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ANIMAL MOVEMENT, MECHANICAL TUNING AND COUPLED SYSTEMS

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Summary

Over the past two decades, there has been a growing interest in developing predictive models of animal movement and force generation in fluids. In a departure from past studies that have asked how prescribed motions of a propulsor (wing or fin) generate lift and thrust during swimming and flying, we are increasingly interested in predicting the propulsor's movement as well as the forces generated by it. This interest, motivated by a need to understand the control and dynamics of locomotion and its applications to robotics and animal physiology, requires that we develop integrative models and analyses of swimming and flying that incorporate neural control and muscle physiology into more traditional biomechanical studies of locomotion in fluids. This approach extends from whole-animal studies to the molecular basis of force generation. In this paper, we explore mechanical tuning from the level of the whole animal to the proteins driving force generation in muscle.

Key words: mechanical tuning, muscle, force, cross-bridge mechanics, locomotion, coupled systems.

Introduction

Animal movement through a fluid medium emerges from the complex interactions among neural control systems (e.g. pattern generators and feedback components), active force generation by muscle, elastic and inertial deformations of passive structures and the dynamics of the reactions from the fluid surrounding an animal (see Fig. 1). While understanding each of these components requires significant resources, there has been for some time a growing interest in understanding how such systems interact to affect motion in animals. This interest is spurred, in part, by a need to develop predictive tools for understanding the control and dynamics of moving animals and, in part, because we seek a deeper understanding of how robust or tuned an animal may be for specific modes of locomotion.

This issue of how all the component parts of a moving animal interact to affect some controlled movement is key to a variety of exciting new questions that arise from integrative studies. Thus, we might ask: what propulsor motions emerge from a given paradigm of muscle activation? Alternatively, what patterns of activation and feedback underlie the motions we observe? What are the consequences of changes in patterns of activation or feedback? How does the design of muscle – its geometry and the mechanics of its protein components – determine movement in animals?

A central issue here is that the various subsystems that constitute a moving animal are highly coupled. Thus, the force generated by muscle depends upon the length of that muscle and its shortening velocity. If the muscle is attached to some propulsive structure immersed in a fluid, we also know that the force acting on the muscle is an additional function (determined by the Navier–Stokes equations) of the position of the propulsor, its velocity and its acceleration (for reviews, see Daniel, 1995; van Leeuwen, 1992; Full, 1993; Williams et al., 1995; Jordan, 1996; Sigvardt and Williams, 1992). The precise details of each of these functional relationships depend upon the type and size of the muscles involved in creating motions, their attachments to skeletal and soft tissue elements, and the dynamics of fluid motions about some relevant surface. Thus, the motions that we predict must be both physically and physiologically feasible.

There is more than a mechanical world to worry about here. This issue of coupling also occurs from the standpoint of control. For example, pattern generators that control the program of motor output delivered to locomotor muscles are sensitive to the vast range of sensory information flowing in a moving animal. Feedback from eyes, from wind or flow receptors and from proprioceptors profoundly affects patterns of muscle activity by modulating the pattern generator (Pearson, 1993; Sigvardt and Williams, 1992).

Among the many problems we now face in understanding how these systems interact are a set of rather elusive issues pertaining to muscle function and its coupling to appendage motion. In this paper, we show how coupled mechanical systems in muscle give rise to tuned (resonant) behaviors. We do so with a brief foray into the dynamics of force generation in muscle, asking how muscle systems may be tuned by the mechanics that drive muscle contraction.

The components of the problem

As we suggest above, analyses of the movements of any swimming or flying animal require a combination of fluid

dynamics, neurobiology, muscle physiology and solid mechanics. Indeed, to develop predictive models of how muscle activation is manifest as propulsor movements that generate lift and thrust, we must grapple with all aspects of this problem. We divide this review into a brief glimpse of the internal world of an animal, its control and the dynamics of muscle force generation, and the external world, including the fluid and inertial elements that characterize animal movement.

The internal world

Periodic motions in swimming and flying animals have their basis in pattern generation (Arbas et al., 1997; Pearson, 1993; Sigvardt and Williams, 1992). The specific details of this pattern generation depend quite strongly on an impressive suite of sensory information flowing into the central nervous system. Multimodal sensory information, such as a combination of visual, mechanical and chemical cues, strongly modulates patterns of activation. Indeed, recent work by Chan et al. (1998) and the long history of analyses of the control of locust flight (Pearson, 1993) point to an important role of such multimodal sensory input in modulating movement *via* direct effects on central pattern generators. The flow of the visual world, the magnitude of fluid forces on rheoreceptive structures and the proprioceptive signals that arise from wing motions all affect the pattern of motor control. The central point here is that sensory information, which depends upon the motion of the animal, modulates pattern generation (Fig. 1).

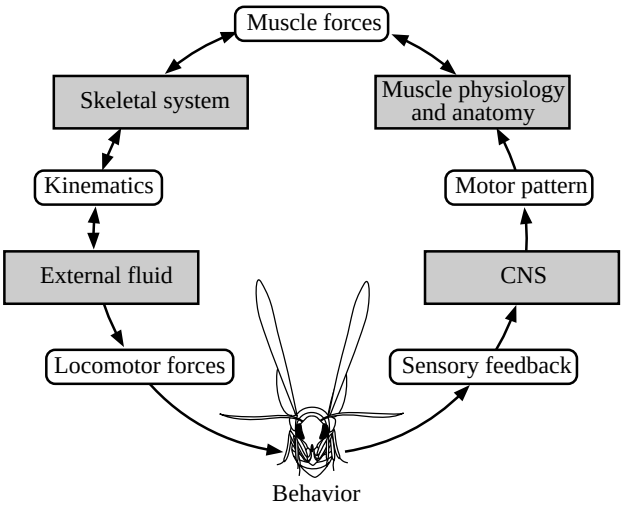

Fig. 1. Flight and swimming behavior involve complex interactions between neural control systems, muscles, skeletal mechanics and fluid/solid interactions in the external environment. Within the context of the fluid medium (air or water), variation in structural properties, and therefore tuning to produce specific behaviors, can occur at multiple levels (shaded boxes). These levels are coupled either mechanically or through sensory feedback. CNS, central nervous system.

With this coupling between sensory information and motor pattern generation as a backdrop, we need to know how any particular motor program is manifest as some temporal pattern of force generation. Several fundamental concepts about muscle and its organization guide our thinking about this issue. First, the geometric arrangement of muscle fibers within an animal is a key determinant of muscle force generation. For example, Alexander's (1969) study of the arrangement of muscle fibers in the myotomes of teleosts and elasmobranchs highlights this crucial role of fiber geometry in determining the design of muscles for either contractile force or speed production. Second, we know from the classic work of Gordon et al. (1966) that force depends upon myofilament overlap, giving rise to the classic length–tension relationship that shows a maximum force at a myofilament overlap of 100 %. In addition to muscle length, contractile forces depend inversely upon muscle shortening velocity by Hill's (1938) relationship, which McMahon (1984) conveniently expresses as a relationship between isometric stress and maximum shortening velocity (see also Daniel, 1995).

Muscle force also depends upon the time following activation, with several important temporal lags determining the time history of force production. These include the dynamics of Ca^{2+} fluxes out of intracellular stores and the dynamics of cross-bridge attachment to binding sites along the thin filament (e.g. Campbell, 1997). These delays, often quite pronounced, greatly modulate the temporal characteristics of force production. Similarly, during deactivation, delays associated with Ca^{2+} sequestration in intracellular stores and in cross-bridge detachment lead to pronounced lags in the decline of force with time. A cautionary note here is that a muscle may be electrically active with little force production (e.g. early in a twitch) or electrically quiescent with significant force remaining (e.g. late in a twitch).

We see, therefore, several important modulators of force production: (1) muscle fiber arrangement and cross-sectional area, (2) muscle length, (3) shortening velocity, (4) the pattern of activation, and (5) the internal dynamics (e.g. Ca^{2+} release, sarcomere length, myofilament overlap). The extent to which each of these factors contributes to the total dynamics of force production will depend quite strongly on the type of muscle involved in propulsion.

Because these measures of force generation are often derived under isotonic or isometric conditions, some caution needs to be exercised in blindly applying a combination of length–tension and force–velocity relationships to predictions of *in vivo* force production. This uncertainty, in part, motivated the development of the 'work-loop' approach (Josephson, 1985) as a means of measuring the work and energy production of muscle under conditions that were neither isometric nor isotonic. In this scheme, muscles are oscillated at frequencies and strains that approximate physiological conditions and are activated at a phase in the cycle of oscillation that may mimic the programmed motor output. Such methods have proved invaluable in determining how effectively muscles operate *in vivo* (e.g. Altringham and Johnston, 1990; Marsh et al., 1992; Tu and Dickinson, 1994; Biewener, 1998; Shadwick et al., 1998).

From a predictive standpoint, however, we are still a bit

limited by current approaches. The work-loop method, while a powerful quantifier of muscle power output, cannot be used to predict the motion of a propulsor since it demands a paradigm of prescribed muscle motion. Similarly, despite the promise shown by the models of van Leeuwen (1992) and Daniel (1995), because of assumptions about either isometric or isotonic conditions, there should still be some healthy skepticism about the direct application of force–velocity and length–tension relationships to predictive models of flight and swimming.

The external world

Movement in fluids requires the transfer of momentum from some propulsive surface to the surrounding fluid. In general, the equations that govern this transfer of momentum in a fluid (the Navier–Stokes equations) show a strong dependence on the geometry, speed and acceleration of propulsive surfaces (for reviews, see Spedding, 1992; Daniel et al., 1992; Ellington, 1995; Dickinson, 1996). With exceedingly important fluid dynamic details, such as delayed stall and the roles of rapid wing rotations in insect flight only now emerging (e.g. Ellington et al., 1996; Dickinson, 1996), we must be wary of more naive approaches for modelling the fluid dynamics around wings and fins that generate lift and thrust. However, until more tractable methods for accounting for instantaneous forces are available, we may resort to simplified models for examining the interactions between fluid-dynamic loads and muscle forces.

Recent progress in this area includes the development of new computational approaches for understanding the forces developed by complex wing, fin or body motions in fluids (e.g. Fauci, 1996; Peskin and McQueen, 1995; Liu et al., 1996). The good news here is that, with ever faster and more accessible programs, we may more readily account for details of the fluid dynamics of animal swimming and flight. The bad news is that we are still far from being able to incorporate such flows into three-dimensional problems for which very complex internal mechanical rules affect the motions.

Putting the pieces together: emergent behaviors at the level of the whole organism

Even in the context of the aero- and hydrodynamic details that are currently emerging, crude models of flight or swimming have nevertheless proved useful as a basis for integrative analyses of animal locomotion (e.g. Daniel, 1995). As in terrestrial systems, there is the strong possibility of mechanical tuning. By this, we mean that the mechanical design of the musculo-skeletal system can be adjusted to maximize force production, to minimize energy requirements for motion or to minimize the time required for specific movements. A classic example of such mechanical tuning occurs in resonant systems that rely on elastic energy storage (e.g. Alexander, 1988). Unlike such simple spring-mass systems, however, the non-linear relationships between fluid forces and the velocity and acceleration of the fluid make identifying resonance frequencies challenging. The same would be true for elastic components that show strong non-linear relationships between stress and strain. Some previous examples that have shown such tuning include studies of jellyfish and notonectid swimming (Daniel, 1995) and vertebrate swimming (Long and Nipper, 1996). In insect flight, we also note that tuning (or resonance) emerges from the mechanics of the thorax and flight muscles (Alexander, 1988).

At the core of our concerns about mechanical tuning lie several factors: (1) the presence of inertial (fluid and solid) components and elastic restoring forces introduces the possibility of resonant and tunable behavior, (2) the strongly non-linear components of the passive materials can lead to multiple resonant frequencies, and (3) the nature of damping (external fluid forces) in aquatic and aerial systems is exceedingly non-linear and may also lead to many possible resonant frequencies.

Delving deeper: tuning twitches and new cross-bridge mechanics

Thus far, in our analyses of movement, we have emphasized rather traditional measures of muscle contractile performance (whole-muscle mechanical properties). In reality, the temporal dynamics of activation, as well as the length–tension, force–velocity and work-loop measures of force, all follow from the dynamics of cross-bridge binding and force generation. Ideally, we would like to account for the design of the myofilament lattice in our analyses of locomotion. By doing so, we could relax many of the assumptions implicit in our application of muscle mechanics to locomotion. To probe still deeper into the problem of how muscle force generation plays a critical and tunable role in locomotion requires analyses that integrate aspects of the cross-bridge cycle into mechanical models of animal movement.

Until quite recently, we have assumed that cross-bridges operate as independent actuators. That is, each cross-bridge binds to a thin filament, generates force and, *via* ATP hydrolysis, releases from the thin filament to repeat the cycle of force generation in a way that is entirely independent of its neighbors (see Fig. 2). This approach is exceedingly convenient for developing models of cross-bridge force generation because we only need to worry about what the average cross-bridge does rather than account for the behavior of each individual cross-bridge. Indeed, the classic models of Huxley and Simmons (1971) and the more recent ones of Pate and Cooke (1989) and Campbell (1997) all follow from the tenets of mass-action kinetics. Such systems of differential equations are used to predict the time history of force generation by the ‘average cross-bridge’, the response of a system of cross-bridges to rapid length perturbations and the relationship between force and velocity in contracting muscle. These models provide the framework for the exciting possibility of analysing animal movement from the standpoint of the signals activating muscles and the arrangement and kinetics of the cross-bridges driving force production.

Having thin and thick filaments that are inextensible is

absolutely critical in models that assume independence among cross-bridges. This is because any compliance in the filaments would lead to the awkward situation in which thin filament binding sites would move about in response to cross-bridge forces. Thus, the likelihood of a cross-bridge finding a binding site might well depend upon what other cross-bridges are active and have moved their thin filament binding locations. Since a very small fraction of binding sites are commonly available for binding (new estimates are around 10–20 %), movements of binding sites mediated by cross-bridge forces would be critical (Howard, 1997).

This issue was brought into sharp focus by several studies showing the presence of a significant amount of compliance in the thin (and thick) filaments (Huxley et al., 1994; Goldman and Huxley, 1994). As much as 70 % of the total compliance of the sarcomere resides in the filaments rather than in the cross-bridges (as had been previously assumed). The idea that cross-bridges are independent is therefore seriously challenged; cross-bridges may be mechanically coupled through elastic (not rigid) thin filaments. This idea of coupling gives rise to the exciting possibility of mechanical tuning for force generation at the level of cross-bridges. Just as the coupling of elastic to inertial (time-lagged) processes leads to tuning and resonance in whole-animal systems, elastic coupling between cross-bridges may also lead to tuned muscle mechanics.

To account for filament compliance, Daniel et al. (1998) developed a spatially explicit model of cross-bridges in which a simple three-state model characterizes the cycle of attachment and detachment (Fig. 2). The various rate constants that underlie the transitions from state to state depend on the distortion of each cross-bridge (Pate and Cooke, 1989; Daniel et al., 1998). When any one cross-bridge binds, it generates a local force on the thin filament which, in turn, deforms in response to that force. This deformation results in a realignment of binding sites on the thin filament, a process that affects the transition rates of other bound or unbound cross-bridges. Thus, cross-bridges are mechanically coupled through the deformations of the thin filament.

Two biomechanical principles are central to our understanding of how cross-bridges generate force. First, their ability to bind to thin filaments depends on how far from a cross-bridge a thin filament binding site may lie: closer sites will have higher rates of binding to cross-bridges. Second, the likelihood that a bound cross-bridge will undergo some state transition (e.g. release or generate more force) depends upon the distortion of the cross-bridge. This is because transition rates depend on the energy in a cross-bridge – a term that depends, in part, on the amount of distortion borne by a bound cross-bridge. This mechanical energy is, for a linearly elastic molecule, proportional to the square of the distortion (Daniel et al., 1998; Pate and Cooke, 1989; Huxley and Simmons, 1971).

We can analyse this using a simple force balance for each individual cross-bridge and thin filament binding site (Fig. 3). In this scheme, the force generated by a cross-bridge is a function of its spring constant (approximately $1\,\mathrm{pN\,nm^{-1}}$) and deformation when bound. That force acts on a piece of the thin filament that stretches by an amount determined by its spring

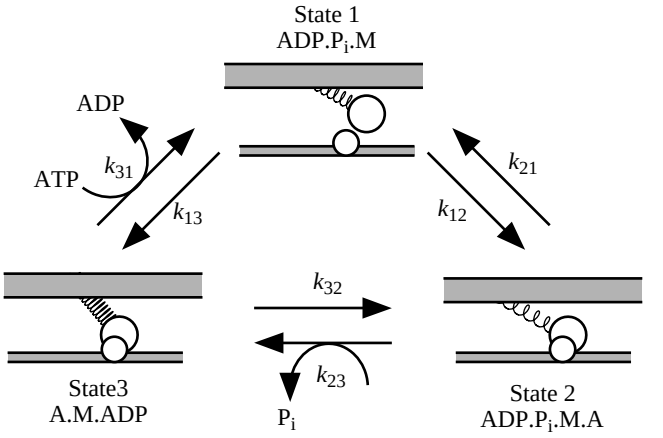

Fig. 2. Three-state model of cross-bridge cycling. State 1: detached myosin (M) cross-bridge with ADP+P_i. State 2: myosin weakly bound to actin-binding site (A). State 3: myosin strongly bound to actin following release of phosphate. The probability of cross-bridge binding depends on the distance from the myosin to the nearest actin-binding site, and the rate constants governing the state transitions depend on cross-bridge distortion. The rate constants k_{ij} represent the transition from state i to state j. The probability of undergoing a transition in time dt is k_{ij}dt. These mechanical dependencies of cross-bridge cycling couple cross-bridges along the length of a sarcomere.

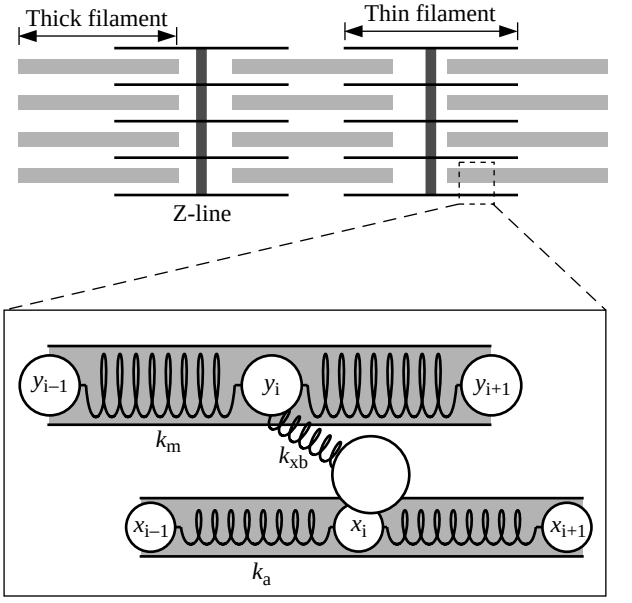

Fig. 3. Spatial model of interacting crossbridges. We calculate filament and cross-bridge deformations on the basis of a force balance at each cross-bridge (y) along the thick filament and at each actin-binding site (x) along the thin filament. k_m, thick filament spring constant; k_a, thin filament spring constant; k_{xb}, cross-bridge spring constant. Values of the various parameters and details of the computational methods are given in Daniel et al. (1998).

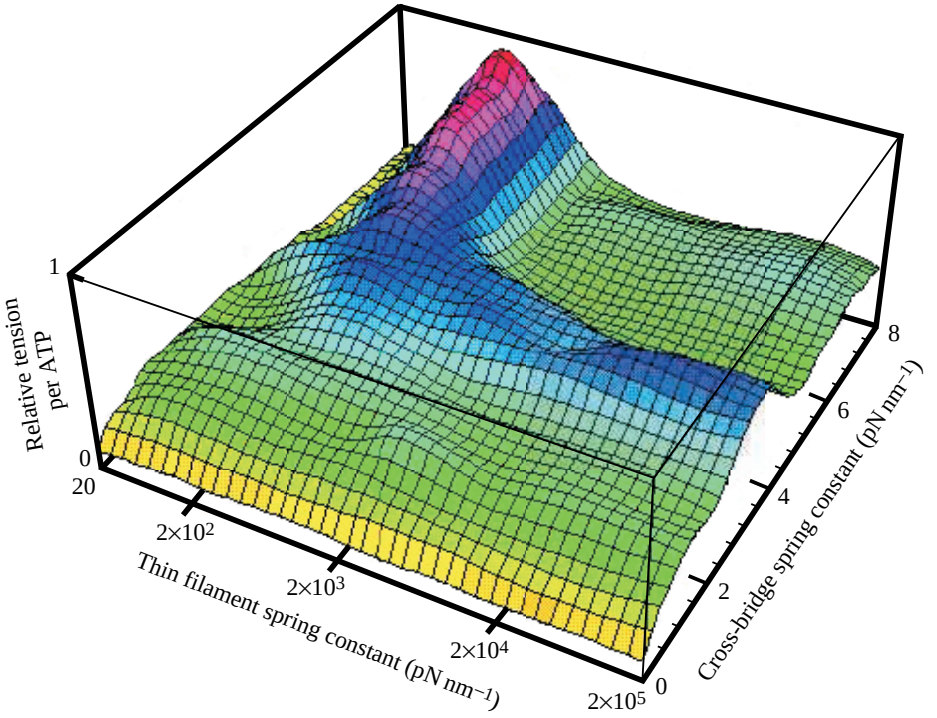

Fig. 4. There is a unique value for the spring constant of cross-bridges and thin filaments that maximizes the total tetanic tension generated for each ATP molecule consumed by the system of cross-bridges. This tuning of the filament lattices suggests that sarcomeres can be designed to maximize contraction efficiency. Unlike Daniel et al. (1998), this figure shows a single maximum for the tension developed per ATP molecule consumed by the lattice of cross-bridges. Current estimates of the thin filament spring constant place it at approximately 1700 pN nm^{-1}. Cross-bridge spring constants may exceed 1 pN nm^{-1} (see Daniel et al., 1998).

constant. This balance of forces can be calculated for each cross-bridge and each binding site. As Daniel et al. (1998) have shown, this can be modeled as a Monte-Carlo Markov chain process of many stochastically forced cross-bridges attaching to their nearest thin filament binding site.

By accounting for the mechanics of the filaments in this way, we can explicitly examine how filament geometry and mechanical properties determine (1) the magnitude of the total force generated, (2) the efficiency of force generation (force per ATP), and (3) the timing of tension rise and twitch duration. Each of these may be optimized for a given sarcomere design.

Mechanical coupling between cross-bridges leads to the exciting possibility that the design of the filament lattice can be tuned to produce maximal forces. Underlying this tuning problem is the idea that cross-bridges and their binding sites are not well aligned. The cross-bridges facing a single thin filament are approximately 43 nm apart, whereas the thin filament binding sites for these are approximately 37 nm apart. Thus, only approximately 10 % of the cross-bridges are within 1 or 2 nm of a binding site – a maximal distance for binding. Herein lies the core of this tuning problem: filament deformations in response to cross-bridge forces permit realignment of binding sites, bringing them closer to cross-bridges that might otherwise find them inaccessible. However, it is possible that filaments could be either too stiff or too compliant. Those that are very compliant permit significant alignment of binding sites but yield low forces by relieving the strain in cross-bridges. Those that are too stiff may permit the maintenance of high cross-bridge strain but recruit relatively few cross-bridges. Accordingly, peak force production occurs at intermediate levels of filament compliance – those quite close to published estimates (Daniel et al., 1998).

The presence of a peak force reported by Daniel et al. (1998) failed to account for the total energy produced by cross-bridge cycling. There is, therefore, the interesting possibility that flexibility in the filaments could well affect the total cycle time of a cross-bridge and, therefore, the total amount of ATP consumed in developing tension. In extending this analysis to account for the efficiency of force generation (in the sense of force generated by ATP), we note that there is a sharp tuning peak for the total tension per molecule of ATP (Fig. 4).

The relationship between peak tetanic force and filament (and cross-bridge) compliance provides a first glimpse into how the geometry and mechanics of a sarcomere affect steady-state force production. Of equal interest, particularly in fast locomotor systems, is the issue of whether the mechanics of the filaments affects the dynamics of a single twitch – both the duration of the twitch and the magnitude of the total force.

In, for example, the hawkmoth *Manduca sexta*, two powerful muscles drive wing motions: the dorsolongitudinal and dorsoventral muscles. These are controlled by a pattern generator that operates at approximately 25 Hz (though probably modulated by sensory feedback). Upon depolarization, these muscles undergo a single twitch in each wing beat, deforming the thorax in a way that either elevates or depresses the wing. Thus, trains of single twitch forces characterize this indirect and synchronous flight system. Is it possible that this is a tunable system at all levels of the mechanics of thorax deformation and at the protein filament level? Elastic energy storage in the thorax and flight muscles together with the inertial dynamics of the wings and unsteady air flow past them provide a mass-elastic system that is potentially tunable. The timing of force generation itself may

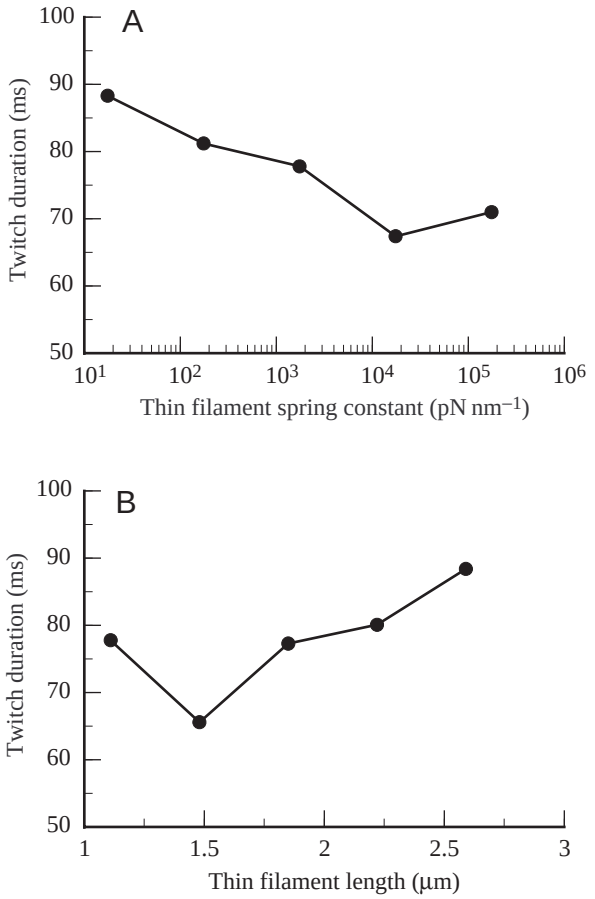

Fig. 5. (A) Predicted twitch duration depends on the spring constant (a measure of the compliance) of the thin filament. Very stiff filaments lead to shorter twitch durations, with a minimum occurring at a spring constant of 1.7×10^4 pN nm^{-1}. (B) Twitch duration also depends upon the length of the thin filament (the number of springs in series), with a minimum occurring at a filament length of 1.5 μm (approximately 30 % longer than that in vertebrate muscle). The points plotted are the mean durations from 50 simulations, and are derived from predictions based on the kinetic rate constants outlined in this paper and in Daniel et al. (1998).

also be tunable, a result that emerges from the effect of coupled cross-bridges acting in a compliant system of filaments.

To explore a part of this question, we used the spatially explicit model to compute the time history of a twitch as a function of the thin filament compliance (Fig. 5). We did so by varying the compliance of the filament over four orders of magnitude with Ca^{2+} fully available to all binding sites. Interestingly, there is a unique value for the filament compliance that minimizes the twitch duration. This is particularly important in antagonistic muscle pairs operating at high frequencies. Twitch durations that are too long lead to significant temporal overlap of the forces generated by pairs of muscles.

There are two ways in which the total spring constant of a thin filament can change. One is by a change in the modulus of elasticity of F-actin. This is unlikely, except that we note that the presence of other regulatory proteins (e.g. tropomyosin) can increase the stiffness of the filament (Isambert et al., 1995). A second, more common, way in which filament compliance may be increased is by increasing the length of the thin filament. This essentially adds additional springs in series to reduce the overall spring constant. Both effects, summarized in Fig. 5, show minima for the predicted twitch duration.

Conclusions

A major challenge in understanding the dynamics of locomotion lies in the problem of accounting for the coupling between neural control, musculoskeletal mechanics and the physics of motion in a fluid environment. Part of this requires that we probe quite deeply into the mechanisms of force generation at all levels of biological organization, from the organism to the molecule.

As a complement to more traditional organism and tissue-level approaches, we have developed a model that accounts for elastic energy storage in the lattice of filaments within a muscle. As in studies of whole organisms, we note a crucial role for elasticity in mechanical tuning for muscle function. Our model suggests that measures of muscle performance such as the total magnitude of force generation, the efficiency and the time course of force generation can exhibit distinct maxima or minima that depend explicitly on molecular-level geometry and mechanical properties.

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