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Release of fascial compartment boundaries reduces muscle force output

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Abstract

Most limb muscles operate within a compartment defined by fascial layers that enclose a muscle or groups of muscles within a defined space. These compartments are important clinically, because fluid accumulation can cause ischemia and tissue necrosis if untreated. Little is known, however, about how fascial enclosures influence healthy muscle function. One previous study showed that removing a fascial covering reduced the force output of a muscle under maximal stimulation. We hypothesized that such reduction in force output was due to a change in the muscle length following fasciotomy and that a reduced force output could be explained by the length-tension relationship of muscle. Thus we predicted that the maximum force across a range of lengths would be unchanged following fasciotomy. We measured maximal tetanic force output in a wing muscle in wild turkeys both before and after removal of fascia that enclosed the muscle in a compartment. Our hypothesis was not supported. The length-tension curve of this muscle showed that removal of fascia reduced maximum force output to $72 \pm 10\%$ of the prefascial release condition. Thus a reduction in muscle force following fasciotomy was not explained by a change in muscle length. The mechanism underlying reduction in force is unclear, but it suggests that the assumption underlying most isolated muscle experiments, i.e., removal of a muscle from its situation in vivo does not influence its maximal mechanical output, may need reexamining.

NEW & NOTEWORTHY Most limb muscles are enclosed within compartments bound by robust fascial sheets. The mechanical significance of the close packing of muscle and fascia is largely unexplored. We used an animal model to show that removal of a fascial covering reduces the maximal force developed during contraction. These results raise questions about the use of isolated muscles to estimate muscle performance and suggest that a muscle's mechanical surrounding influences performance by mechanisms that are not understood.

Keywords: compartment, fasciotomy, intramuscular pressure, muscle, tendon

INTRODUCTION

Muscle contractile function is most often studied in isolated muscles or muscle fibers (e.g., Refs. [6](#), [12](#), [14](#), [19](#)). It is generally assumed that such measurements accurately define the limits of muscle function *in vivo*; for example, isotonic force-velocity measurements can define a muscle's peak isometric force and peak power output ([18](#), [21](#)). It is understood that the actual force and power output of a muscle *in vivo* will reflect (likely complex) patterns of activation and recruitment, but measurements on isolated muscles should provide an indication of the limits of muscle performance under conditions of maximal activation.

In life, muscles operate within an environment of other muscles, bones, and connective tissue wrappings that may constrain muscle motion during contraction. Interaction among these elements is significant in a clinical setting through compartment syndrome. The compartments enclosed by fascial sheets can accumulate fluid due to a bleed or edema. With no outlet for the enclosed fluid, fluid pressures within the compartment can rise to levels that impede blood flow, inhibiting function and often requiring emergency "fascial release" surgery to prevent ischemic damage ([8](#), [15](#)).

The spatial constraints imposed by muscle compartments have the potential to influence muscle function because muscles must bulge when they contract. Shortening of a muscle must be associated with radial bulging of muscle fibers because muscles are essentially isovolumetric over short time scales ([3](#)). It is unknown the extent to which adjacent structures (e.g., bones, fascia, other muscles) present resistance to such bulging, or whether such resistance could influence muscle mechanical function. At least one study provides evidence that fascial enclosures affect muscle mechanical output. In the tibialis anterior muscle in dogs, maximum force production decreased following fascial release (incision of the fascia enclosing the muscle compartment) by 10–15% ([10](#)). This result suggests that lateral spatial constraints may be somehow beneficial to force production. At least one other study provides somewhat indirect support for this conclusion. After enclosing isolated frog muscles in a glass tube that limited lateral bulging, Mozan and Keagy ([17](#)) measured increased deflection of a muscle lever, which they interpreted as an indicator of increased force. However, other studies have demonstrated a reduction in the muscle force output under conditions of lateral (orthogonal to the line of force production) loads. Transverse loading of rat gastrocnemius with a plunger apparatus reduced muscle force output in shortening contractions ([22](#)). Model and experimental data show that muscle shortening and work, but not force, are reduced in the presence of a rigid constraint that limits bulging ([2](#)).

The question of whether enclosing fascia influences muscle function is important for a few reasons. First, models and experimental work aimed at understanding muscle function *in vivo*, for example, during locomotion, rely on existing metrics of muscle performance derived from measures on isolated muscles. If adjacent tissues influence muscle force output, such an influence should be accounted for in models. Fascial boundaries are also important clinically. The "release" of fascial compartments via the incision of enclosing fascia is a common procedure to treat compartment syndrome ([8](#)). It has been demonstrated that the capacity for force production of muscles within the affected compartment is significantly reduced following fasciotomy. Such reduced capacity likely reflects damage to tissues during an ischemic period in the affected compartment, but the results of Garfin et al. ([10](#)) would suggest that it may also be due in part to a direct influence of enclosing fascia on force output.

The goal of the present study was to reexamine whether fascial enclosures influence muscle mechanical output. We identified the ventral interosseus (VI) muscle in the wing of turkeys as an ideal study model for this question. The VI sits within a compartment bound on two sides by a fascial covering and on two sides

by the fused carpo-metacarpals of the “hand” region of the wing. It is the only muscle within this compartment. Because we could not identify a mechanism to explain a decrease in muscle force as a result of removal of adjacent structures (10), we hypothesized that there would be no change in peak muscle force following fasciotomy. We further hypothesized that a shift in muscle operating length following fasciotomy would provide a possible explanation for the postfasciotomy reduction in muscle force observed previously (10). Because force was measured at only one muscle-tendon unit length in the previous study, a small shift in position of the muscle following fascial release may have allowed for a change in muscle fiber length, resulting in a change in muscle force output as the muscle operated on a different (lower force) region of its length-tension relationship. To test these hypotheses, we measured force at a number of lengths in the VI muscle to identify peak isometric force (i.e., force at the optimal length) both before and after fasciotomy.

METHODS

Animals and study muscle. Wild turkeys (*Meleagris gallopavo*) were obtained from a licensed breeder and maintained at the Brown University Animal Care Facility. Food and water were provided ad libitum. Three male and two female adult animals were used for this study, average body mass 6.07 ± 0.74 kg. All experimental protocols were approved by the Brown University Institutional Animal Care and Use Committee.

The ventral interosseus (VI) muscle sits within a bony valley formed by the fused carpometacarpus of the distal wing (Fig. 1). The muscle belly is bound by a single fascia on its dorsal surface and a single fascia overlying the muscle’s ventral surface. These fascial sheets attach to the bones on either side of the VI muscle. The muscle is bipennate, with a long distal tendon inserting on the distal phalanx.

In situ force measurements. To determine the influence of enveloping fascia on muscle force, we measured force during maximal isometric contractions both before and after removing the ventral fascia. A servomotor was used to measure muscle force and adjust muscle length to allow for measurement of force over a range of lengths.

All procedures were performed in anesthetized (inhaled isoflurane, 2–4%) animals. Heart rate, oxygen saturation, expired CO₂ levels, and body temperature were monitored, and ventilation rate and isoflurane percentage were adjusted accordingly. A heating pad was used to help maintain normal body temperature.

Once an animal was anesthetized, the ramus superficialis, a distal branch of the ulnar nerve, was isolated on the dorsal surface of the radial carpal bone. Surrounding connective tissue was gently removed and a cuff enclosing a bipolar stimulating electrode was attached to the nerve. The nerve was severed proximal to the cuff. The isolated region of the nerve was distant from the site of the muscle; thus exposing the nerve did not risk damage to the fascia enclosing the muscle. The wing was fixed to a rigid custom-made aluminum frame by way of metal clamps attached to the carpometacarpal junction and the distal phalanx of digit III (Fig. 2). The distal tendon of the muscle was isolated on the dorsal surface of digit III. Bone cutters were used to cut around the distal tendon’s insertion site, leaving a small piece of bone attached to the tendon upon removal. This bone fragment anchored a length of Kevlar thread tied to the tendon on one end and to the servomotor lever on the other (Fig. 2). An advantage of this muscle for the purposes of this study is that the tendon insertion is some distance from the muscle’s compartment, allowing for access to the tendon for force measurements with no disruption to the muscle compartment.

Force was measured with a servomotor (Aurora Instruments 310B-LR; Aurora Scientific, London, ON, Canada). The servomotor was also used to adjust muscle length. Force measurements were recorded via an analog-to-digital converter (National Instruments model NI-6259A) operated with the software application Igor Pro (Wavemetrics; Lake Oswego, OR).

A series of contractions was performed to determine a supramaximal stimulation voltage. A Grass Instruments (West Warwick, RI) S48 stimulator generated a 0.2-ms pulse. The muscle's twitch force was monitored as the stimulation voltage was increased by 1-V increments. The voltage that resulted in maximum twitch force was increased by 50% and used to stimulate the muscle supramaximally. All tetanic stimulations were at 100 pulses/s.

Muscles were stimulated supramaximally under isometric conditions. Contractions were performed at different muscle lengths for five to seven contractions to characterize a length-tension curve (see [Fig. 5](#)). The muscle was allowed a minimum of 5 min between contractions to prevent fatigue.

Following measurement of a length-tension curve with the compartment intact, a fasciotomy was performed via a skin incision on the ventrum of the distal wing followed by a longitudinal incision along the ventral fascia overlaying the ventral interosseous. The fascia was carefully elevated above the muscle via forceps before the incisions to minimize the possibility of damaging underlying muscle fibers during fascia release. Following fascial release, the ventral interosseous was once again subjected to approximately five to seven contractions at different muscle lengths to characterize a postfascial release length-tension relationship. Repeat isometric contractions at the same muscle length were used to assess muscle fatigue. Throughout all measurements, muscle temperature in an adjacent muscle, the extensor digiti tertii manus distalis, was recorded. Temperatures in this muscle remained within a range of 35–41°C.

Data analysis and statistics. Data from individual contractions were analyzed using Igor Pro. For each of the five individuals, isometric force production before and after fascial release was quantified at a range of muscle lengths; a length-tension curve was characterized. To construct a single curve for all animals, force was normalized to the maximum isometric force measured for each individual. A third order polynomial was fit to the length tension data, and the maximum of this curve was taken as the optimal muscle length, L_0 . Resting muscle fascicle length was measured via calipers at the length that elicited maximum force. Length and force-normalized data for each animal were averaged by binning over regions of $0.2 L/L_0$.

To determine the influence of fascial release on force, a mixed-model ANOVA was used with length (L/L_0) and fascial condition (pre or post) as fixed effects and animal as a random effect.

RESULTS

Morphological measurements of muscles used in the study are presented in [Table 1](#). The interosseus is a bipennate muscle. At resting length, measured pennation angle averaged $22.4 \pm 2.1^\circ$ from the line of the central tendon.

In fixed-end contractions, the ventral interosseus typically reached a plateau value of tetanic force at 200–300 ms following stimulation ([Fig. 3](#)). The relatively slow rate of force rise in this muscle was presumably due to the effects of series compliance present in the long distal tendon. [Figure 3A](#) shows sample

contractions measured at the same length, before and after fascial release. These contractions are representative of the decline in force observed following fascial release, which is apparent at all lengths for a representative animal (Fig. 3B).

The decline in muscle force following fasciotomy was consistent across all animals studied (Fig. 4). The maximum force measured in the postfasciotomy condition was 0.72 ± 0.10 (means \pm SD) that of the prefasciotomy condition. A post hoc test following ANOVA indicated a significant difference in force in the pre- and postfasciotomy conditions for all lengths except the shortest length measured.

Repeat contractions indicated there was little fatigue over the course of the series of contractions used to characterize the length-tension relationship. Repeat contractions near optimal length taken after five to seven length-tension contractions over an ~30-min period generated 0.98 ± 0.04 of the force developed at the beginning of the series of contractions (Fig. 3). This suggests that force output was not affected by either number of contractions or time elapsed.

DISCUSSION

We found that removal of the fascia overlying the turkey ventral interosseous significantly reduced the maximum force the muscle was capable of developing. The observation that force is reduced by >20% following fasciotomy did not support our hypothesis, which was that there would be no decline in maximum active force developed when the influence of muscle length was accounted for. Our results support those of Garfin et al. (10), who showed a 15% decline in maximum force following fasciotomy in the anterior compartment of the dog hindlimb. Together these results are significant because they suggest that estimates of muscle force from traditional and common in vitro measurements may underestimate muscle force capacity. The results are also worth considering carefully because given our existing understanding of the mechanics of muscle contraction, a mechanism to explain a reduction in force following removal of adjacent fascia is not obvious.

Garfin et al. (10) included measures of intramuscular pressure in their study and found a reduction in fluid pressure within the muscle compartment from the pre- to postfasciotomy condition. They concluded that it was this reduction in intramuscular pressure that explained the reduction in muscle force following fasciotomy. This conclusion is intriguing but problematic, because we are not aware of an existing theory of muscle contraction that predicts a causal relationship between intramuscular pressure and developed force. While there appears to be a correlation between developed muscle force and intramuscular pressure under some conditions (7), existing theories suggest that the pressure results from the force developed by contractile elements (13). A mechanism whereby developed force would be reduced by a reduction in the pressure developed is, to our knowledge, not present in existing models of muscle contraction.

Studies on isolated muscle fibers have used pressure chambers to experimentally alter the hydrostatic pressure of contracting muscles and measure the effect on force output (4, 5, 11, 23). These studies show that an applied hydrostatic pressure can influence tetanic force. The mechanism explaining these changes is uncertain, but it has been proposed that changes in tetanic force with pressure result from either changes in rates of cross-bridge cycling or force per cross bridge (23). The observation of a link between force and pressure in isolated fibers does not likely support the idea that pressure changes explain a drop in force following fasciotomy for two reasons. First, in most studies tetanic force is reduced at higher pressures (11, 23), while after fascial release lower forces correlate with lower pressures (10). In some cases, an increase in tetanic force with increased pressure has been observed (9, 23). However, most relevant for the present

results is that the pressures studied are orders of magnitude greater than the expected physiological range. In the study of compartment release in dogs, for example, interstitial fluid pressure measured in the intact condition was ~80 mmHg, and it was ~40 mmHg postfasciotomy (10). This change of ~5 kPa would not be expected to effect a measurable change in force output based on single fibers studies. Frog muscle fibers at 21°C increase tetanic force output under pressure at a ratio of 0.4% mPa⁻¹ (23), a ratio that would predict only a 0.002% change in force for the change in pressure observed by Garfin et al. (10).

We considered a number of possible issues of measurement error or artifact that might explain the decrease in force following fasciotomy. The possibility exists that the muscle was damaged during the process of fascia removal, either through the dissection process itself or due to structural connections between the fascia and the muscle. Our subjective judgement is that fascial removal was possible with negligible muscle damage. It was quite easy to elevate the fascia from the muscle, and there were no obvious substantial physical connections between the two structures. Removing the fascia was a process of only a few seconds and was done with a single cut along the midline of the elevated fascia (with forceps used to elevate the fascia away from the muscle). The fascia and the muscle both originate from the fused carpometacarpals. We examined these origins under magnification post mortem and determined that the connections of the fibers and the fascia to the bone are in close proximity. The possibility exists that some fibers originate on the fascia and that disrupting the fascia reduces active force output because it disrupts some fiber origins. While we cannot completely exclude this possibility based on our observations of anatomy alone, we do not believe it explains the reduction in active force output observed following fasciotomy. If the reduction in active force was due to a disruption of fiber origins, we would expect a decrease in passive force following fasciotomy that was proportional to the observed decrease in active force. However, passive force at the lengths measured did not change significantly following fasciotomy (Figs. 3 and 4). In addition to measuring passive force statically in each bird, in a few birds we also applied a ramp stretch to passive muscle both before and after fasciotomy. Such measurements confirmed negligible change in passive force from the pre- to postfasciotomy condition as shown for a representative trial in Fig. 5. These observations support the idea that the reduction in active force following fasciotomy was not due to damage to the muscle or muscle fiber origins.

Fasciotomy uncovered the muscle and potentially exposed it to cooling and dehydration that could influence force output. We endeavored to keep the muscle moist by covering it with warmed saline-soaked gauze between contractions. While we were not able to measure temperature in the study muscle, we found that an adjacent muscle maintained temperatures above 35° under all conditions. Maximum isometric tension in vertebrate muscles is relatively insensitive to temperature. For example, peak isometric tension in rat soleus and extensor digitorum longus decreases by <5% from 35 to 25° (20). We do not think the VI cooled significantly during experiments, and even substantial cooling would not likely explain the magnitude of force reduction observed following fasciotomy in our experiments. The reduction in force following fasciotomy in dogs was measured in a preparation in which the skin was closed over the muscle following fasciotomy, presumably maintaining muscle hydration and temperature (10).

A decline in muscle force due to fatigue also does not appear to explain the reduction in force following fasciotomy. Across five to nine contractions over a ~30-min period in the prefasciotomy condition, we found no reduction in maximum force output at optimal length. While force output can often decline steadily across a series of contractions in an in vitro preparation (1), the intact blood supply and homeostatic mechanisms in an in situ preparation result in our experience in performance that is quite consistent and resistant to fatigue.

The length-tension relationship measured for the ventral interosseus was much broader than expected for a vertebrate muscle fiber. We attribute the relative high force at short lengths to the fact that we measured muscle length, rather than fiber length, in our preparation. The ventral interosseus has a long tendon, and the stretch of this series elasticity will distort a length-tension relationship determined from the position of a servomotor. It is possible that regional variation in fiber length following fascial release could influence fiber length heterogeneity and influence the measured force-length curve, including maximum force. Given that the pre- and postrelease curves are similar in shape, and not shifted along the length axis, we think such heterogeneity is an unlikely explanation for the change in maximum measured force following fasciotomy.

The results from the present study suggest that fasciotomy can result in a significant change in force. It is difficult to know whether a similar magnitude of effect might be expected in other muscles. The ventral interosseus is bound by bone on two sides: muscle/fascia dorsally and fascia on its ventral aspect. Most compartments contain several muscles and are most commonly adjacent bone on only one side. The decline in force of ~20% in the ventral interosseus is similar to the 15% decline measured for dog tibialis anterior (10), a muscle that sits with several other muscles within the anterior compartment of the leg.

The ways in which surrounding structures influence muscle force in vivo require further study. It has been shown convincingly in animal models that force can be transferred from one muscle belly to an adjacent belly, and thus we know that the force produced at any given tendon might not be accurately predicted from the properties and dimensions of the muscle to which it directly attaches (16, 24). The observation of a reduction in force following fasciotomy suggests that adjacent structures might influence force output through other mechanisms. The nature of these mechanisms requires further study.

GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

R.J.R., D.A.S., and T.J.R. conceived and designed research; R.J.R., D.A.S., and T.J.R. performed experiments; R.J.R. analyzed data; R.J.R., D.A.S., and T.J.R. interpreted results of experiments; R.J.R. and T.J.R. prepared figures; R.J.R. drafted manuscript; R.J.R., D.A.S., and T.J.R. edited and revised manuscript; R.J.R., D.A.S., and T.J.R. approved final version of manuscript.

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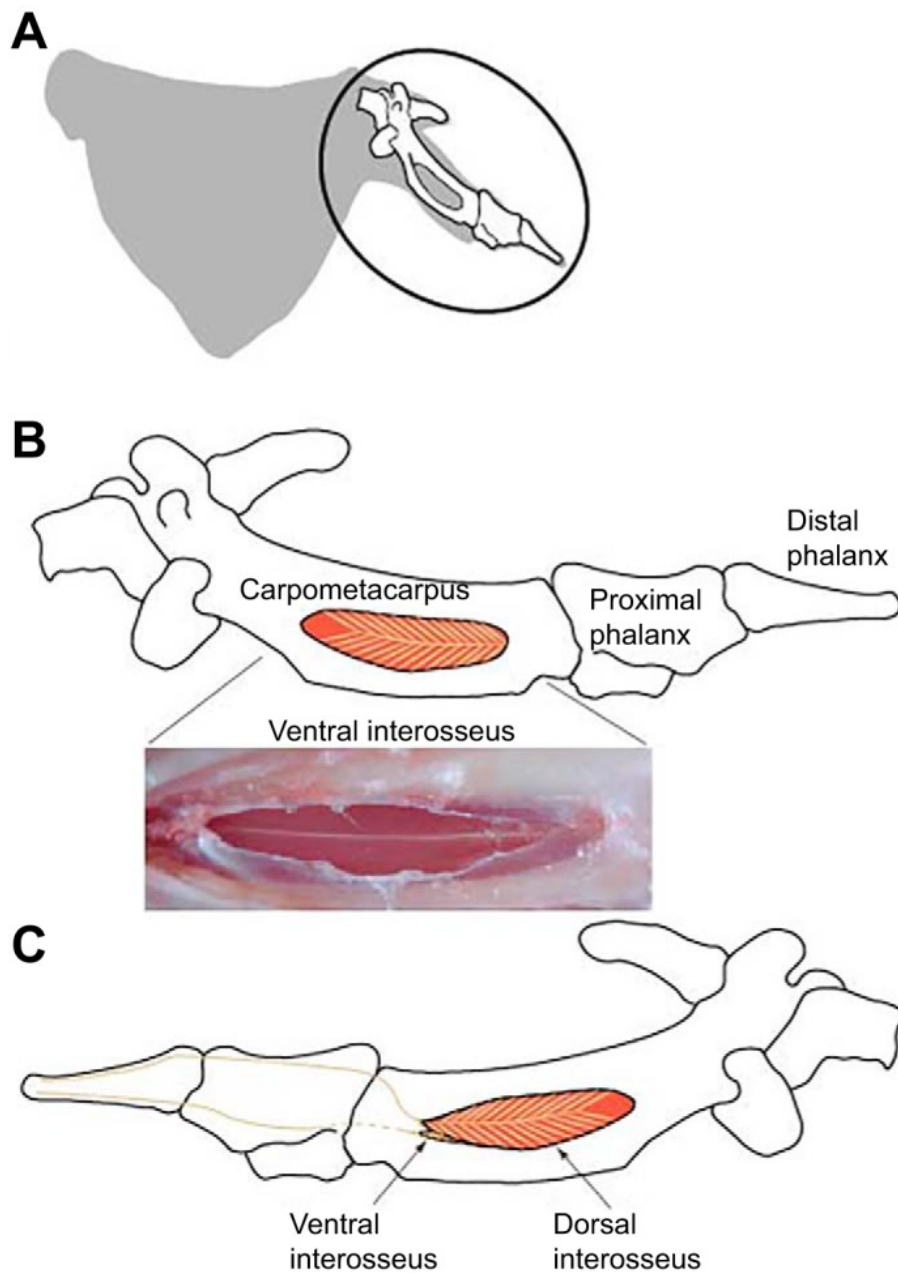
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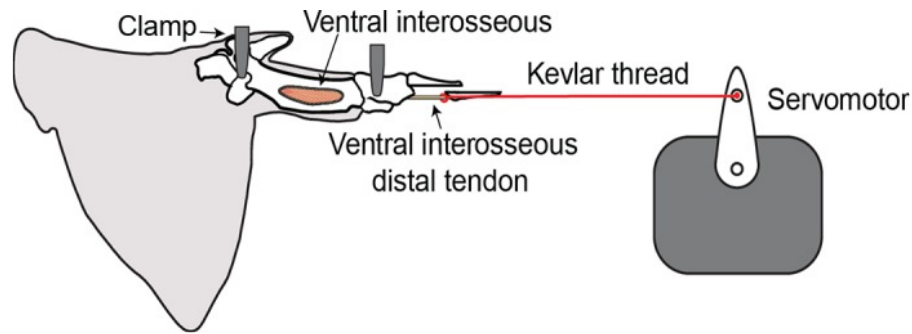
Figures and Tables

Fig. 1.



Location of the region of interest within the wing (A) and ventral (B) and dorsal (C) views of turkey interosseous muscles. The muscles sit within a bony canal formed by the fused carpometacarpus and are encased by a fascial layer (not shown) on the dorsal and ventral surfaces. Muscle fibers insert on a long central tendon that attaches at the dorsal surface of the distal phalanx of digit III. *Inset*: photo shows the ventral interosseus with the cut edges of the fascia after fascial release.

Fig. 2.



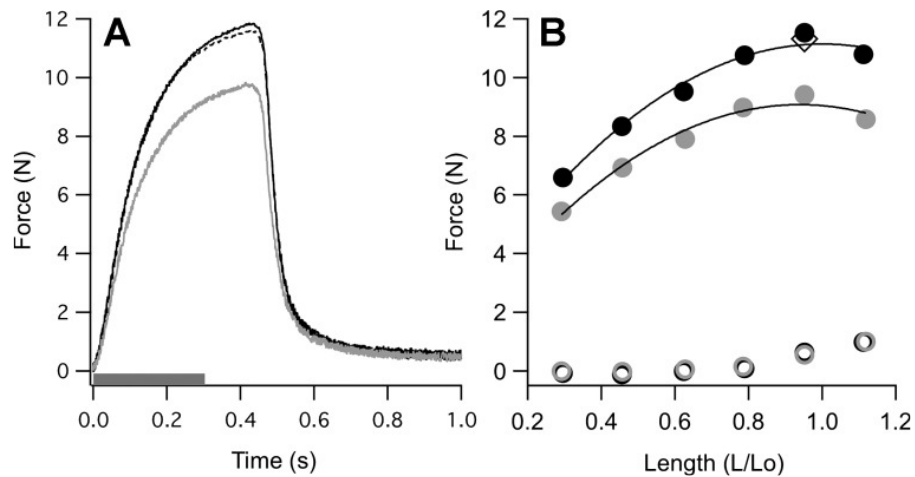
Schematic of in situ preparation. The distal tendon of ventral interosseous was attached to a servomotor lever via Kevlar thread (red line). The wing was secured via clamps.

Table 1.

Dimensions of muscles studied

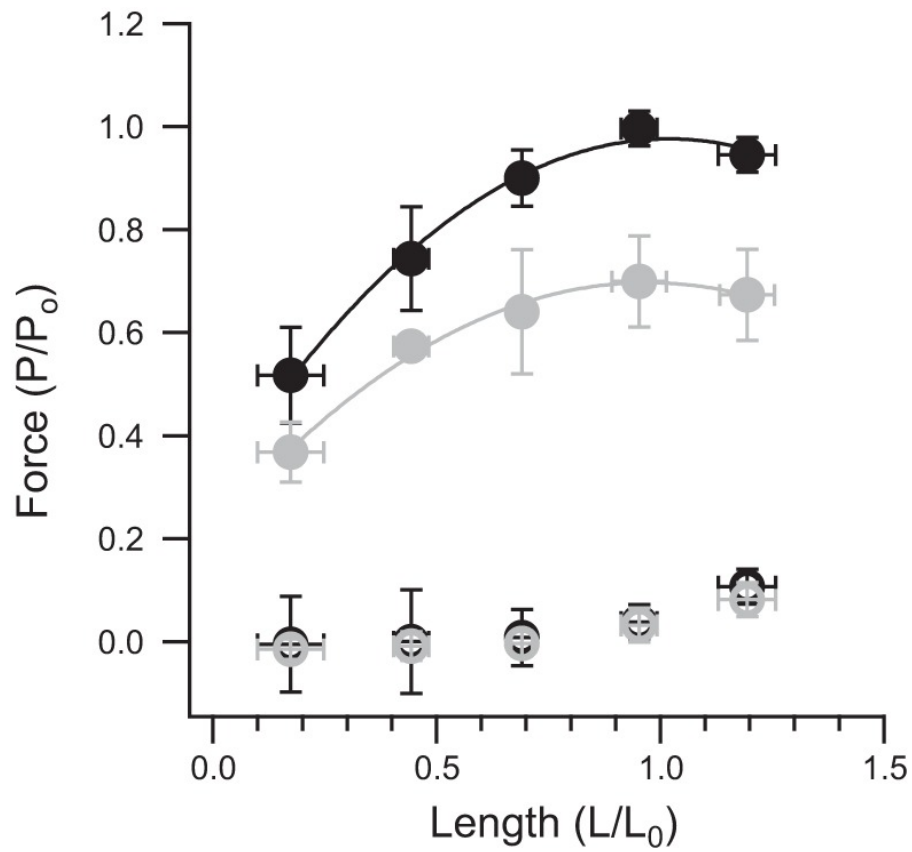
Animal	Muscle mass, g	Optimal Fiber Length, L_0 , mm	Pennation Angle, °
<i>Bird 1</i>	0.344	6.13	25
<i>Bird 2</i>	0.489	6.36	21
<i>Bird 3</i>	0.651	7.21	22
<i>Bird 4</i>	0.553	6.47	20
<i>Bird 5</i>	0.601	6.89	24
Means \pm SD	0.527 ± 0.119	6.61 ± 0.433	22.4 ± 2.1

Fig. 3.



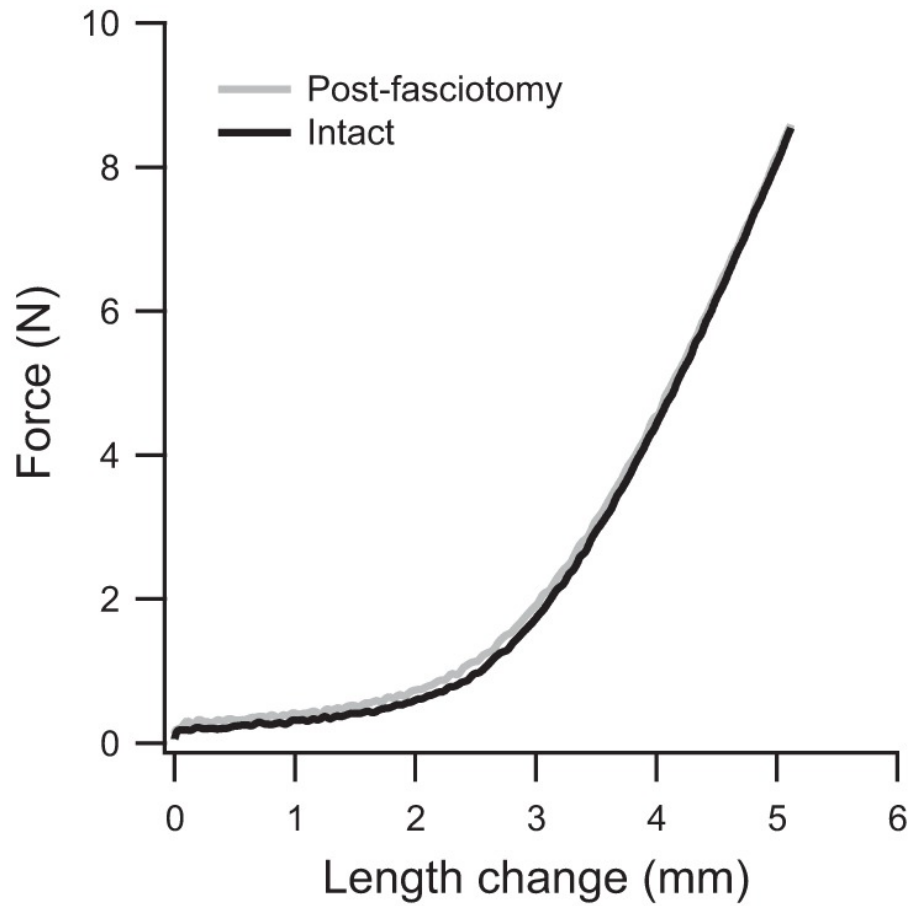
Sample contractions and length-tension curve. *A*: single contractions at optimal length show a decline in force from the intact condition (solid black line) to the postfasciotomy condition (gray line). The dotted line indicates the change in force in the intact condition from the first contraction to the last intact contraction. *B*: force produced across a range of lengths for the same individual (closed circles active; open circles passive), with the intact condition indicated by black and the postfasciotomy condition indicated by gray. The open diamond symbol is a repeat contraction, just before fasciotomy. The gray bar at *A*, *bottom*, indicates the time of stimulation.

Fig. 4.



Length-tension curves for intact and postfasciotomy conditions. Fascial release was associated with decreased muscle tension in active (closed circles) but not passive (open circles) conditions. Black symbols represent intact values, and gray represent postfasciotomy. Values are means \pm SD for 5 animals.

Fig. 5.



Passive force-length curves for a single muscle during a ramp stretch before (black) and immediately after (gray) fasciotomy.