

Exercise-induced modulation of skeletal muscle force

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Unil

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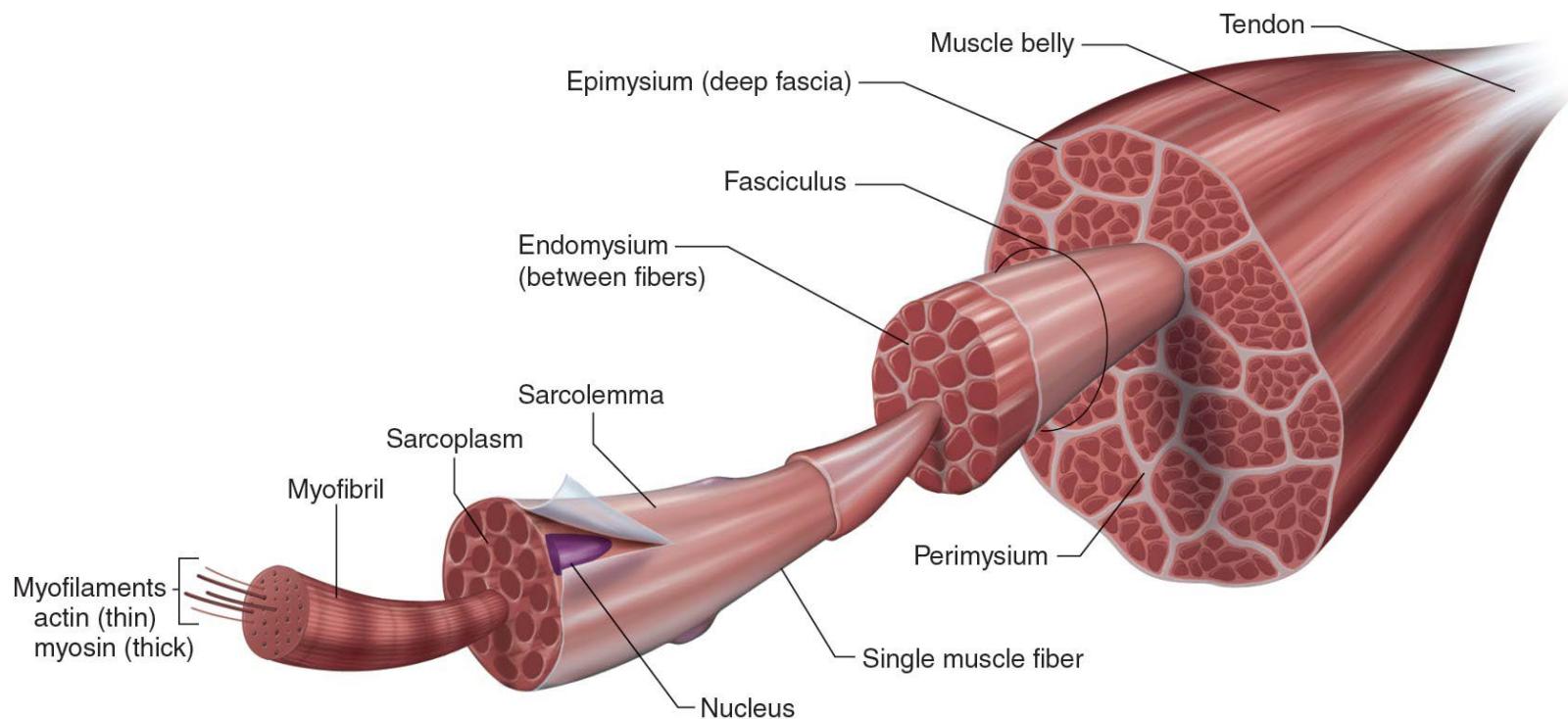
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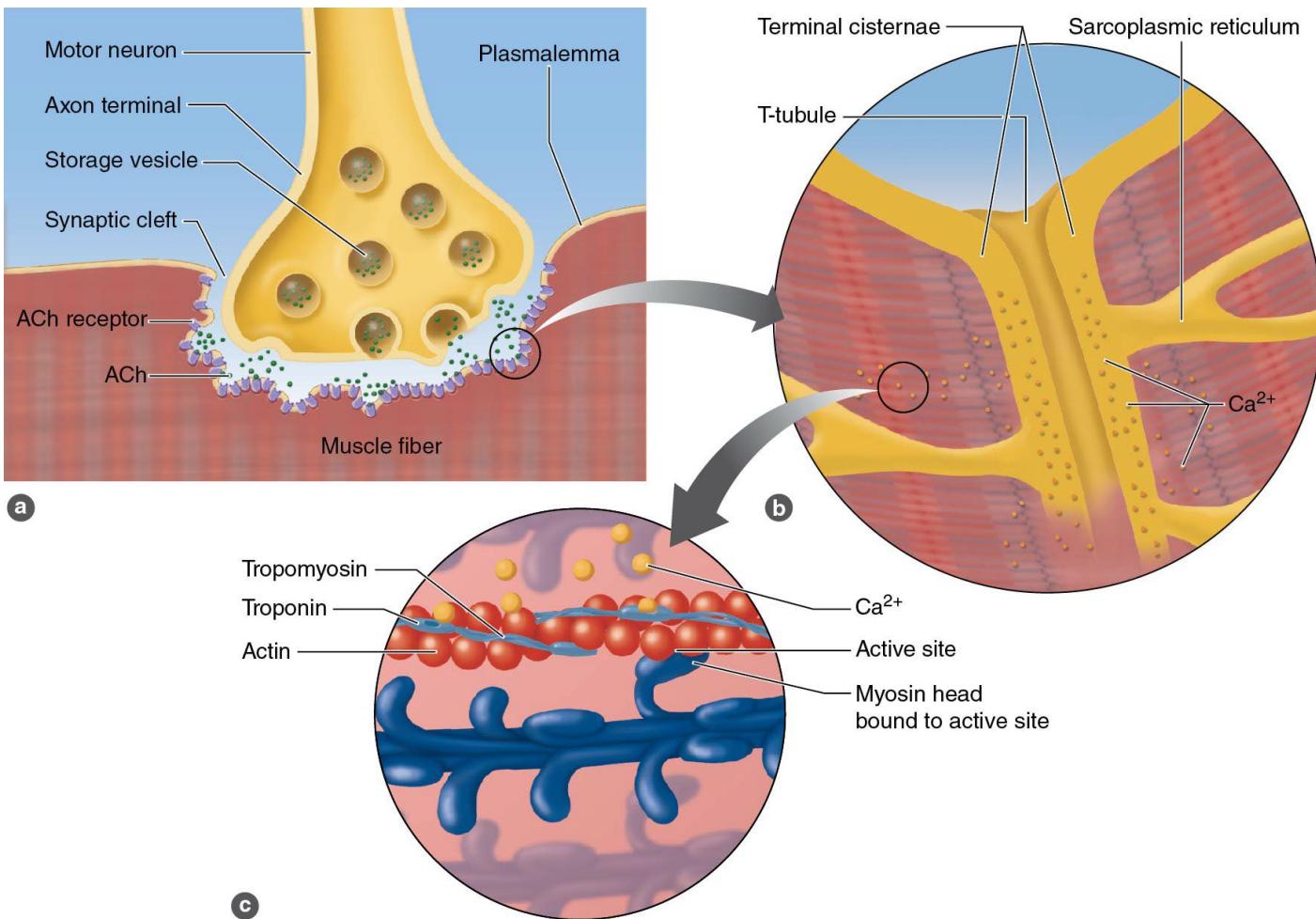
Our program today

- Different types of muscle fibres
- Motor unit properties
- Force generation and measurement
- Modulation of muscle force
 - Acute adaptations (fatigue)
 - Chronic adaptations (resistance training)

Skeletal muscle



Muscle contraction



Different muscle fibre types in humans

TABLE 1.1 Classification of Muscle Fiber Types

FIBER CLASSIFICATION			
System 1 (preferred)	Type I	Type IIa	Type IIx
System 2	Slow-twitch (ST)	Fast-twitch a (FTa)	Fast-twitch x (FTx)
System 3	Slow oxidative (SO)	Fast oxidative/glycolytic (FOG)	Fast glycolytic (FG)
CHARACTERISTICS OF FIBER TYPES			
Oxidative capacity	High	Moderately high	Low
Glycolytic capacity	Low	High	Highest
Contractile speed	Slow	Fast	Fast
Fatigue resistance	High	Moderate	Low
Motor unit strength	Low	High	High

TABLE 1.2 Structural and Functional Characteristics of Muscle Fiber Types

Characteristic	FIBER TYPE		
	Type I	Type IIa	Type IIx
Fibers per motor neuron	≤300	≥300	≤300
Motor neuron size	Smaller	Larger	Larger
Motor neuron conduction velocity	Slower	Faster	Faster
Contraction speed (ms)	110	50	50
Type of myosin ATPase	Slow	Fast	Fast
Sarcoplasmic reticulum development	Low	High	High

Adapted from Close, 1967.

Physical activity & Muscle fibre type

- Most muscles contain both type I and type II muscle fibres.
- The relative distribution depends on genes, muscle function and training level
- Sprinters have more type II muscle fibres
- Endurance sports = more type I
- Only few changes in fibre type with training (< 10%?)
- Preferential loss in type II muscle fibres in ageing

Physical activity & Muscle fibre type

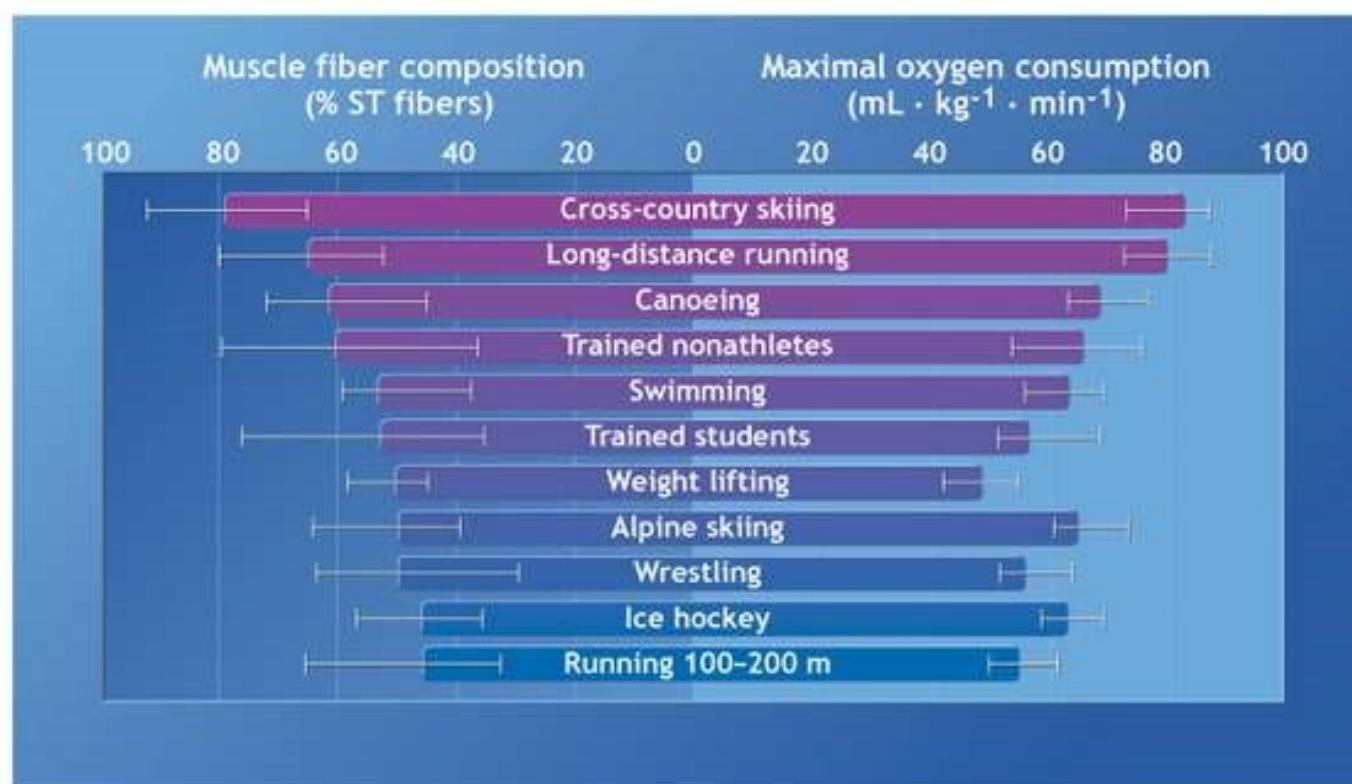


Figure 18.17. Muscle fiber composition (% slow-twitch fibers, *left side*) and maximal oxygen consumption (*right side*) in athletes representing different sports. The outer, white bars denote the range. (From Bergh U, et al. Maximal oxygen uptake and muscle fiber types in trained and untrained humans. *Med Sci Sports* 1978;10:151.)

How to determine muscle fibre type?



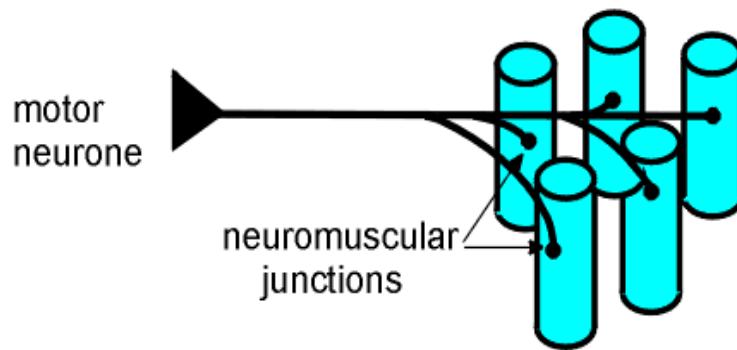
Courtesy of the authors.



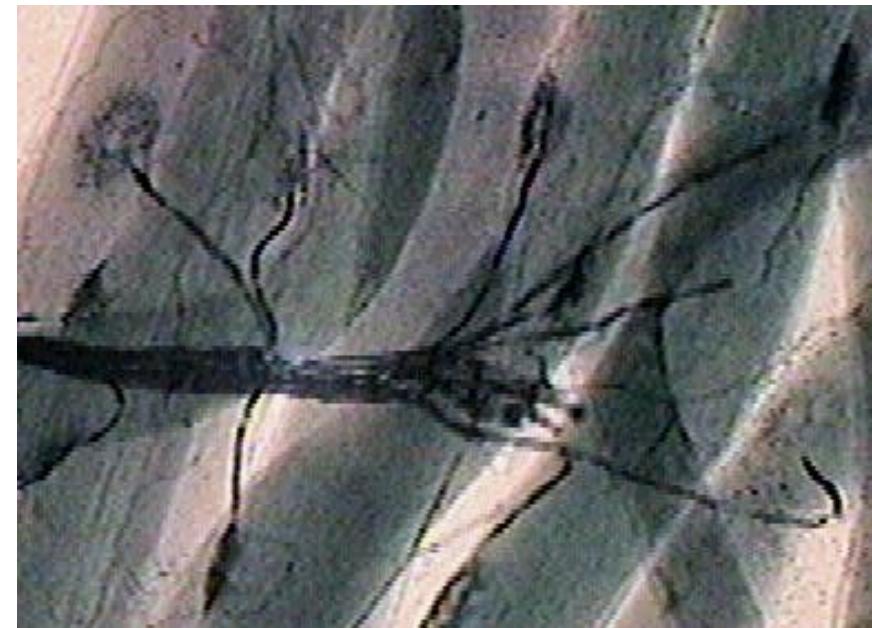
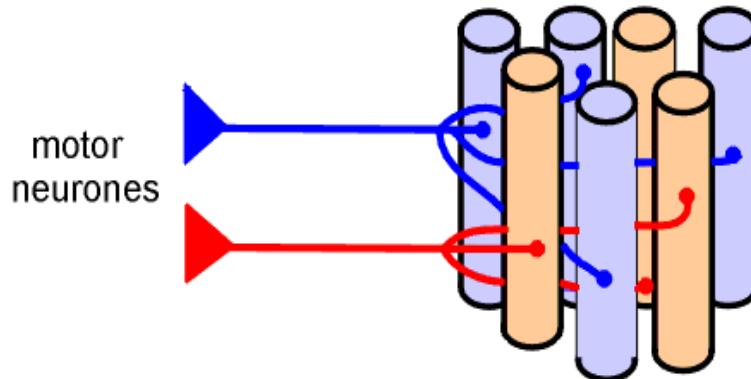
Courtesy of the authors.

Motor units

Single motor unit



Two motor units



Motor units & fibre type

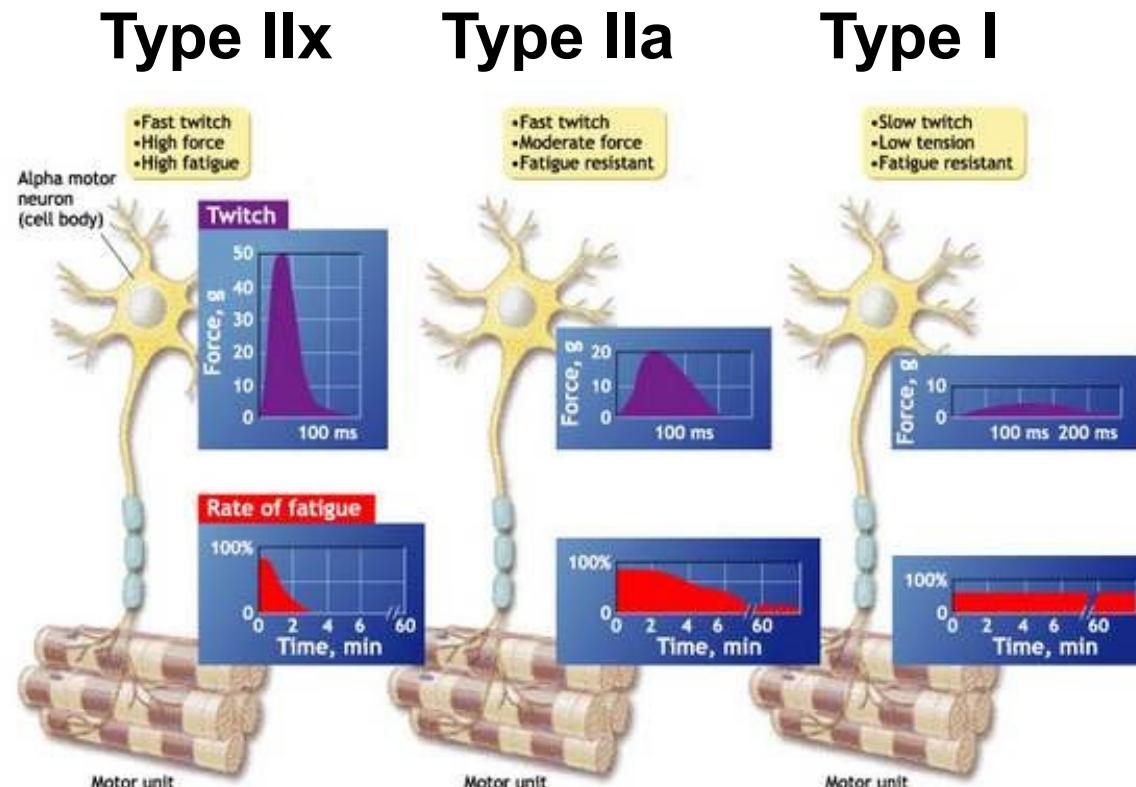


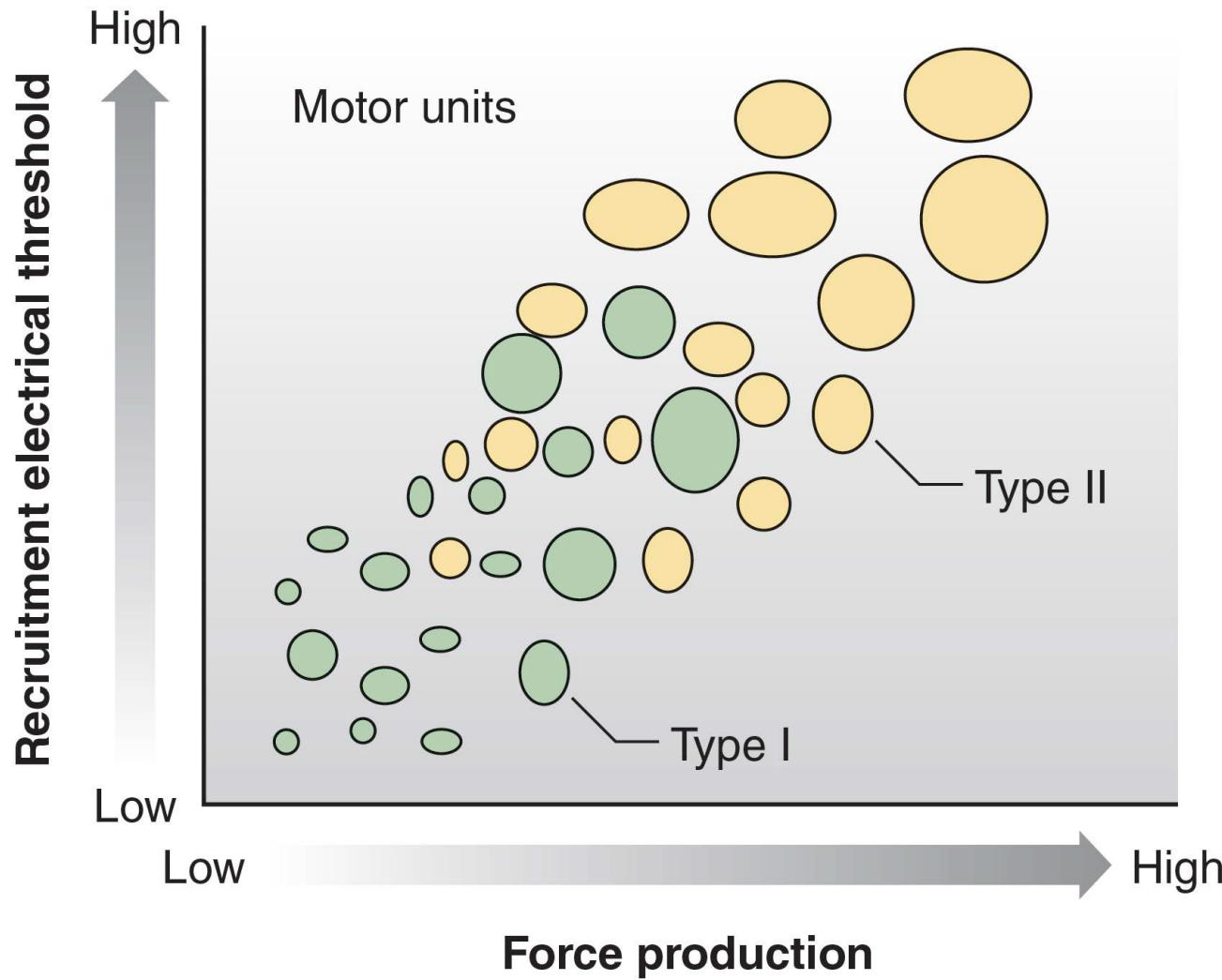
Figure 19.13. Speed, force, and fatigue characteristics of motor units. "Phasic" motor neurons fire rapidly with short bursts; "tonic" motor neurons fire slowly but continuously.

Force modulation – motor unit recruitment

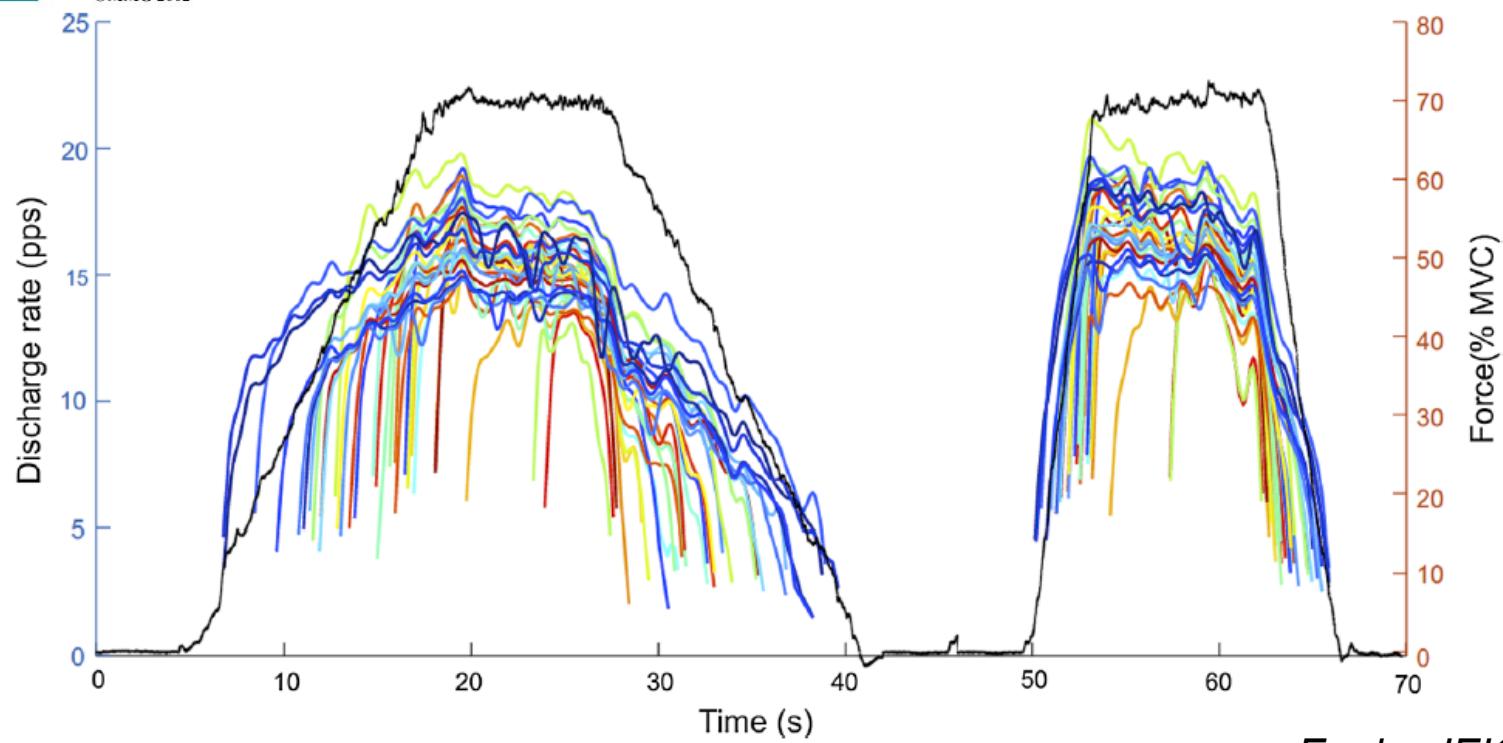
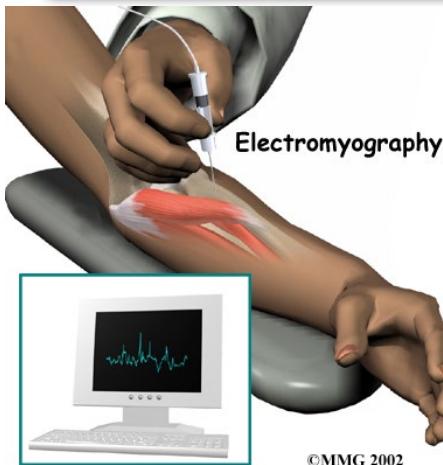
How is muscle force regulated ?

- Spatial recruitment (more motor units)
→ ‘size principle’ (*Henneman, 1965*)
- ‘All or Nothing principle’
- Temporal recruitment (change in MU firing rate)

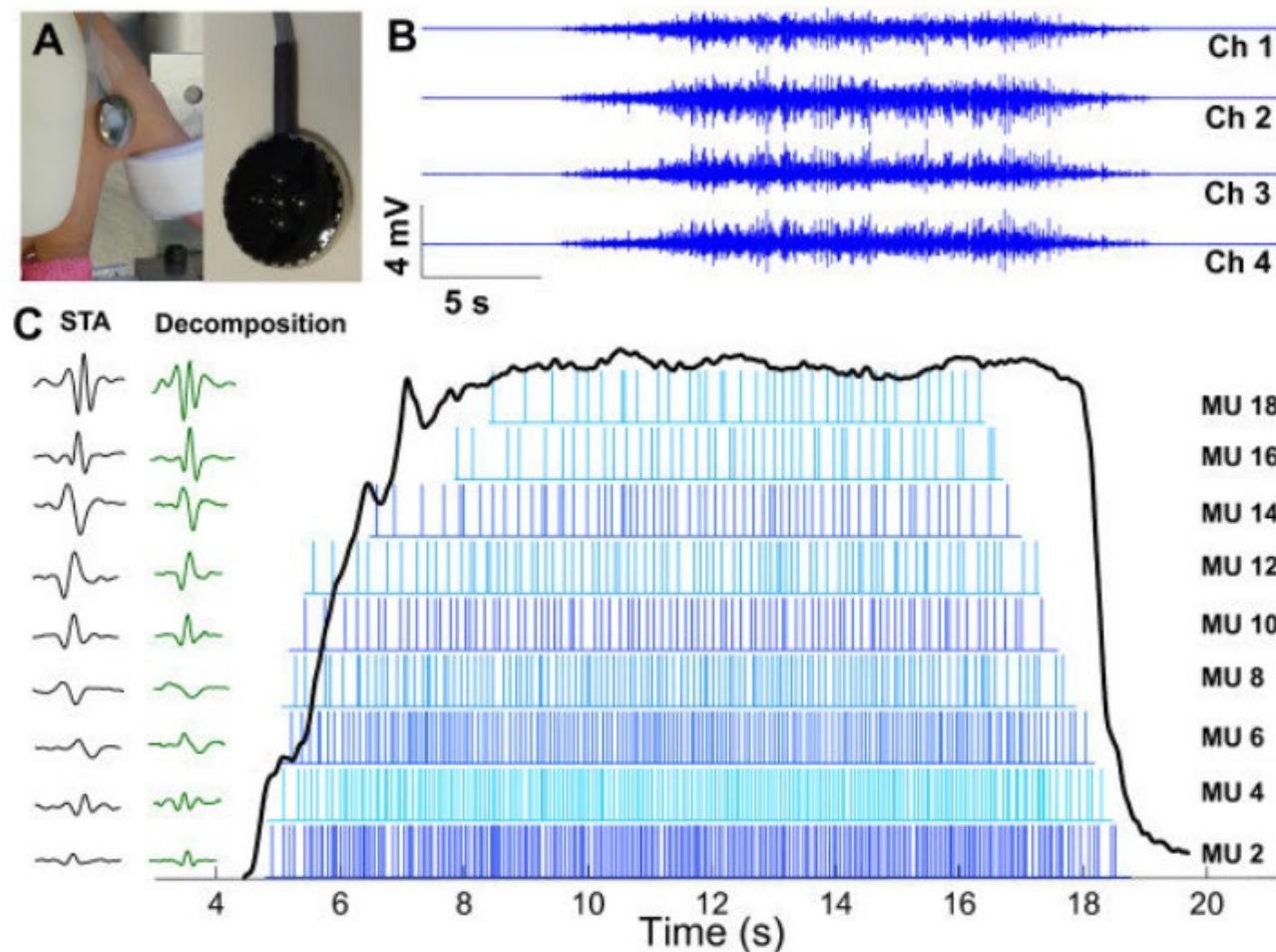
Illustration of the size principle



MU firing frequency



MU firing frequency



Measurement of muscle performance

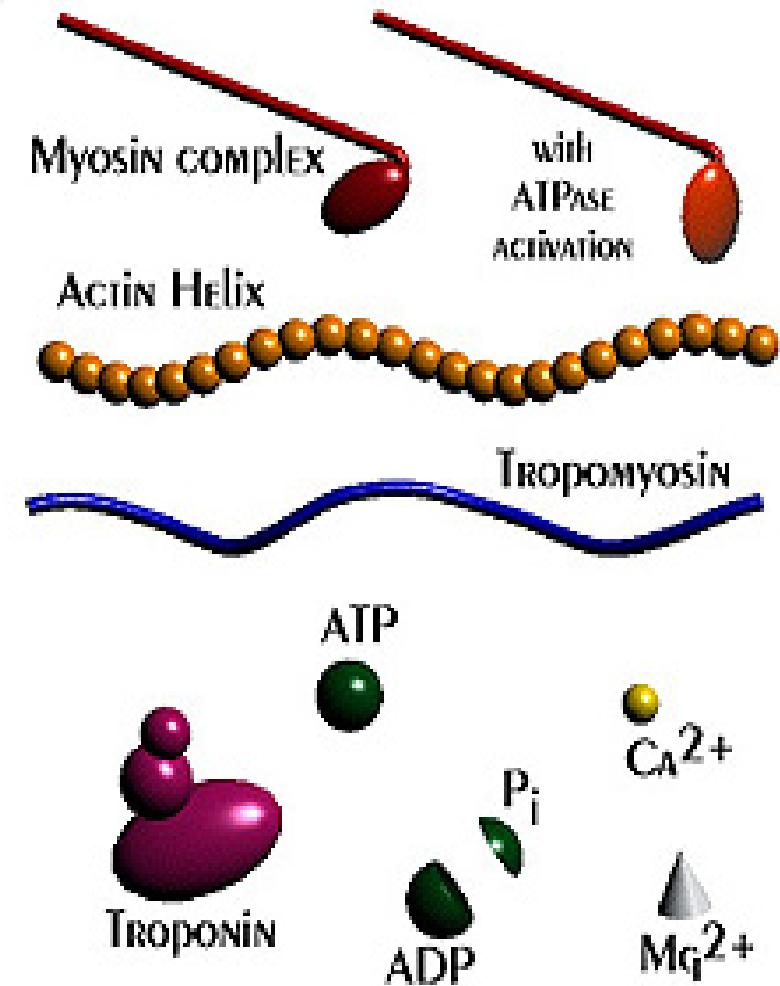
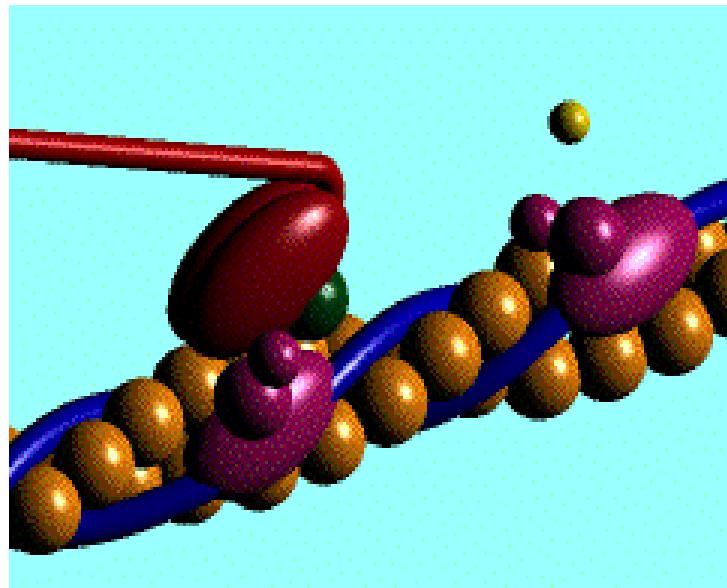
Force — maximal load that a muscle / muscle group can produce

Power — the product of force and movement speed
→ essential in sport

Muscular endurance — ability to sustain (i) repeated muscle actions or (ii) a submaximal isometric contraction

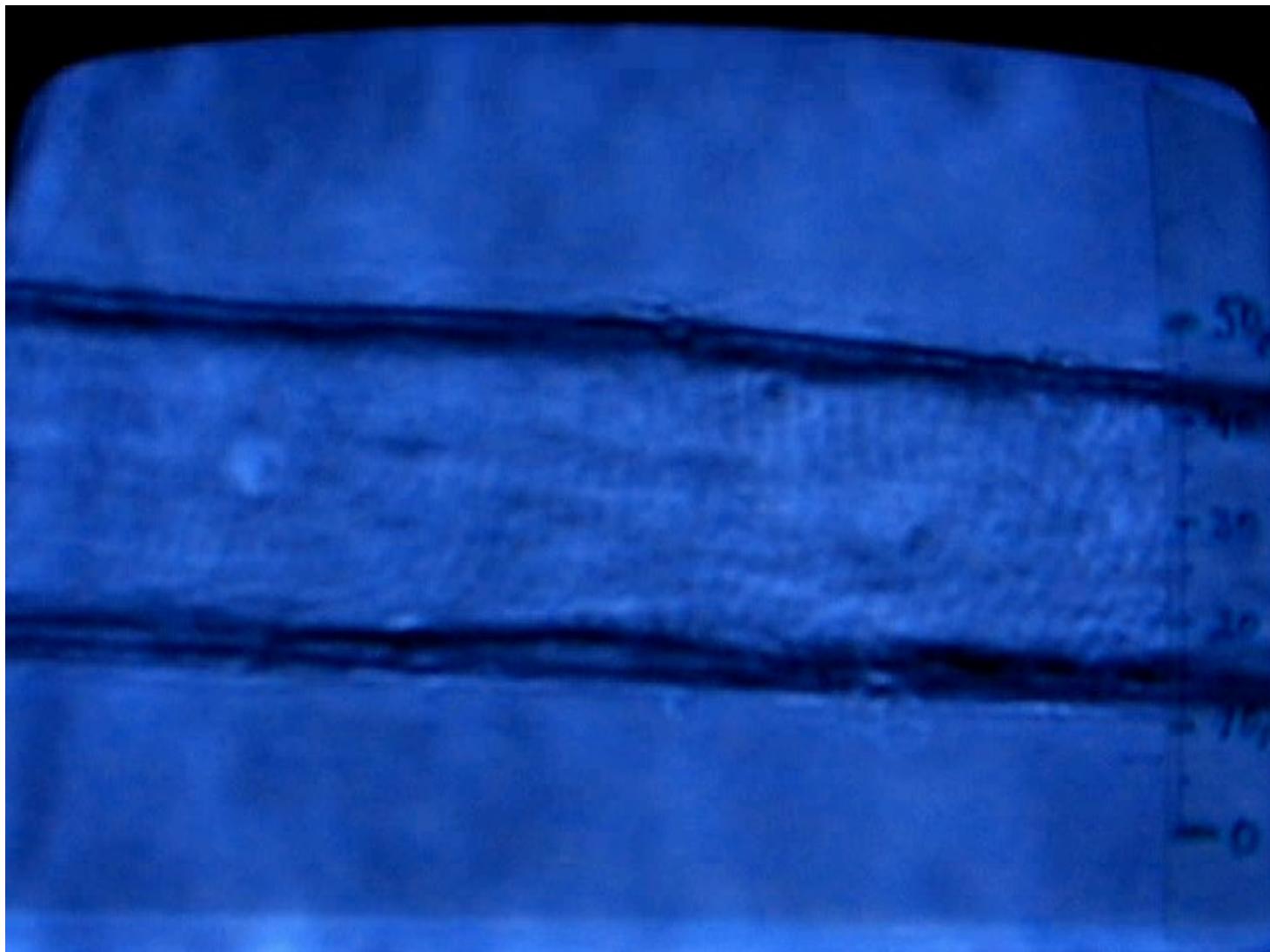


How force is generated ?



Source: UCSD

Mouse single FDB fibre, 70 Hz tetani



How can we measure muscle force in humans?

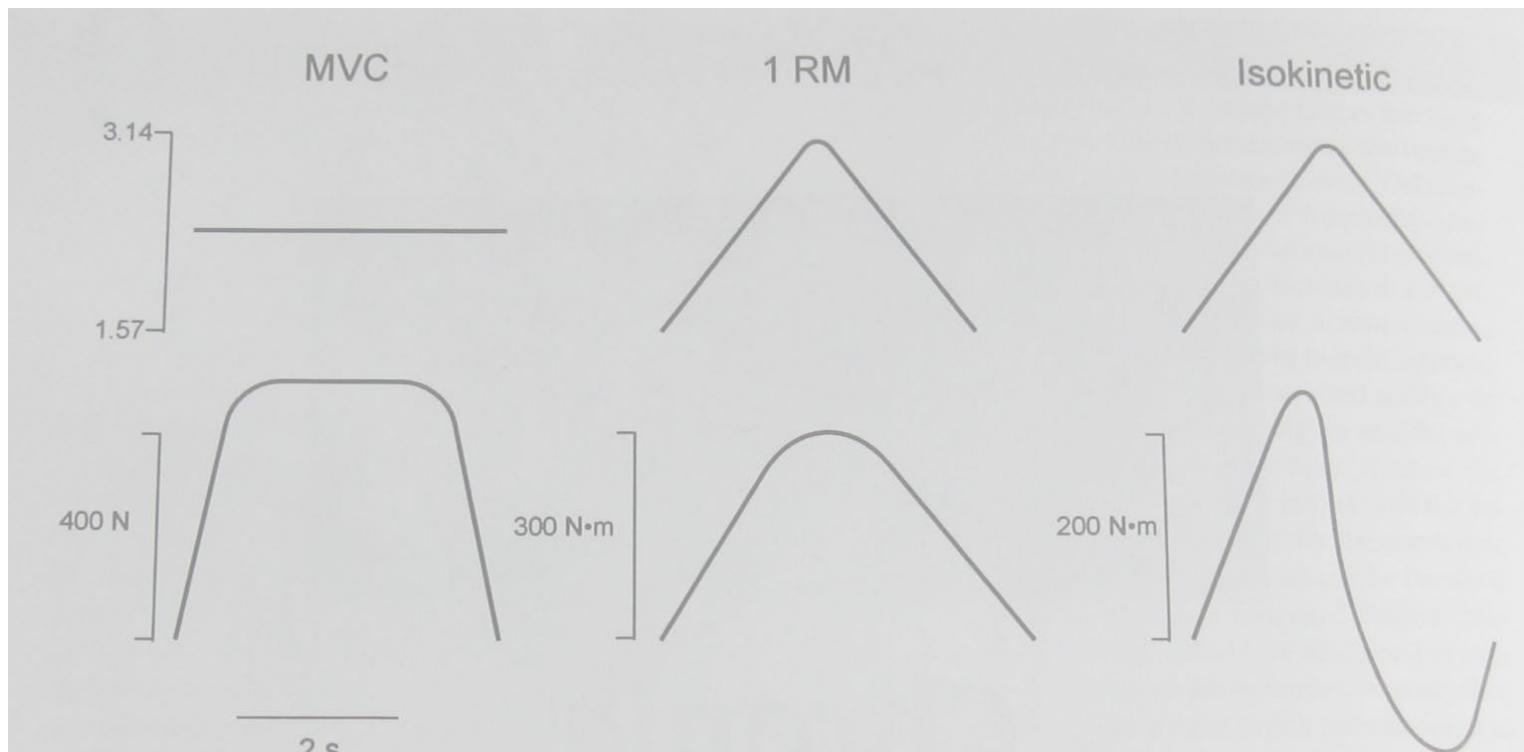


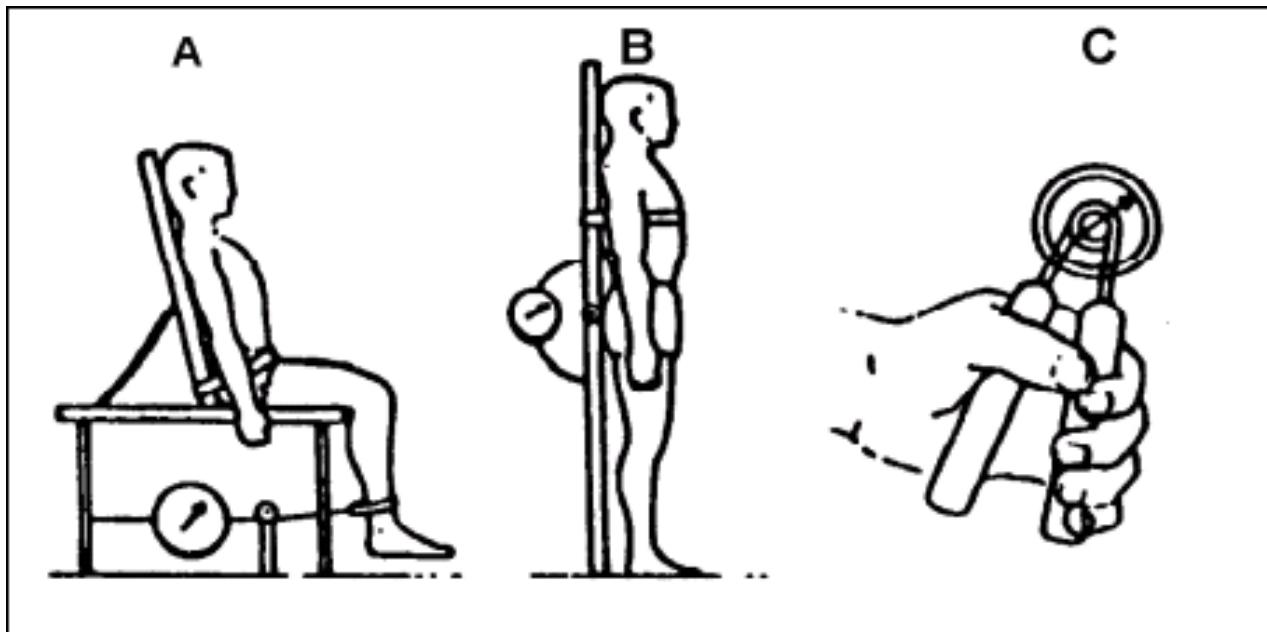
Figure 9.1 Idealized performances of a maximum voluntary contraction (MVC), a one repetition-maximum (1-RM) lift, and an isokinetic contraction with the knee extensor muscles. The upper row indicates knee angle (3.14 rad = complete extension). The lower row shows force for the MVC but the resultant muscle torque for the 1-RM and isokinetic tasks.

Force transducer,
Isometric ergometer

Free weights

Isokinetic
Ergometer

Isometric dynamometer



Measurement of maximal voluntary contraction force (MVC)

Isometric dynamometer



Measurement of maximal voluntary contraction force (MVC)

Free weights



- Usually 1-RM (concentric action) is considered as the index of muscle force
- Maximal weight that can be lifted only once

Isokinetic ergometer

Measures joint torque in isometric, concentric or eccentric condition

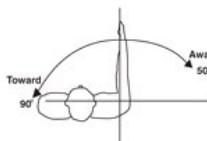


→ Predetermined constant angular velocity

→ Torque produced by the engine (measured) is the opposite of the one developed by the subject

SHOULDER

EXTERNAL/INTERNAL ROTATION
(MODIFIED NEUTRAL POSITION)



Axis alignment is longitudinal through the head of the shaft of the humerus in a horizontal plane.

AWAY: External Rotation
TOWARD: Internal Rotation
READY POSITION: Full Internal Rotation



DYNAMOMETER ORIENTATION: 20°

DYNAMOMETER TILT: 50°

ATTACHMENT: ELBOW/SHOULDER ATTACHMENT WITH CUFF

SEAT ORIENTATION: 15°

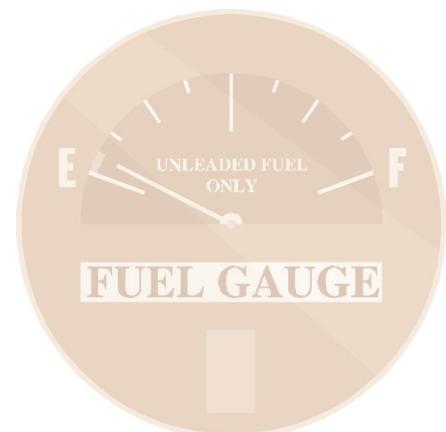
Alteration of the force generating capacity

I – Acute: Fatigue

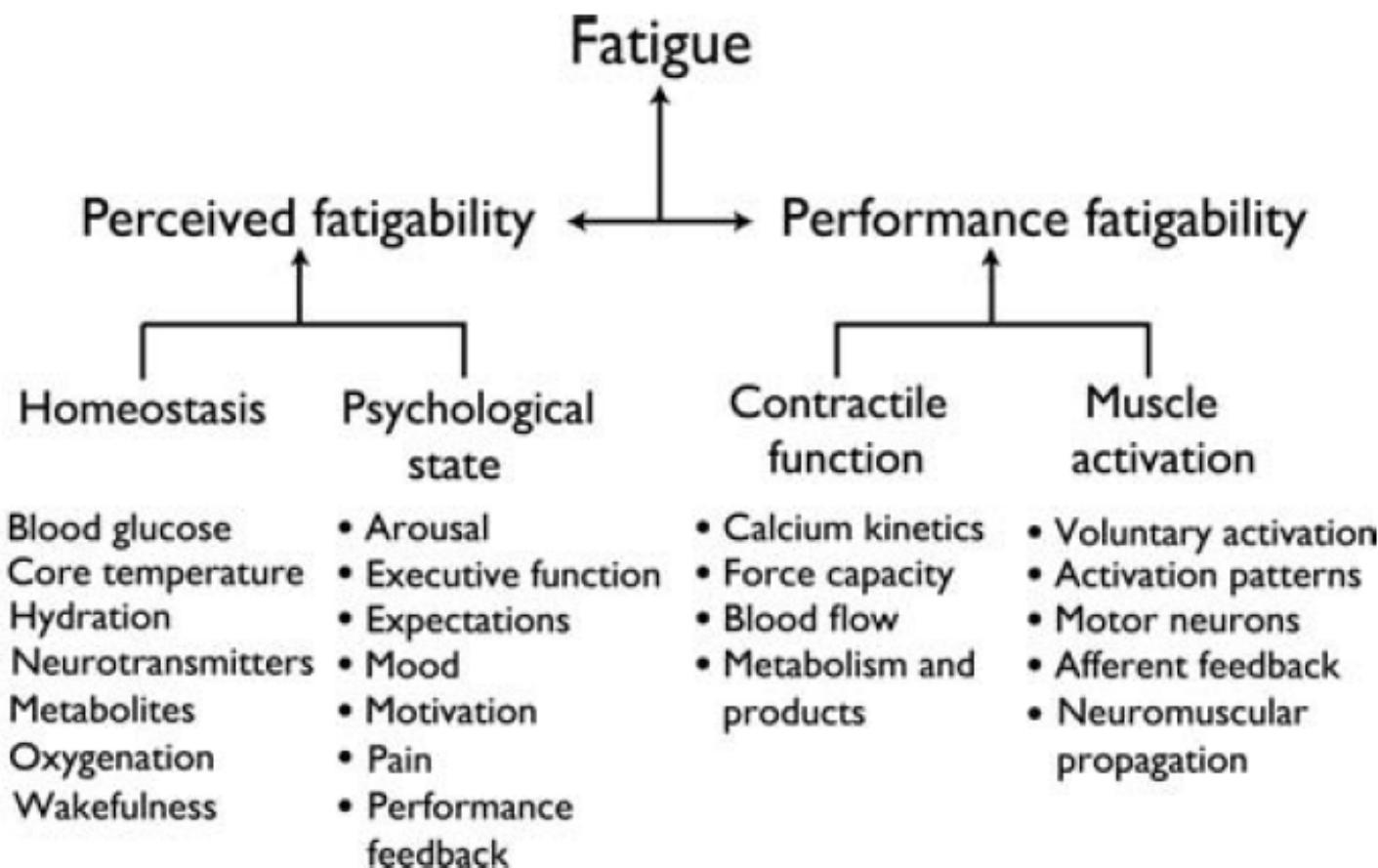
II- Chronic: Strength training

I- Muscle fatigue

- Inability to sustain muscular work/force/power to a given intensity
- Reduction in the maximal force generating capacity (MVC)
(*Gandevia 2001*)



Muscle fatigue: definition



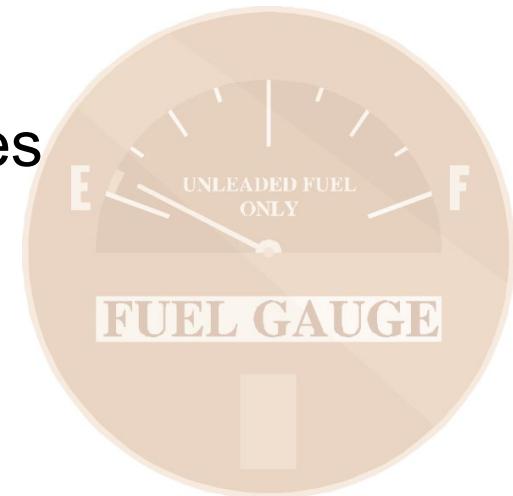
Different models to assess muscle fatigue

Table 1. Advantages and disadvantages of various approaches to the study of fatigue

	Advantages	Disadvantages
Muscle in vivo 	All physiological mechanisms present Fatigue can be central or peripheral All types of fatigue can be studied Stimulation patterns appropriate for fiber types and stage of fatigue	Mixture of fiber types Complex activation patterns Produces correlative data; hard to identify mechanisms Experimental interventions very limited
Isolated muscle 	Central fatigue eliminated Dissection simple	Mixture of fiber types Inevitable extracellular gradients of O ₂ , CO ₂ , K ⁺ , lactic acid Mechanisms of fatigue biased by presence of extracellular gradients Drugs cannot be applied rapidly because of diffusion gradients
Isolated single fiber 	Only one fiber type present Force and other changes (ionic, metabolic) can be unequivocally correlated Fluorescent measurements of ions, metabolites, membrane potential, etc. possible Easy and rapid application of extracellular drugs, ions, metabolites, etc.	Dissection difficult Environment different to in vivo K ⁺ accumulation and other in vivo changes absent Prone to damage at physiological temperatures Small size makes analysis of metabolites difficult
Skinned fiber 	Precise solutions can be applied Possible to study myofibrillar properties, SR release and uptake, AP/Ca ²⁺ release coupling Metabolic and ionic changes associated with fatigue can be studied in isolation	Relevance to fatigue can be questionable May lose important intracellular constituents Relevant metabolites to study must be identified in other systems

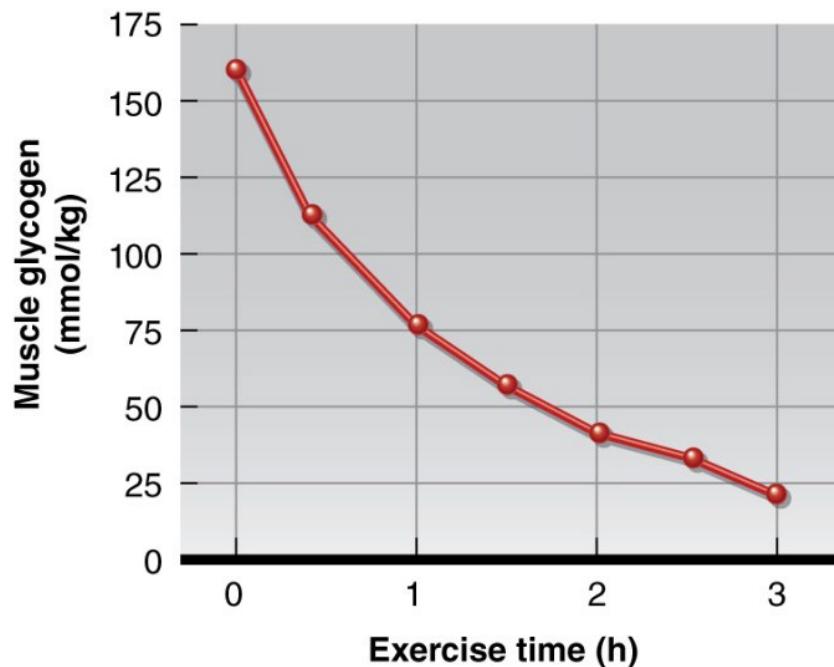
Causes of muscle fatigue

- Phosphocreatine (PCr) depletion
- Glycogen depletion (especially for activities lasting > 30-60 min)
- Accumulation of metabolites (Pi, ROS) → E-C coupling alteration
- Neural and contractile processes
- Decreased motivation, cognitive processes

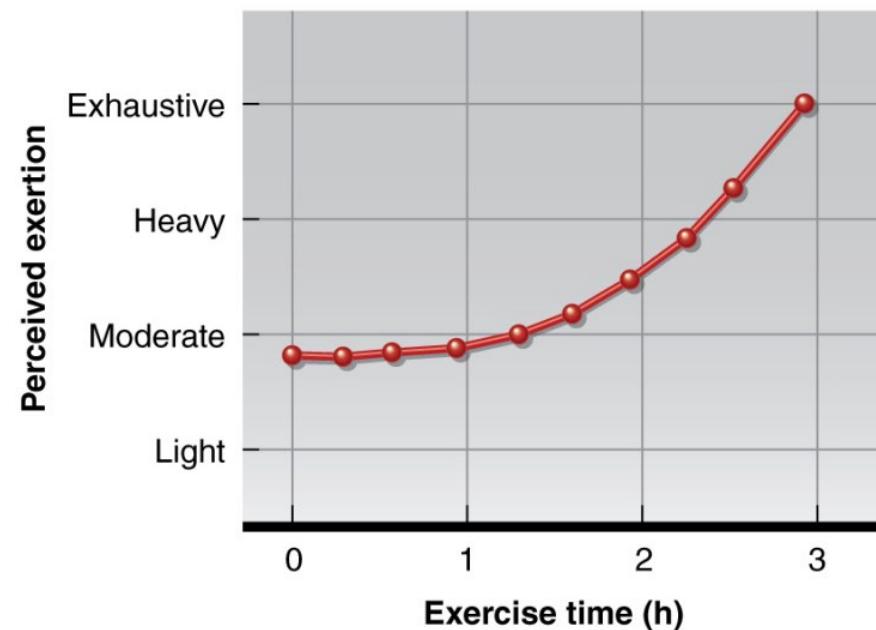


Muscle glycogen utilization during exercise

3h 70% $\dot{V}O_2\text{max}$ on a treadmill



a



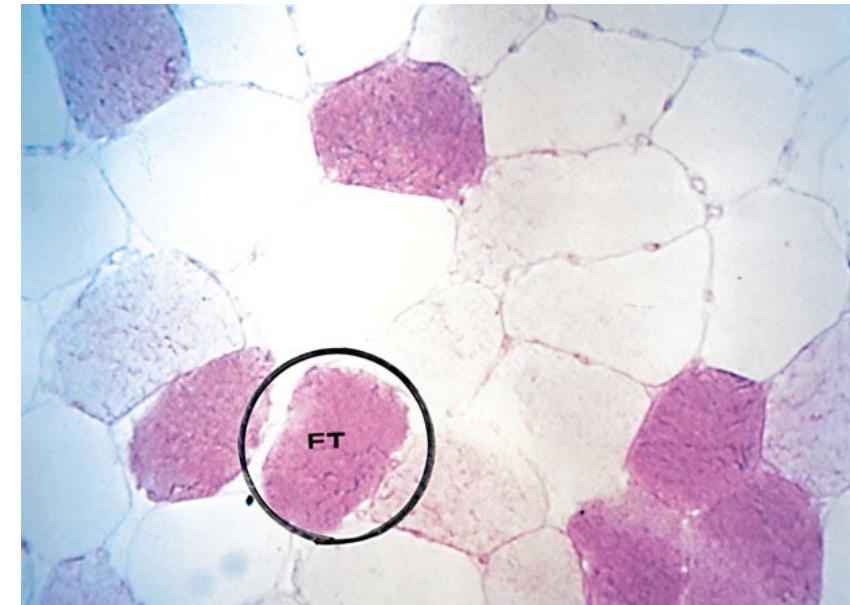
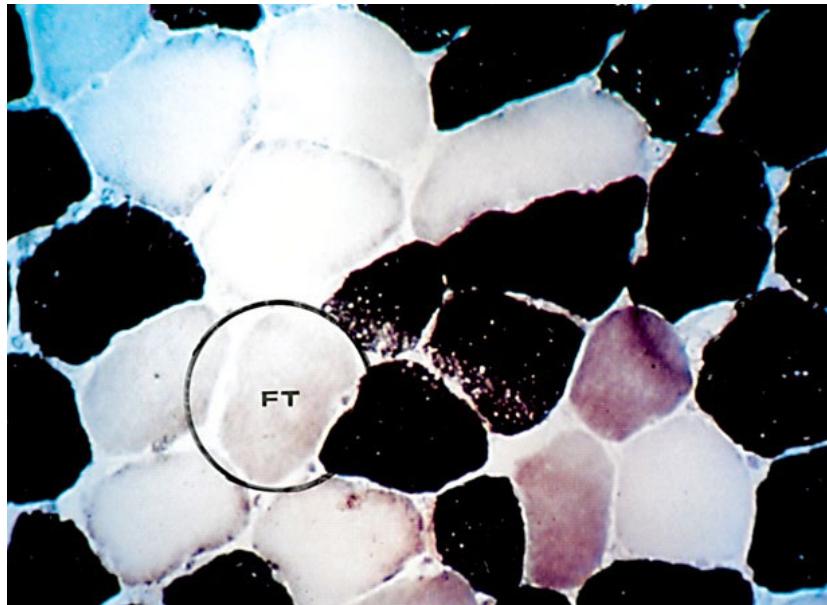
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Adapted by permission from Costill 1986.

Glycogen depletion \rightarrow increased RPE despite a constant speed

Glycogen depletion & fibre type

30-km race

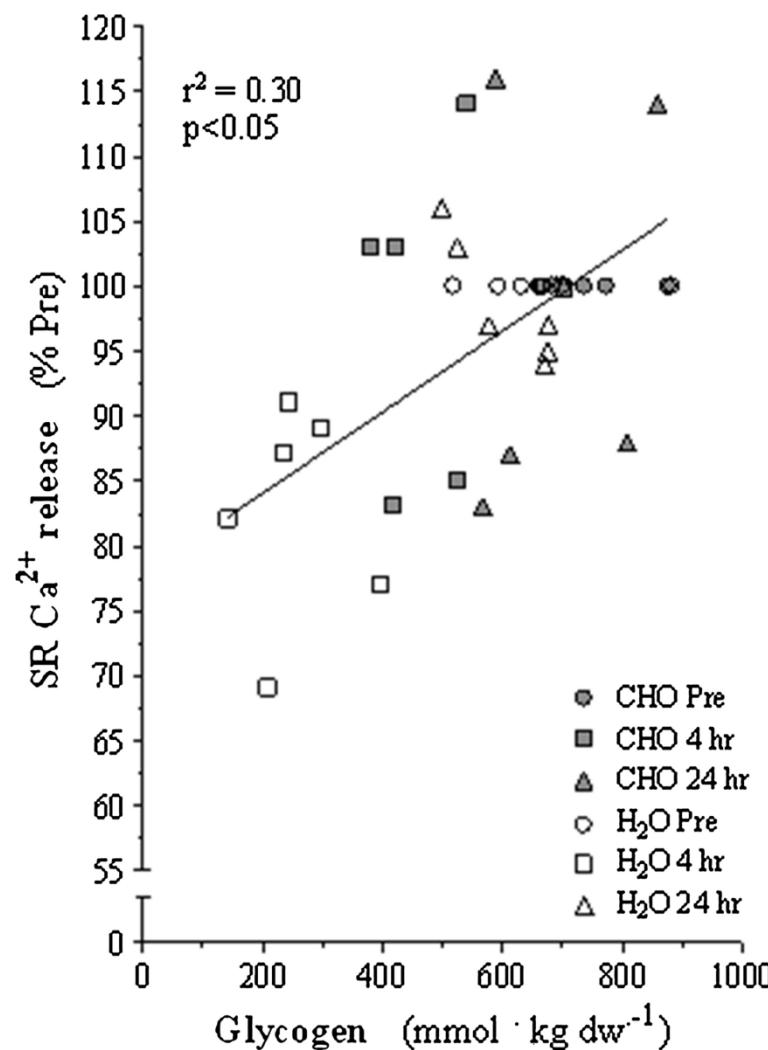


(Histochemical staining for muscle glycogen)

Glycogen content and Ca^{2+} release

Muscle Glycogen Content Modifies SR Ca^{2+} Release Rate in Elite Endurance Athletes

KASPER DEGN GEJL¹, LARS GRØNDAHL HVID¹, ULRIK FRANDSEN¹, KURT JENSEN^{1,2},
KENT SAHLIN³, and NIELS ØRTENBLAD^{1,2}

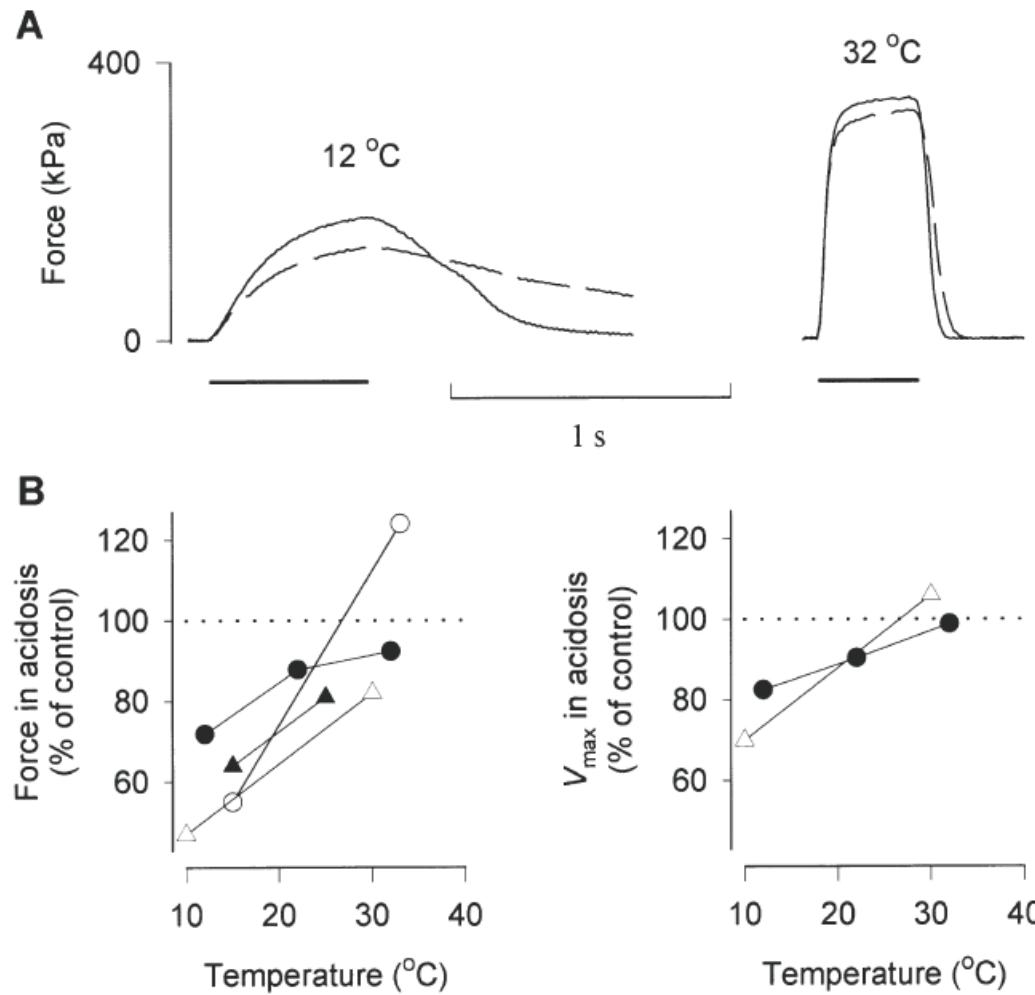


Metabolites & fatigue

- Short and intense activities rely on anaerobic glycolysis and lead to production of lactate and protons (H^+).
- Lactate can be oxidized in type I muscle fibres or in the heart, and/or converted to glucose by the liver (neoglucogenesis)
- Cells buffer H^+ with bicarbonate (HCO_3^-) to maintain pH between 6.4 and 7.1.
- If the pH value reaches 6.4, H^+ ions would stop glycolysis. The contribution of H^+ in muscle fatigue is debated.
- Pi (ATP by-product) might precipitate with Ca^{2+} ions and limit Ca^{2+} release from the SR.
- Reactive oxygen species (ROS) can also influence Ca^{2+} release and contractile protein sensitivity to Ca^{2+} .



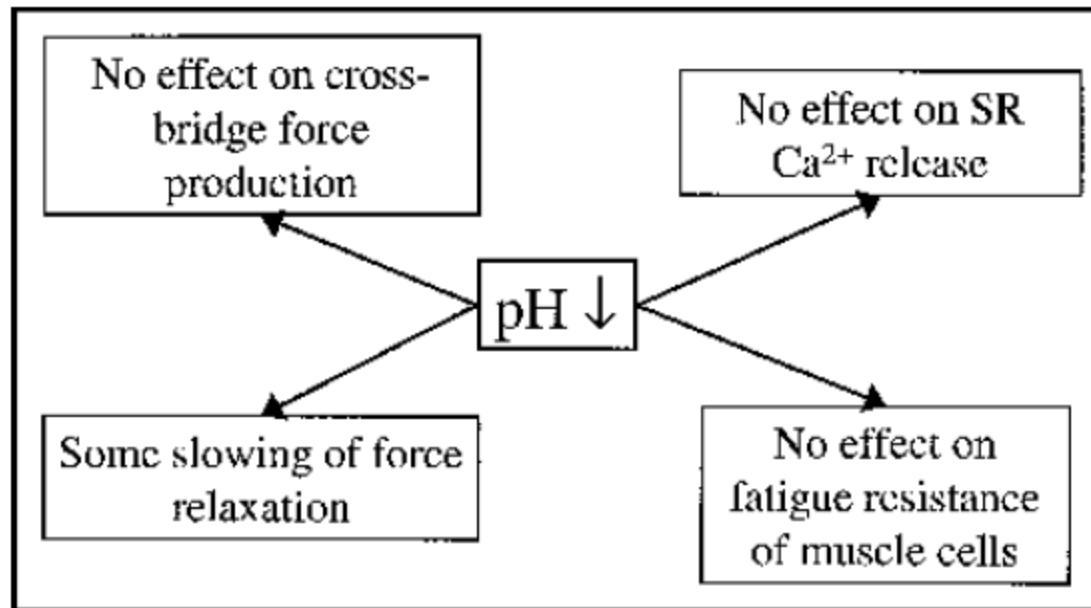
Acidosis & contractile properties



Westerblad et al. 2002

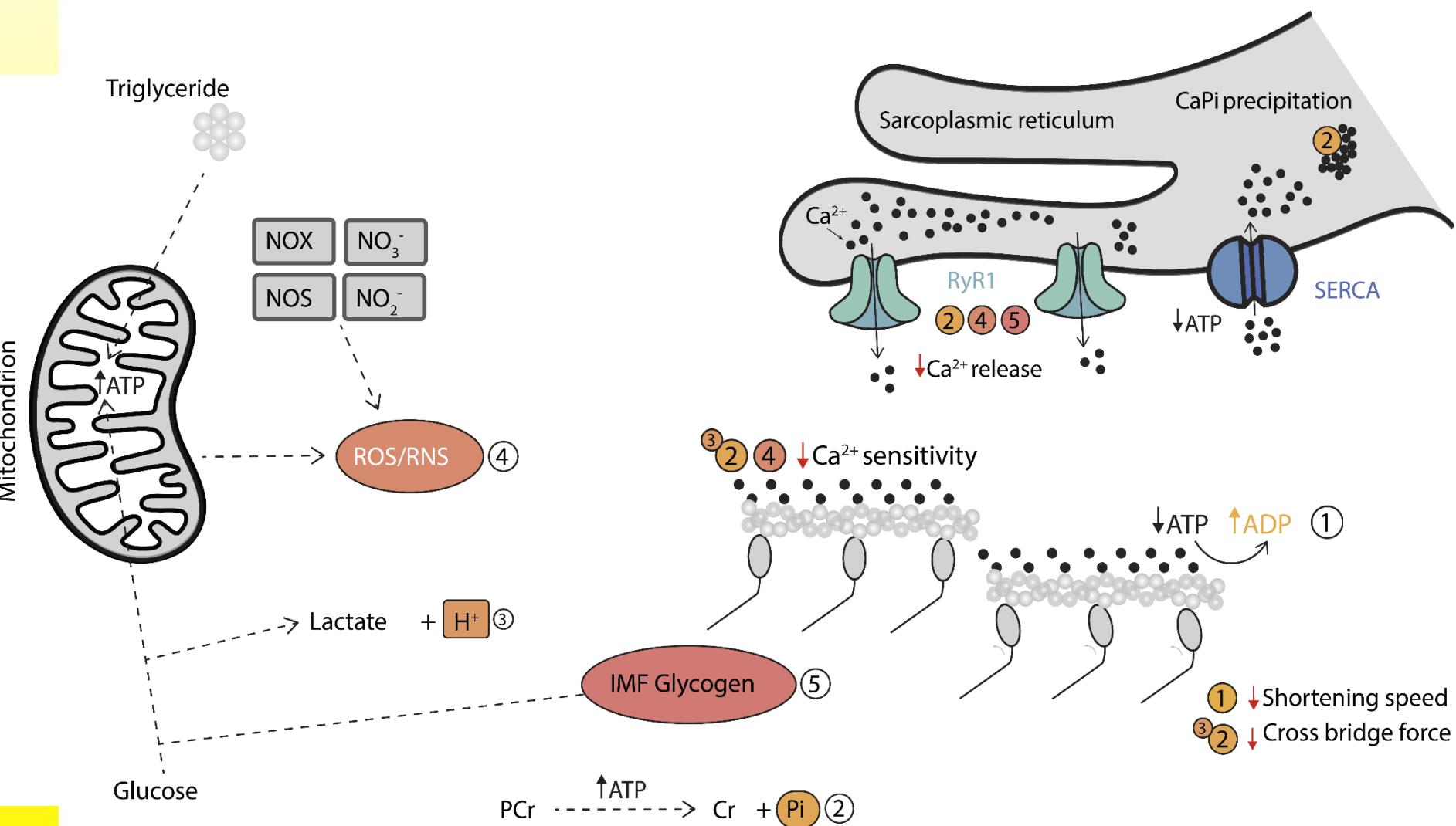
Acidosis & contractile properties

Figure 2. Decreased intracellular pH and contractile function of skeletal muscle cells.



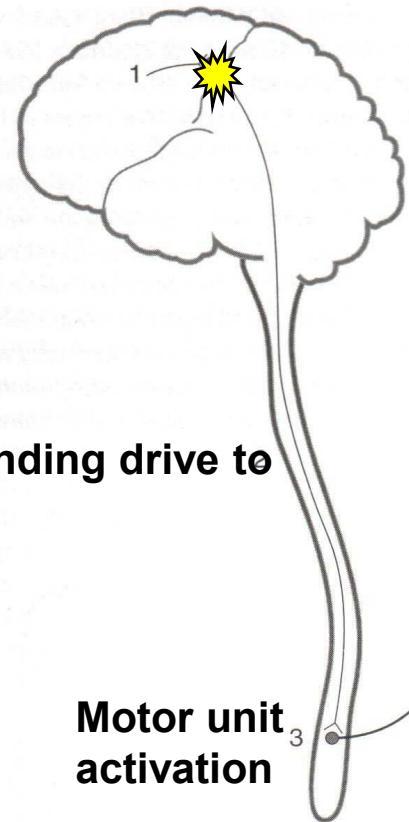
Decreased intracellular pH has little direct effect on the contractile function of skeletal muscle cells.

Metabolites & fatigue



Neuromuscular fatigue

Motor cortex activation



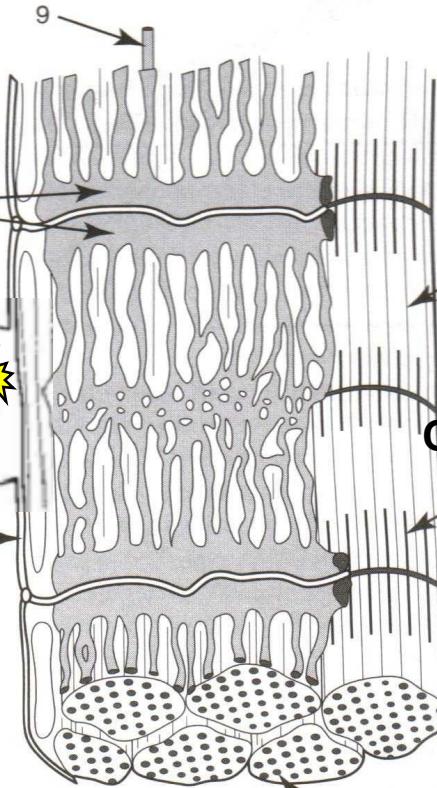
Descending drive to the Mn

Motor unit activation

Muscle blood flow

E-C coupling

Neuromuscular propagation



Intracellular milieu

Contractile apparatus

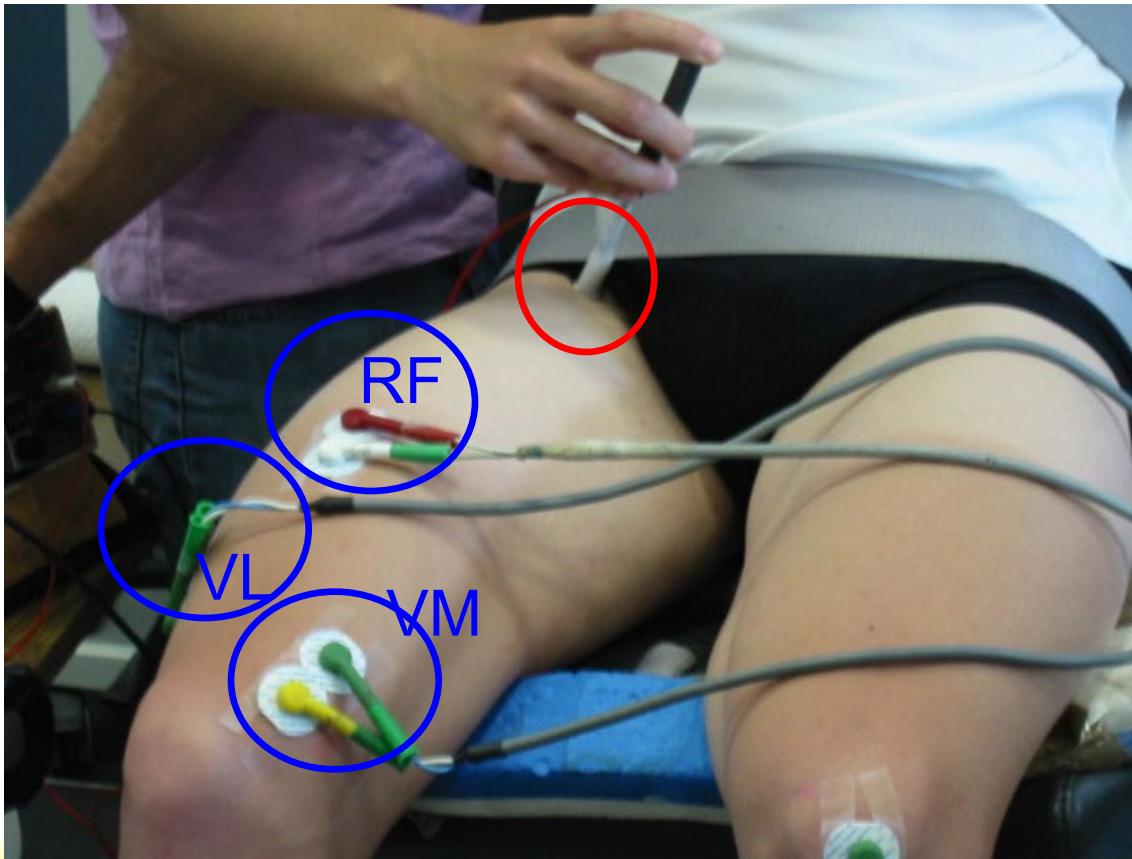
Metabolic substrates availability

Central fatigue

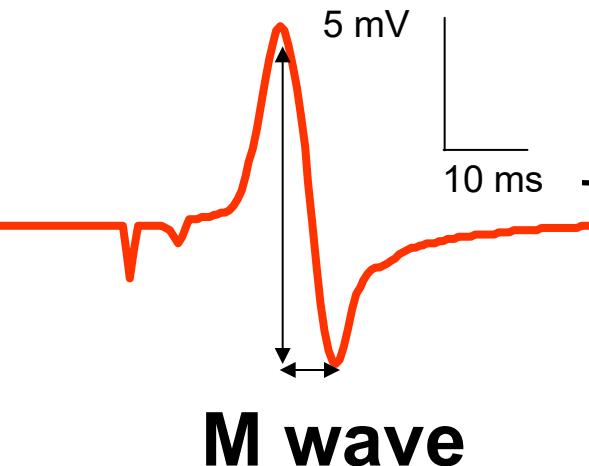
Peripheral fatigue

Non-invasive techniques to assess neuromuscular function

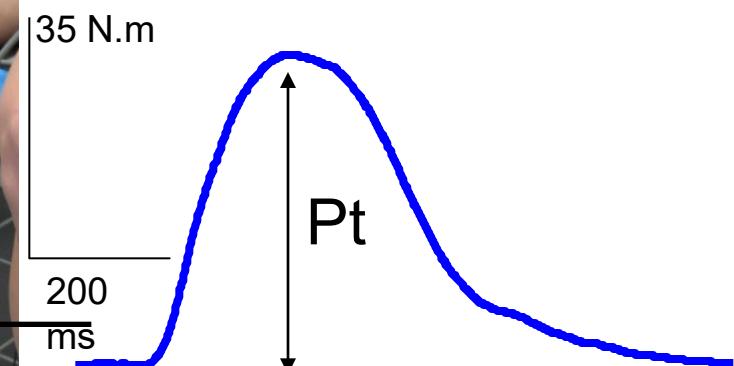
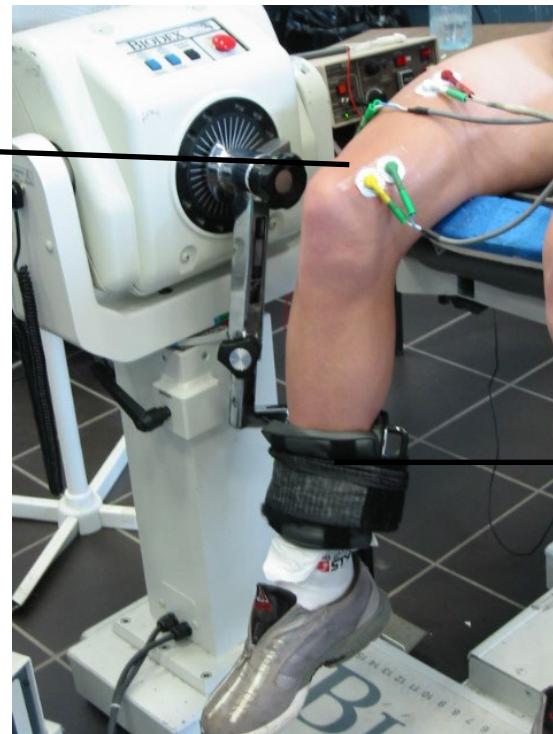
- Transcutaneous stimulation
- Surface electromyography (EMG)



Peripheral fatigue



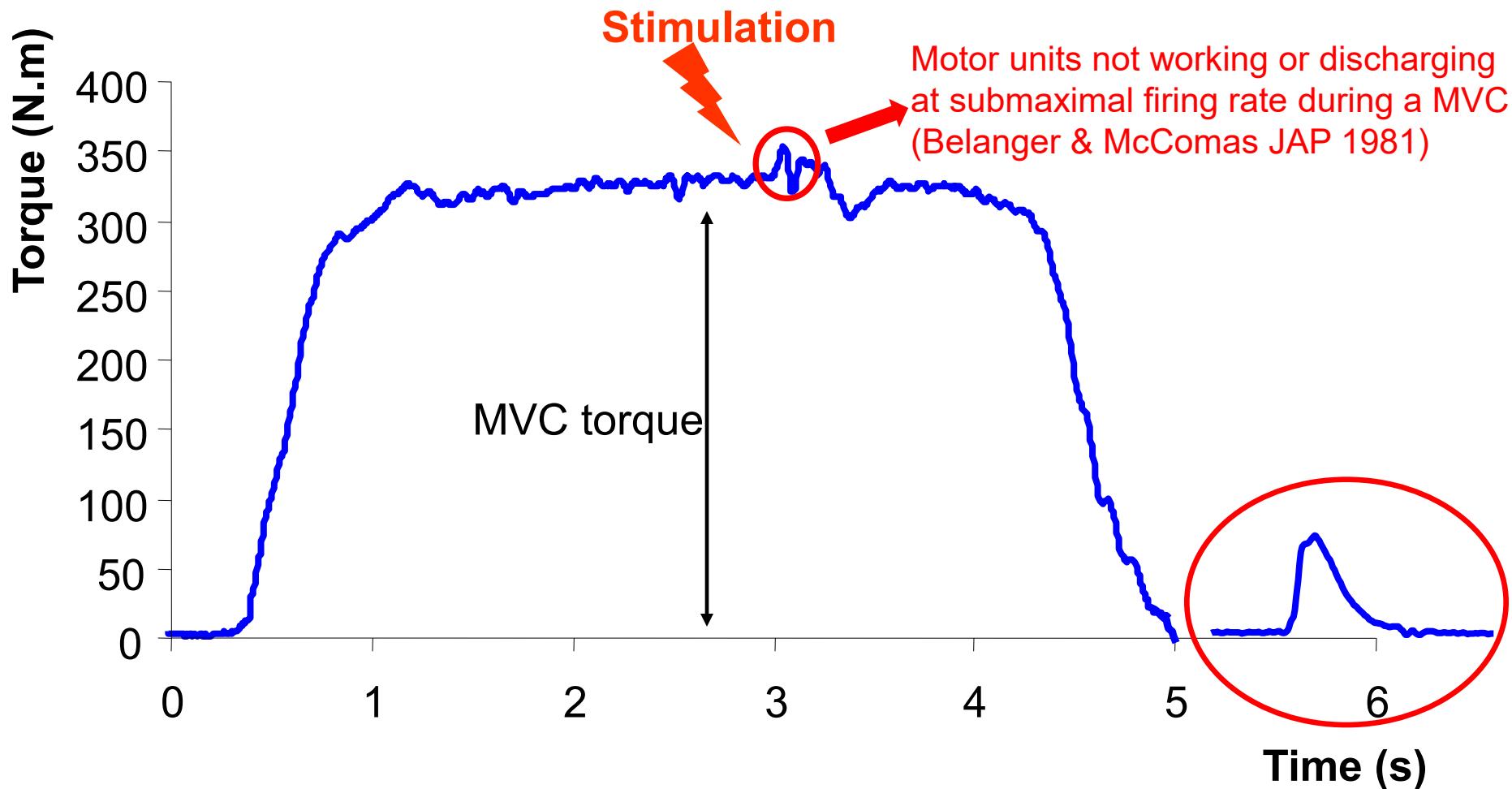
AP
conduction/transmission
failure



Muscular twitch
Contractile failure

Excitation - Contraction coupling failure

Central activation: Twitch Interpolation Technique



$$\text{VAL (\%)} = [1 - (\text{superimposed twitch} / \text{potentiated twitch})] \times 100$$

Allen et al. Muscle Nerve 1995

Key points

Causes of fatigue

- Fatigue may result from PCr or glycogen depletion, limiting ATP production
- Metabolic by-products may impact muscle force production
- Neuromuscular transmission, action potential propagation may be altered (decreased release of ACh, extracellular K^+ accumulation,...).
- Ca^{2+} handling failure has been shown to play an important role
- Central fatigue also contributes to the reduced force generating capacity

II- Resistance training

- How is muscle strength increased ?
- What does happen in the muscle ?
- What is the role of the CNS ?

Did you know ...?

Resistance training programs can lead to strength increase of 25-100% after 3-6 months

→ « Neuromuscular plasticity »



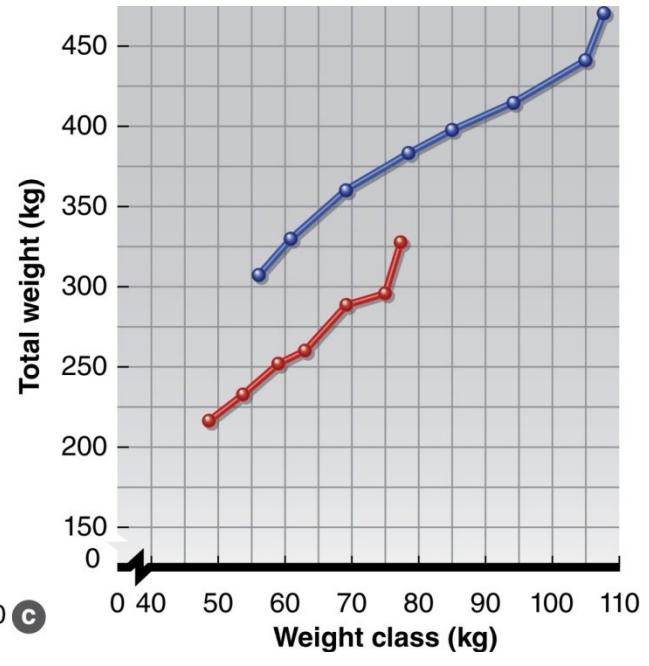
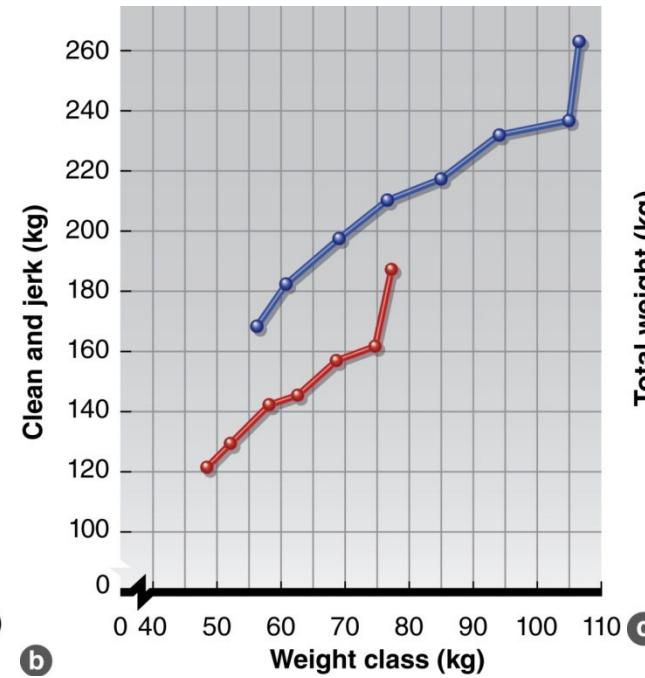
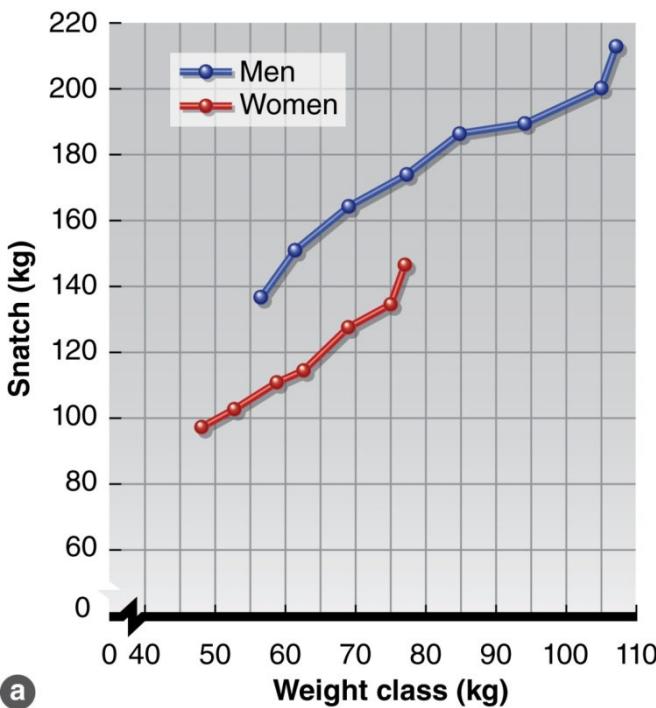
Muscle volume

- **Muscle hypertrophy** = muscle volume increases
- **Muscle atrophy** = reduction in muscle volume
- Muscle strength doesn't only depend on muscle volume.

Ex: . Early stage of a training program
. Women / children



Weightlifting world record & sex

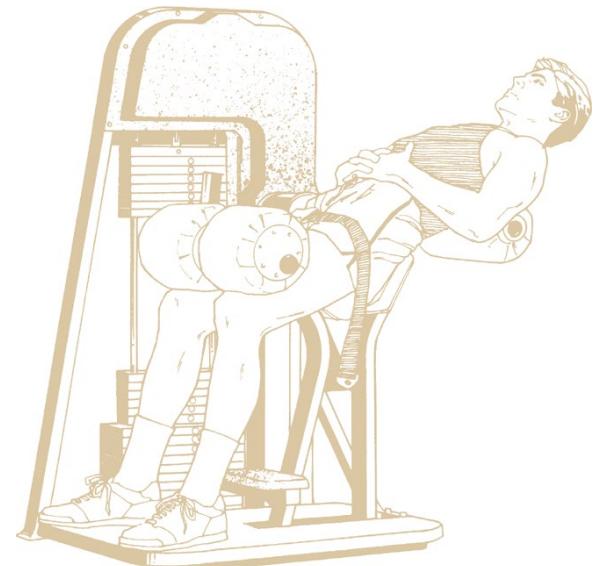


Resistance training consequences

- Hypertrophy
- Change in neural drive to the muscles



Force can increase without hypertrophy but not without neural adaptations



Possible neural adaptations to increase muscle force: hypotheses

- Additionnal motor units recruited ✓
- Increased motor unit firing rate ?
- Reduced antagonist coactivation ✓
- Reduced autogenic inhibition (protective mechanism) ?
- Changes at the neuromuscular junction level ✓
- Motor unit synchronization ✗

Muscle hypertrophy

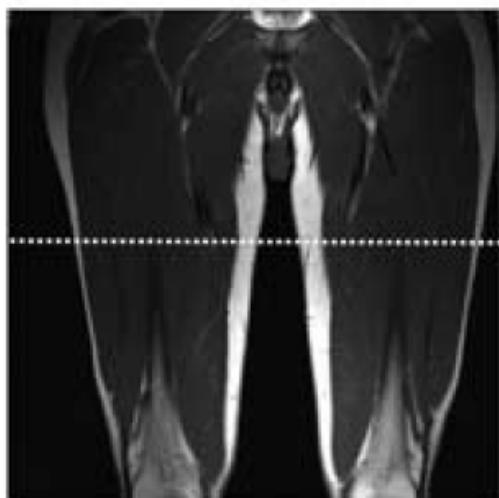
1 – transient — liquid accumulates in the interstitial space after exercise

2 - Chronic — increase in muscle volume after training is due to an increase in the size of muscle fibres (hypertrophy) and/or in the number of muscle fibres (hyperplasia)

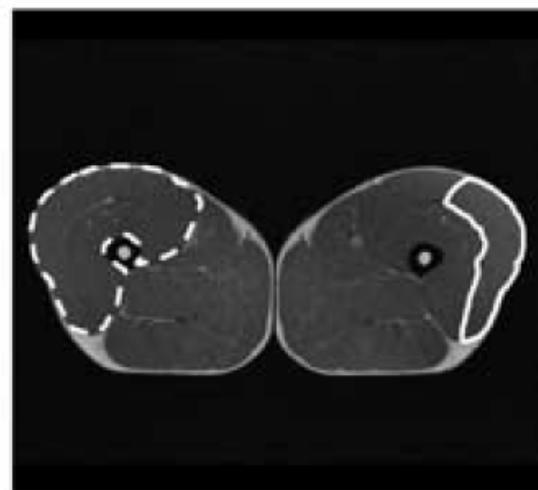
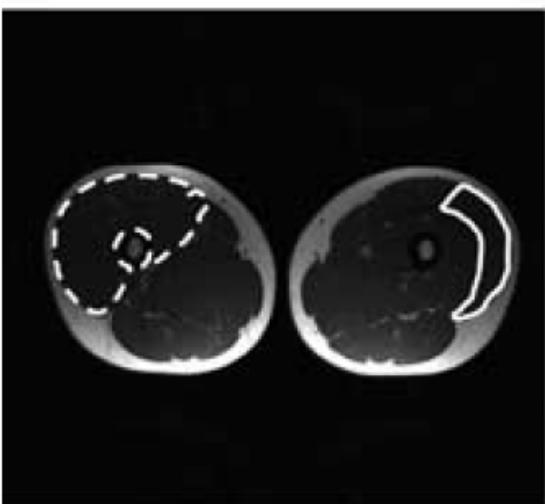
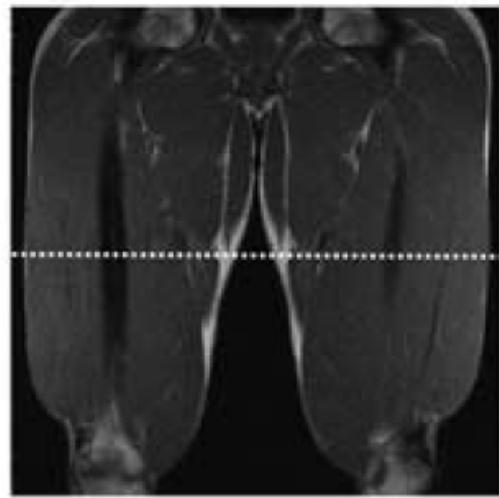


Muscle hypertrophy

CTRL

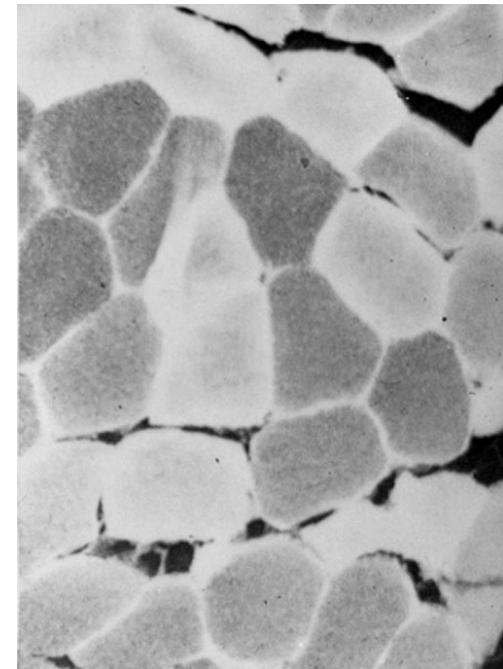
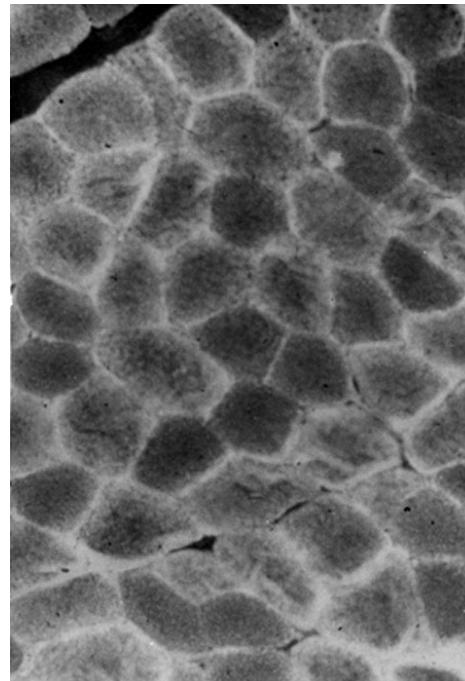


Body builder



Hypertrophy of muscle fibres

- More myofibrils → more actin and myosin filaments → more cross-bridges → more force
- Protein synthesis increases after exercise
- Testosterone influences muscle hypertrophy
- High intensity training → more gains than mid-intensity
Ex: 10 x 10 RM

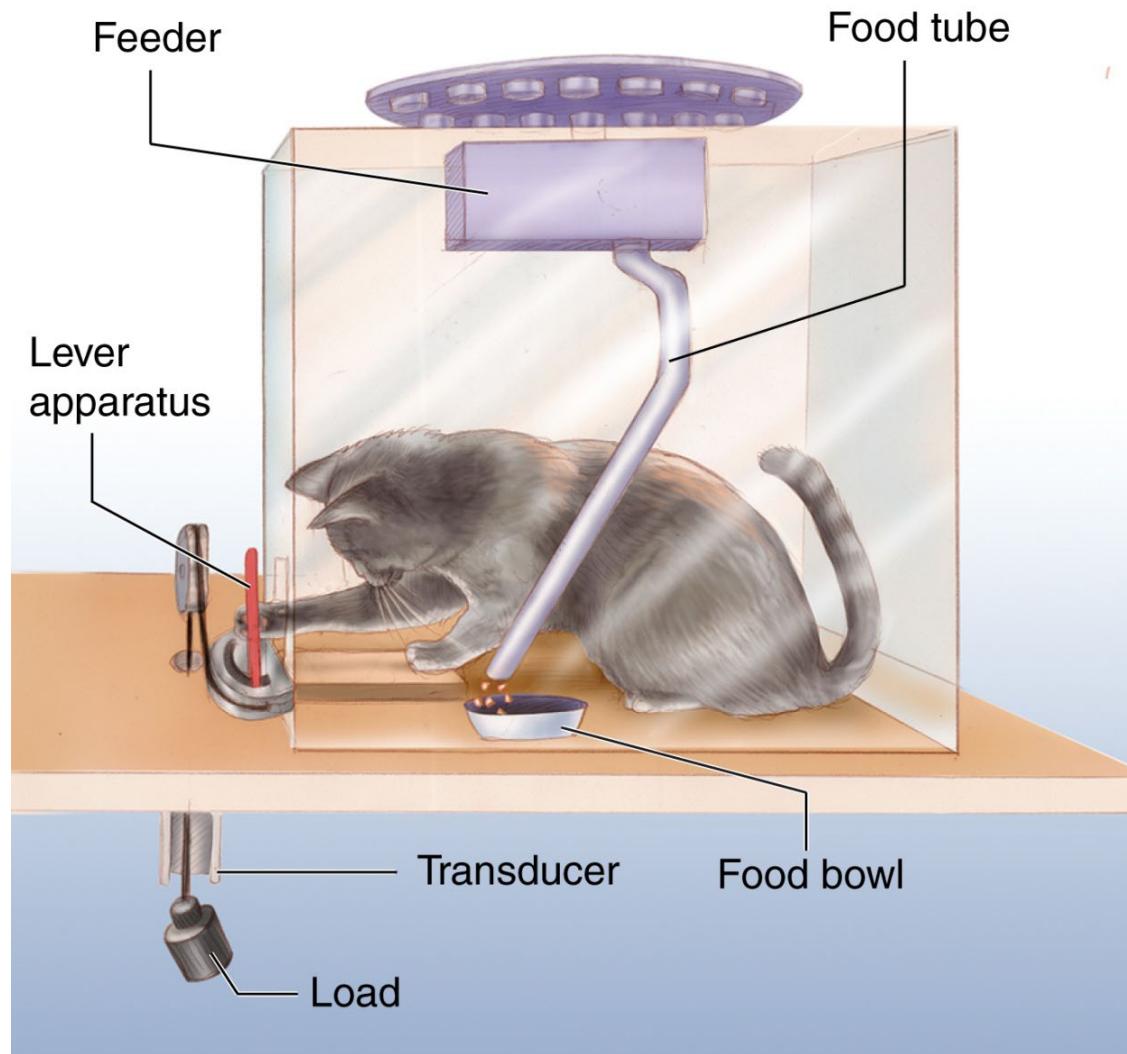


pre –post 6 months of resistance training

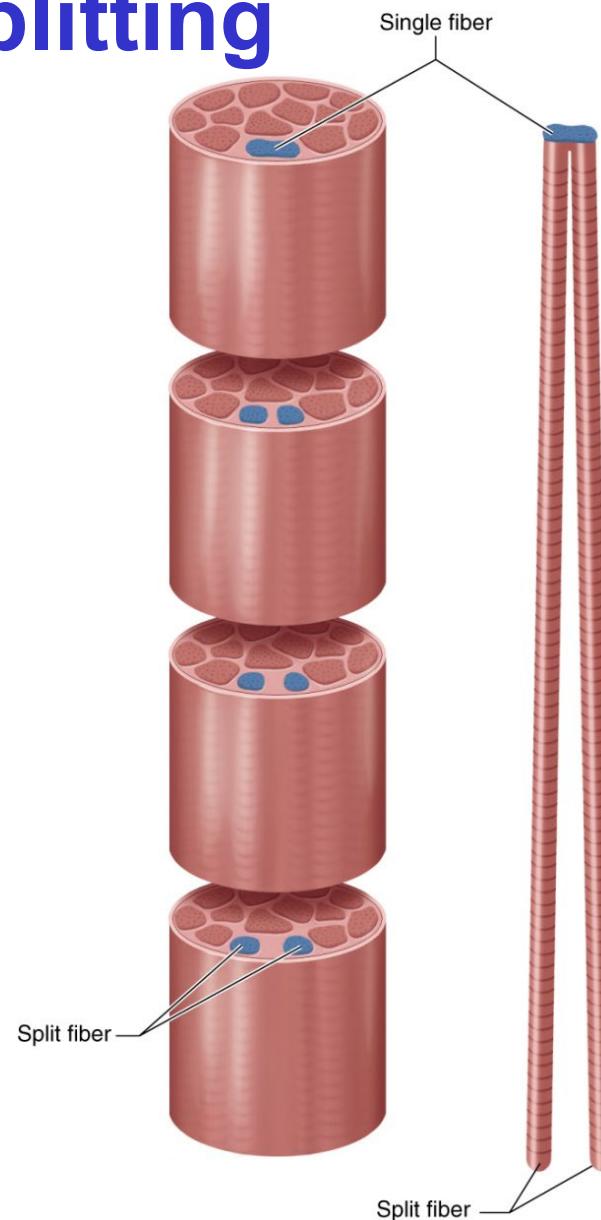
Fibre hyperplasia

- Animal: muscle fibres divide into 2 parts with intensive training
- Each half increases in size until reaching the size of the parent fibre
- Satellite cells (myoblasts) can also be recruited and differentiated
- Well-described phenomenon in animal, no consensus in human

Resistance training for a cat



Muscle fibre splitting



Hyperplasia: meta-analysis 17 studies (animal models)

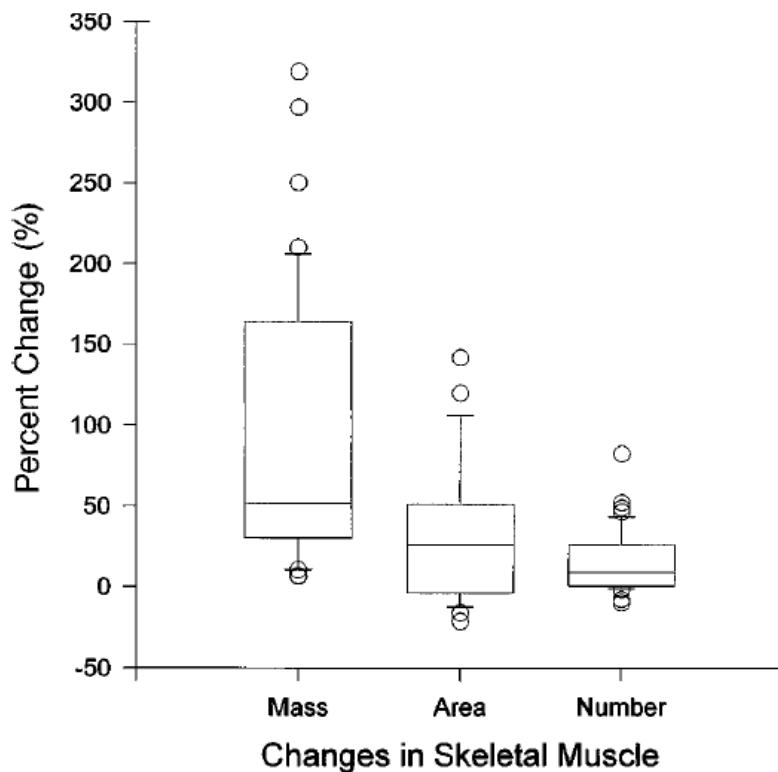


Fig. 1. Percent changes in skeletal muscle mass ($n = 37$), fiber area ($n = 25$), and fiber number ($n = 37$). ○, Outliers beyond 10th and 90th percentiles. Percent change calculated as $(\text{treatment} - \text{control})/\text{treatment} \times 100$.

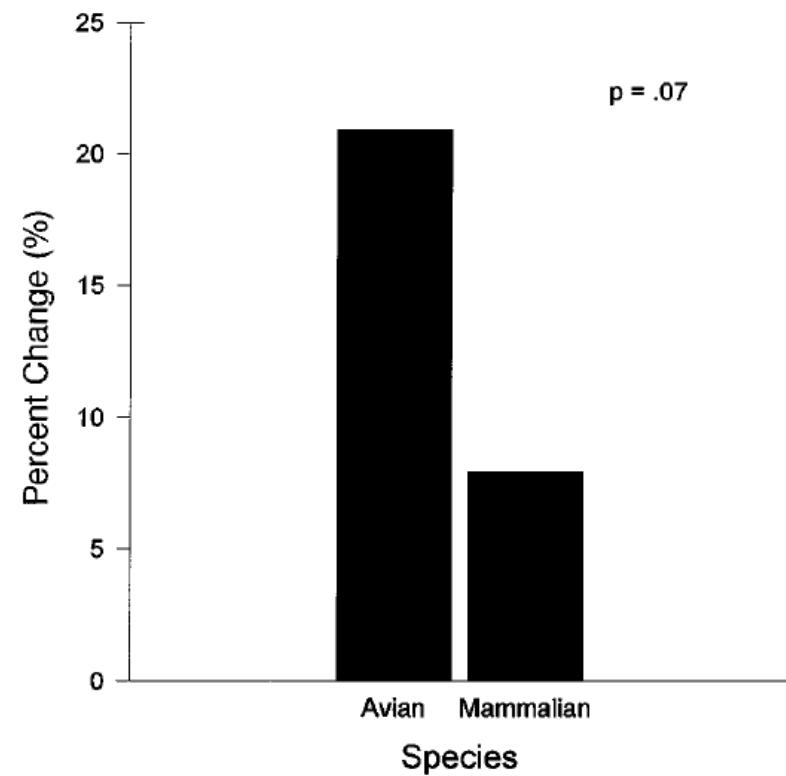


Fig. 3. Percent increases in muscle fiber number according to whether species was avian ($n = 20$) or mammalian ($n = 17$). Percent change calculated as $(\text{treatment} - \text{control})/\text{treatment} \times 100$.

Hypertrophy vs. hyperplasia in human

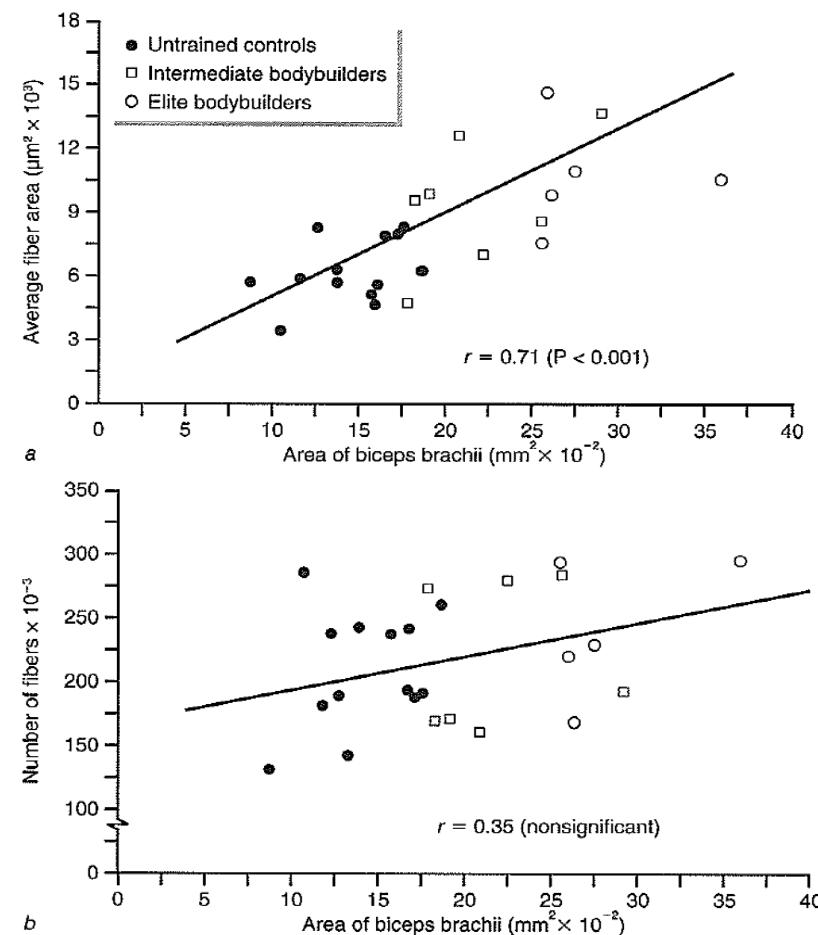


Figure 9.2 Correlations (a) between biceps brachii area and average muscle fiber area and (b) between biceps area and fiber number. Biceps area was determined using computerized tomographic scanning, and fiber areas were determined from biopsy material. The size of the biceps is better related to the size of the individual fibers than it is to the number of fibers.

Reprinted by permission from MacDougall et al. 1984.

MacDougall et al. 1984

Neural activation and fibre hypertrophy

- Early increase in force mostly due to neural adaptations
- The contribution of structural parameters to force gain appears later (training lasting > 8 weeks).



MODEL OF NEURAL VS. MUSCULAR ADAPTATIONS TO STRENGTH TRAINING

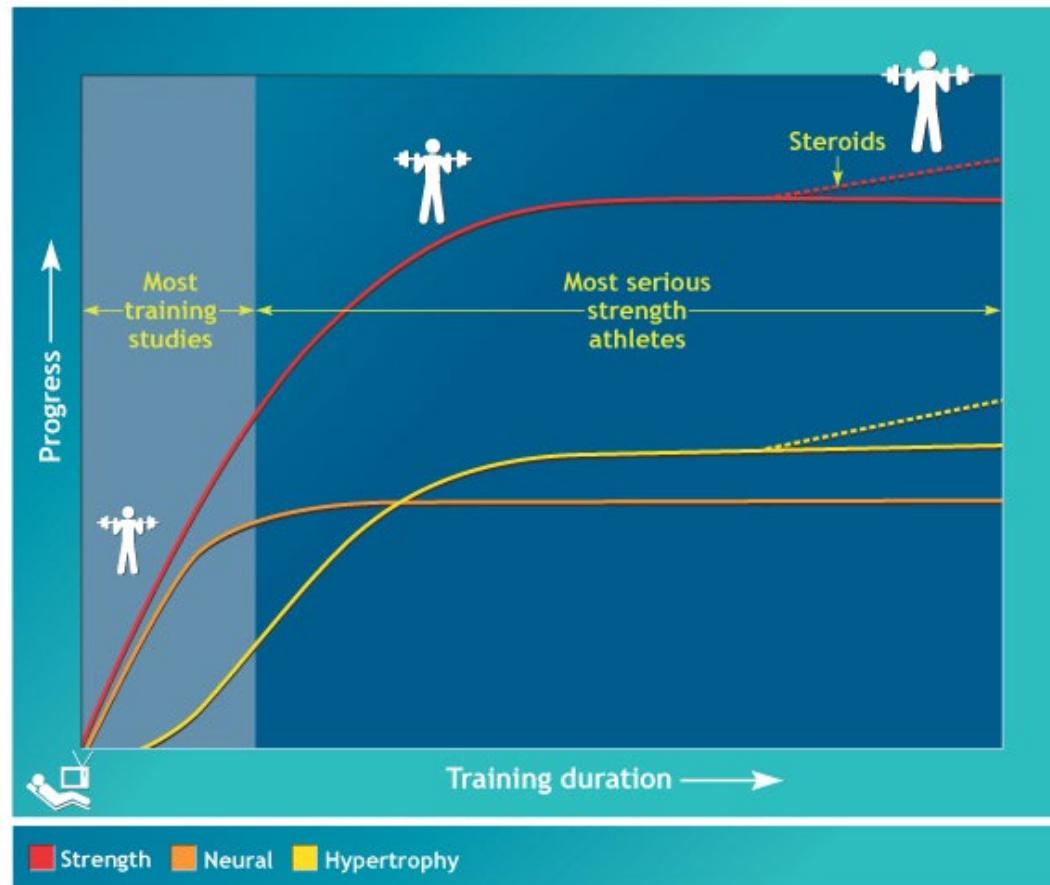
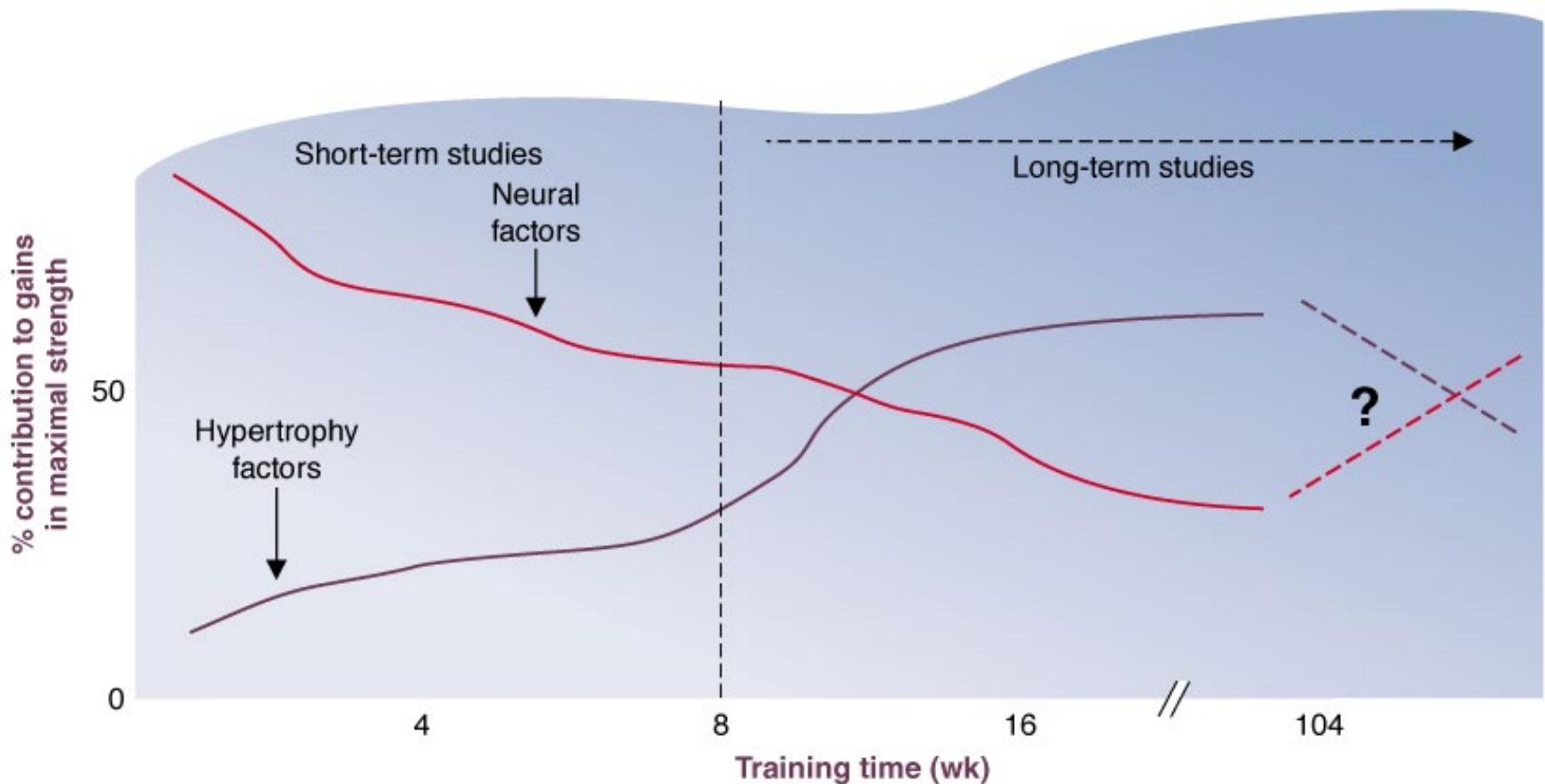


Figure 22.21. Relative roles of neural and muscular adaptations in strength improvement with resistance training. Note that neural adaptations predominate in the early phase of training (this phase also encompasses most training studies). Hypertrophy-induced adaptations place the upper limit on longer-term training improvements. This tempts many athletes to use anabolic steroids and/or human growth hormone (dashed line) to induce continual hypertrophy if training alone fails. (From Sale DG. Neural adaptation to resistance training. *Med Sci Sports Exerc* 1988;20:135.)

MODEL OF NEURAL VS. MUSCULAR ADAPTATIONS TO STRENGTH TRAINING



Did you know...?

Once your objective in strength gain is attained, you can reduce frequency, intensity or duration of the training sessions and maintain this force level.



Key points

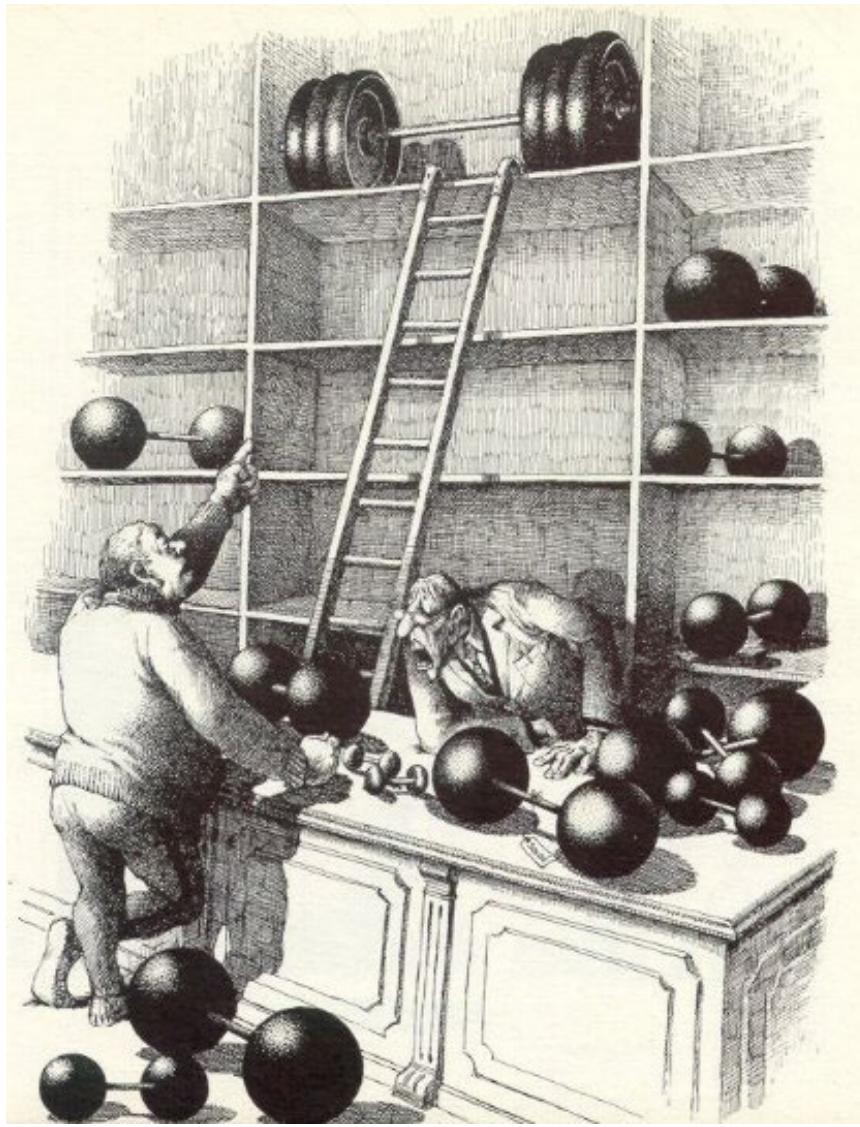
Resistance training

- Always leads to neural adaptations, not necessarily to hypertrophy
- Transient hypertrophy results from an accumulation of liquid in the interstitial space
- Chronic hypertrophy results from resistance training (usually >8 wks) and is due to structural changes in the muscle

Key points

Resistance training

- Muscle hypertrophy is due to increased size of muscle fibres with a possible (?) rise in their number.
- Inactive muscle → atrophy. A reduction in training stimulus could be enough to prevent muscle mass loss.







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