatial constraints while preserving the functional and anatomical integrity of both the muscle and surrounding tissues.

One example of this comes from the adductor mandibulae of *Creatochanes*, a teleost fish with big eyes (Alexander, 1964, 1983). In this species, the main jaw-closing muscle exhibits a crescent-like, concave shape that partially wraps around the posterior and ventral surfaces of the eyeball (Alexander, 1964). If this muscle had a parallel-fibred architecture, contraction would cause it to bulge radially and compress the adjacent orbital tissue. If the muscle had a pennate configuration, it could be designed in a way that contraction would cause muscle expansion predominantly in the opposite direction of the eye, avoiding compression of ocular structures. In reality, the adductor mandibulae muscles exhibit a pennate architecture with fibres inserting obliquely onto a central subocular tendon lying along the inner concave edge of the muscle, adjacent to the eye. Thus, the contracting fibres pull the tendon away from the eyeball without requiring the muscle belly to expand toward the orbit (Alexander, 1983; Datovo & Vari, 2013). In effect, this muscle architecture offers both a mechanical and spatial

solution to optimise force production while protecting sensitive surrounding structures from compression.

A comparable example in humans involves the MG and LG, which act synergistically during plantarflexion. These muscles differ in shape, fibre orientation, and deformation patterns (Randhawa & Wakeling, 2018; Wakeling *et al.*, 2011; Kelp *et al.*, 2021; Kelp *et al.*, 2023). While the MG tends to reduce in thickness during contraction, the LG increases in thickness. This reciprocal deformation pattern may represent a spatially efficient and energetically favourable strategy in which the muscles coordinate shape changes to avoid competing for space and generating unnecessary internal work to deform each other, thus maximising net external output. Overall, these examples support the broader hypothesis that muscle architecture, and its associated changes in shape during contraction, has evolved to optimise both mechanical performance and anatomical compatibility within tightly constrained anatomical environments. Understanding these interactions could inform the design of bioinspired actuators or therapeutic strategies that consider both force output and spatial constraints. Nonetheless, further research is warranted to fully understand the trade-offs involved.

VI. Clinical and practical implications

There are several benefits to improving our understanding of muscle gearing within the clinical context, including potentially gaining insights into muscle functional loss with ageing, neurological disorders, and physical training and detraining.

Ageing is typically accompanied by (i) reductions in muscle strength and power (Freitas *et al.*, 2024), (ii) a reduction in resting pennation angle (Morse *et al.*, 2005; Narici *et al.*, 2003; Tomlinson *et al.*, 2014; Randhawa & Wakeling, 2013), (iii) increased longitudinal and transverse ECM stiffness resulting from alterations in ECM geometry and increased cross-

1130 linking (Wood *et al.*, 2014; Kragstrup, Kjaer & Mackey, 2011), (iv) increased intra- and inter-
1131 muscular fat content (Addison *et al.*, 2014; Kelp *et al.*, 2023), (v) reduced intramuscular fluid
1132 with reductions in intracellular and maintenance of extracellular water (Yamada *et al.*, 2010;
1133 Iwasaka *et al.*, 2023), and (vi) irregular myofiber shape (Soendenbroe *et al.*, 2023). Alterations
1134 in one or more of these structural factors have the potential to influence gearing. Growing
1135 evidence suggests that muscle gearing is influenced by biological age (Holt *et al.*, 2016;
1136 Randhawa & Wakeling, 2013; Kelp *et al.*, 2023), or at least the increase in inactivity that comes
1137 with ageing. In both rats (Holt *et al.*, 2016) and humans (Randhawa & Wakeling, 2013), older
1138 muscles have been found not to change gear as much in response to load (Holt *et al.*, 2016) or
1139 velocity (Randhawa & Wakeling, 2013) changes. The inability to change gear in response to a
1140 task's mechanical demands (e.g., force or velocity) may at least partly explain the age-related
1141 decline in both muscle strength and power. As illustrated in Figure 11, the age-related
1142 difference in *fibre* shortening peak power is smaller than the difference in *whole muscle* peak
1143 power. Furthermore, and perhaps of greater functional importance, there is substantial
1144 narrowing of the *whole muscle* power−velocity relation due to the inability of muscles to vary
1145 gear in response to changes in load (see Figure 3 and 11B). These results were observed in
1146 aged rats and are similar to those demonstrated in single fibre preparations from human muscles
1147 (Grosicki, Zepeda & Sundberg, 2022) as well as whole muscle peak power and width of the
1148 power−velocity relation *in vivo* (Alcazar *et al.*, 2023). Thus, alterations in variable gearing
1149 capacity offers a promising mechanistic explanation for some of the age-related decline in the
1150 power and strength in older adults, although further studies in humans are necessary to test this
1151 hypothesis.

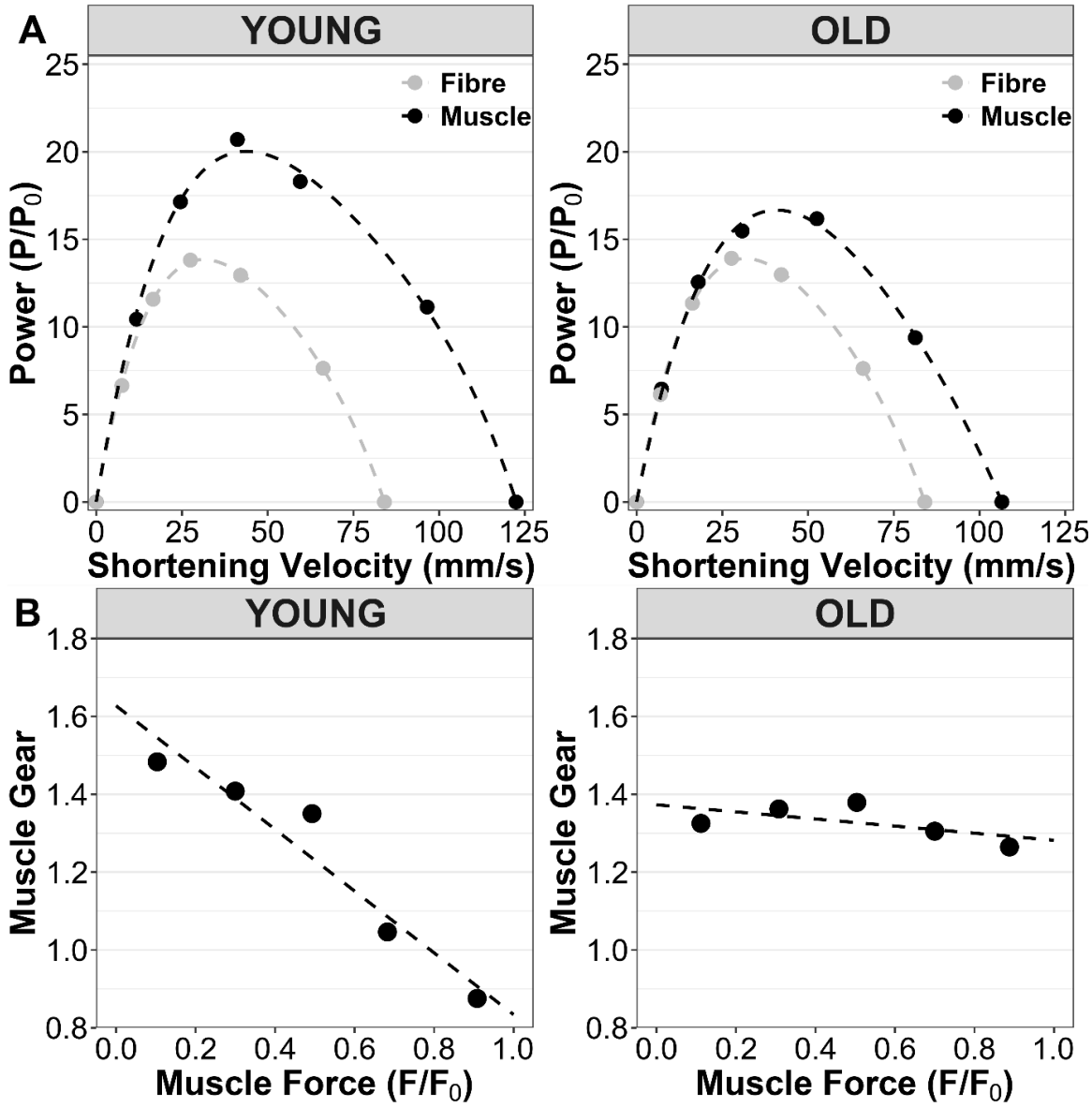

Figure 11. (A) Differences in fibre and whole muscle power-velocity relation in young and old rats. **(B)** Muscle gear in young (left) and old (right) rats. Gearing varies with force in young but not old rats. Old rats work at similar gears in the high force and low-force contractions, and this is hypothesised to be affected by increases in stiffness of both aponeurosis and the extracellular matrix surrounding fibres and fascicles. Figure adapted from Holt *et al.* (2016). Power was calculated from the available shortening velocity and whole muscle force (normalised to peak isometric force). Values were estimated using an open available graph digitiser (WebPlotDigitiser, https://apps.automeris.io/wpd/).

Second, several musculoskeletal disorders as well as recovery from muscle injury are associated with substantial alterations in the quantity and structure of the ECM and intramuscular fat. For example, increased collagen content (fibrosis), altered collagen architecture, and increased collagen cross-linking have been observed in Duchenne muscular

1166 dystrophy (Wohlgemuth *et al.*, 2023; Brashear *et al.*, 2021) and cerebral palsy (Smith *et al.*,
1167 2011; Lieber & Fridén, 2019) as well as following muscle strain injuries (Reurink *et al.*, 2015)
1168 and both ligament tears and reconstruction (Noehren *et al.*, 2016; Peck *et al.*, 2019). In contrast,
1169 classic-like Ehlers-Danlos Syndrome, a genetic connective tissue disorder caused by
1170 tenascin-X deficiency in the ECM, leads to reduced collagen fibril density and length in the
1171 endomysium and more compliant connective tissue within and between muscles (Huijing *et*
1172 *al.*, 2010; Voermans *et al.*, 2007). Accumulation of intra- and inter-muscular fat content are
1173 also a common response to skeletal muscle injury and disease (D'Souza *et al.*, 2020; Akinci
1174 D'Antonoli *et al.*, 2021; Leroy-Willig *et al.*, 1997), with both persistent fatty infiltration in the
1175 interfascicular space and within myofibrils even after a short-term 3-month resistance training
1176 (Bayer *et al.*, 2021). The internal work needed to deform these non-contractile tissues may
1177 detract from the external work that the muscle can do (Rahemi *et al.*, 2015; Wakeling *et al.*,
1178 2020; Konno *et al.*, 2021b) and alter the way the muscle fascicles can change in length and
1179 pennation. It is therefore possible that the persistent muscle strength, speed reductions, or even
1180 altered metabolic cost and muscle efficiency after injury and disease may be influenced by
1181 altered muscle gearing.

1182 Third, changes in muscle gearing could play a role in the changes in voluntary and
1183 involuntary (electrically-induced) contractile capacity in response to exercise-based warm-up,
1184 which has been shown to enhance performance (Blazevich & Babault, 2019). This is because
1185 brief bouts of muscle contraction can trigger substantial fluid shifts into the muscle (Sjøgaard,
1186 Adams & Saltin, 1985; Ploutz-Snyder, Convertino & Dudley, 1995), which should then
1187 enhance contractile capacity by (i) affecting the interactions between collagenous ECM tissues
1188 and the fluid to increase muscle stiffness (Sleboda & Roberts, 2017; Sleboda *et al.*, 2019), (ii)
1189 increase intramuscular pressure, (iii) increase resting pennation and subsequent rotation, and
1190 (iv) possibly reduce the distance between each contractile unit (fibres and fascicles), which all

impact gearing. It has been hypothesised that post-warm-up fluid shifts should increase muscle gear and benefit fascicle force–velocity and force–length properties during contraction (Blazevich & Babault, 2019). Indeed, Bourgeois, Duchateau & Baudry (2022) recently demonstrated small yet significant increases in peak twitch and doublet forces accompanied by increases in medial gastrocnemius fascicle rotation and shortening. These changes occurred in the absence of detectable alterations in resting architecture and are unlikely to be attributed to changes in series elastic compliance (Gago *et al.*, 2014) after performing the conditioning 6-s maximal voluntary contraction. Such finding is consistent with an acute contraction-induced alteration in gearing.

Lastly, many physical exercise interventions, whether combined with nutritional strategies or not, often immediately reduce (fatigue) or enhance (potentiation) muscle force. Some studies also report post-exercise changes in muscle fluid (Sjøgaard *et al.*, 1985; Ploutz-Snyder *et al.*, 1995; Beis *et al.*, 2011; Antonio *et al.*, 2021; Ribeiro *et al.*, 2020; Francaux & Poortmans, 1999), ECM remodelling (Csapo, Gumpenberger & Wessner, 2020; Guzzoni *et al.*, 2018), intra- and inter-muscular fat (Langleite *et al.*, 2016; Ramírez-Vélez *et al.*, 2021), and resting pennation angle (Kawakami *et al.*, 1995; Timmins *et al.*, 2016), which may all influence muscle gearing. Nonetheless, the extent to which physical exercise interventions acutely or chronically affect muscle gearing remains uncertain, and it is unclear whether the observed changes in muscle force and function following these interventions can be attributed to alterations in muscle gearing.

VII. Conclusions

(1) Skeletal muscles exhibit a broad range of contractile behaviours that are strongly influenced by their internal architecture. Historically, our understanding of the structure–function relation has been largely derived from muscle structure measurements

obtained at rest, through anatomical dissections or imaging techniques, and its function determined during isometric or concentric contractions. However, muscles typically operate under dynamic conditions involving both active and passive shortening and lengthening, during which substantial structural and shape changes occur. These structural deformations critically influence the most fundamental properties of muscle contraction (i.e., force–velocity and force–length relationships) and cannot be accurately predicted from examination of resting (non-contracting) architectural features alone.

(2) Structural changes during muscle contraction have been documented for over a century (since at least the 1920's), yet only recently has the functional significance of fascicle rotation and its influence on muscle shape changes during contraction been recognised. Fascicle rotation serves as a gearing mechanism, partially decoupling fascicle velocity from whole-muscle velocity and thereby providing a mechanical (velocity) advantage. Gearing enables muscle fibres to operate closer to their optimal force–length and force–velocity relations while facilitating greater whole-muscle level length change or contraction velocity. Importantly, in most cases the extent of this decoupling is not fixed; it varies dynamically with the mechanical demands of the task, allowing muscles to modulate gear ratios in real-time, effectively broadening their functional range and mechanical efficiency.

(3) Recent studies have investigated the mechanisms underpinning variable gearing, most often using bioinspired muscle models or isolated muscle preparations subject to supramaximal, short-duration electrical stimulations. These investigations have examined factors including the roles of pennation angle, directional components of contractile elements (fibres) forces, three-dimensional muscle expansion, intrinsic and extrinsic tissue properties, intramuscular pressures, external pressures, and mechanical work on the muscle surface. Variable gearing is a multifactorial phenomenon but yet appears to be an

emergent property of muscle function, and emerges from the complex, non-linear interactions among all these factors.

(4) The computational modelling introduced herein integrates these mechanisms and demonstrates that the general features of muscle gearing emerge from the physical properties of the contractile elements and the muscle tissue properties alone, without requiring extrinsic structures such as aponeurosis or other connective tissue structures. The simulations reproduced the shape changes and gearing effects reported in maximally activated animal muscles and extended these predictions to more nuanced behaviours observed under submaximal activation levels typical of human movement. The modelling framework showed that force, velocity, and activation level independently modulate muscle gearing, with the most pronounced effects occurring at higher contraction velocities, where reducing fibre shortening velocity can enhance force output.

(5) Real-time changes in muscle gear in response to contractile conditions have been documented in both human and animal pennate muscles. While many animal studies suggest a decrease in gear with increasing force, human *in vivo* studies have shown more variable responses of gear changes, including increases with force or velocity, decreases with force or torque, or no change at all. These discrepancies may reflect both methodological and biological differences, such as whether muscle force is held constant during measurement, the mechanical influence of compliant tendons, and the complex deformation patterns associated with synergistic muscle behaviour *in vivo*; such conditions differ markedly from those of controlled animal experiments.

(6) Muscle architecture and shape changes during contraction may offer functional advantages beyond influencing variable gearing. Because muscles operate in confined anatomical spaces, their deformation patterns may be tuned not only for force or velocity production but to prevent interference with adjacent tissues. Coordinated shape changes such as

reciprocal deformation in synergists or directional bulging in pennate muscles may represent strategies to maximise spatial efficiency and protect anatomical structures. These spatial-functional adaptations represent an elegant solution to spatial constraints within functional systems and may have implications for both biological understanding and translation applications.

(7) A better understanding of variable gearing is particularly important in clinical contexts. Many conditions involving disuse, whether due to immobilisation, injury, ageing, or neurological or musculoskeletal experiments, lead to substantial alterations in muscle architecture and tissue properties and are often accompanied by rapid declines in muscle power and functional capacity. Other physiological states may conversely enhance muscle performance via changes in tissue hydration or stiffness. Evidence suggests that ageing and disuse can attenuate or abolish fascicle rotation and impair variable gearing, while acute electrically induced maximal contraction appears to enhance performance and increase the magnitude of fascicle rotation. As such, influences on variable gearing may help explain these changes and offer a framework for interpreting muscle function loss across a range of conditions, including ageing, disease, and physical training or detraining. Advancing our understanding of these phenomena may provide a basis for understanding human muscle function and movement performance, and reveal novel targets for future intervention through exercise, nutrition, or pharmacological strategies.

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