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The effects of passive warming on muscle injury*

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ABSTRACT

This study investigated the effects of passive warming on the biomechanical properties of the musculotendinous unit. Paired tibialis anterior (TA) and extensor digitorum longus (EDL) muscles in the rabbit hindlimb were passively heated to different temperatures and then subjected to controlled strain injury. The parameters examined were: 1) percent increase in length to failure, 2) force to failure, 3) energy absorbed by the musculotendinous unit to failure, and 4) site of failure.

Warmed ($39^{\circ}\text{C} \pm 0.5^{\circ}\text{C}$) TA ($P \leq 0.01$) and EDL ($P \leq 0.05$) muscles achieved a greater increase in length from rest before failing than did their contralateral controls at $35^{\circ}\text{C} \pm 0.5^{\circ}\text{C}$. In both the TA and EDL the force at failure was greater at 35°C than at 39°C , although the difference was significant for only the EDL ($P \leq 0.05$). The energy absorbed (area beneath the length-tension curve) by both the TA and EDL was greater at 39°C , but these differences were not significant. All muscles failed at the distal musculotendinous junction. These data suggest that passive warming increases the extensibility of the musculotendinous unit and may thereby reduce its susceptibility to strain injury.

Despite the marvelous and usually effective mechanism by which muscles transmit their forces to the skeleton, the structures involved in this transmission constitute a frequently injured unit, and one that is generating much interest in the area of injury prevention. It has been commonly accepted that a period of warmup prior to exercise, often

using active muscular contraction followed by stretching, increases the flexibility of the connective tissue^{1,16} and thereby decreases the risk of injury to the musculotendinous structures during the subsequent activity.

Although it is well accepted that the musculotendinous tissues do in fact become more flexible after a period of active contraction and stretching, the relative contribution to this increased flexibility from muscle warming and/or muscle facilitation-stretching is still in question. Our laboratory investigations have shown that passive repetitive stretching of musculotendinous units causes the tension in these units to decrease.¹² In addition we have shown that muscles that are preconditioned by a single active contraction are able to tolerate greater force and length increases than are unconditioned muscles when pulled to failure.¹⁰ The present study was designed to investigate the effects of passive heating on the following parameters of the musculotendinous unit: increase in length to failure, force to failure, energy absorbed to failure, and site of failure. These were chosen as the most representative for the effects of passive (noncontractile) heating.

MATERIALS AND METHODS

Two muscles from each hindlimb of 10 New Zealand White rabbits (average weight, 2.6 ± 0.14 kg) were tested. After intramuscular injection of an anesthetic mixture of ketamine, 100 mg/kg (Ketaset, Aveco Co., Fort Dodge, IA), Xylazine, 12.5 mg/kg (Mobay Corp., Shawnee, KS), and Acepromazine, 3 mg/kg (Aveco), the hindlimbs were shaved. A latex rubber cuff was affixed to the skin of each of the two thighs using Hollister Medical Adhesive (Hollister, Inc., Libertyville, IL), leaving a portion of the cuff free for attachment to the warming chamber. A skin incision was then made, extending proximally from the dorsal surface of the foot to the upper border of the patella, and the skin reflected. The lengths of both the tibialis anterior (TA) and extensor

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digitorum longus (EDL) muscles were recorded with the knee in full extension by measuring from the proximal most point of origin to the crural ligament on the distal tibia using a dial caliper. Sutures were then tied to each tendon immediately distal to the cruciate ligament on the dorsal surface of the foot, and the tendons transected distal to the suture. The TA and EDL muscles were dissected free of one another and of their investments of connective tissue (as required to allow individual testing) with their neurovascular supplies carefully preserved. The muscles were kept moist throughout these procedures using normal saline irrigation.

The muscles of the left hindlimb were warmed in a $35^{\circ}\text{C} \pm 0.5^{\circ}\text{C}$ saline bath and then pulled to failure using the procedures outlined below. In order to immobilize the entire hindlimb Kirschner wires were driven through the distal femur and the proximal tibia. The hindlimb was then passed through the opening in the bottom of the warming chamber and the latex cuff pulled over the lip of the opening to provide a waterproof seal. The Kirschner wires were fixed to the testing frame by means of four tubular clamps that passed through rubber grommets in the wall of the warming chamber, again guarding against leakage. The testing frame was then fixed to the crosshead of an Instron Universal Testing Instrument, Model TM (Instron Corporation, Canton, MA) using C-clamps. Flexible supply and return lines from a HAAKE circulating water bath were connected to the warming chamber, and the chamber filled with circulating normal saline at $35^{\circ}\text{C} \pm 0.5^{\circ}\text{C}$, covering the TA and EDL muscles (Fig. 1). After 2 minutes in the saline bath the TA tendon was connected to the Instron load cell by means of a pneumatic clamp (the clamp pressure was set to 30 psi and the clamping surfaces covered with Dermicel tape to prevent slipping of the tendons) and the musculotendinous unit pulled to failure at a rate of 10 cm/min. Failure was defined as complete disruption of the muscle-tendon unit with a loss of continuity or resistance to further stretch.

Temperature within the chamber was monitored through-

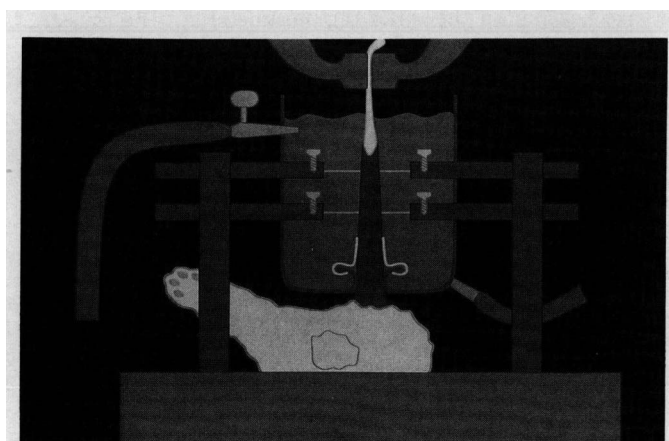


Figure 1. The hindlimb was stabilized by a wire in the femur and tibia. The limb was "sealed" through the use of a latex cover glued to the limb and pulled over the metal rim of the chamber. A constant temperature bath supplied inflow superiorly to maintain the desired temperature.

out testing. The length and tension changes were recorded on the adjacent Instron chart recorder. The EDL remained in the saline bath until 4 minutes had elapsed, and it was then pulled to failure in a similar fashion. The load cell clamp held the tendon of each muscle at the approximate position of the crural ligament, assuring that the recorded length changes could be related directly to the previously measured muscle lengths. The TA and EDL muscles in the right hindlimb were prepared in an identical fashion, but were heated in a $39^{\circ}\text{C} \pm 0.5^{\circ}\text{C}$ saline bath for 2 minutes (TA) or 4 minutes (EDL) before being pulled to failure. The testing required approximately 1 minute; hence, the 2 and 4 minute pattern of testing.

Following the experiment in five of the animals the proximal muscle fragments were carefully dissected from their origins, blotted dry, and weighed to the nearest 0.01 gm on a Mettler PE 360-Delta Range (Mettler Instrument Corp., Hightstown, NJ) balance along with the distal fragments of the same muscles. The animals were euthanized with 1 cc of Letalis (Barber Veterinary Supply, Richmond, VA).

Statistical analyses of the change in length to failure, maximal force to failure, and relative energy absorbed to failure were performed on the left and right leg pairs of both the TA and EDL muscles using a matched pairs Student's *t*-test to eliminate interindividual variation.

RESULTS

Length

Figure 2 shows the average percent increase in length at failure in TA and EDL muscles warmed to 35°C and 39°C for 2 minutes (TA) or 4 minutes (EDL). The 35°C TA muscles achieved a $31.5\% \pm 3.3\%$ length increase before failure, while the contralateral TA muscle at 39°C achieved

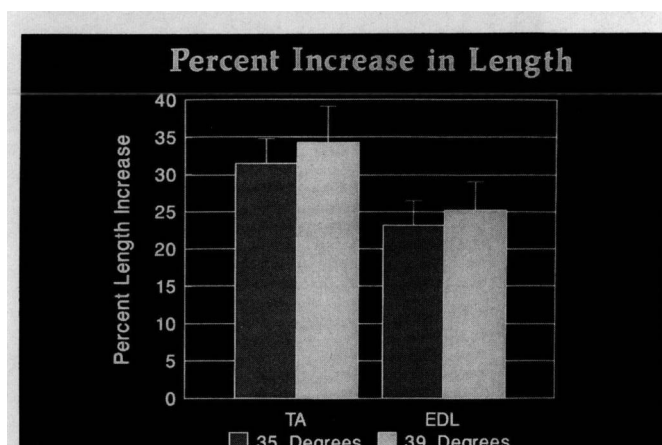


Figure 2. Average percent increase in length before failure in 35°C and 39°C TA and EDL muscles. All values are mean \pm SD ($N = 10$).

	35°C	31.5 ± 3.3%	23.2 ± 3.3%
	39°C	34.3 ± 4.8%	25.2 ± 3.8%
Difference:		2.8	2.0
		$P \leq 0.01$	$P \leq 0.05$

a $35.3\% \pm 4.8\%$ length increase before failing (difference, 2.8%; $P \leq 0.01$). The average increase in length for the 35°C EDL muscle was $23.3\% \pm 3.3\%$, while that for the 39°C EDL was $25.2\% \pm 3.8\%$ (difference, 2.0%; $P \leq 0.05$).

Force

Figure 3 shows the average force recorded at failure for TA and EDL muscles warmed to 35°C and 39°C for 2 minutes (TA) or 4 minutes (EDL). The 35°C TA muscles required a force of 3.83 ± 0.64 kg, to failure, while the contralateral TA muscle at 39°C required a force of 3.73 ± 0.56 kg (difference = 0.10 kg; $P \leq 0.1$). The average force to failure for the 35°C EDL was 8.29 ± 0.92 kg, while the 39°C EDL required 8.06 ± 0.90 kg (difference = 0.23 kg; $P \leq 0.05$).

Absorbed energy

Figure 4 shows the average relative energy absorbed to failure by the TA and EDL muscles warmed to 35°C and 39°C for 2 minutes (TA) or 4 minutes (EDL). This can be defined as the area under the load-deformation curve generated by the muscle-tendon unit as force is measured while it is stretched to ultimate failure. The shape of the curve is sensitive to extensibility and stiffness. The 35°C TA muscles absorbed an average of 224 ± 40 units of energy, while those at 39°C averaged 242 ± 58 units. The average energy absorption for the 35°C EDL muscles was 291 ± 54 units, while the 39°C EDL muscles averaged 298 ± 54 units. In neither case was the difference in energy absorption at the different temperatures statistically significant.

Site of failure

The site of failure for all of the muscles tested was the area of the distal musculotendinous junction. Table 1 shows the

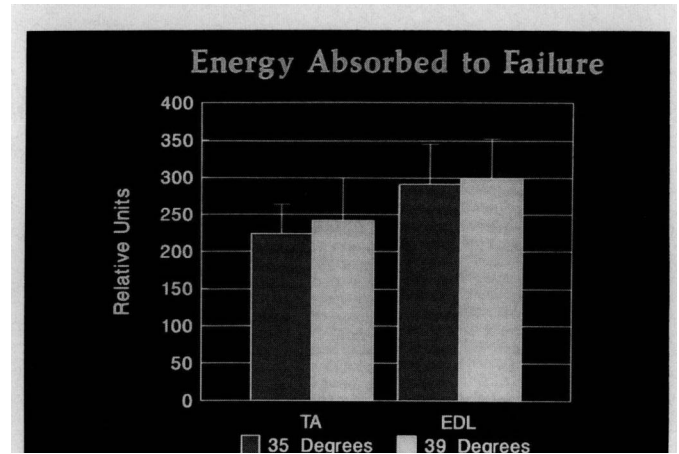


Figure 4. Average relative energy absorbed to failure by 35°C and 39°C TA and EDL muscles. All values are mean ± SD ($P = 10$).

35°C	224 ± 40	291 ± 54
39°C	242 ± 58	298 ± 54
NS		NS

TABLE 1
Average relative contribution of the weight of the distal musculotendinous fragment to the total weight for 35°C and 39°C TA and EDL muscles in five of the experimental animals

Hindlimb	Avg. % of total weight	Range (%)
Left (35°C)		
TA	11.4	4-25
EDL	11.8	5-23
Right (39°C)		
TA	9.0	2-23
EDL	18.4	7-32

average relative contribution of the distal fragments of the TA and EDL to the total weights of these muscles in the left (35°C) and right (39°C) hindlimbs of five of the experimental animals. In the left hindlimb the distal fragments averaged 11.4% (range, 4% to 25%) of the total muscle weight for the TA and 11.8% (range, 5% to 23%) for the EDL. In the right hindlimb the distal fragments averaged 9.0% (range, 3% to 23%) of the total muscle weight for the TA and 18.4% (range, 7% to 32%) for the EDL.

DISCUSSION

The results of our study support the concept that temperature elevation in muscle increases the extensibility of the musculotendinous unit. The passive tension exhibited by a muscle is frequently attributed primarily to the connective tissues by which it is attached to its tendon. It has also been suggested that structures within the sarcomere may be responsible for much of the resting tension, especially at shorter sarcomere lengths.⁷ As these structures have not been identified, and their physical properties investigated, the discussion of our results will be confined to the effects of heat on the more widely studied connective tissue elements of the musculotendinous unit. Before entering into

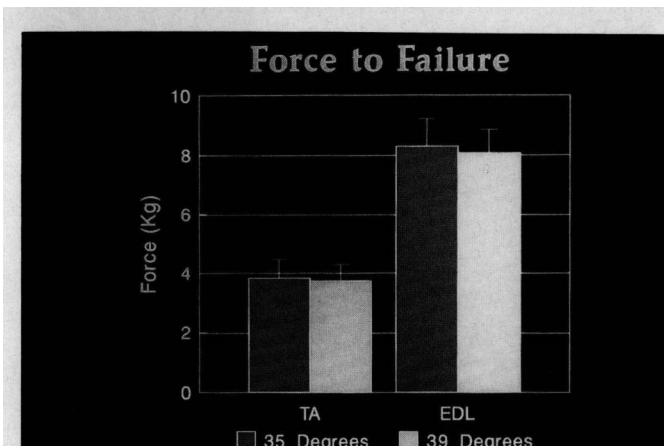


Figure 3. Average force to failure for 35°C and 39°C TA and EDL muscles. All values are mean ± SD ($N = 10$).

35°C	3.83 ± 0.64 kg	8.29 ± 0.92 kg
39°C	3.73 ± 0.56 kg	8.06 ± 0.80 kg

Difference:	0.10	0.23
	$P \leq 0.1$	$P \leq 0.05$

this discussion we would like to present the rationale used in selecting the temperatures to which the muscles were exposed.

The muscles in the left leg were heated in a 35°C bath to simulate their approximate temperature in an intact limb at rest. A series of thermoprobe measurements beneath the skin covering the TA and EDL muscles prior to the skin incision revealed a nearly constant temperature of 33.5°C. The thermoprobe was not inserted into either the TA or EDL muscle bellies for direct measurement because we felt the resulting injury to the muscle would alter its physical properties in an unpredictable way. Lehmann et al.⁶ presented information on the temperature distribution in the human thigh which showed a 3°C difference in temperature between the skin surface and tissues at a depth of 1.0 cm. We assumed the difference in temperature between the subcutaneous tissues and the TA and EDL muscles at a depth of about 1.0 cm to be about half of this value, or 1.5°C. Our estimate of the TA and EDL muscles temperatures at rest was, thus, 33.5°C + 1.5°C, or 35°C.

The temperature to which the right leg muscles were exposed (39°C) was chosen to approximate the body temperature of the New Zealand White rabbit, which ranges from 38.6°C to 40.1°C.¹⁵ Lehmann et al.⁶ showed that the application of an external hot pack at 48°C to the human thigh did not cause the tissues at a depth of 1 cm to reach body temperature until 10 minutes had elapsed. Thus, by immersing the muscles in a 39°C bath we hoped to mimic the warming effects of an externally applied hotpack on an intact limb while keeping the muscles available for immediate testing when the warming period ended.

It has been shown in a number of studies that dense connective tissue, composed primarily of collagen fibers, becomes more extensible as its temperature is increased within normal physiologic/therapeutic limits.^{2, 5, 9, 13, 17} La-Ban⁴ showed that canine calcaneal tendon subjected to a constant stress demonstrated an initial elongation beyond its resting length of 5.9% at 37°C, 6.7% at 39.8°C, and 7.6% at 42.5°C. He suggested that this response may have been due more to the properties of the ground substance of the tendon than to the collagen. Warren et al.¹⁴ studied the effects of temperature on rat tail tendon extensibility, concluding that the viscoelastic response of this tissue increases with temperature increase. As tendons under identical stress were warmed in a series of experiments in which temperatures ranged from 39°C to 45°C, the length of time required to reach 2.6% strain decreased significantly, indicating a marked increase in extensibility in the warmer tendons. The higher temperatures were also associated with lower levels of tissue damage. Safran et al.¹⁰ suggested that temperature increase within muscles due to active contraction may have an effect on the extensibility and force-resisting ability of the connective tissues that the muscles contain. It was found that muscles which had been preconditioned with a tetanic contraction, and consequently warmed an average of 1°C, attained greater increases in length and force when pulled to failure than did muscles which were not preconditioned.

Our study supports the findings of these authors concerning the effects of increased temperature on connective tissue extensibility.

The effect of heating muscles to 39°C on maximum force obtained before rupture is shown in Figure 3. In both muscles warming caused a decrease in maximum force, but this difference was significant for only the EDL. The amount of energy absorbed was higher for warmed muscles, but this difference was not significant. The energy absorbed considered as the area under the force-length curve as muscle is pulled to failure, should be sensitive to both the maximum force and length of muscle pulled to failure. Although the length is increased in both muscles, the force is decreased. These effects combined to make energy absorption differences insignificant between the two groups. There was a trend for more energy absorption in both warmed muscles.

Although all of the failures occurred in the vicinity of the distal myotendinous junction, there was quite a bit of variability in the amount of muscle that remained with the distal tendon (Table 1). This phenomenon was thus observed and evaluated during testing on the last five rabbits. The low figures in the range measurements represent tendon with little or no attached muscle, and these values were subtracted from the high figures to give an estimate of the amount of muscle contained in the distal fragment. Thus, for the TA from 0% to 21% of the total muscle tissue was attached to the tendon, while for the EDL the weight of the attached muscle was from 0% to 25% of the total muscle weight. No trends in these values may be identified due to the small sample size and variability among specimens, although the TA generally left less muscle tissue with the distal tendon than did the EDL. Also, the 39°C EDL left relatively more muscle with the distal tendon than did the 35°C EDL. Usually the pattern seen on the left leg was also found on the right, suggesting that a component of individual muscular or connective tissue variation may be involved.

There is considerable interest among sports medicine personnel in understanding factors that may relate to the susceptibility of muscle to injury. It has often been stated that cold muscles may be more susceptible to stretch injury than warm muscles.^{8, 10, 11, 16} Accordingly, athletes are often encouraged or taught to warm up prior to high intensity athletic participation. There are really very few scientific studies which bear on the effects of temperatures on the susceptibility of muscle to injury.

It is known that internal temperature of the muscle affects the ability of the muscle to generate active force.³ Warm muscles generate more force than do cold muscles. However, little is written regarding temperature and susceptibility of muscles to strain injury. Safran et al.¹⁰ have shown that a single isometric muscle contraction can alter the amount of stretch a muscle can withstand prior to failure. The single contraction resulted in a warming of the muscle by 1°C and, therefore, the altered biomechanics may have been influenced by temperature. However, muscle activation was also involved and other factors, such as metabolic or viscoelastic influences, may have affected the muscle.

These experiments have attempted to investigate the temperature effect alone. These experiments suggest that protection of a muscle from strain injury may be afforded by the increased length to which a muscle can stretch before causing injury. However, other factors may well be present in physiologic settings. For example, since muscle generates more force at a warm temperature than at a colder temperature, it is possible that activated warm muscle is better adapted to absorb more energy than cold muscle when stretched to failure.² It has been shown that differences exist in energy absorption and energy strain to failure and this may be dependent on the degree of activation and force production of the muscle. If warm muscle can generate more force while being stretched, it may well allow for increased energy absorption.

CONCLUSIONS

The susceptibility of muscle to strain injury has been investigated in passively stretched muscles maintained at different temperatures. Elevation of the muscle temperature by 4° (from 35°C to 39°C) for a brief period increases the amount of elongation which can occur without rupture. The maximum force developed was significantly higher for the cold EDL but not for the TA muscle. Energy absorption was not different between the warm and cold muscles. These data show that warmed muscles are more extensible than cold muscles. The increased extensibility could afford some protection from strain injury for warmed muscles.

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DISCUSSION

Thomas L. Wickiewicz, MD, New York, New York: The authors have put forth in their paper the premise that passive warming would in some way reduce the potential for muscle injury. They have attempted to put science into a common training technique based primarily on tradition. But before the practitioner interprets this data and tries to make the jump to human training techniques, let us look carefully at their results.

Although the muscles tested did show increased extensibility in response to warming, a result the authors attribute to the connective tissue elements, there was no ability of the muscle to absorb more energy to any significant degree. Nor was there a strong statistical significant difference in the force to failure difference between the two temperatures. The two test temperatures chosen were to a large extent arbitrary, reflecting rest and normal core temperature of the rabbit. Although I agree with their desire not to damage the muscle with the temperature probe, I think there is a need to better define, in this model, whether the temperatures that they selected are appropriate. Larger differences may be seen if the muscles are warmed to temperatures that can be seen during physiologic activity. The protective effects of sarcomere activation prior to stretch are more complex than just intrinsic warming of the muscle.

The authors are encouraged to contribute further with this interesting and important work.