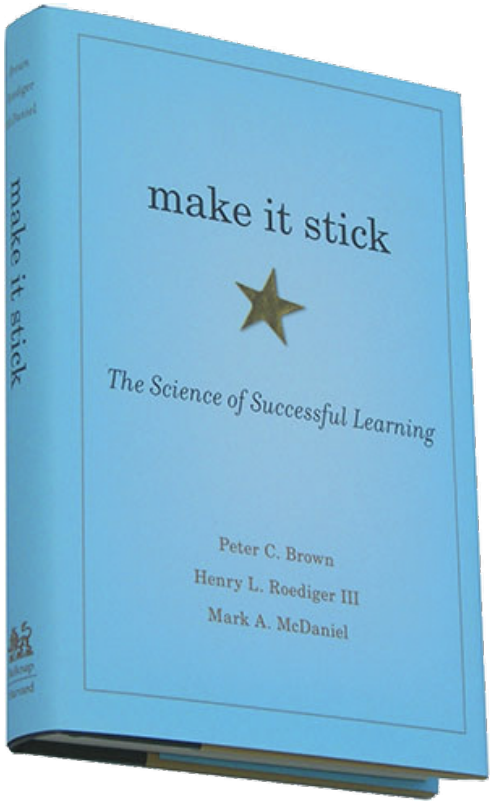
HESP 147
SECTION ONE

REVIEW
FOR EXAM 1

Helpful information that I won't ask about. Helpful for exam prep though:



Active practice. Think specificity of adaptation. What makes you do better on exams? Recalling things. Reading notes isn't effective test preparation unless the tests insists that you read notes.

Spaced vs. massed practice. Cramming = less helpful.

Mixed practice. Don't train/study one thing at a time.

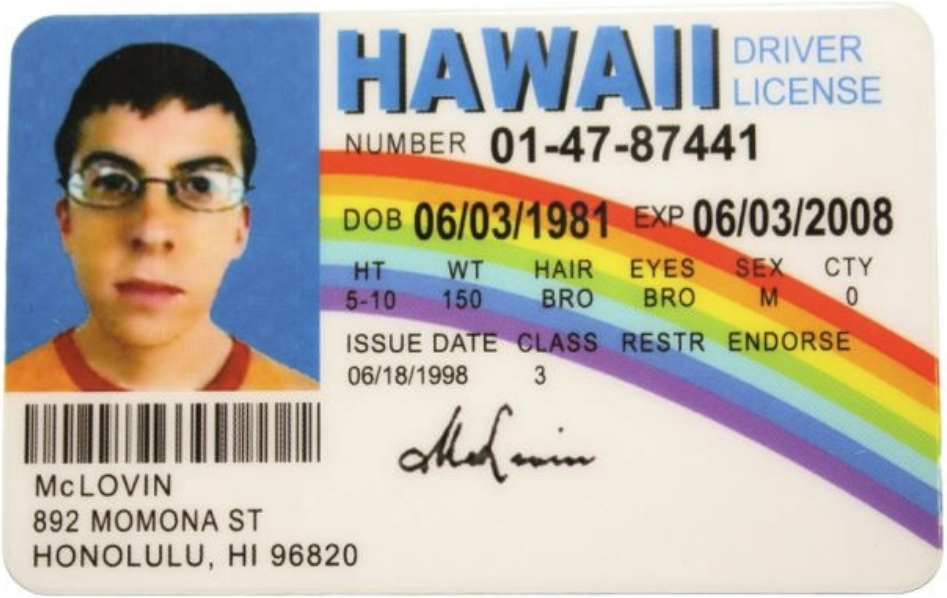
Sleep. Do it. Studying without sleeping is nearly pointless.

Effort. The harder it is, the better you remember it.

** Remember the Dunning-Kruger effect: Appraise your effort, knowledge, and capacities objectively.*

What's the difference between a license and a certification?

≠



What's the difference between a license and a certification?

Licensure vs. Certification

A statement or declaration that one has completed a course of study, passed an examination, or met some other criteria (somehow) for "certification".

It is not permission to act, but a statement of qualification (or at least completion of a "qualifying" challenge).

It does not involve state or federal agencies (which is what a license does); a certification is a *private* matter.

What are the CVD risk factors?

Physical Activity Readiness Questionnaire - PAR-Q (revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO		
☐	☐	1.	**Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?**
☐	☐	2.	**Do you feel pain in your chest when you do physical activity?**
☐	☐	3.	**In the past month, have you had chest pain when you were not doing physical activity?**
☐	☐	4.	**Do you lose your balance because of dizziness or do you ever lose consciousness?**
☐	☐	5.	**Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?**
☐	☐	6.	**Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?**
☐	☐	7.	**Do you know of any other reason why you should not do physical activity?**

If you answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active – begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal – this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever – wait until you feel better; or
- if you are or may be pregnant – talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____ DATE _____

SIGNATURE OF PARENT or GUARDIAN (for participants under the age of majority) _____ WITNESS _____

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.

CSEP SCPE © Canadian Society for Exercise Physiology Supported by: Health Canada Santé Canada

continued on other side...

2020 PAR-Q+

The Physical Activity Readiness Questionnaire for Everyone

The health benefits of regular physical activity are clear; more people should engage in physical activity every day of the week. Participating in physical activity is very safe for MOST people. This questionnaire will tell you whether it is necessary for you to seek further advice from your doctor OR a qualified exercise professional before becoming more physically active.

GENERAL HEALTH QUESTIONS

Please read the 7 questions below carefully and answer each one honestly: check YES or NO.	YES	NO
1) Has your doctor ever said that you have a heart condition ☐ **OR** high blood pressure ☐?	☐	☐
2) Do you feel pain in your chest at rest, during your daily activities of living, **OR** when you do physical activity?	☐	☐
3) Do you lose balance because of dizziness **OR** have you lost consciousness in the last 12 months? Please answer **NO** if your dizziness was associated with over-breathing (including during vigorous exercise).	☐	☐
4) Have you ever been diagnosed with another chronic medical condition (other than heart disease or high blood pressure)? **PLEASE LIST CONDITION(S) HERE:** _____	☐	☐
5) Are you currently taking prescribed medications for a chronic medical condition? **PLEASE LIST CONDITION(S) AND MEDICATIONS HERE:** _____	☐	☐
6) Do you currently have (or have had within the past 12 months) a bone, joint, or soft tissue (muscle, ligament, or tendon) problem that could be made worse by becoming more physically active? Please answer **NO** if you had a problem in the past, but it **does not limit your current ability** to be physically active. **PLEASE LIST CONDITION(S) HERE:** _____	☐	☐
7) Has your doctor ever said that you should only do medically supervised physical activity?	☐	☐

☑ **If you answered NO to all of the questions above, you are cleared for physical activity. Please sign the PARTICIPANT DECLARATION. You do not need to complete Pages 2 and 3.**

- Start becoming much more physically active – start slowly and build up gradually.
- Follow Global Physical Activity Guidelines for your age (https://apps.who.int/iris/handle/10665/44399).
- You may take part in a health and fitness appraisal.
- If you are over the age of 45 yr and NOT accustomed to regular vigorous to maximal effort exercise, consult a qualified exercise professional before engaging in this intensity of exercise.
- If you have any further questions, contact a qualified exercise professional.

PARTICIPANT DECLARATION

If you are less than the legal age required for consent or require the assent of a care provider, your parent, guardian or care provider must also sign this form.

I, the undersigned, have read, understood to my full satisfaction and completed this questionnaire. I acknowledge that this physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if my condition changes. I also acknowledge that the community/fitness center may retain a copy of this form for its records. In these instances, it will maintain the confidentiality of the same, complying with applicable law.

NAME _____ DATE _____

SIGNATURE _____ WITNESS _____

SIGNATURE OF PARENT/GUARDIAN/CARE PROVIDER _____

If you answered YES to one or more of the questions above, COMPLETE PAGES 2 AND 3.

⚠ **Delay becoming more active if:**

- You have a temporary illness such as a cold or fever; it is best to wait until you feel better.
- You are pregnant - talk to your health care practitioner, your physician, a qualified exercise professional, and/or complete the ePARmed-X+ at www.eparmedx.com before becoming more physically active.
- Your health changes - answer the questions on Pages 2 and 3 of this document and/or talk to your doctor or a qualified exercise professional before continuing with any physical activity program.

The PAR-Q is necessary but insufficient.

TABLE 2.2. Atherosclerotic Cardiovascular Disease (CVD) Risk Factors and Defining Criteria (26,31)

Risk Factors	Defining Criteria
Age	Men ≥45 yr; women ≥55 yr (12)
Family history	Myocardial infarction, coronary revascularization, or sudden death before 55 yr in father or other male first-degree relative or before 65 yr in mother or other female first-degree relative
Cigarette smoking	Current cigarette smoker or those who quit within the previous 6 mo or exposure to environmental tobacco smoke
Sedentary lifestyle	Not participating in at least 30 min of moderate intensity, physical activity (40%–<60% $\dot{V}O_2R$) on at least 3 d of the week for at least 3 mo (22,30)
Obesity	Body mass index ≥30 kg · m^{-2} *or* waist girth >102 cm (40 in) for men and >88 cm (35 in) for women (10)
Hypertension	Systolic blood pressure ≥140 mm Hg and/or diastolic ≥90 mm Hg, confirmed by measurements on at least two separate occasions, *or* on antihypertensive medication (9)
Dyslipidemia	Low-density lipoprotein (LDL) cholesterol ≥130 mg · dL^{-1} (3.37 mmol · L^{-1}) *or* high-density lipoprotein[b] (HDL) cholesterol <40 mg · dL^{-1} (1.04 mmol · L^{-1}) *or* on lipid-lowering medication. If total serum cholesterol is all that is available, use ≥200 mg · dL^{-1} (5.18 mmol · L^{-1}) (21)
Prediabetes[a]	Impaired fasting glucose (IFG) = fasting plasma glucose ≥100 mg · dL^{-1} (5.55 mmol · L^{-1}) and ≤125 mg · dL^{-1} (6.94 mmol · L^{-1}) *or* impaired glucose tolerance (IGT) = 2 h values in oral glucose tolerance test (OGTT) ≥140 mg · dL^{-1} (7.77 mmol · L^{-1}) and ≤199 mg · dL^{-1} (11.04 mmol · L^{-1}) confirmed by measurements on at least two separate occasions (5)
Negative Risk Factors	**Defining Criteria**
High-density lipoprotein (HDL) cholesterol	≥60 mg · dL^{-1} (1.55 mmol · L^{-1})

[a]If the presence or absence of a CVD risk factor is not disclosed or is not available, that CVD risk factor should be counted as a risk factor except for prediabetes. If the prediabetes criteria are missing or unknown, prediabetes should be counted as a risk factor for those ≥45 yr, especially for those with a body mass index (BMI) ≥25 kg · m^{-2}, and those <45 yr with a BMI ≥25 kg · m^{-2} and additional CVD risk factors for prediabetes. The number of positive risk factors is then summed.

[b]High HDL is considered a negative risk factor. For individuals having high HDL ≥60 mg · dL^{-1} (1.55 mmol · L^{-1}), for these individuals one positive risk factor is subtracted from the sum of positive risk factors.

$\dot{V}O_2R$, oxygen uptake reserve.

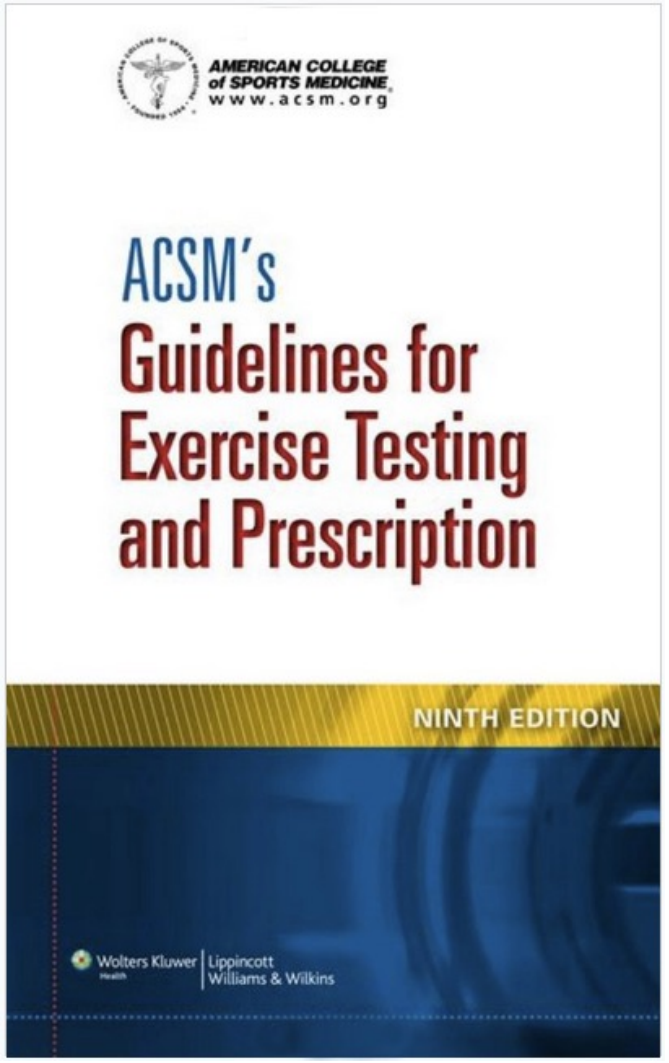


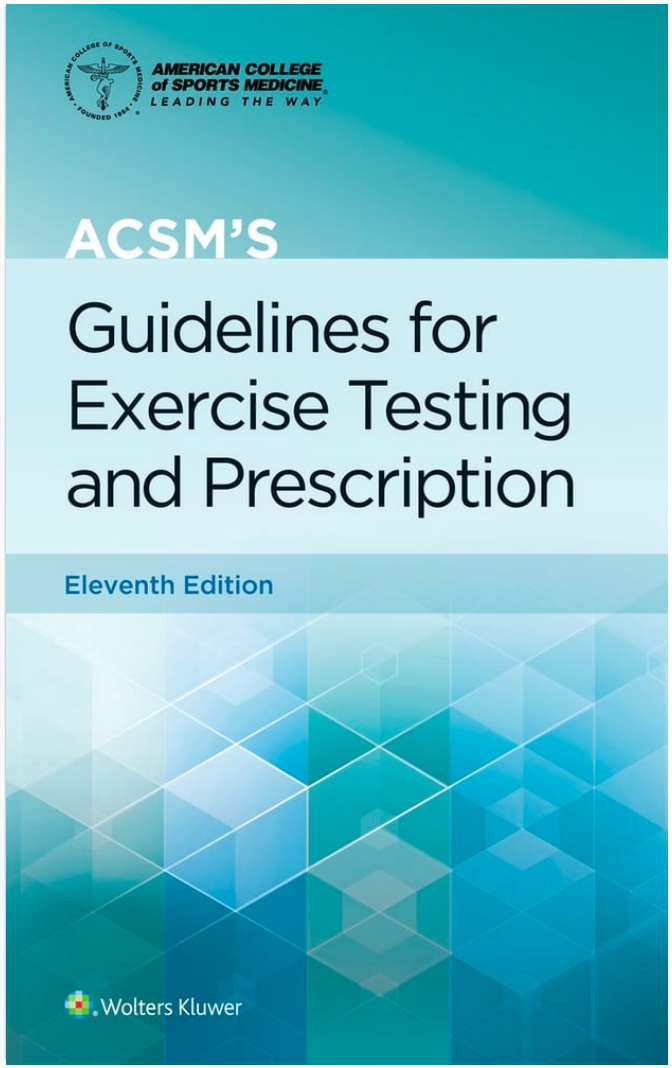
TABLE 2.2. Cardiovascular Disease (CVD) Risk Factors and Defining Criteria

Positive Risk Factors[a]	Defining Criteria
Age	Men ≥45 yr; women ≥55 yr (36)
Family history	Myocardial infarction, coronary revascularization, or sudden death before 55 yr in father or other male first-degree relative or before 65 yr in mother or other female first-degree relative (37)
Cigarette smoking	Current cigarette smoker or those who quit within the previous 6 mo or exposure to environmental tobacco smoke (37,38)
Physical inactivity	Not meeting the minimum threshold of 500–1,000 MET-min of moderate-to-vigorous physical activity or 75–150 min · wk^{-1} of moderate-to-vigorous intensity physical activity (23)
Body mass index/waist circumference	Body mass index ≥30 kg · m^{-2} or waist girth >102 cm (40 in) for men and >88 cm (38 in) for women (39)
Blood pressure	Systolic blood pressure ≥130 mm Hg and/or diastolic ≥80 mm Hg, based on an average of ≥2 readings obtained on ≥2 occasions, or on antihypertensive medication (40)
Lipids	Low-density lipoprotein cholesterol (LDL-C) ≥130 mg · dL^{-1} (3.37 mmol · L^{-1}) or high-density lipoprotein cholesterol (HDL-C) <40 mg · dL^{-1} (1.04 mmol · L^{-1}) in men and <50 mg · dL^{-1} (1.30 mmol · L^{-1}) in women or non-HDL-C <130 (3.37 mmol · L^{-1}) or on lipid-lowering medication. If total serum cholesterol is all that is available, use ≥200 mg · dL^{-1} (5.18 mmol · L^{-1}) (41).
Blood glucose	Fasting plasma glucose ≥100 mg · dL^{-1} (5.5 mmol · L^{-1}); or 2 h plasma glucose values in oral glucose tolerance test (OGTT) ≥140 mg · dL^{-1} (7.77 mmol · L^{-1}); or HbA1C ≥5.7% (42)
Negative Risk Factors	**Defining Criteria**
HDL-C[b]	≥60 mg · dL^{-1} (1.55 mmol · L^{-1}) (41)

[a]If the presence or absence of a CVD risk factor is not disclosed or is not available, that CVD risk factor should be counted as a risk factor.

[b]High HDL-C is considered a negative risk factor. For individuals having high HDL ≥60 mg · dL^{-1} (1.55 mmol · L^{-1}), one positive risk factor is subtracted from the sum of positive risk factors.

HbA1C, glycated hemoglobin; MET, metabolic equivalent; non-HDL-C, total cholesterol minus HDL-C.



The rest of that stuff is in Lecture 1

What are these things?

Organ Systems

Control loops

Positive Injury Principle

Specificity of Adaptation

Steady-State Exercise

Periodization

Dose-Response Curve

Recovery Optimization

Homeostasis

Individuation

Accommodation vs. Adaptation

Overload/Adaptation Principle

FITT

Absolute vs. Relative Intensity

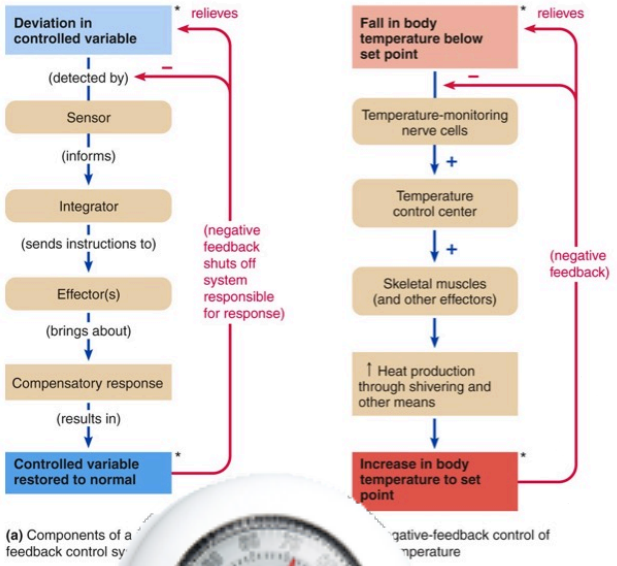
Trainability Principle

Size Principle

Fundamentals of Exercise Physiology:

Control loops:

Negative



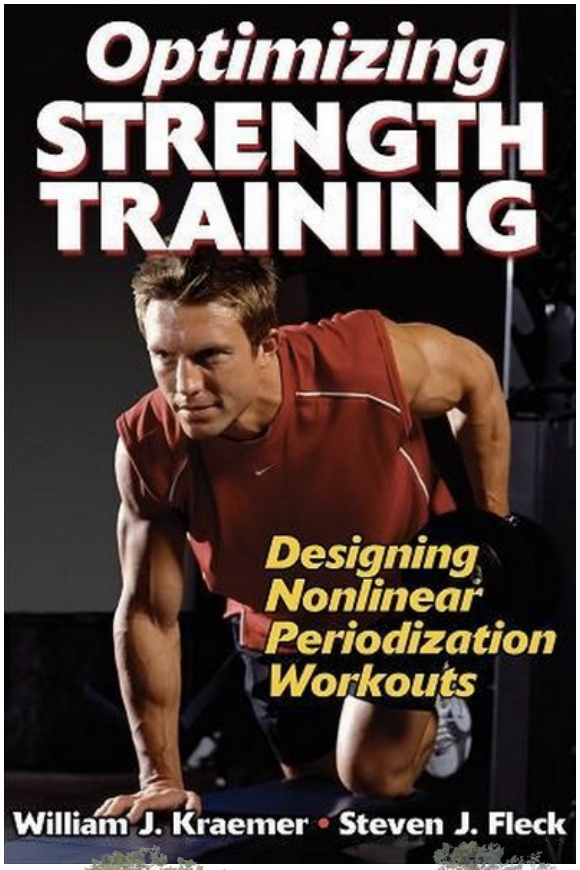
Positive

Fundamentals of Exercise Physiology:

Recovery Optimization:

- Uncontrollable factors such as genetics, sex or age
- FITT characteristics
- Conditioning level of the subject
- Environment (e.g., temperature, elevation)
- Amount of sleep
- Nutritional status
- Hydration
- Medications

Fundamentals of Exercise Physiology:



Macrocycle. Annual plan. Incorporates in-season maintenance and off-season progress toward goals. Phases: preparation, competition, and transition.

Mesocycle. About 2–6 weeks, during which the program emphasizes a particular goal.

Microcycle. A week's training program (heavy days, light days, different body parts, etc.).

Linear Periodization. A similar training intensity and volume (to achieve a core goal) is used during a microcycle and generally throughout a mesocycle.

Nonlinear (or "Undulating") Periodization. Focuses on more than one goal during micro and mesocycles. Characteristics of FITT change more frequently to vary muscle stimuli.

Fundamentals of Exercise Physiology:

Individuation:

A body is not a body: there are important differences between men and women, children and adults, the injured and healthy, the athletic and the sedentary. What are some differences?

Fundamentals of Exercise Physiology:

Overreaching vs. Overtraining:

What's the difference?

Fundamentals of Exercise Physiology:

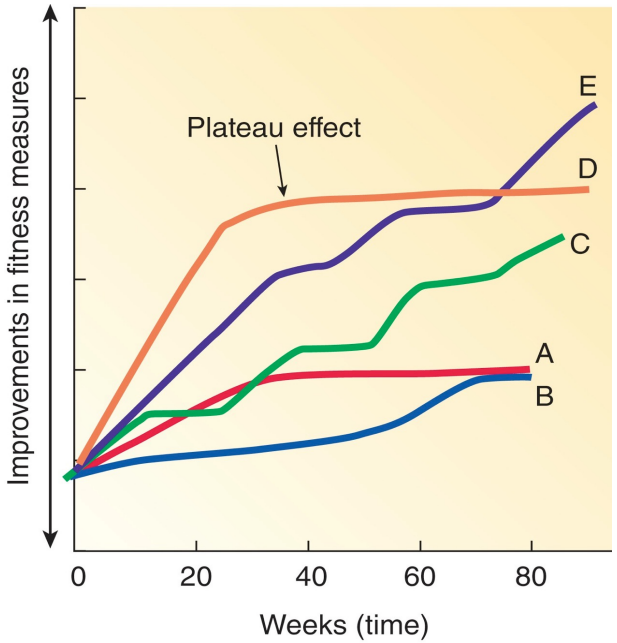
The Positive Injury Principle

Exercise is good for you because it's bad for you.

Fundamentals of Exercise Physiology:

Trainability Principle:

In general, an untrained person, system, or muscle will respond to a training regimen with rapid improvement.



A–E = different individuals

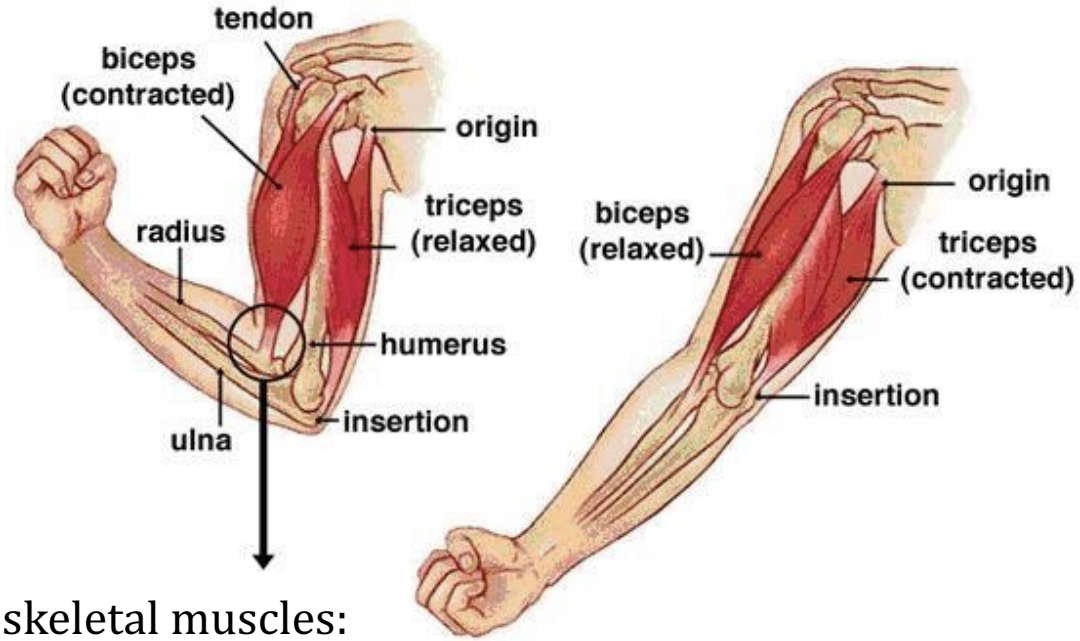
The rest of that stuff is in lecture 2.

Macrostructure:

You have a bunch of skeletal muscles

(>430 according to the book; probably ~640)

Macrostructure:



Basic purpose of skeletal muscles:

Move your bones around.

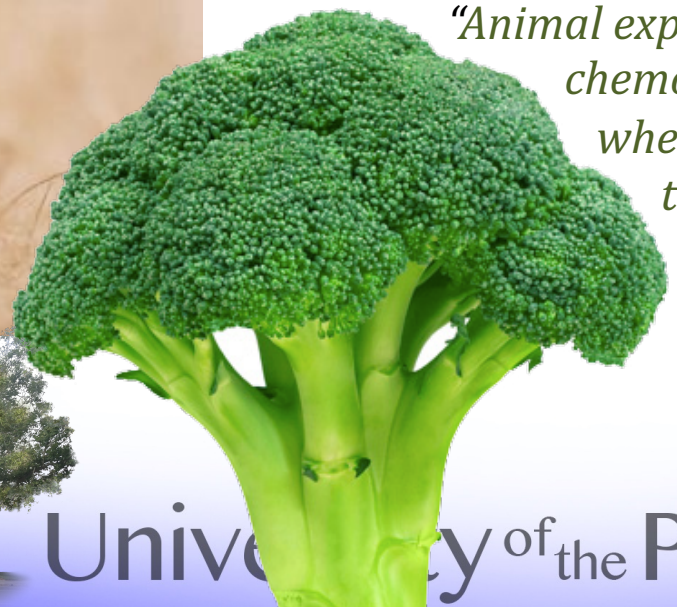
Macrostructure:

Health benefits and possible risks of broccoli – An overview

Klaus Peter Latté, Klaus-Erich Appel, Alfonso Lampen *

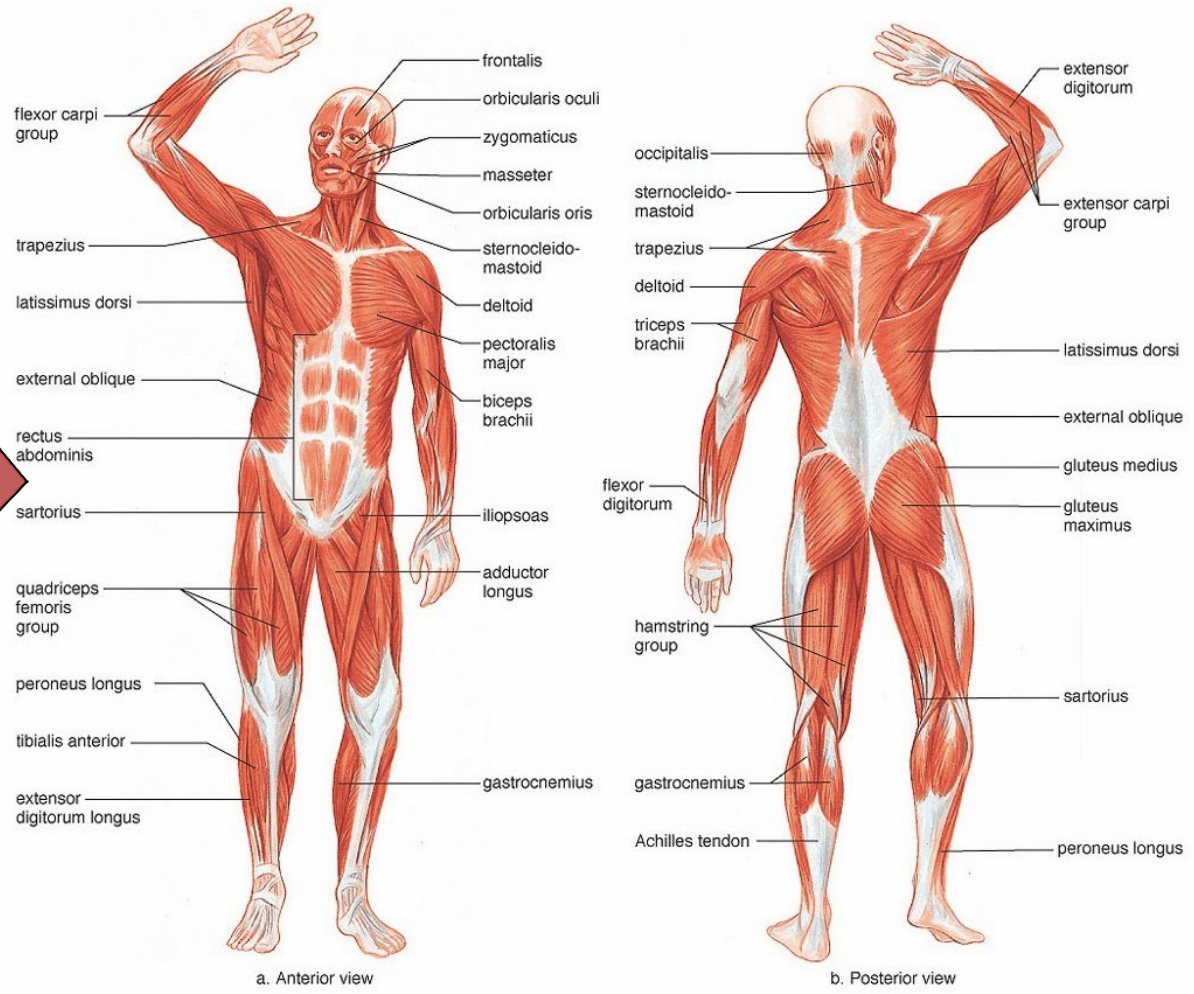
Food and Chemical Toxicology 49 (2011) 3287–3309

"Animal experiments indicate that the chemopreventive effect is highest when broccoli is consumed prior to the exposure of carcinogens."



Macrostructure:

By looking at a diagram, how can you tell what their function is?



Macrostructure:

SHAPE

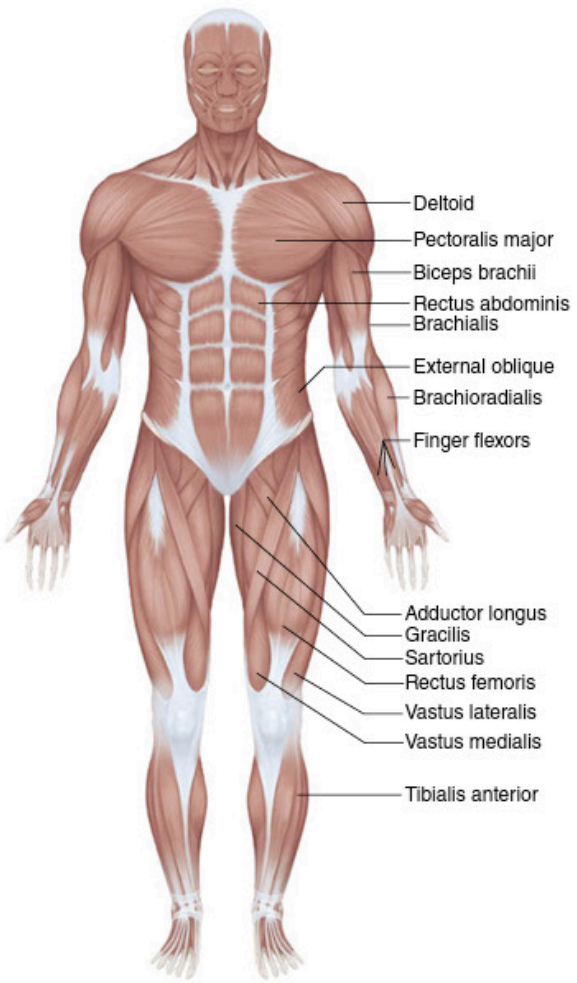
Longer muscle

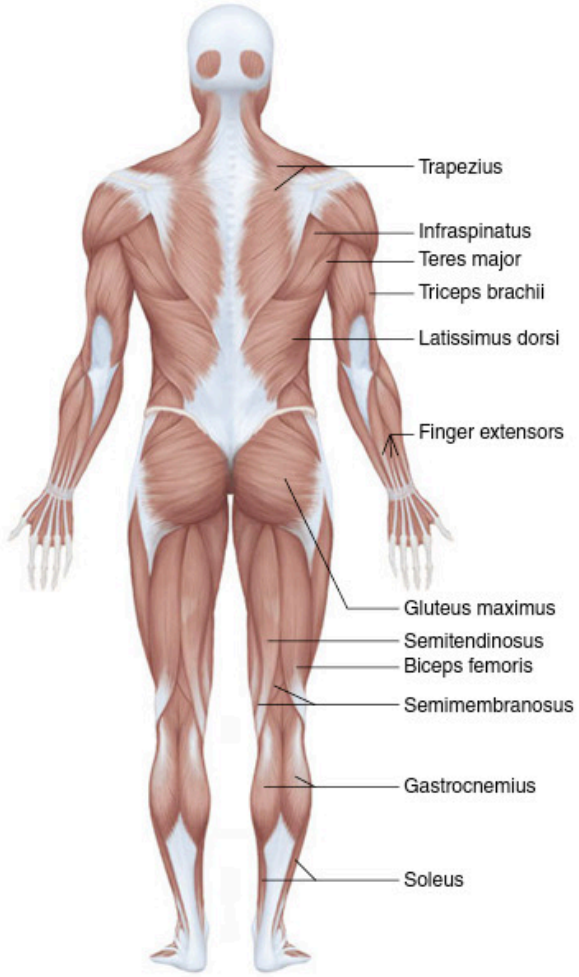
⇧ Amount of shortening

⇧ Speed of shortening

Thicker muscle

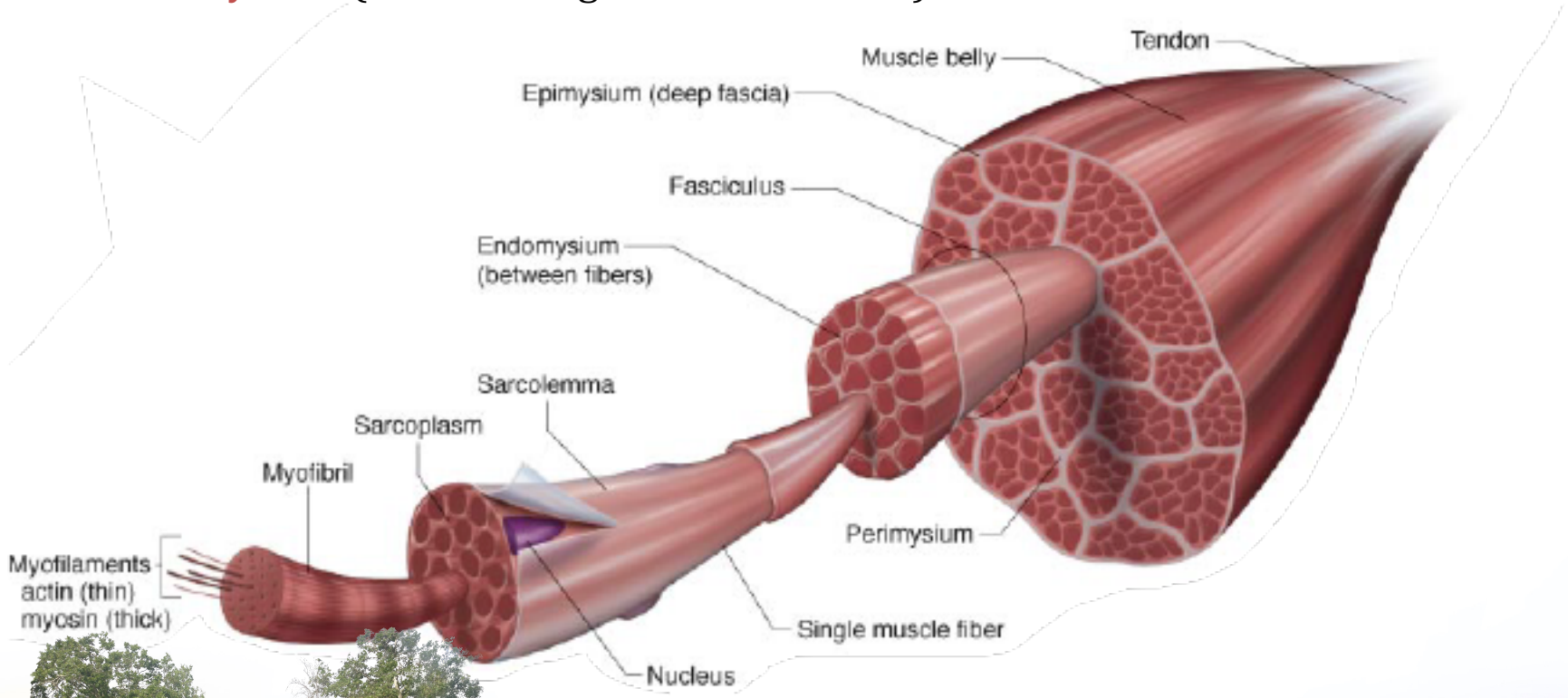
⇧ Force development



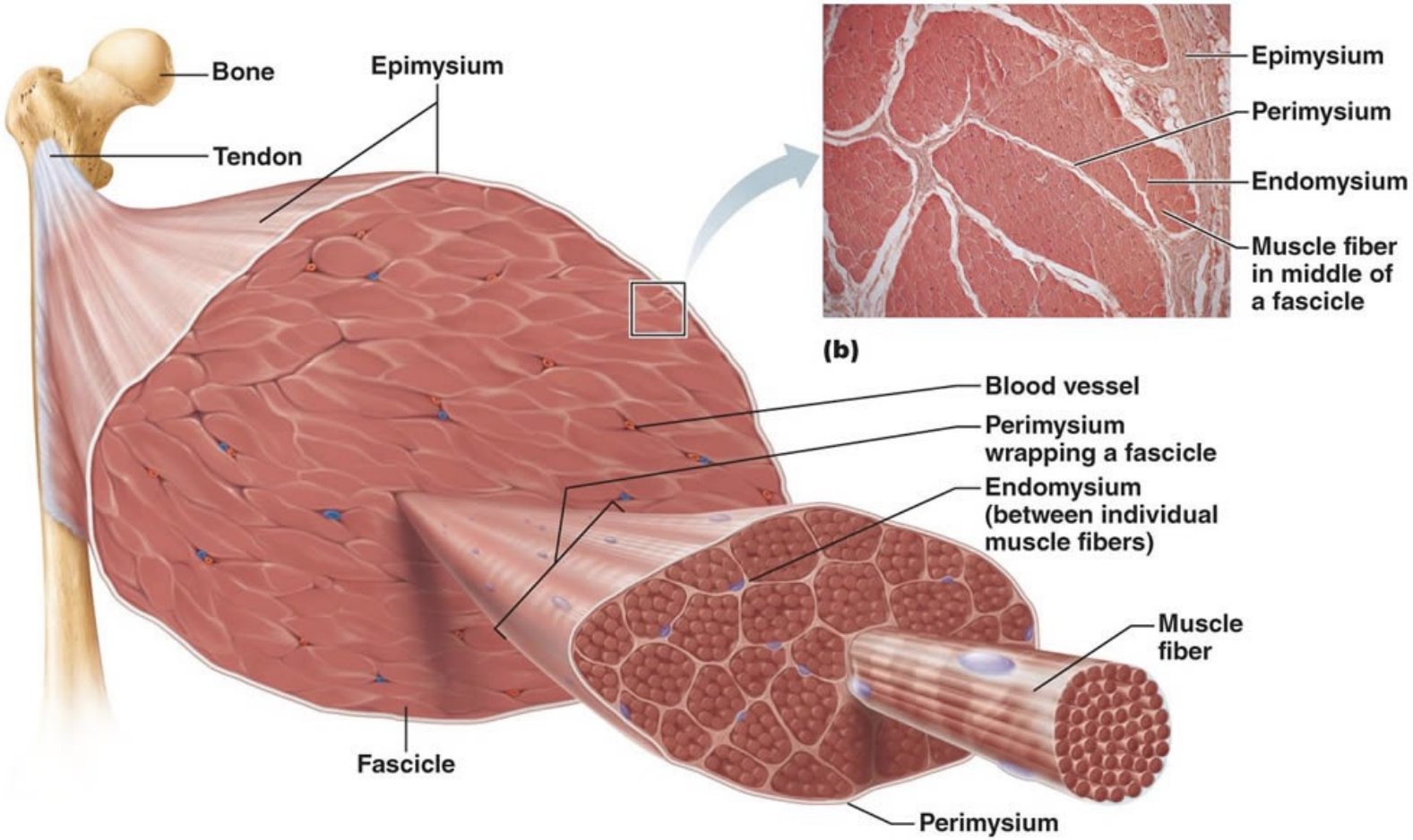


Macrostructure:

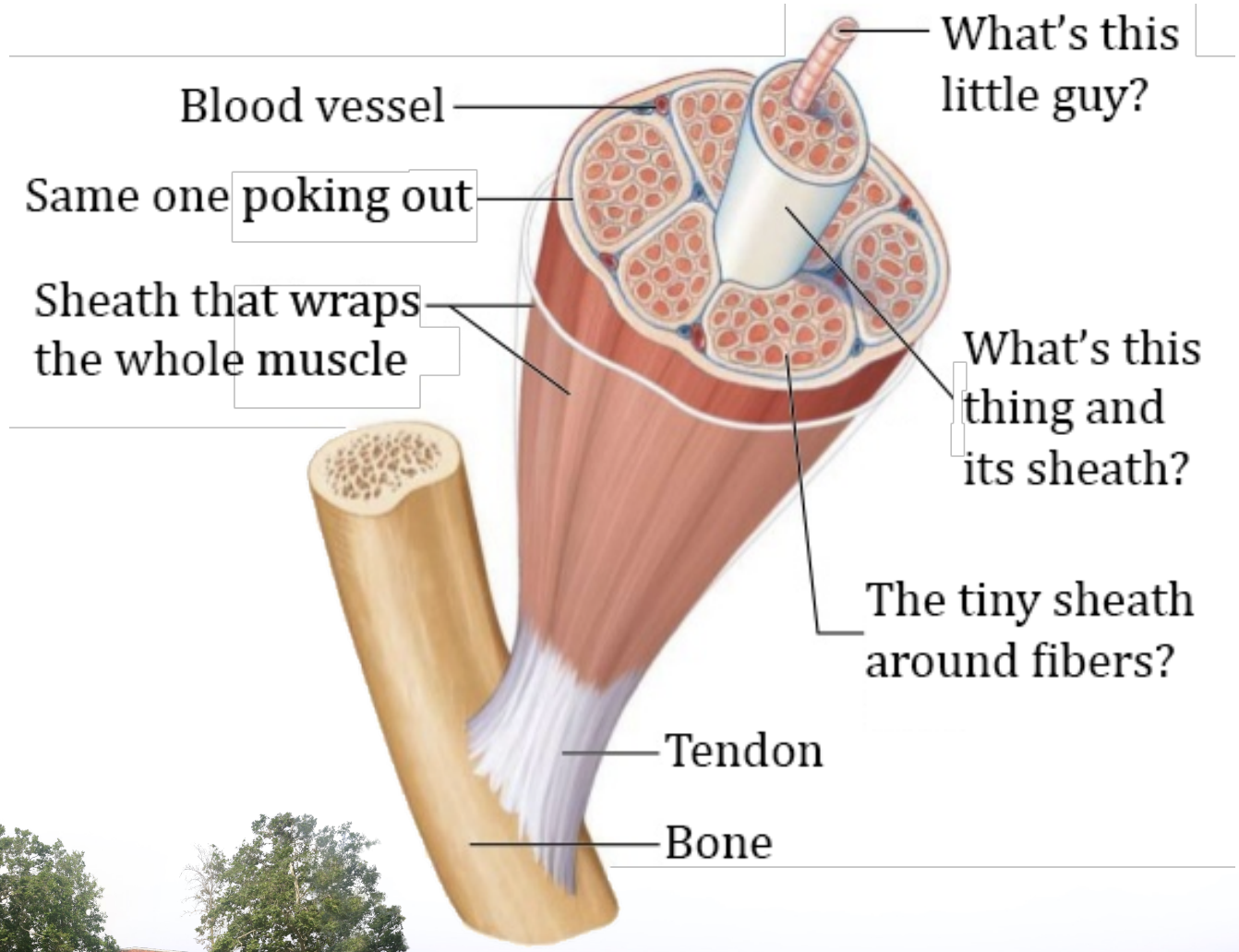
- Epimysium (outer layer, surrounding whole muscle)
- Perimysium (surrounding each fasciculus, i.e., group of fibers)
- Endomysium (surrounding individual fibers)

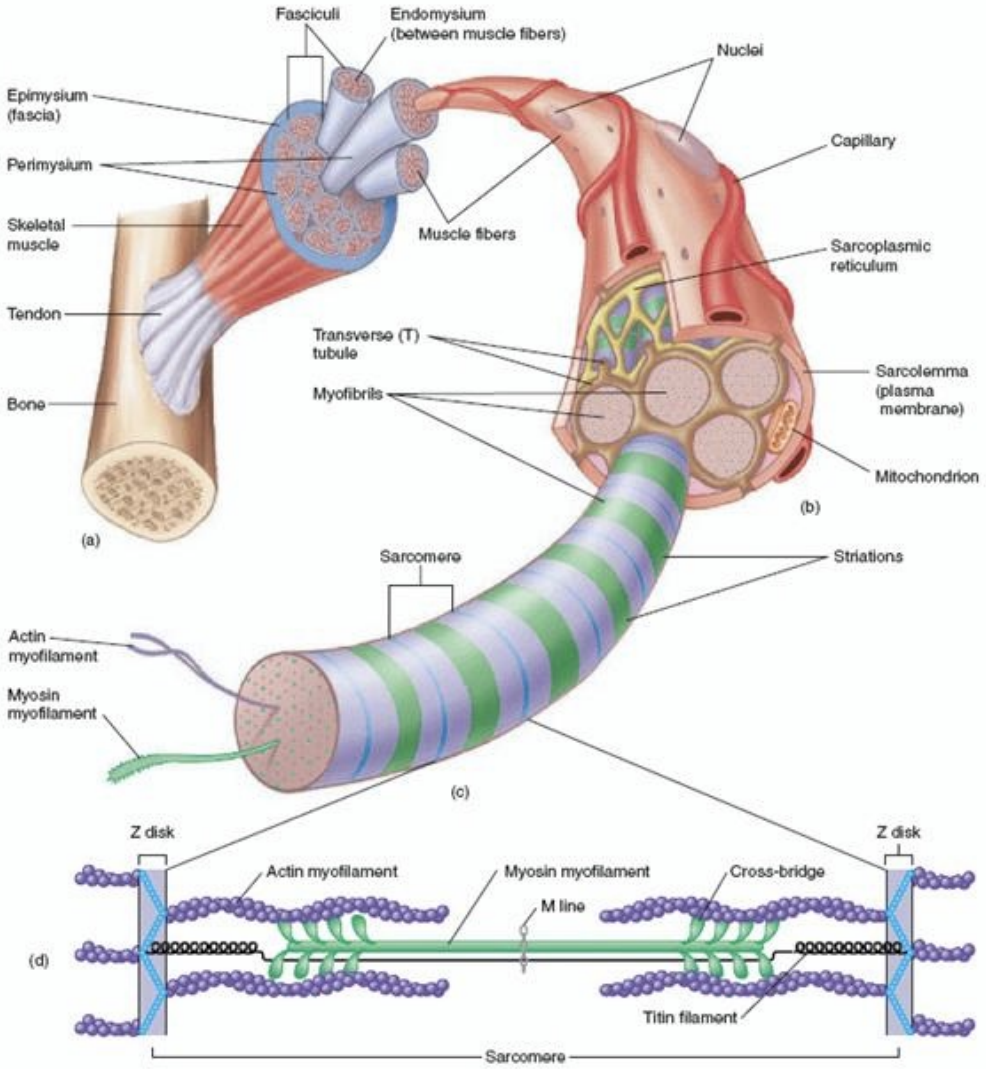


Macro and micro structures:



Macro and micro structures:





Macro and micro structures:

1. Muscle
2. Fascicles (bundles)
3. Muscle Fiber (cell)
4. Myofibril
5. Sarcomere
6. Myofilaments
    1. Actin
    2. Myosin

Structural Hierarchy

Number of fibers in various muscles

Muscle	Number of muscle fibers
First lumbrical	10,250[a]
External rectus	27,000
Platysma	27,000
First dorsal interosseous	40,500
Sartorius	128,150[a]
Brachioradialis	129,200[a]
Tibialis anterior	271,350
Medial gastrocnemius	1,033,000

Note. Results given to nearest 50. [a]Average values. Value for sartorius from MacCallum 1898; all others from Feinstein *et al.* 1955.

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Note. Results given to nearest 50. [a]Average values. Value for sartorius from MacCallum 1898; all others from Feinstein *et al.* 1955.

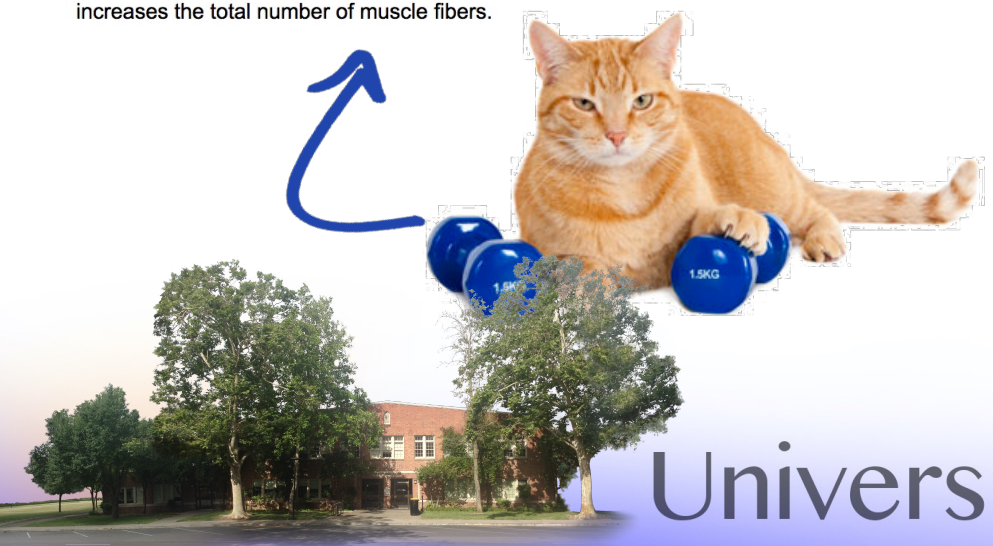
European Journal of Applied Physiology and Occupational Physiology

1986;55(2):137-41.

Gonyea WJ, Sale DG, Gonyea FB, Mikesky A.

Abstract

The effect of weight-lifting, which induced muscular enlargement, on fiber number was tested in the flexor carpi radialis muscle by operantly conditioning 6 cats to flex their right wrist against increasing resistance for an average of 101 weeks. The left was used as a control. At the end of training, the cats were performing "one-arm" lifts with an average of 57% of their body weight. There was an 11% greater muscle weight (P less than 0.01) and 9% (P less than 0.02) more fibers in the exercised muscles from the right limb than in the left. This study using a different method, supports our earlier observations that prolonged weight-lifting exercise significantly increases the total number of muscle fibers.



You probably have – roughly – the same number of fibers throughout your adult life.

0195-9131/93/2512-1333$3.00/0
MEDICINE AND SCIENCE IN SPORTS AND EXERCISE

Skeletal muscle fiber hyperplasia

JOSE ANTONIO and WILLIAM J. GONYEA

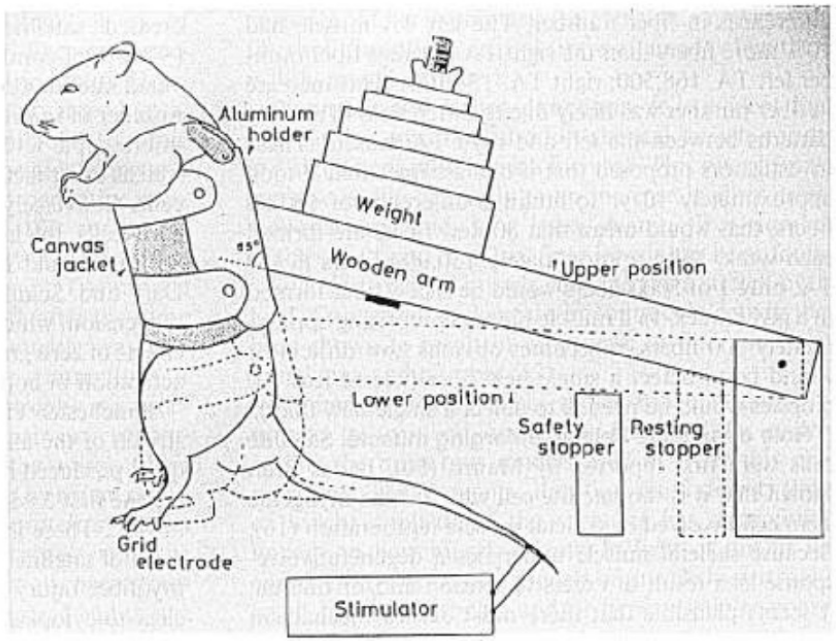
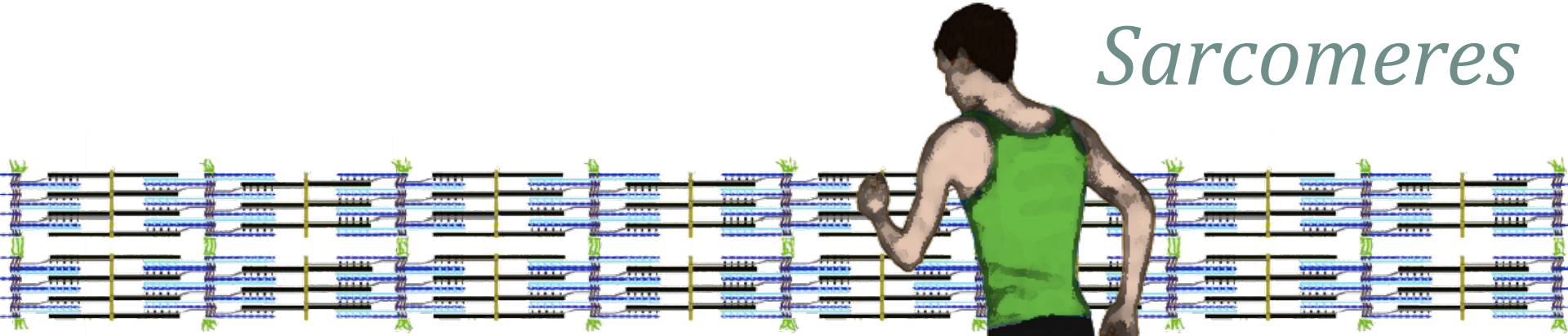
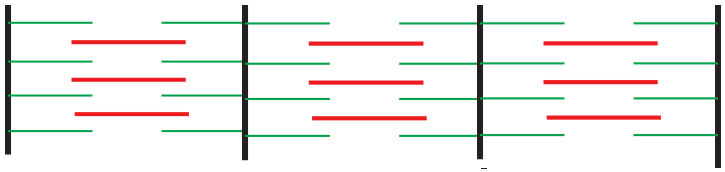
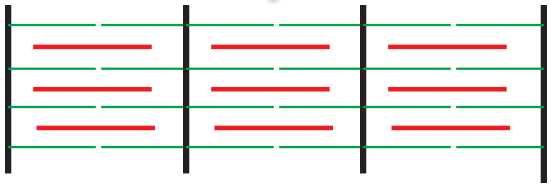


Figure 6—Rat weight lifting model developed by Tamaki et al. (66). Rats were fastened to a wooden arm via a canvas jacket with the resistance or weight placed on the wooden arm. Rats were conditioned to perform a squat movement through the use of electrical stimulation given to the tail. As a result of the electrical stimulation, the rats extended their legs and hips, which would lift the weight attached to the wooden arm. A safety stopper was set to prevent hyperflexion of the knee and ankle while a resting stopper was used to relax the legs during periods of rest. The plantaris muscle of rats trained in this fashion has shown modest fiber hyperplasia (14% increase in fiber number) (66).

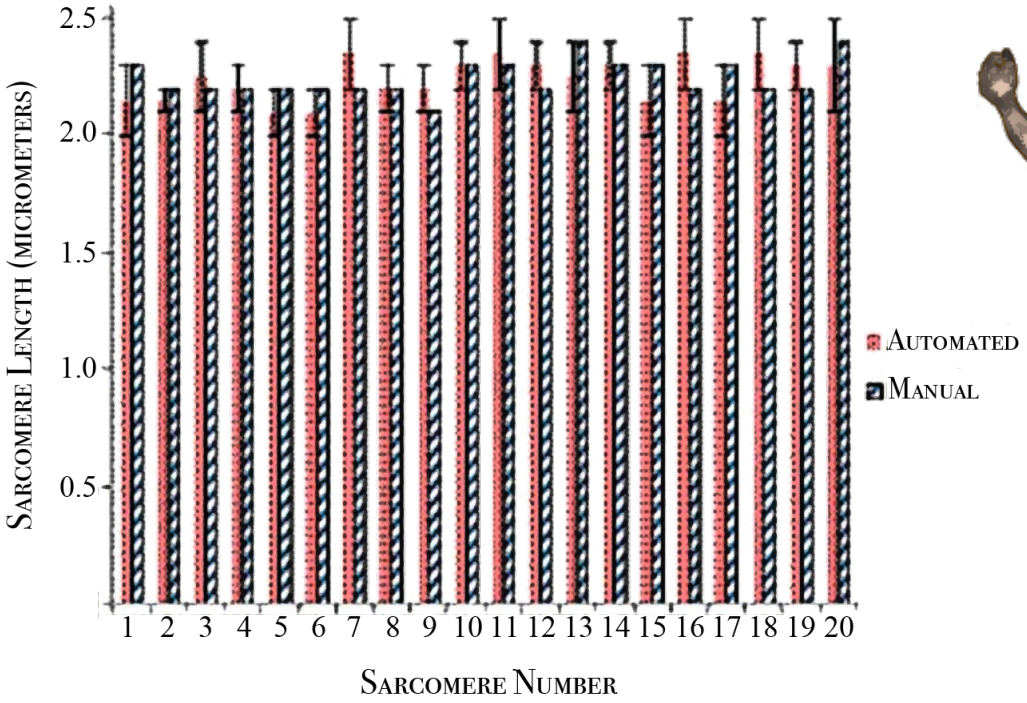
Sarcomeres

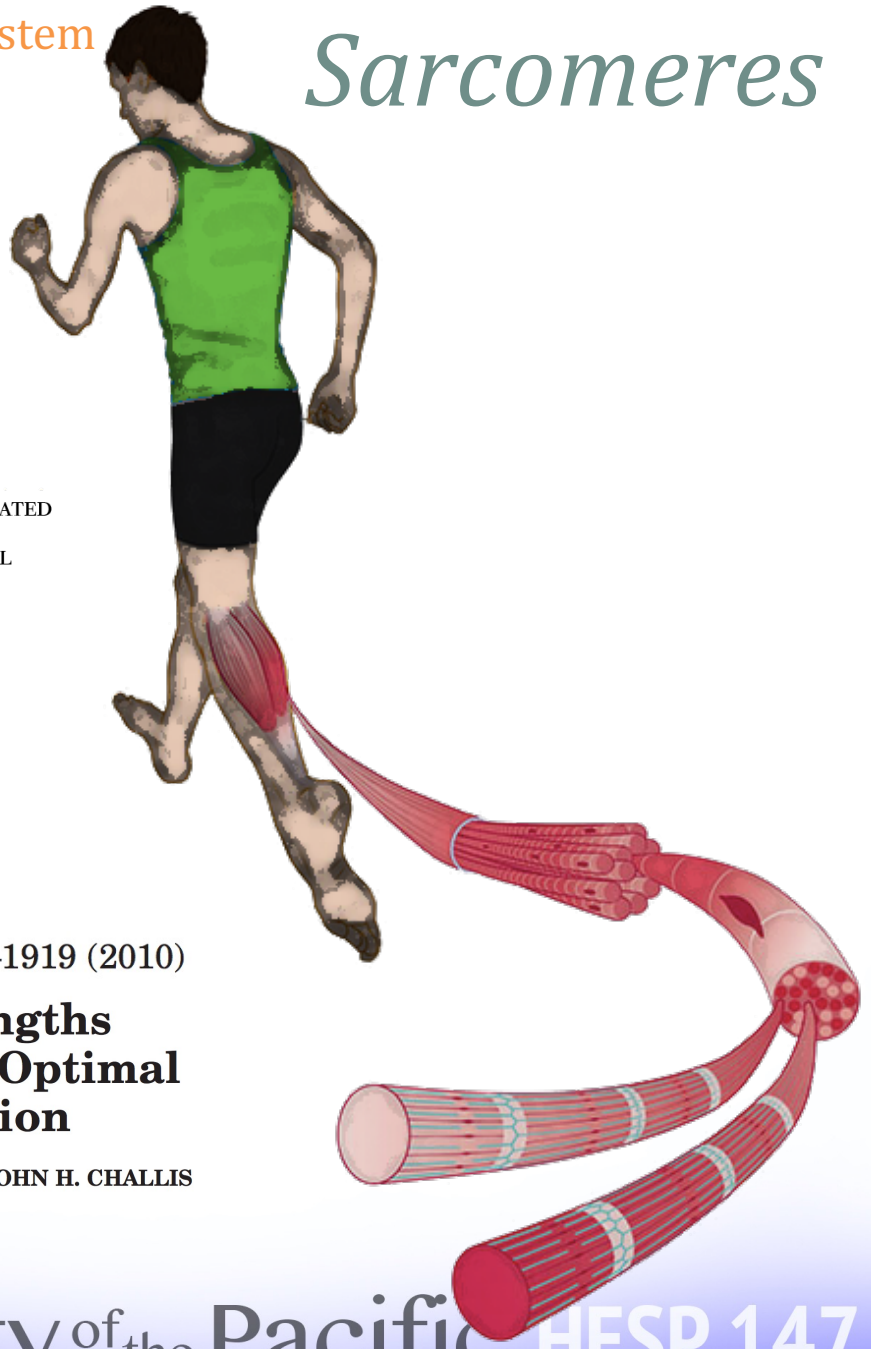






Sarcomeres

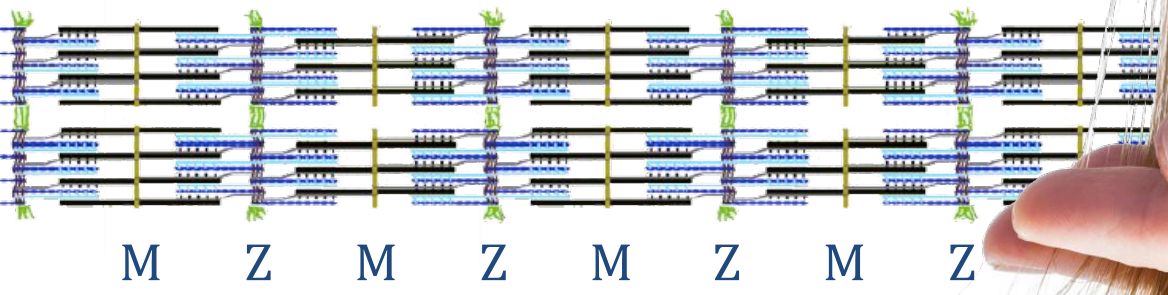




THE ANATOMICAL RECORD 293:1913–1919 (2010)

Individual Sarcomere Lengths in Whole Muscle Fibers and Optimal Fiber Length Computation

BENJA… …OLINO,* MICHAEL J. ELLIS, AND JOHN H. CHALLIS



Each individual sarcomere is approximately 2.2 to 3.3 micrometers in length.

One human hair is *probably roughly* 20-150 micrometers in diameter.

Numbers of Sarcomeres in Human Muscles

	Number of sarcomeres per fiber ($\times 10^4$)		
Muscle	**I**[a]	**II**[a]	**III**[a]
Tibialis posterior	1.1	1.5	0.8
Soleus	1.4	—	—
Medial gastrocnemius	1.6	1.5	1.5
Semitendinosus	5.8	6.6	—
Gracilis	8.1	9.3	8.4
Sartorius	15.3	17.4	13.5

[a]Refers to individual limbs analyzed. The values for the three thigh muscles at the bottom of the table do not take into account the fibrous inscriptions in the belly, and the true values will be rather lower than those stated.

Data from "Muscle Architecture of the Human Lower Limb" by T. L. Wickiewicz, R. R. Roy, P. L. Powell, and V. R. Edgerton, 1983, *Clinical Orthopaedics and Related Research*, 179, p. 277.

~15,000 sarcomeres long.

Medial gastrocnemius	1.6	1.5	1.5

If 15,000 sarcomeres each contract .2 micrometers, that's 3,000 micrometers at the whole muscle (i.e., 3 millimeters, which moves your bones a lot more).

~15,000 sarcomeres long.

Medial gastrocnemius	1.6	1.5	1.5

Muscles get longer: sarcomerogenesis
Muscles get shorter: sarcomerolysis

Physiological Reports ISSN 2051-817X Physiological Reports

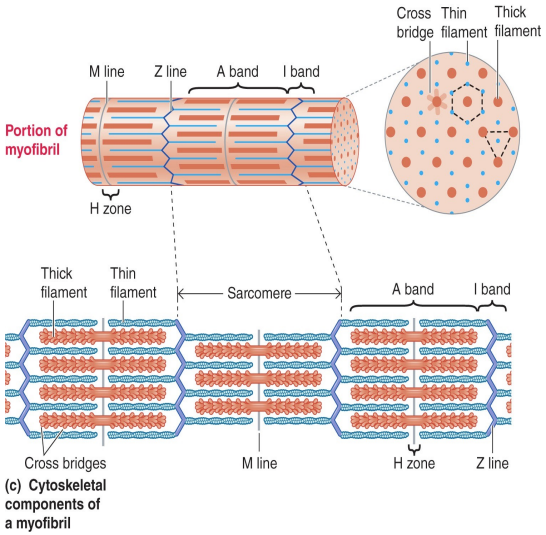
Muscle damage and inflammation after eccentric exercise: can the repeated bout effect be removed?

Nikos V. Margaritelis[1], Anastasios A. Theodorou[2], Vasilios Baltzopoulos[3], Constantinos N. Maganaris[4], Vassilis Paschalis[2,5], Antonios Kyparos[1] & Michalis G. Nikolaidis[1]

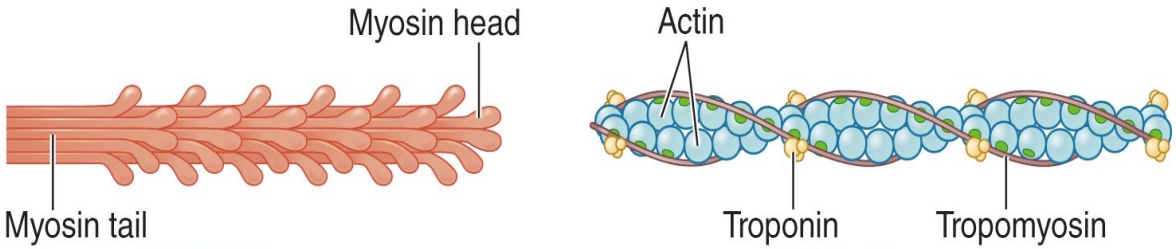
Muscle Anatomy

What's the stuff inside of a sarcomere?

Muscle Anatomy



(c) Cytoskeletal components of a myofibril



(d) Protein components of thick and thin filaments

What are the "myofilaments"

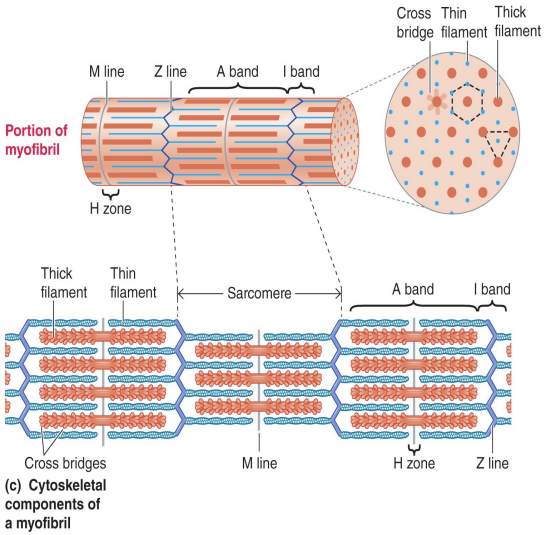
?

Myofilaments:

Actin and Myosin

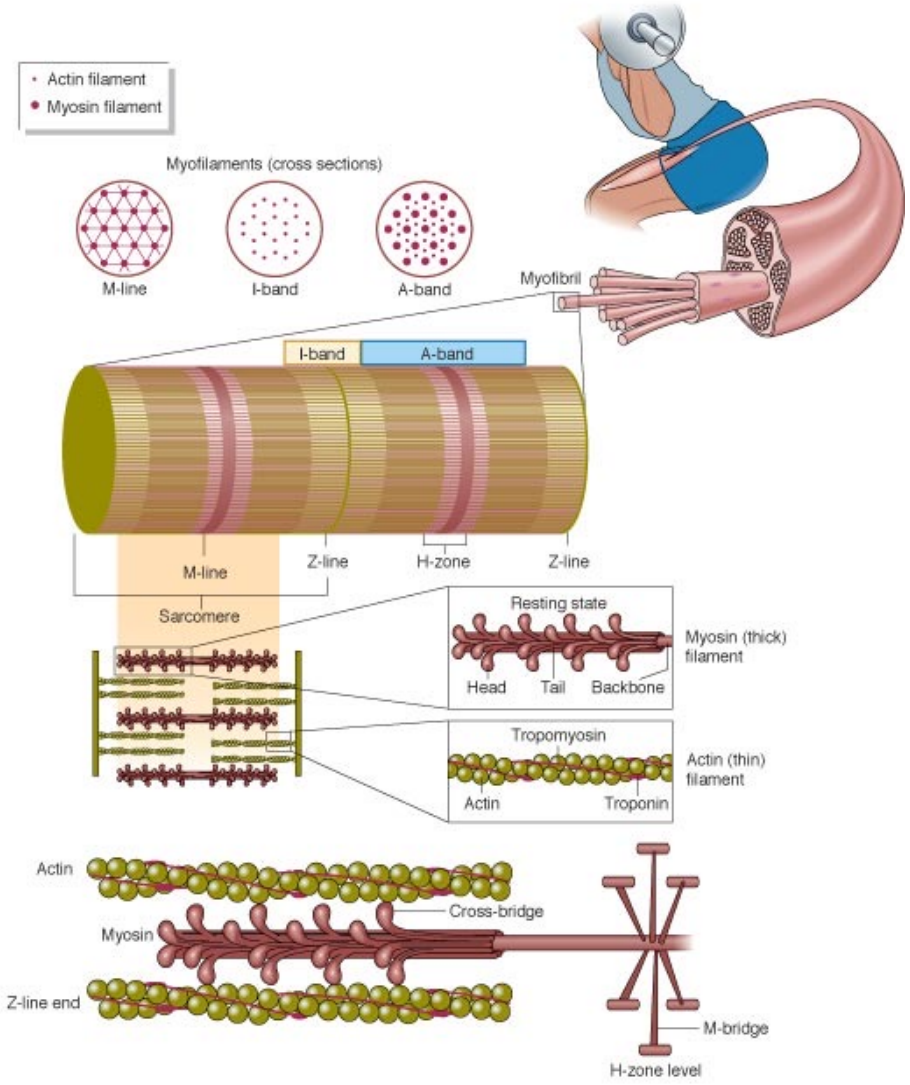
(The things that "slide" in sliding "filament" theory)

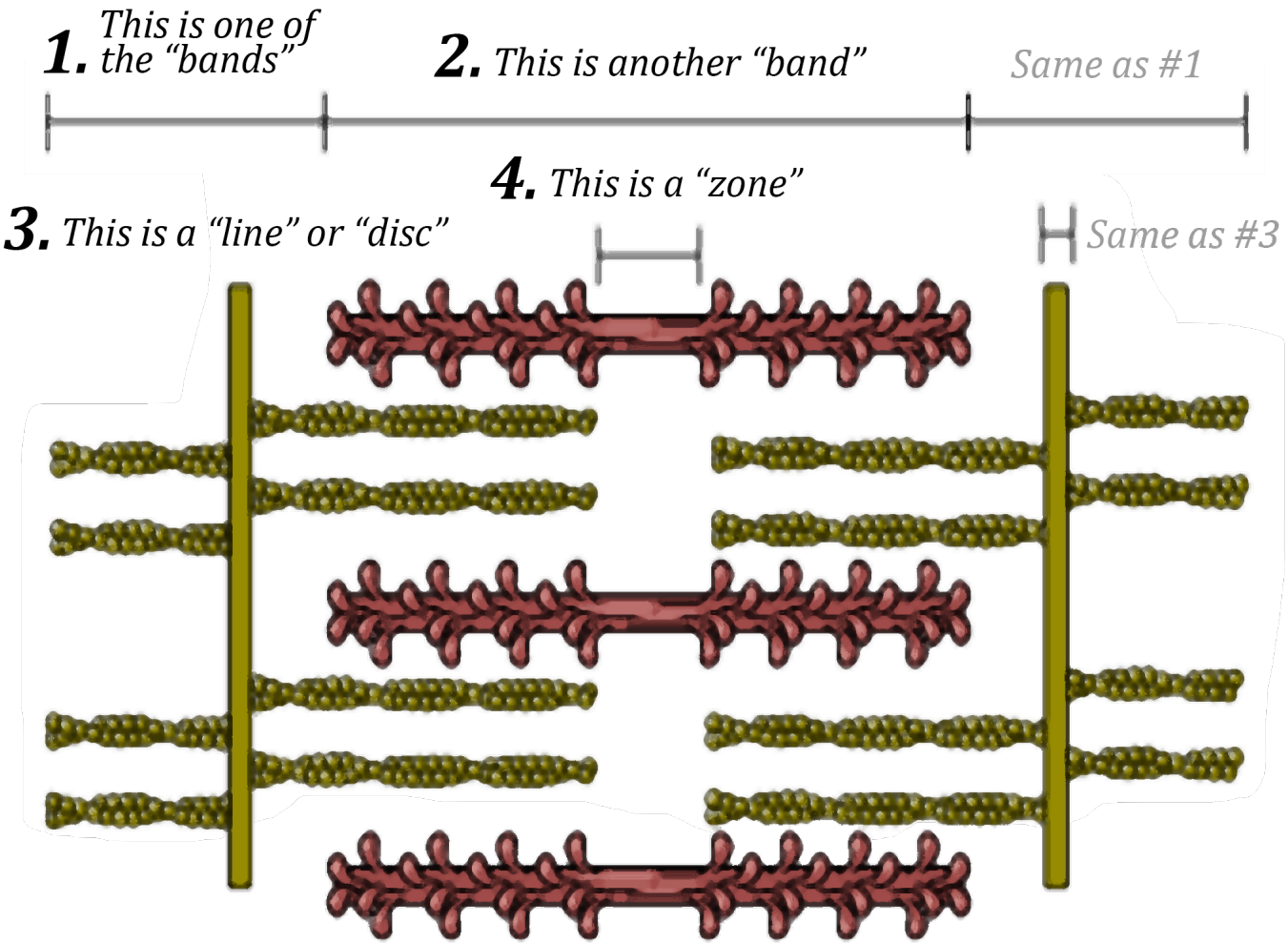
Muscle Anatomy



What are the "myofilaments"

?




1. This is one of the "bands"
2. This is another "band"
Same as #1
4. This is a "zone"
3. This is a "line" or "disc"
Same as #3

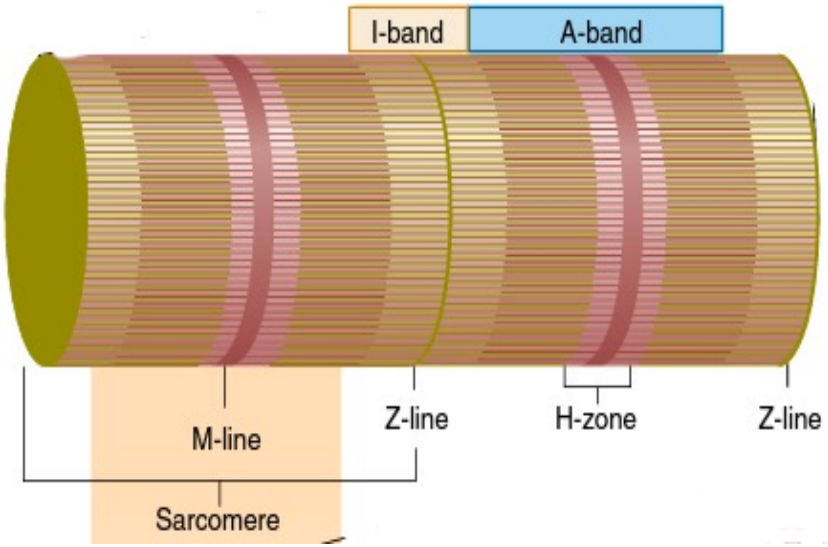
A-Band: Alignment of myosin filaments

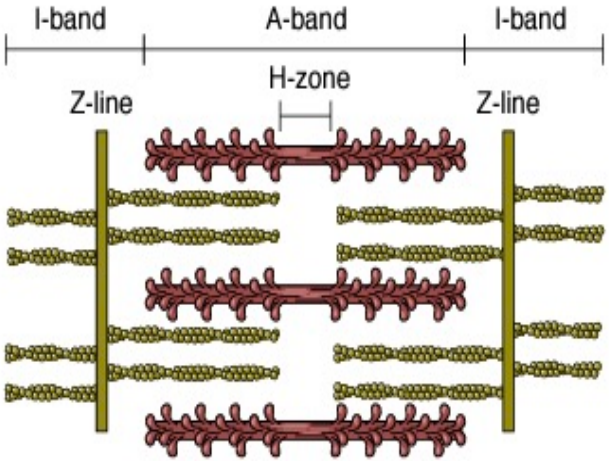
I-Band: Two adjacent sarcomeres that contain only actin filaments

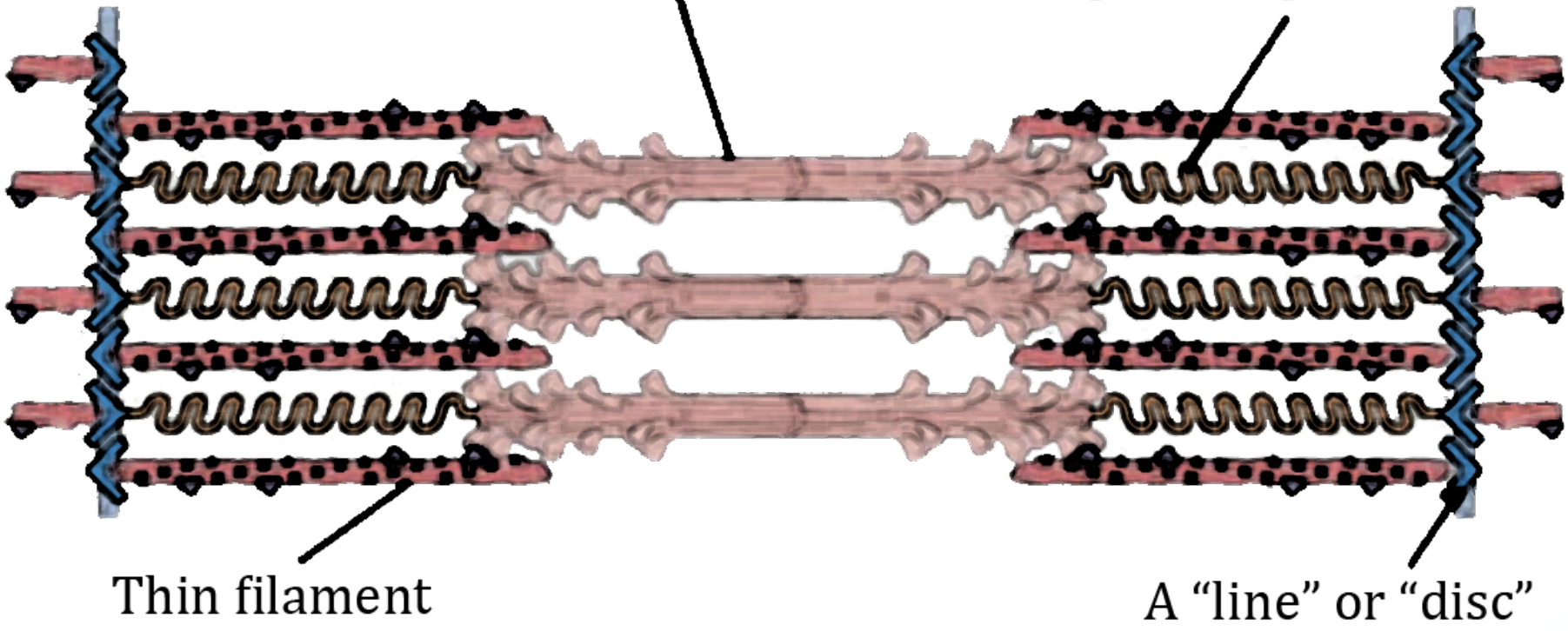
Z-Line: Middle of I-band

H-Zone: Center of sarcomere where only myosin is present

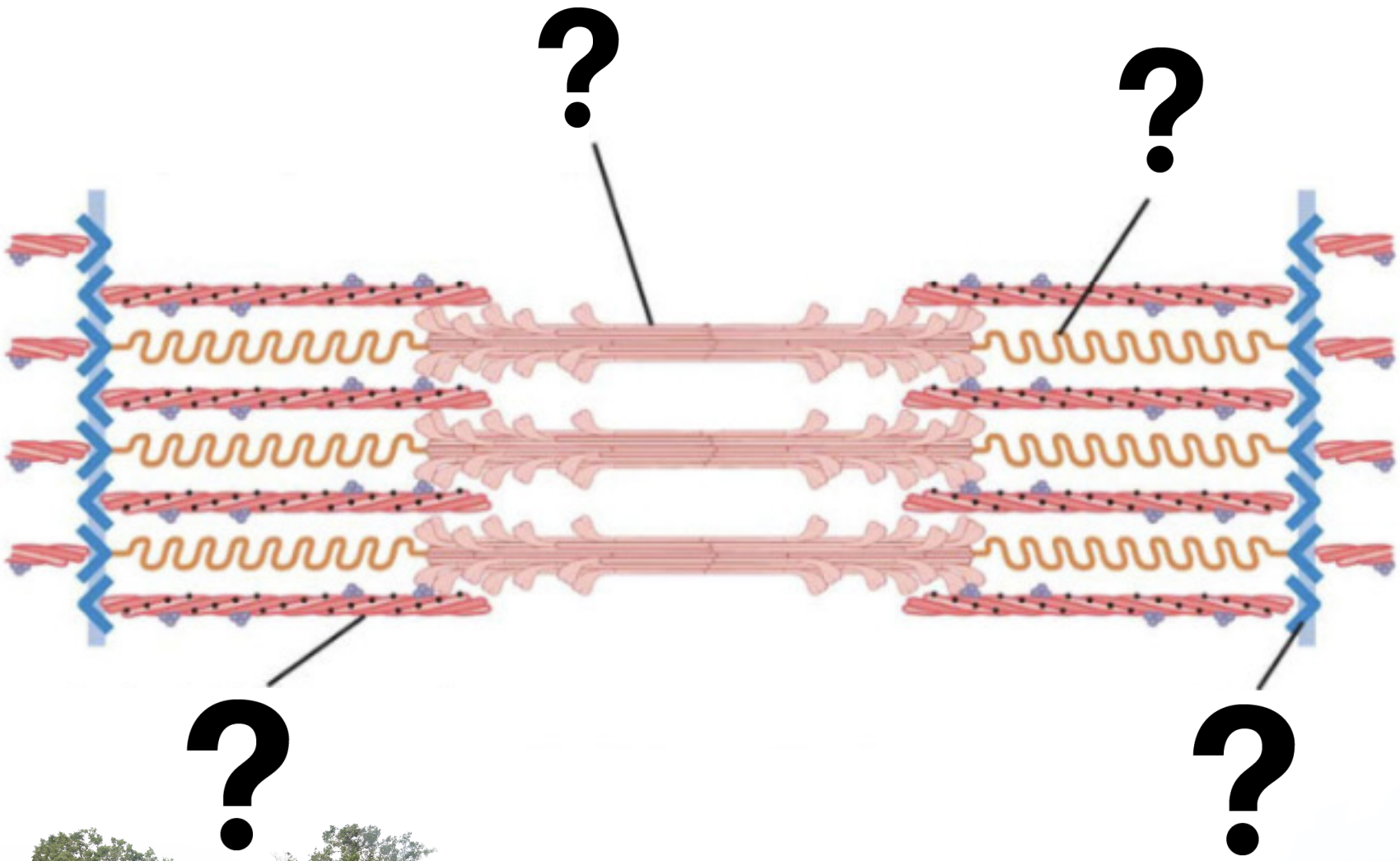
M-Line: Right down the middle of the H-Zone

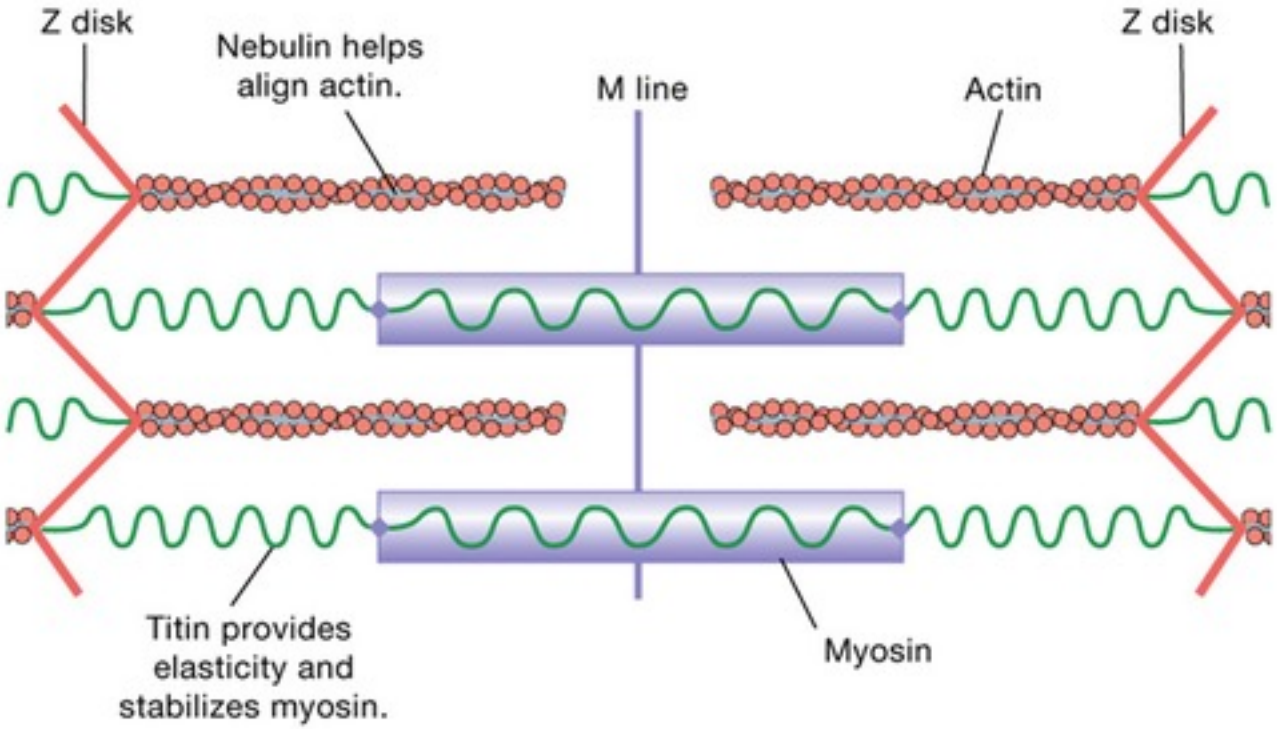


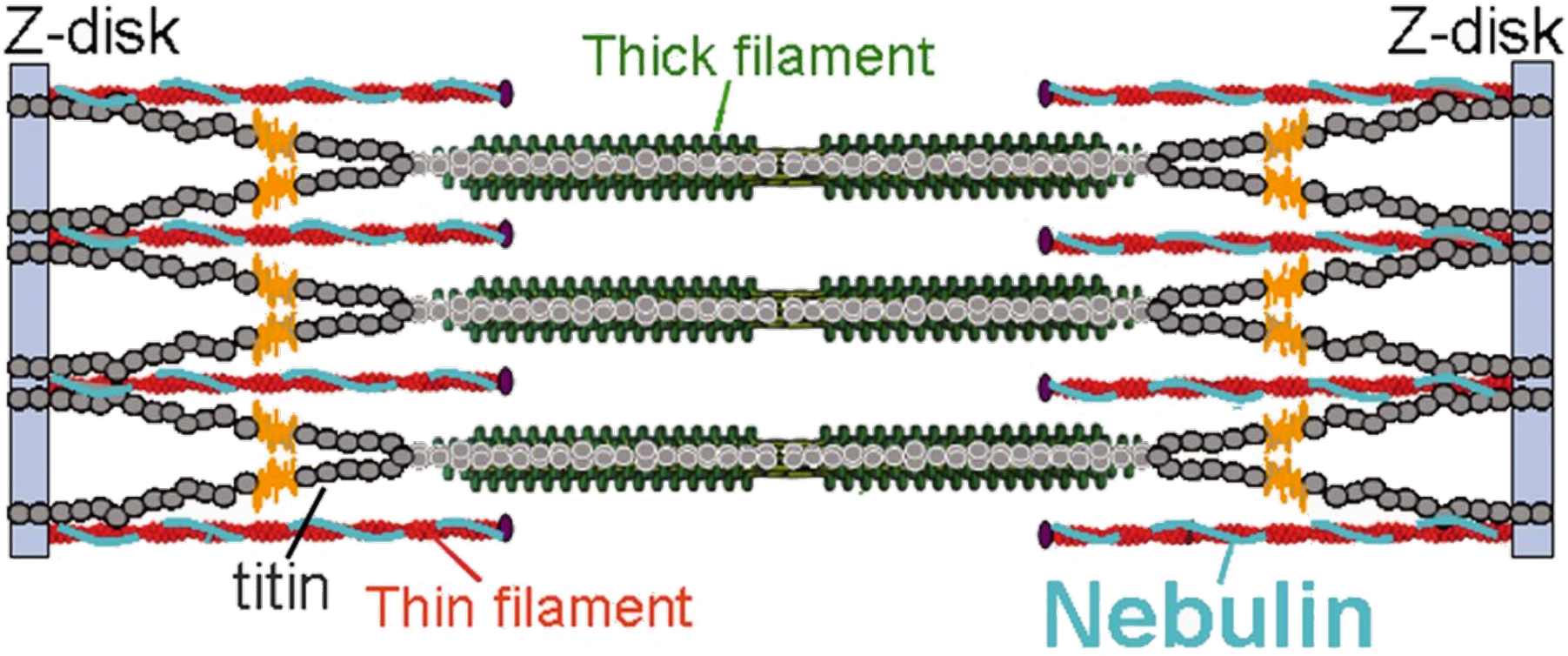


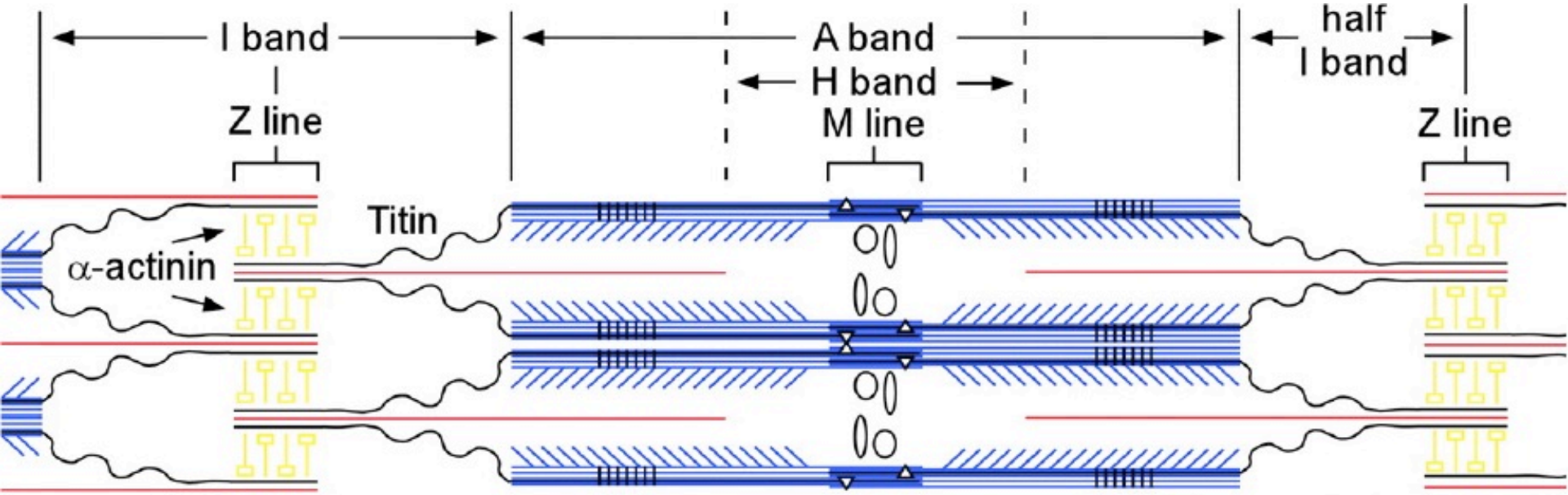

Thick filament
An important protein
Thin filament
A “line” or “disc”

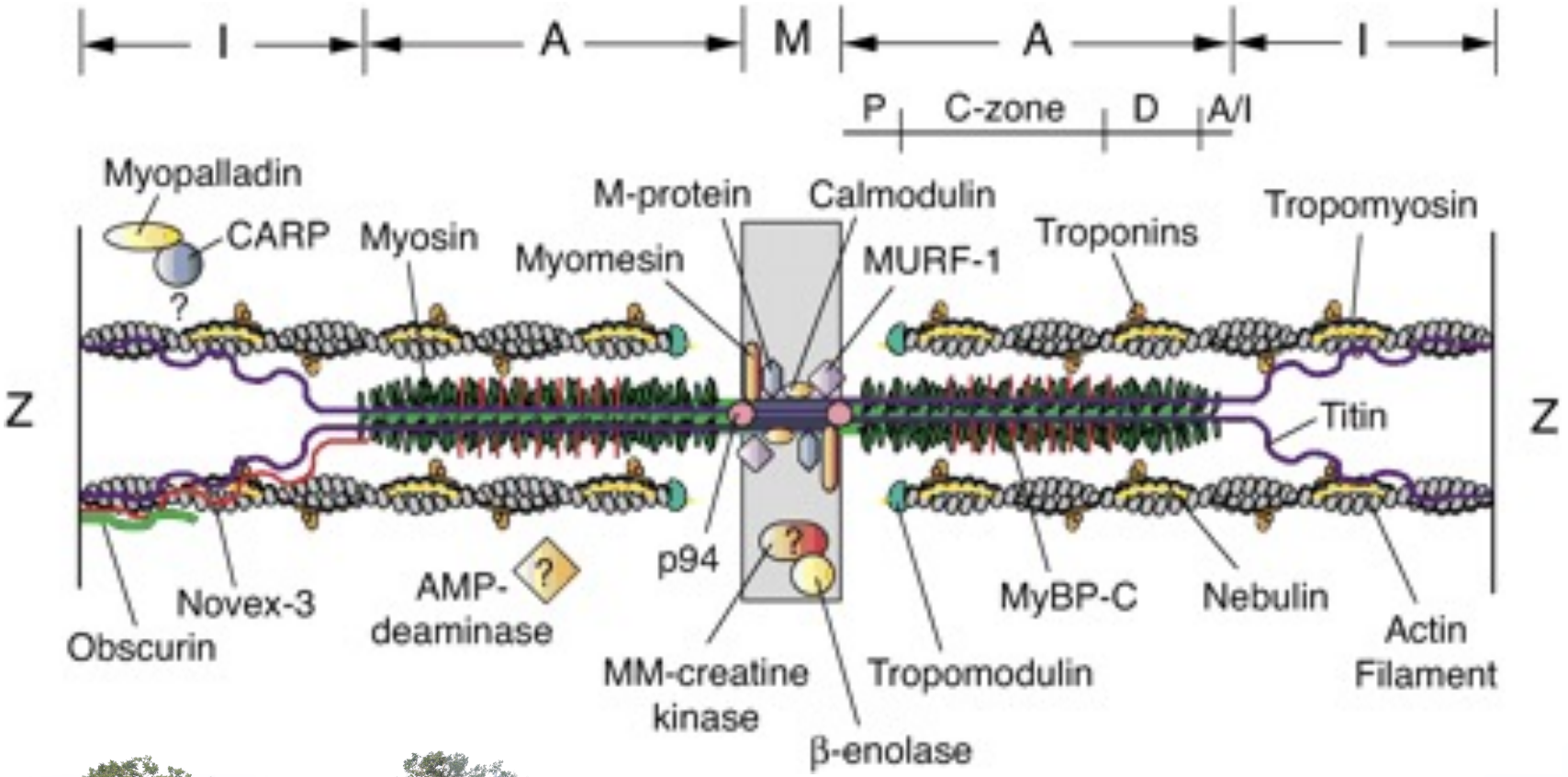
Actin: Myosin: Z-Disc: M-Line: Titin:



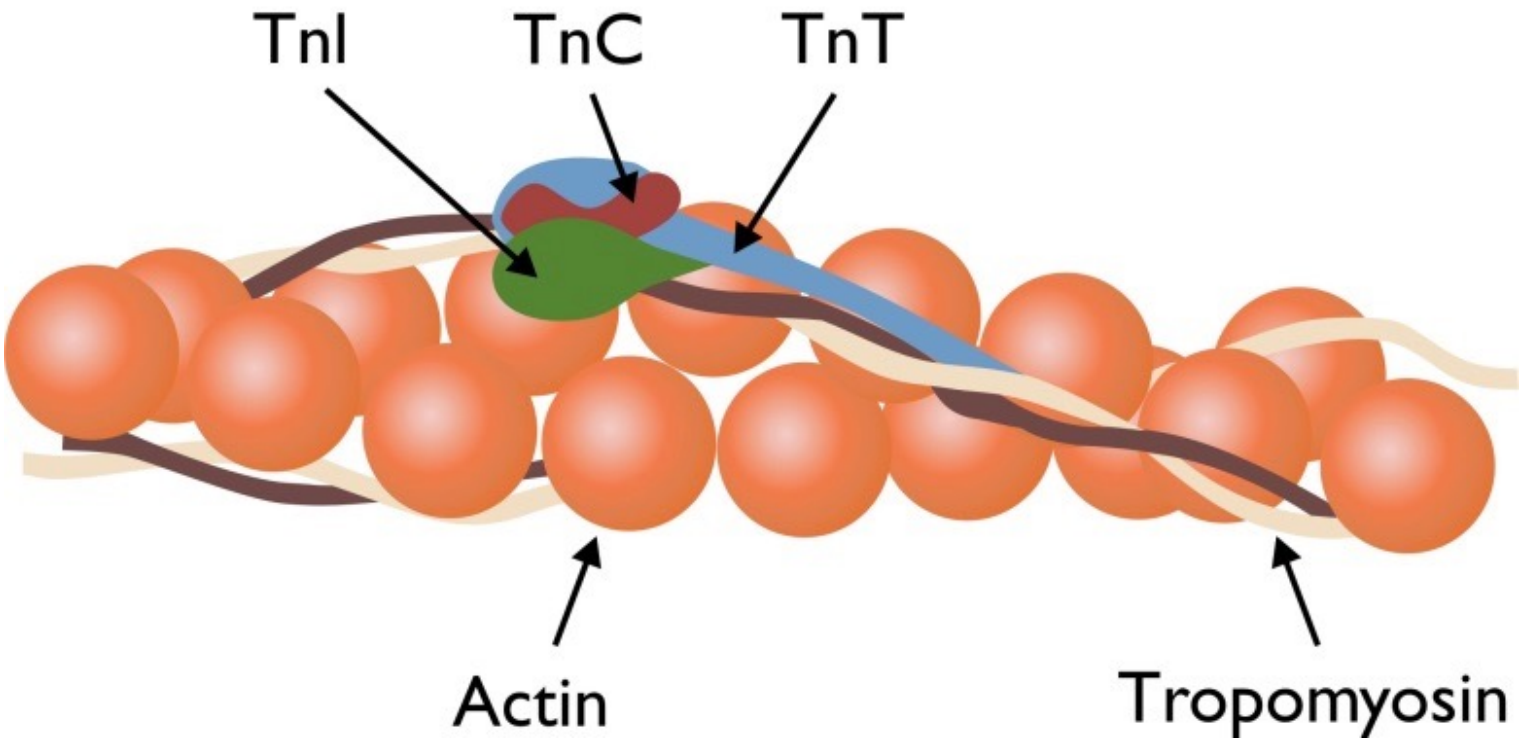

Z disk
Nebulin helps align actin.
M line
Actin
Z disk
Titin provides elasticity and stabilizes myosin.
Myosin

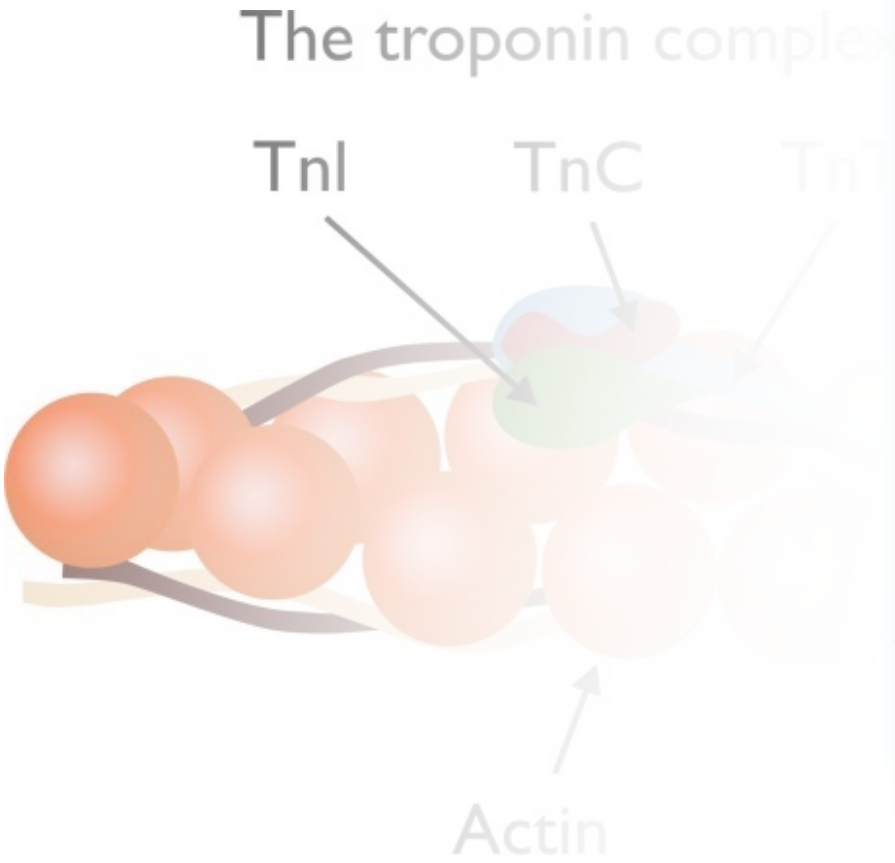

Z-disk
Thick filament
Z-disk
titin
Thin filament
Nebulin

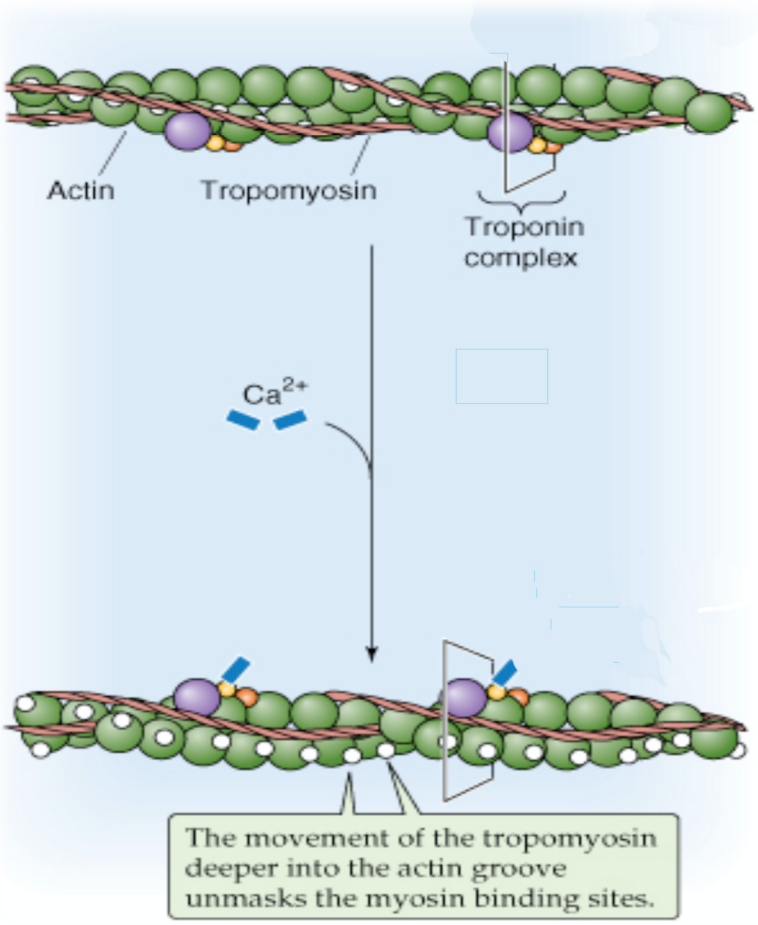

I band
Z line
A band
H band
M line
half
I band
Z line
Titin
α-actinin

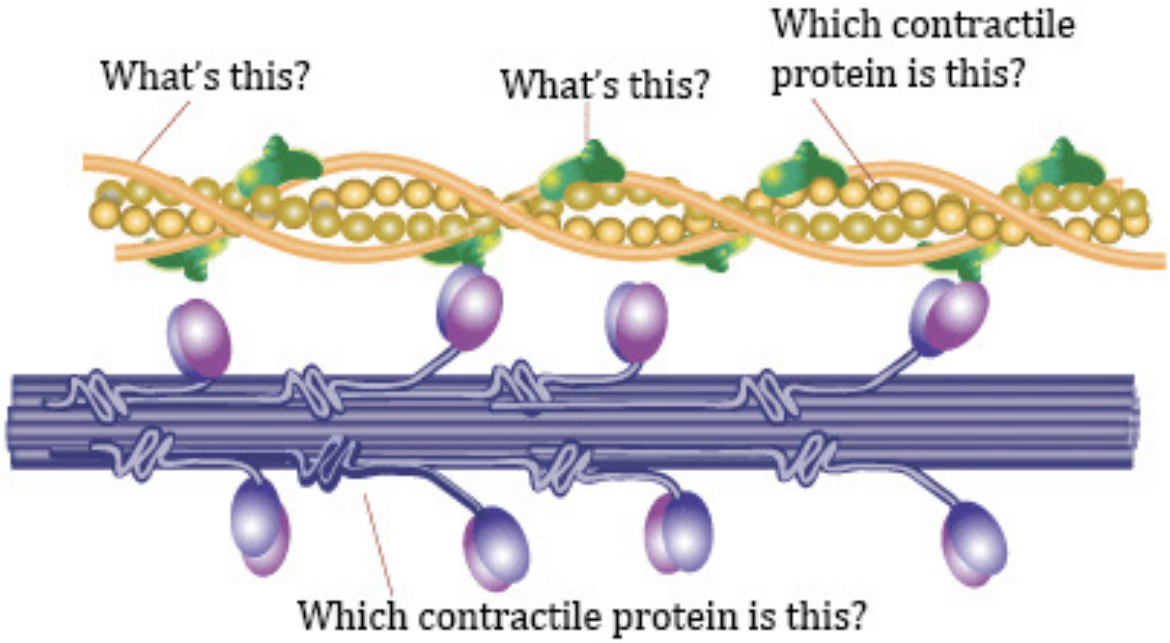

I
A
M
A
I
P
C-zone
D
A/I
Myopalladin
CARP
?
Myosin
Myomesin
M-protein
Calmodulin
MURF-1
Troponins
Tropomyosin
Z
Titin
Z
Obscurin
Novex-3
AMP-
deaminase
?
p94
?
MyBP-C
Nebulin
Actin
Filament
MM-creatine
kinase
Tropomodulin
β-enolase

The troponin complex:

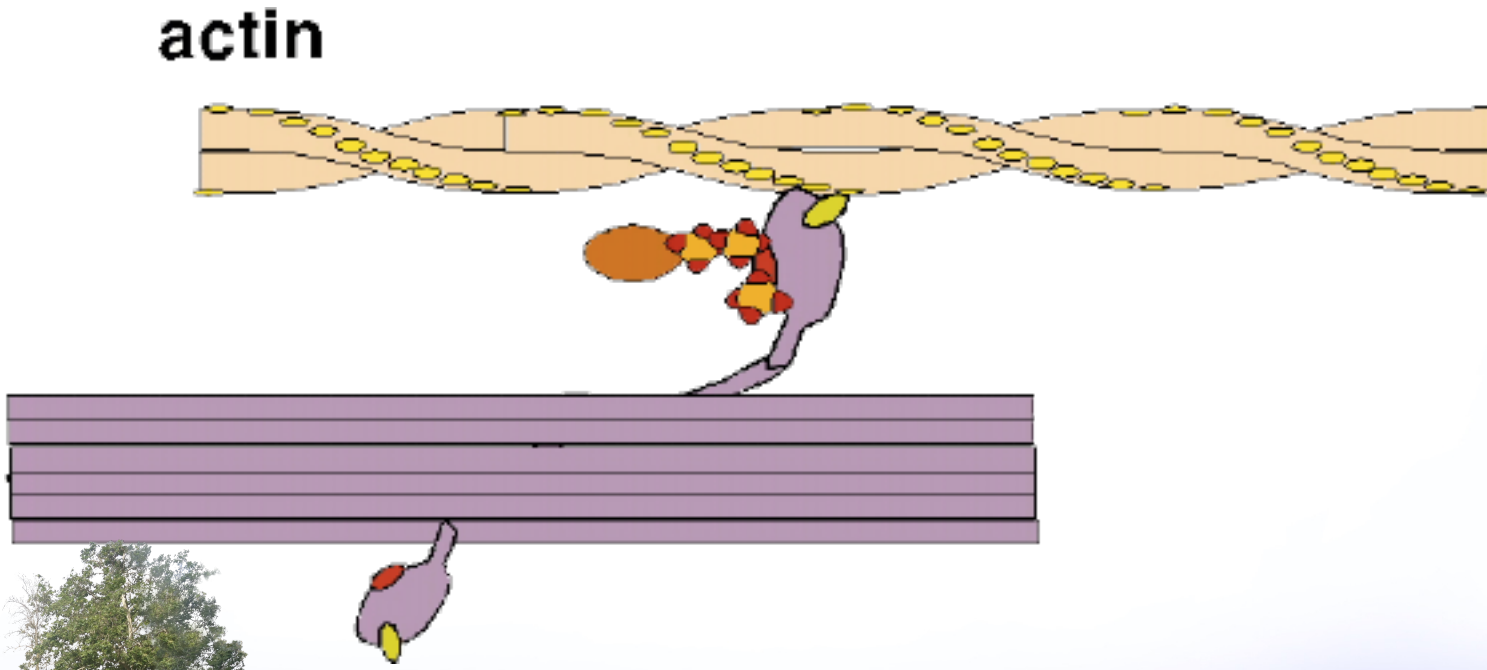



The troponin complex
TnI
TnC
TnT
Actin

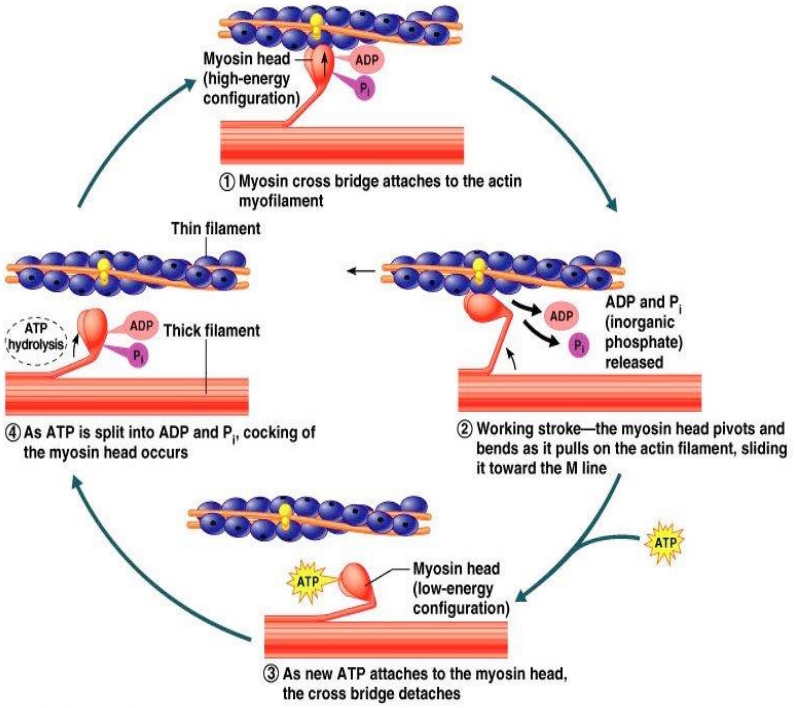

Actin
Tropomyosin
Troponin complex
Ca^{2+}
The movement of the tropomyosin deeper into the actin groove unmasks the myosin binding sites.

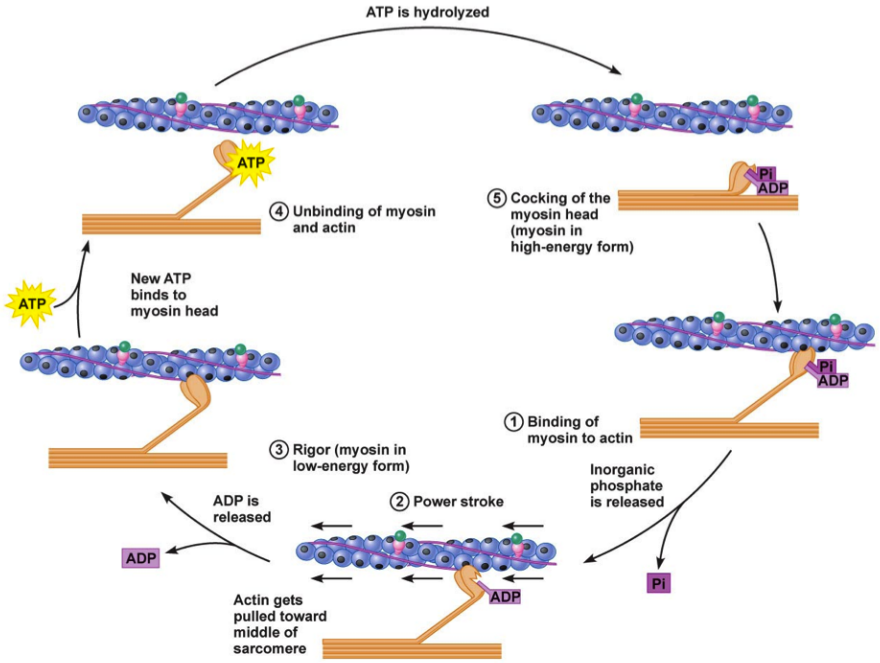

What's this?
What's this?
Which contractile protein is this?
Which contractile protein is this?

$$ATP^{4-} + H_2O \leftarrow ATPase \rightarrow ADP^{3-} + HPO_4^{2-} + H^+$$



Cross-Bridge Cycling





Cross-Bridge Cycling

1. There's a nerve impulse that reaches the muscle.

2. Calcium is released from the sarcoplasmic reticula.

3. That calcium binds to troponin (on the surface of the actin filaments), which tugs the tropomyosin off of the myosin binding sites.

4. Myosin hydrolyzes an ATP molecule, which cocks its head, and it binds to those newly exposed binding sites.

5. Myosin is still hanging onto its ADP and phosphate (byproducts of the ATP hydrolysis reaction), so the bond it has with actin is weak.

Cross-Bridge Cycling

6. Myosin releases its Pi, which activates the strong conformation.

7. The myosin head release its ADP and performs its “power stroke”, which creates movement in the contractile apparatus.

8. A new ATP molecule binds to the myosin head, which switches its bond to the weak conformation.

9. The myosin head is released from the binding site on the actin.

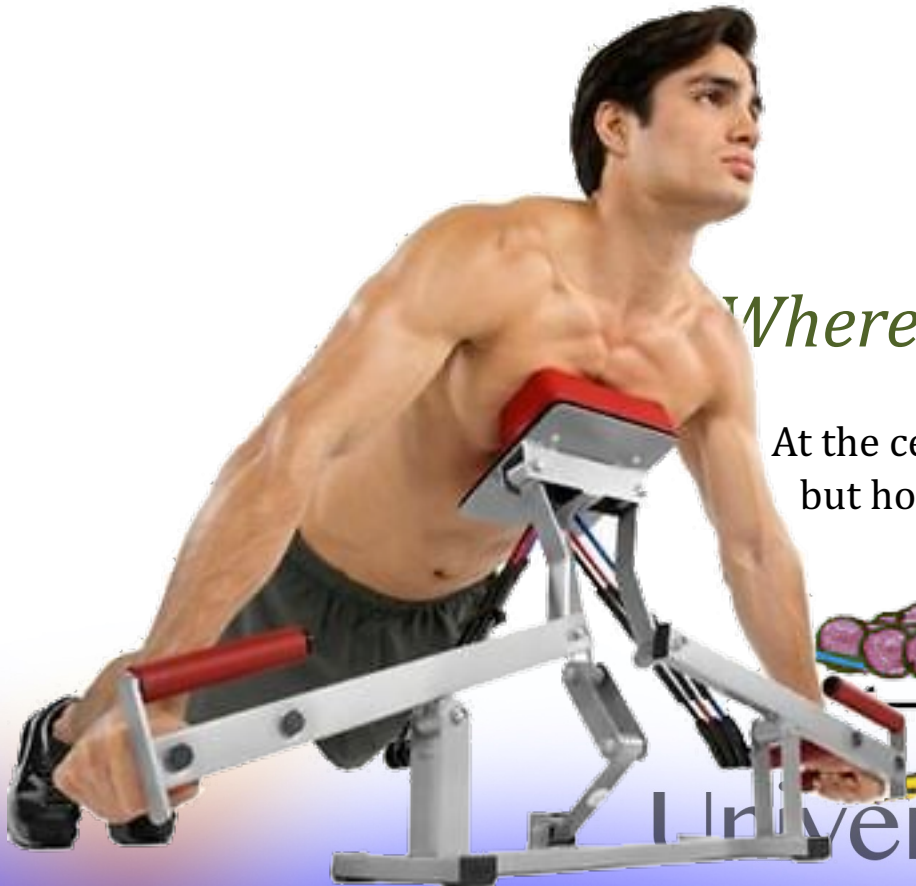
10. Myosin hydrolyzes the ATP, re-cocks its head, and latches onto a new binding site.

And so on. Muscle contraction continues until the calcium leaves.

1. How many total muscle fibers contribute to the contraction.

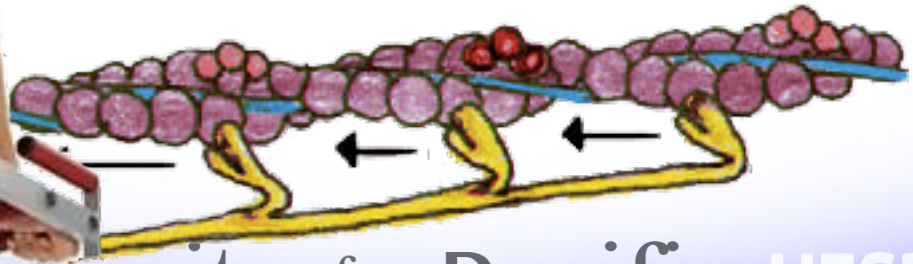
2. How much actin and myosin are in each of those contributing fibers.

3. How well the individual actin-myosin cross-bridges are functioning.

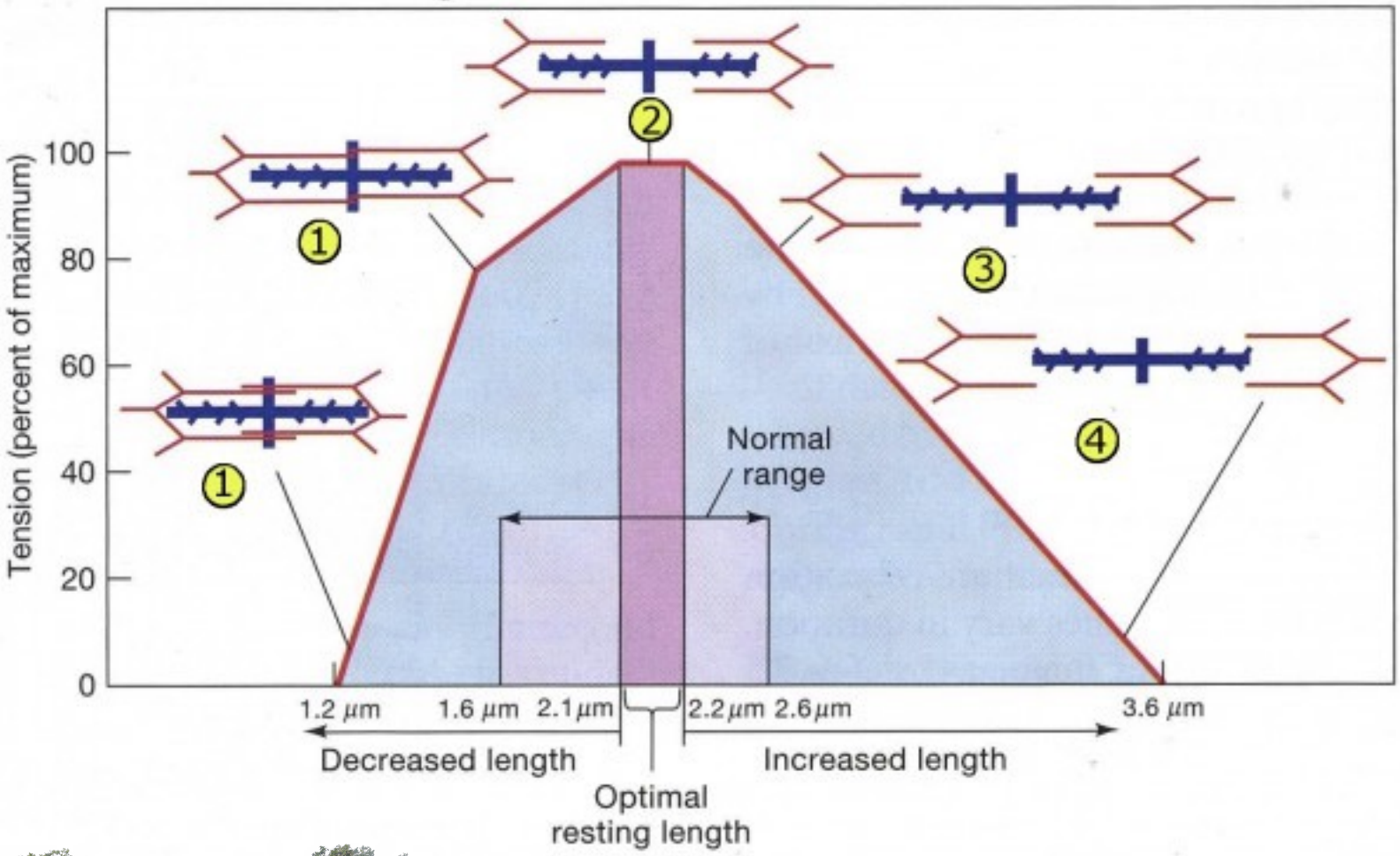


Where do you get your strength

At the cellular level, you produce it with actin-myosin bonds, but how do you lift a light weight versus a heavy weight?



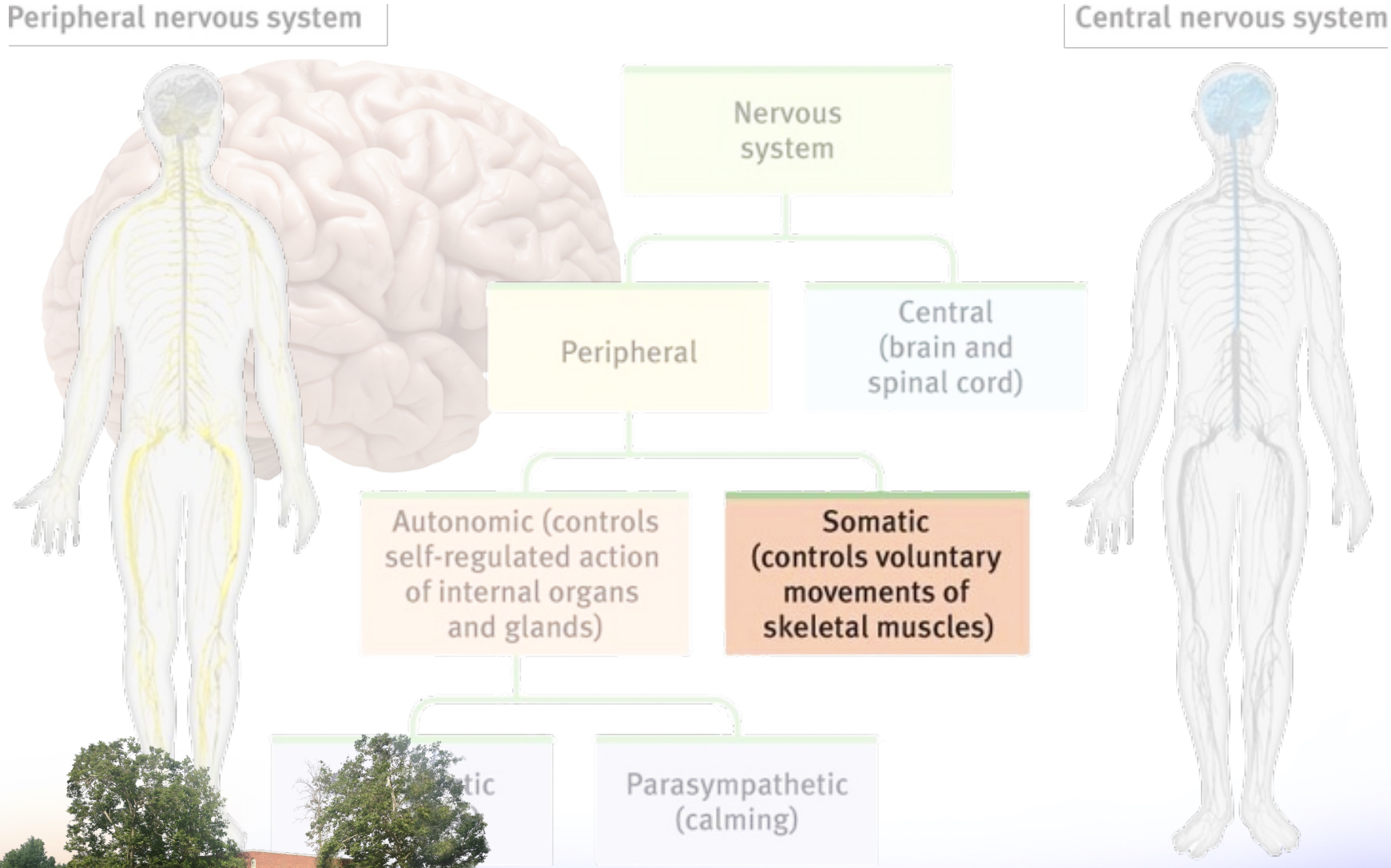
Length-tension relationship:



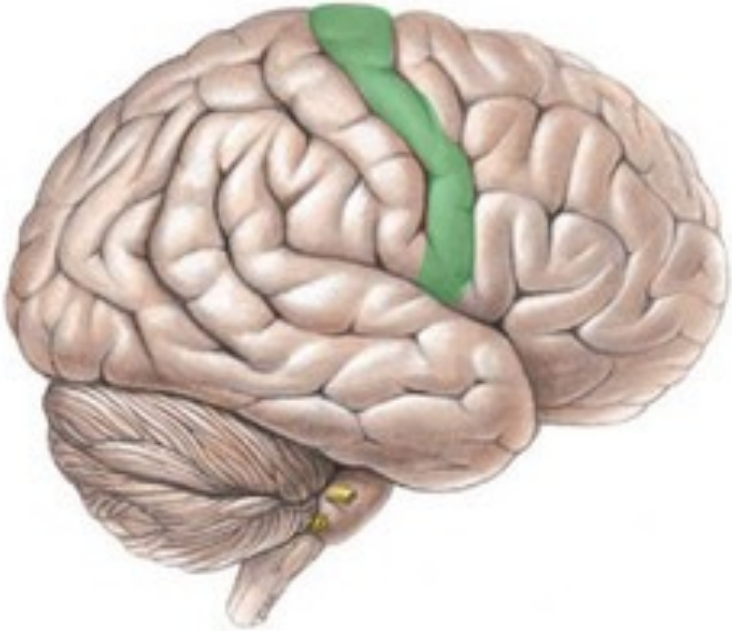
The rest of that stuff is in Lecture 3 (and some in Lecture 4)

Neural Recruitment / Motor Activation:

Neural Recruitment / Motor Activation:



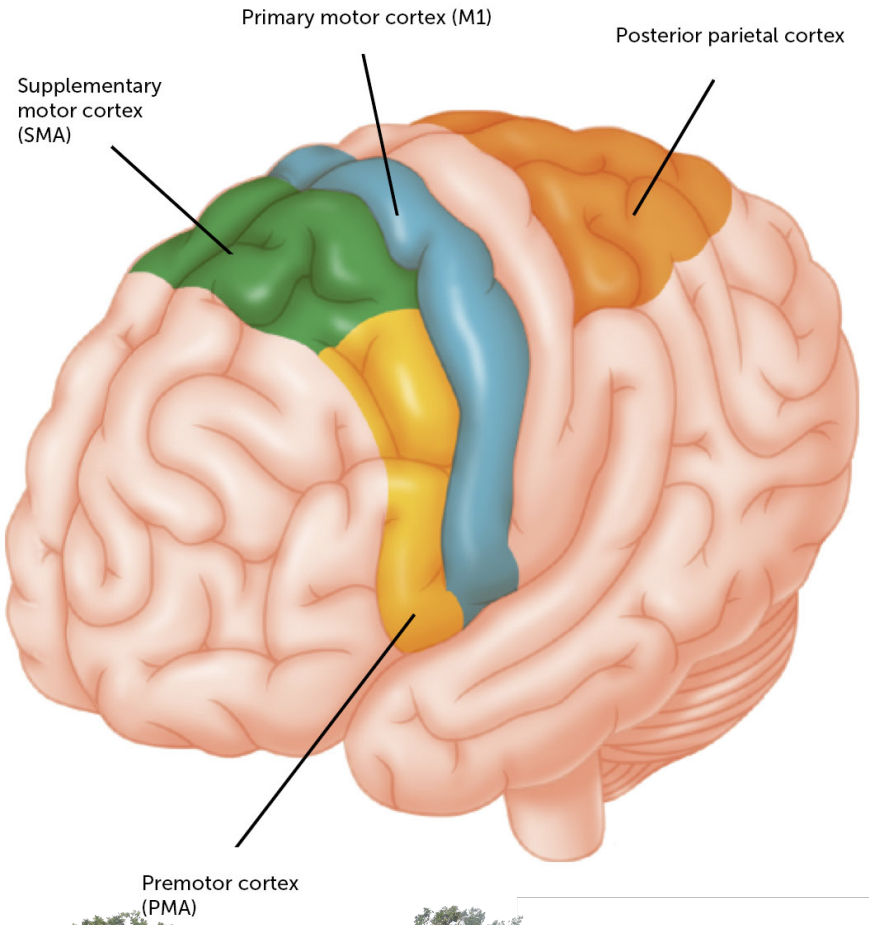
Neural Recruitment / Motor Activation:



Muscle recruitment begins as an electrical event in the motor cortex.

This is the place where your nerve impulses for voluntary muscle contraction are born.

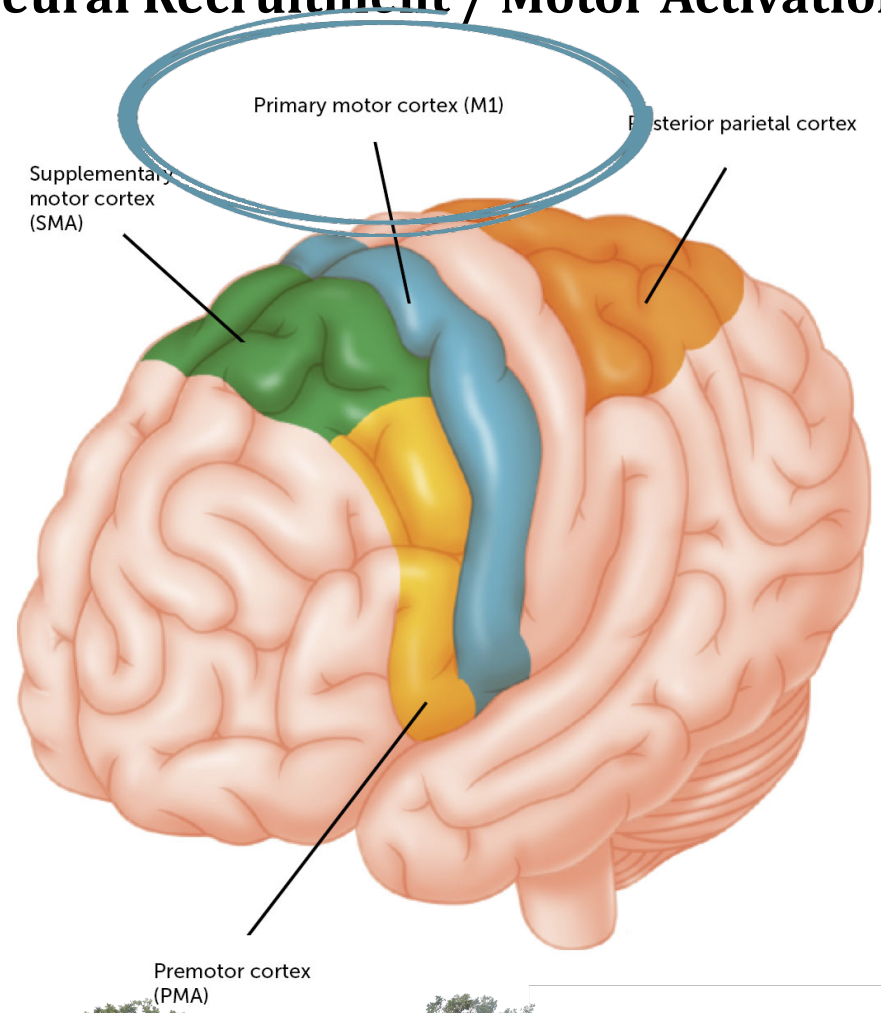
Neural Recruitment / Motor Activation:



There's a *pre*motor cortex and a *primary* motor cortex

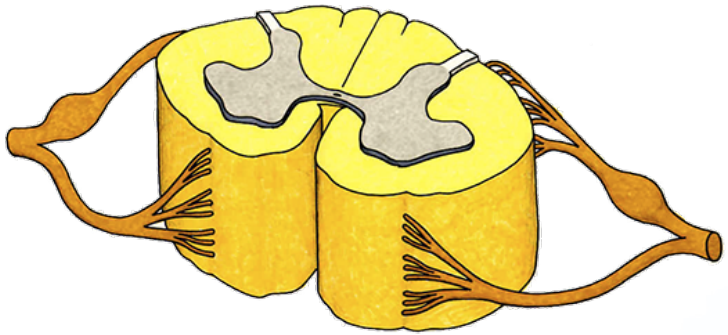
(and there's a supplementary motor area and other stuff)

Neural Recruitment / Motor Activation:

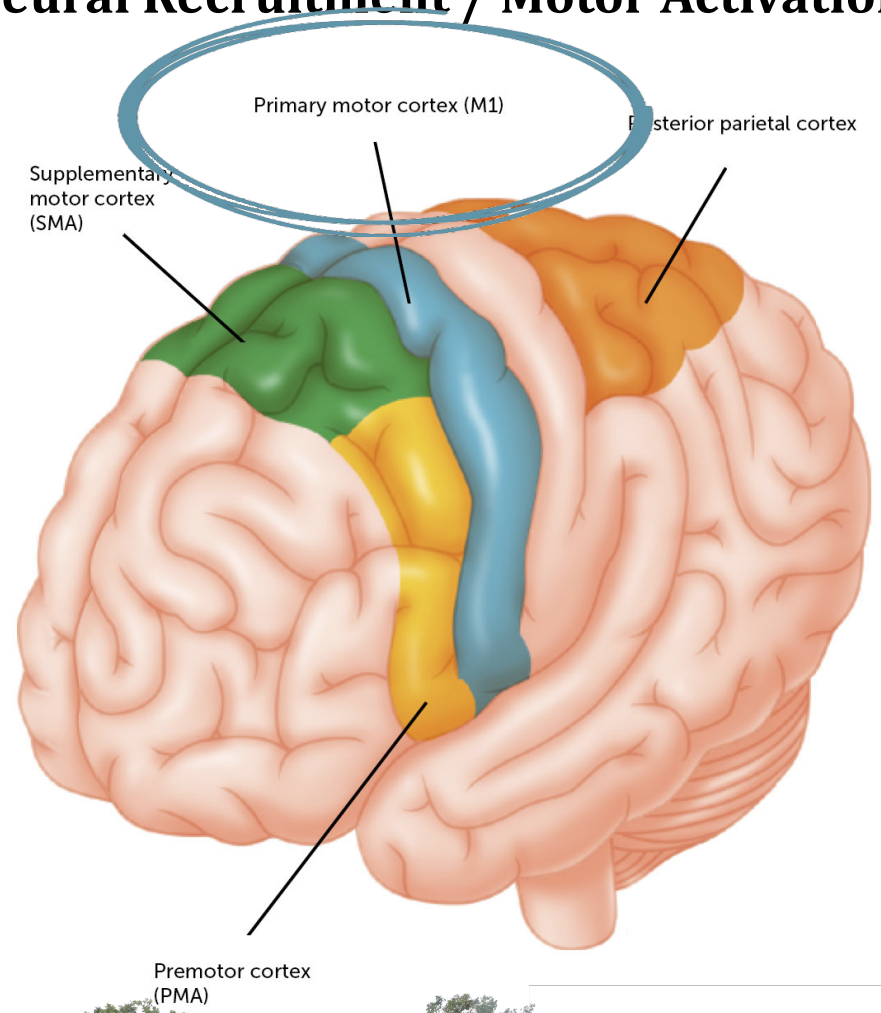


Primary Motor Cortex:

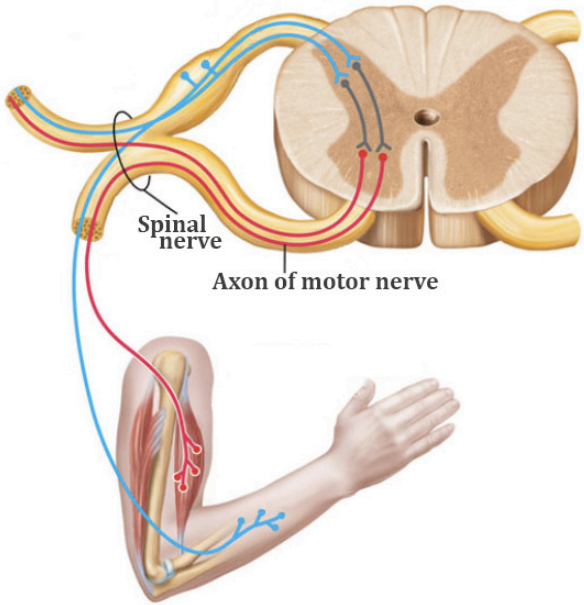
Alpha motor neurons project out of the brain and send their axons down the spinal cord to the ventral horn.



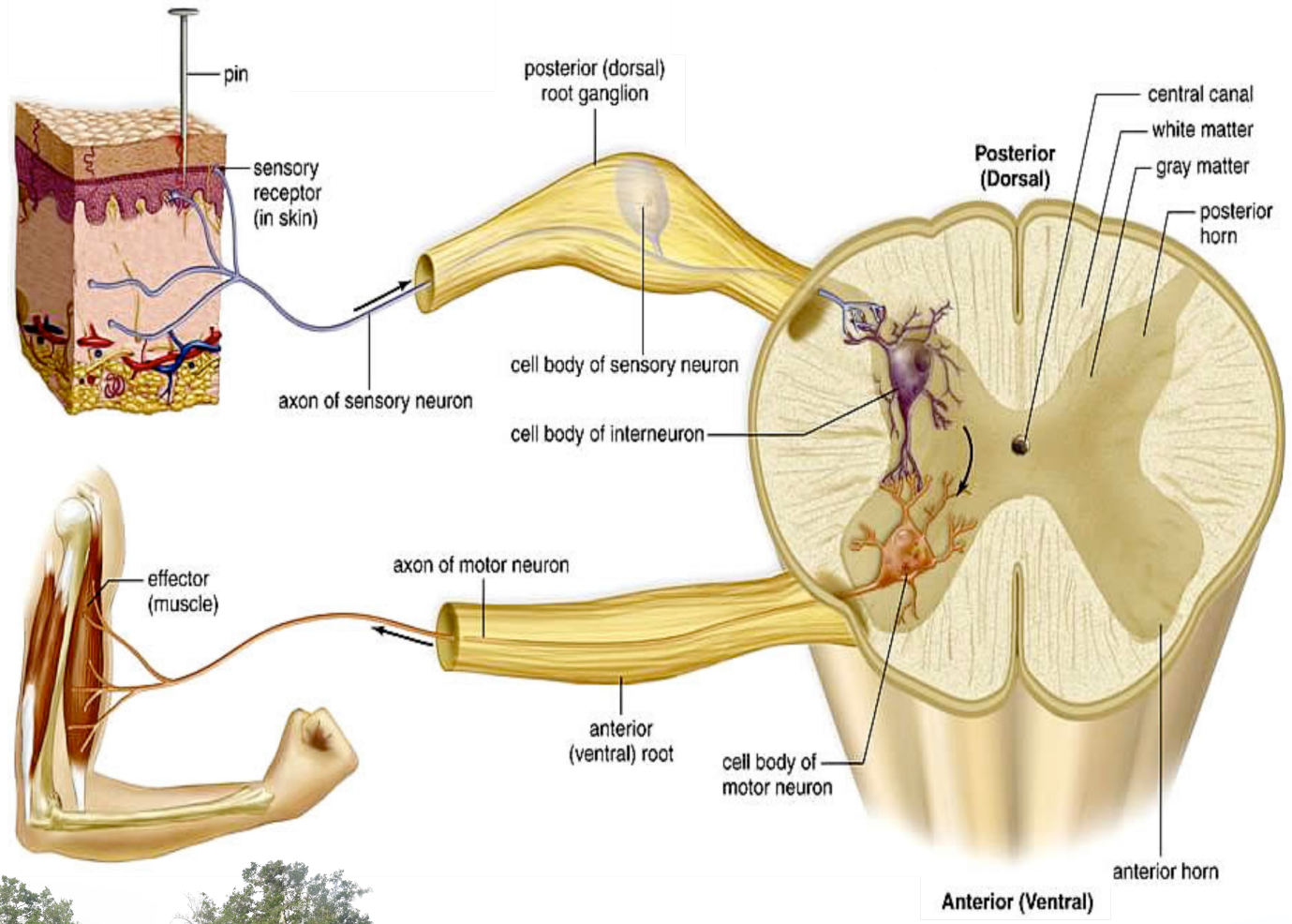
Neural Recruitment / Motor Activation:



Primary Motor Cortex:



Neural Recruitment / Motor Activation:

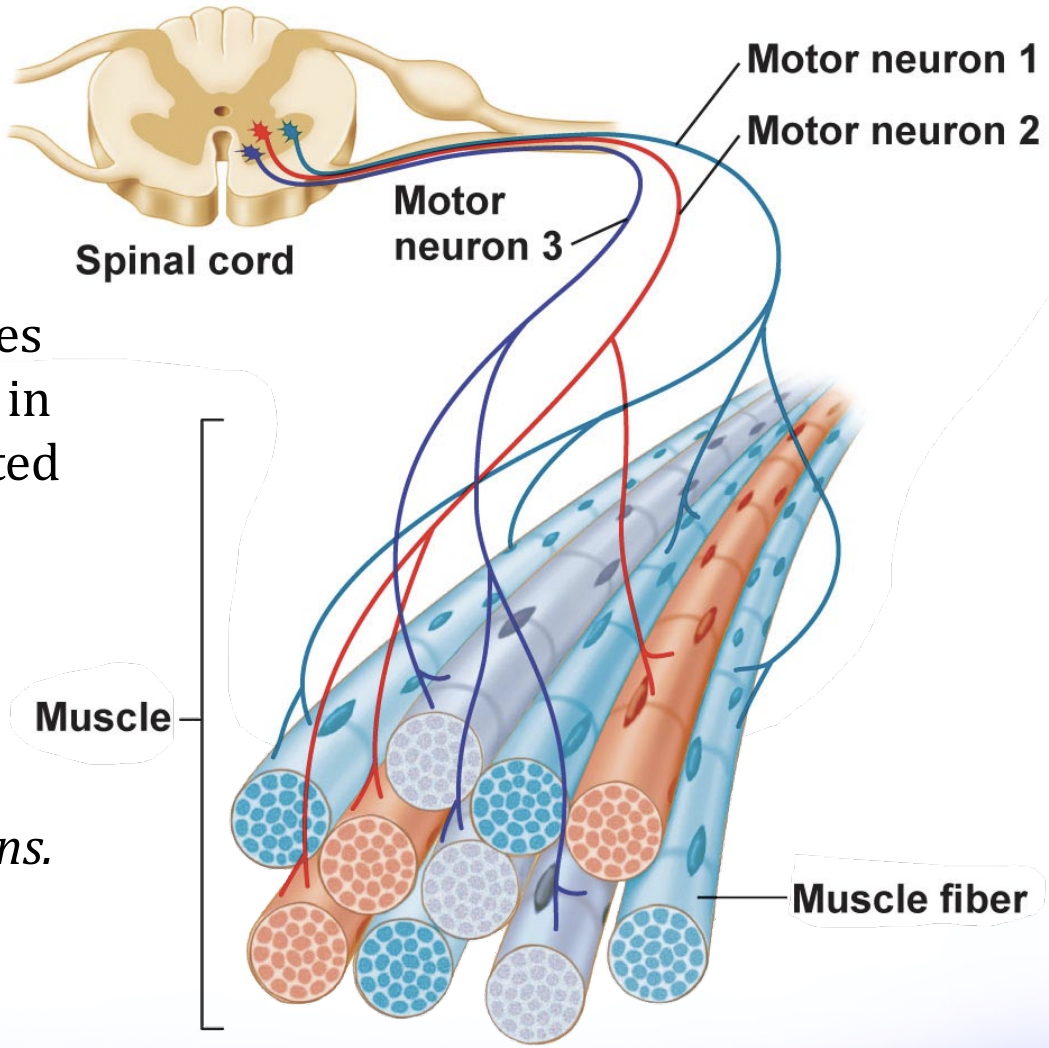


Neural Recruitment / Motor Activation:

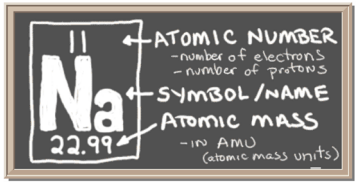
All-Or-None.

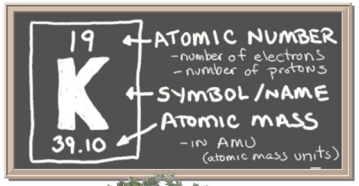
The all-or-none principle states that every single muscle fiber in a specific motor unit is activated maximally if activated at all.

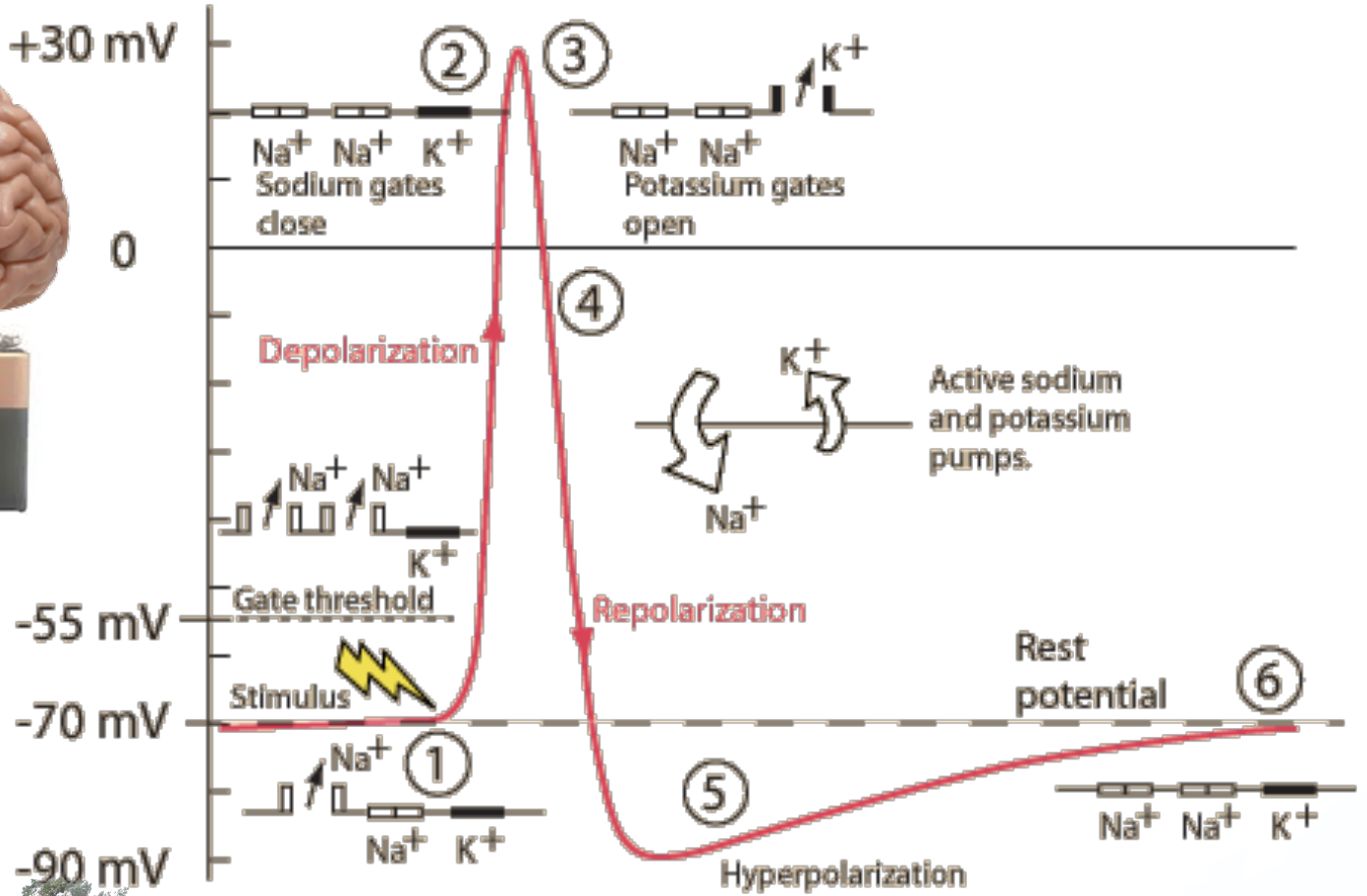
There are no "kinda" activations.

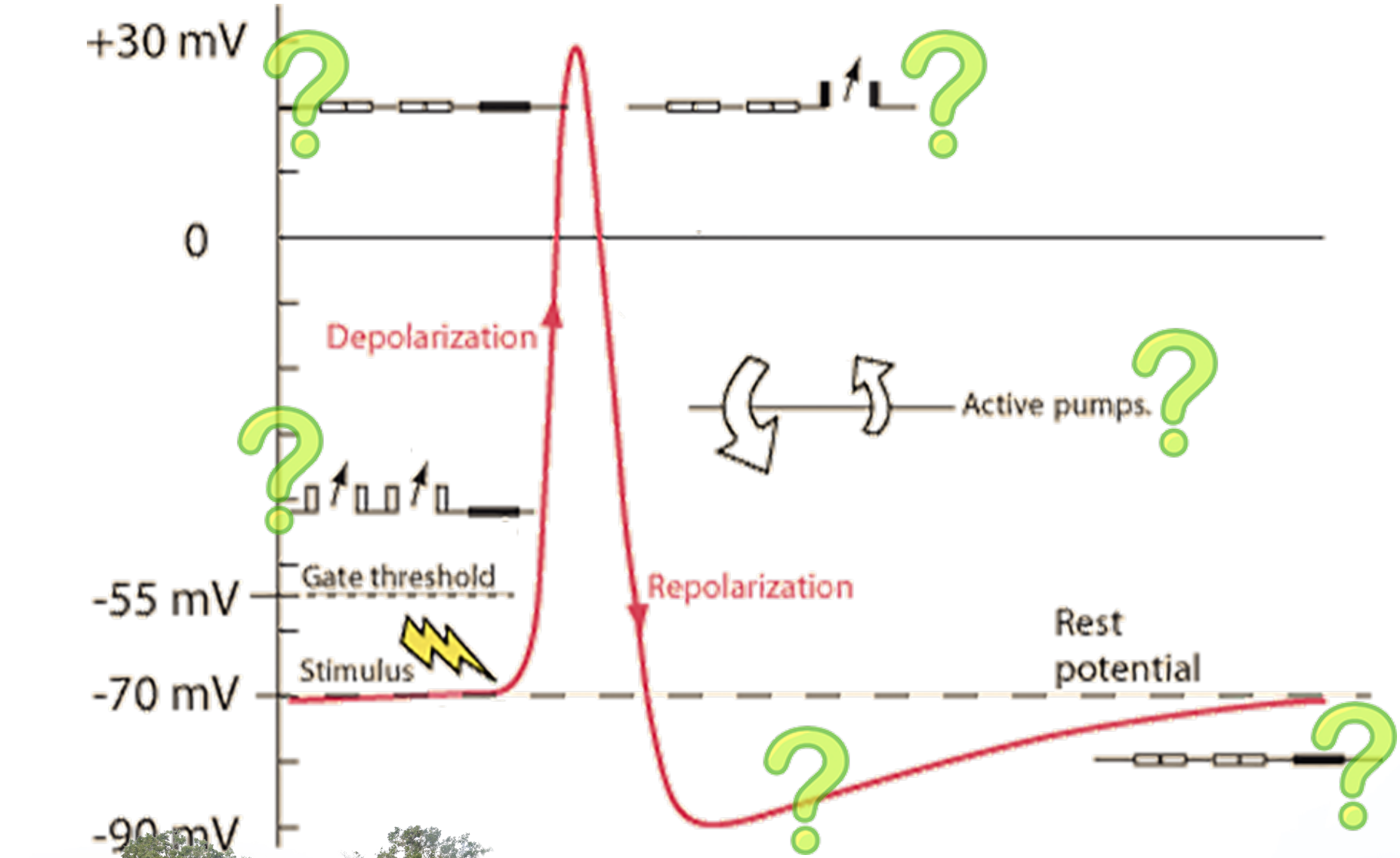


What's this called (and explain it to me)?

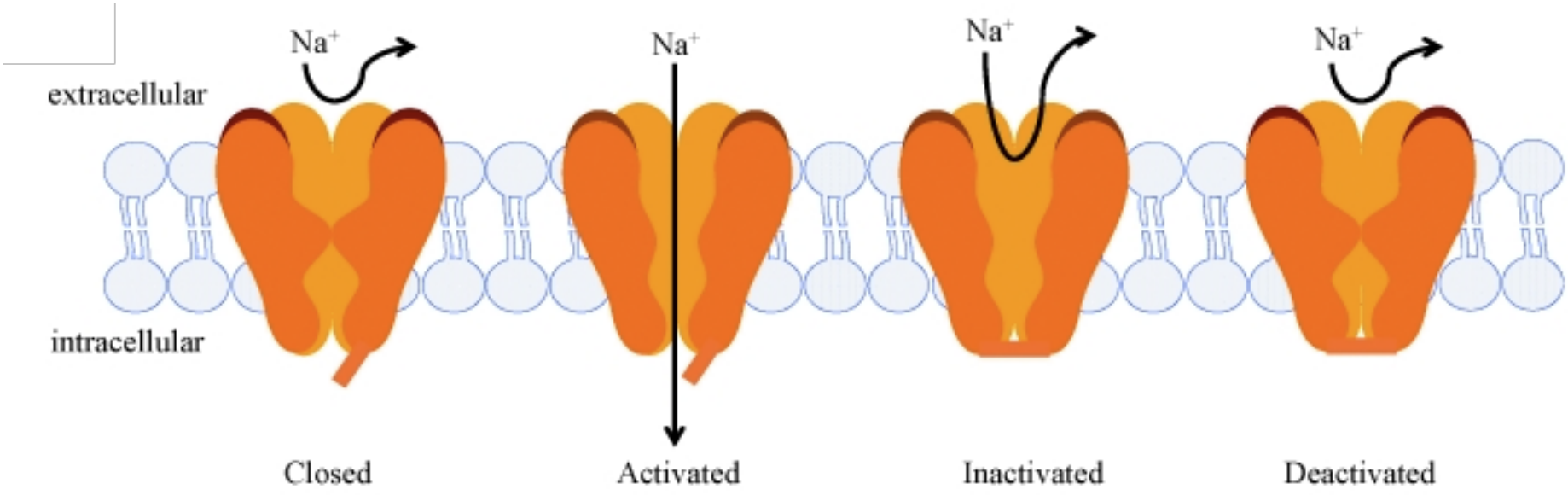







+30 mV
0
Depolarization
Active pumps.
Gate threshold
-55 mV
Repolarization
Rest
potential
Stimulus
-70 mV
-90 mV

Action Potential:



At resting potential. Extracellular side is closed.

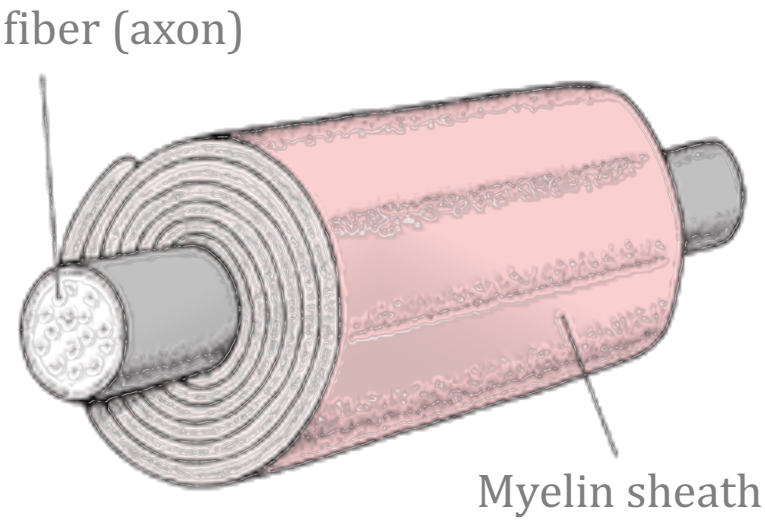
Depolarization: Channel opens.

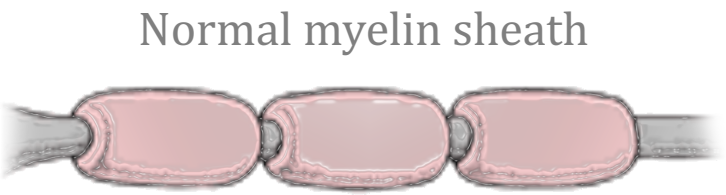
Repolarization phase: Intracellular side closes, unable to be reopened.

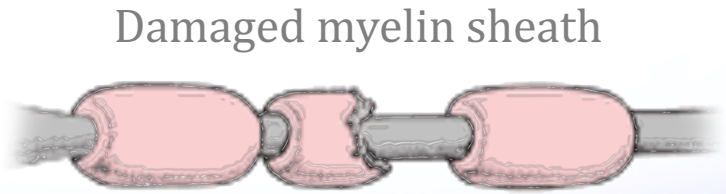
Hyperpolarization: Switching to closed state.

Action Potential:

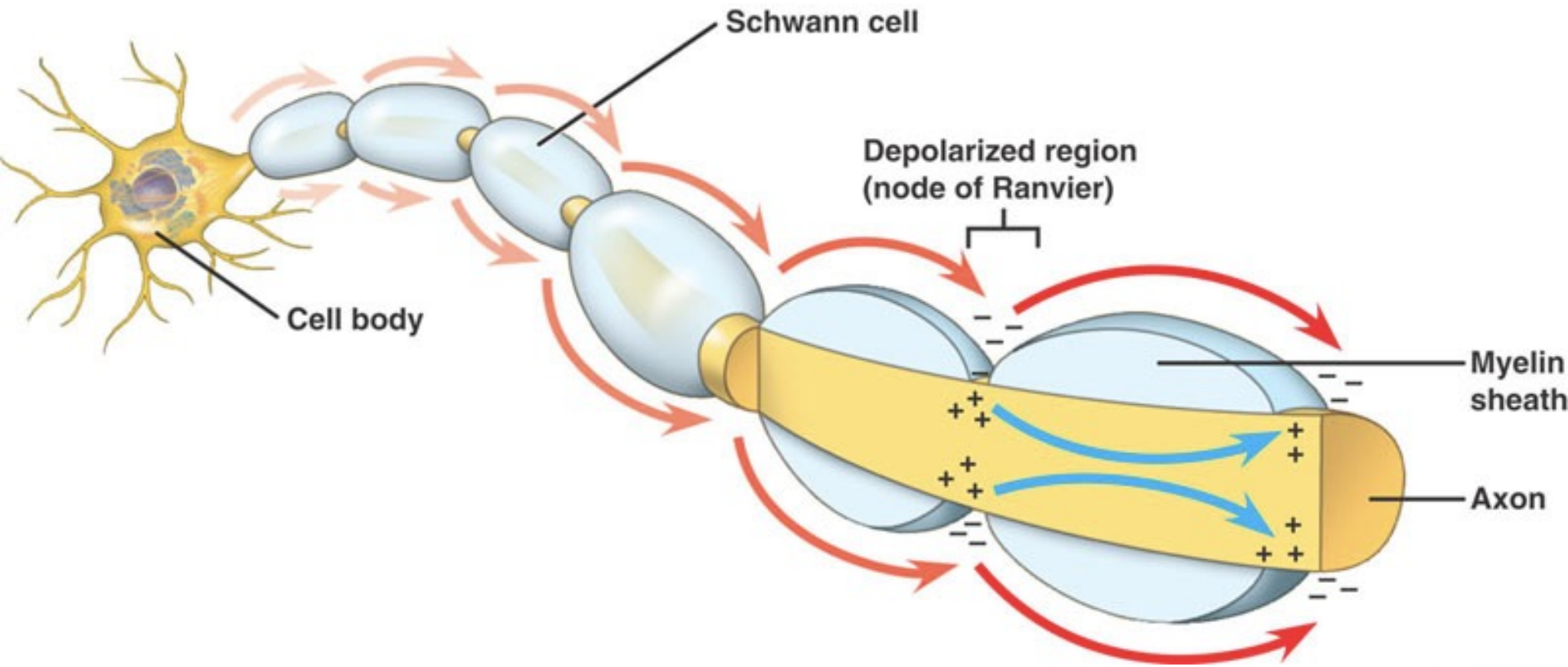
- Myelin
  - Spiral wrappings of tightly packed membranes
- Nodes of Ranvier
  - Action potential generation
  - High concentration of Na^+ and K^+ channels
  - Speedy conduction



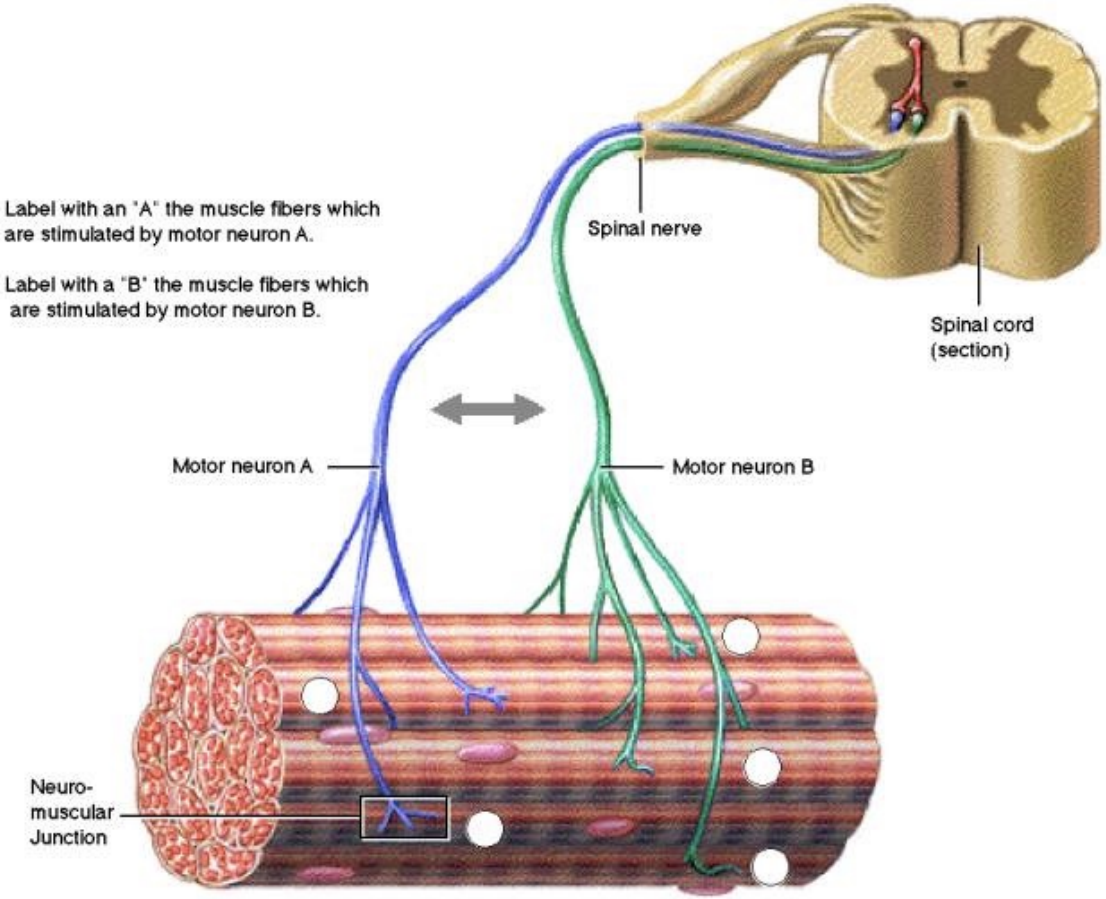


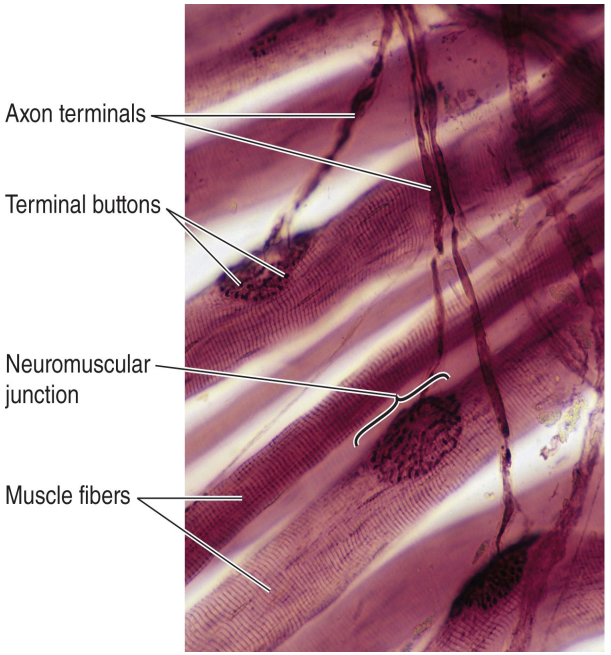


What kind of conduction is this? How does it work?

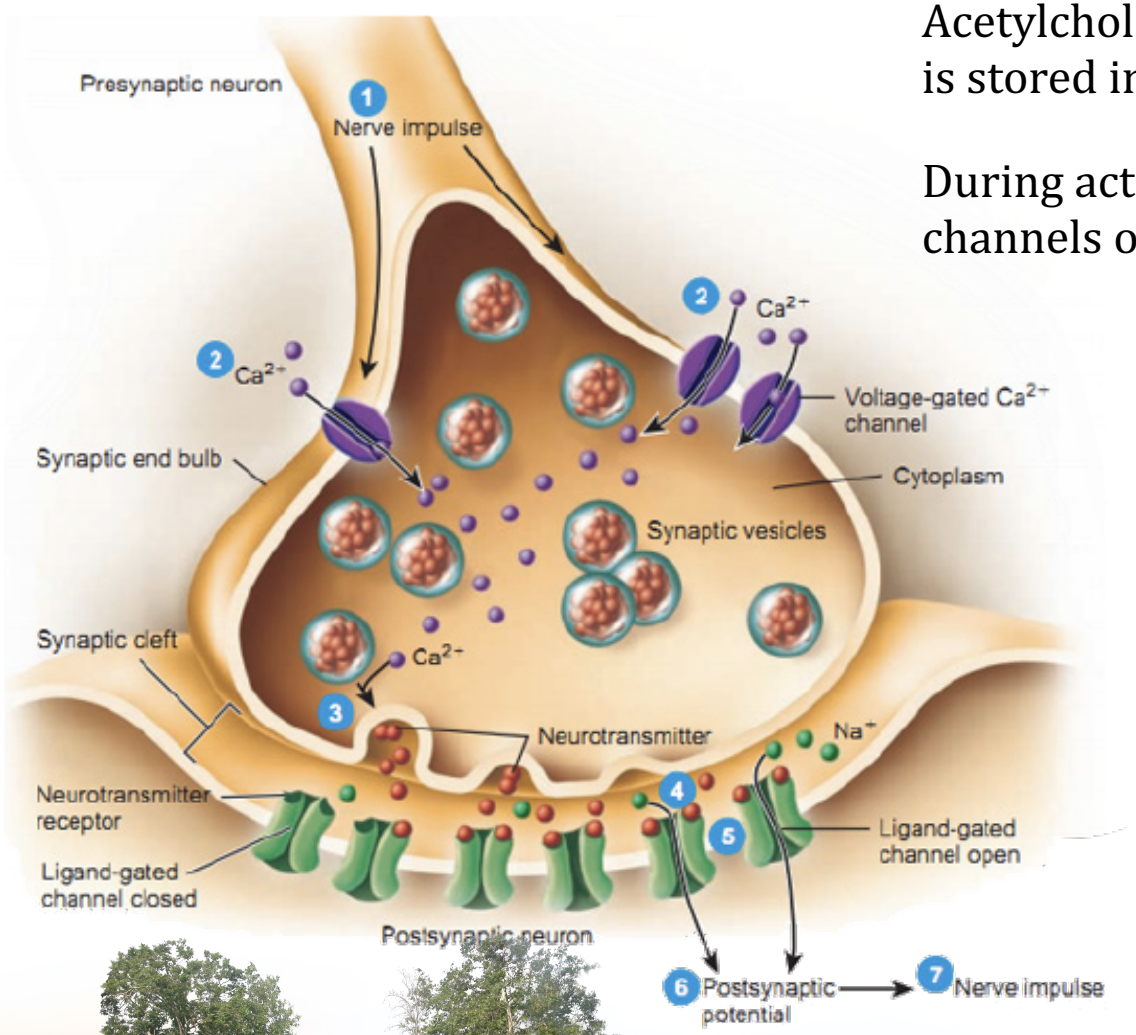


Neuromuscular Junction:





Neuromuscular Junction:



Acetylcholine (neurotransmitter) is stored in little sacs ("vesicles").

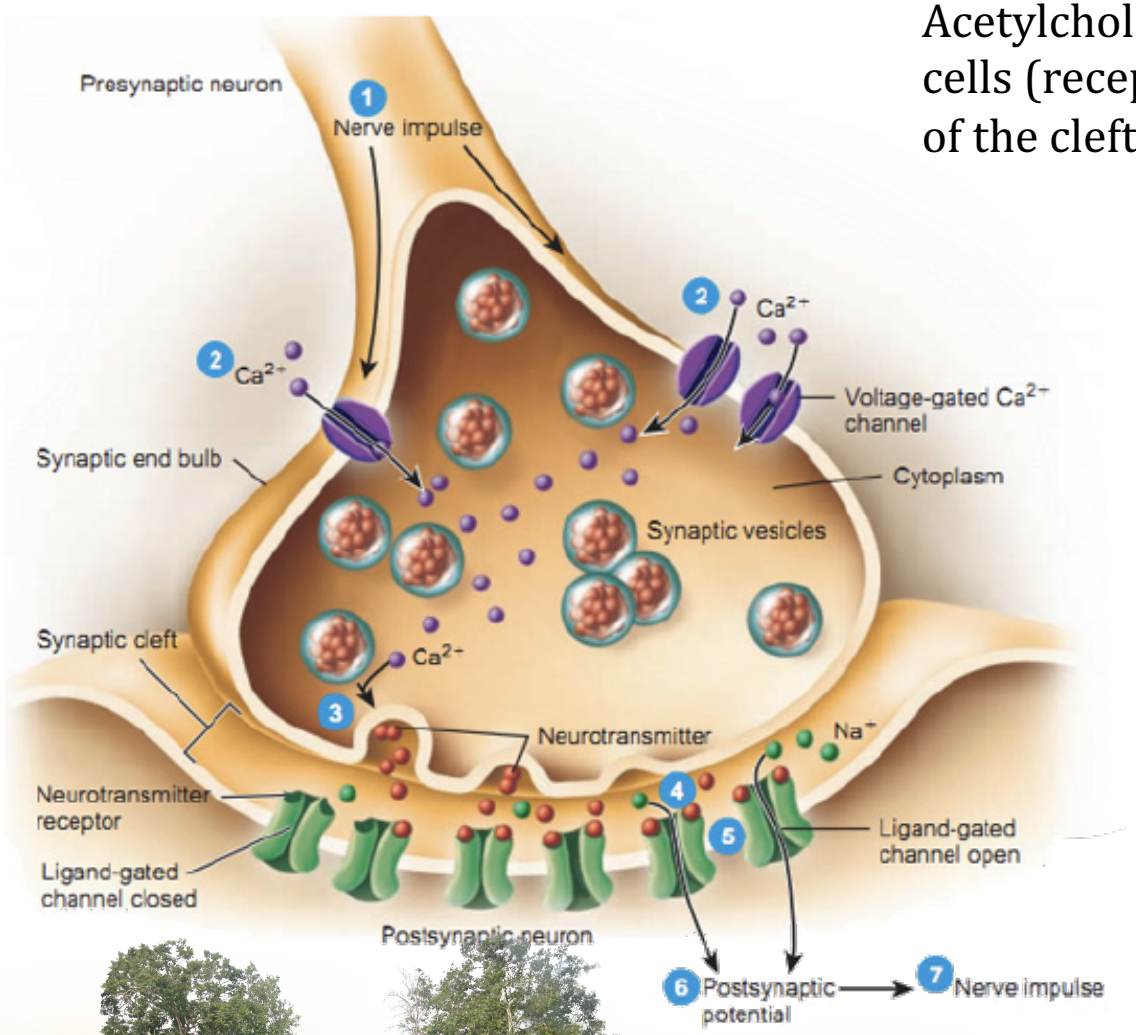
During action potential, calcium channels open and calcium enters.

On the surface of the vesicles there are calcium-sensitive proteins.

When calcium binds to those proteins, the vesicles become activated.

When activated, they dump their acetylcholine into the synaptic cleft.

Neuromuscular Junction:



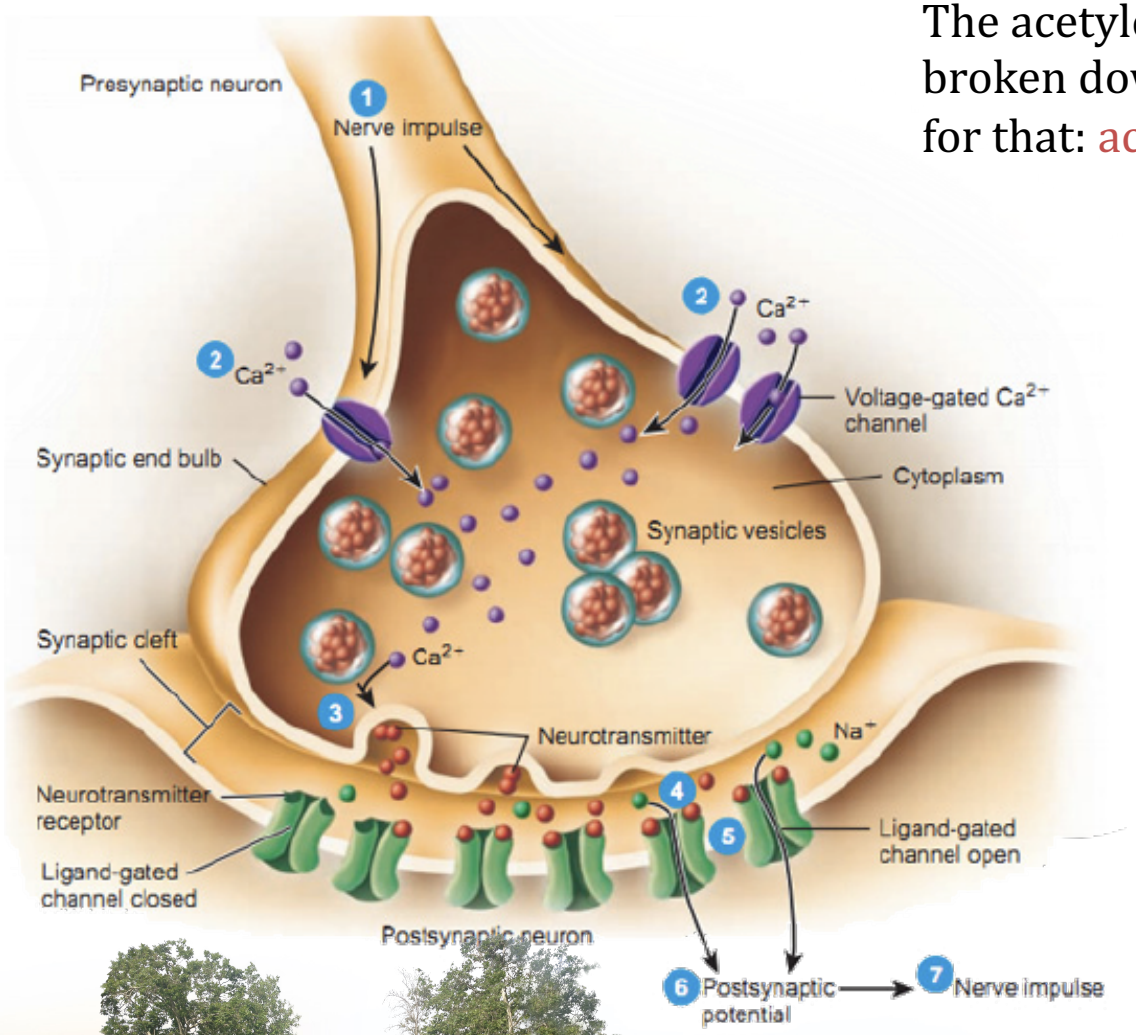
Acetylcholine binds to *post*synaptic cells (receptors) on the other side of the cleft.

The acetylcholine receptors (the postsynaptic cells) are on the plasma membrane (the "sarcolemma") of the muscle fiber.

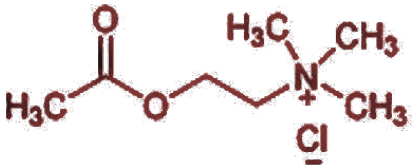
The acetylcholine must then be broken down.

Acetylcholinesterase is the enzyme responsible for that.

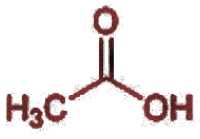
Neuromuscular Junction:



The acetylcholine must then be broken down. Enzyme responsible for that: acetylcholinesterase.

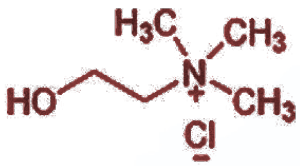


Acetylcholine (ACh)



Acetic Acid

+

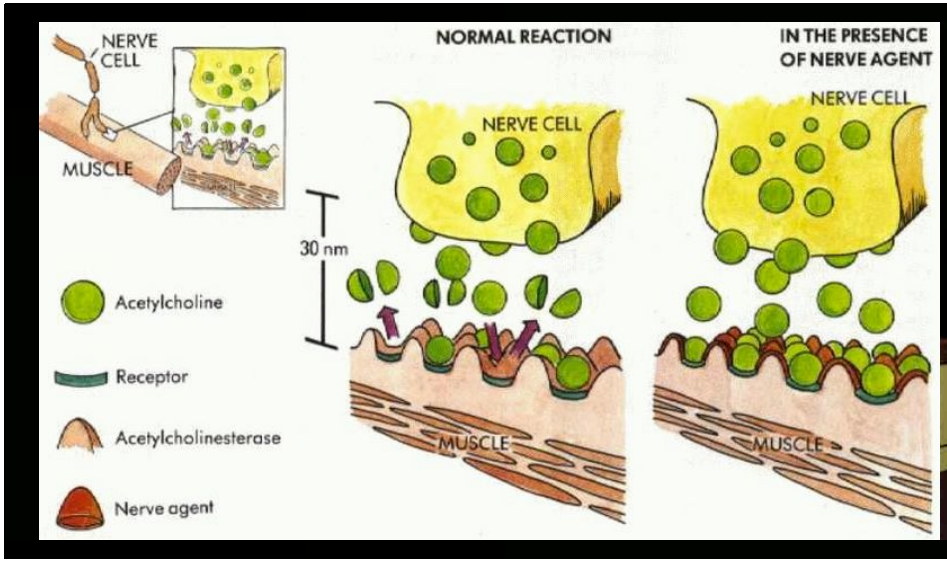


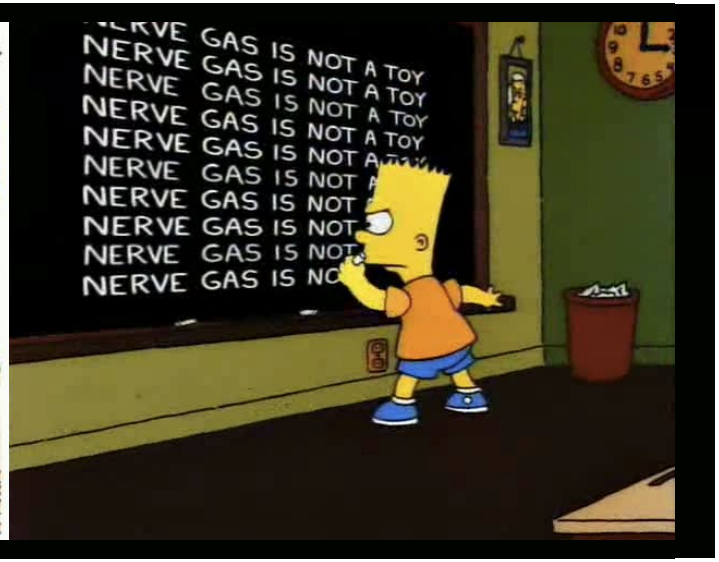
Choline (Ch)

Neuromuscular Junction:

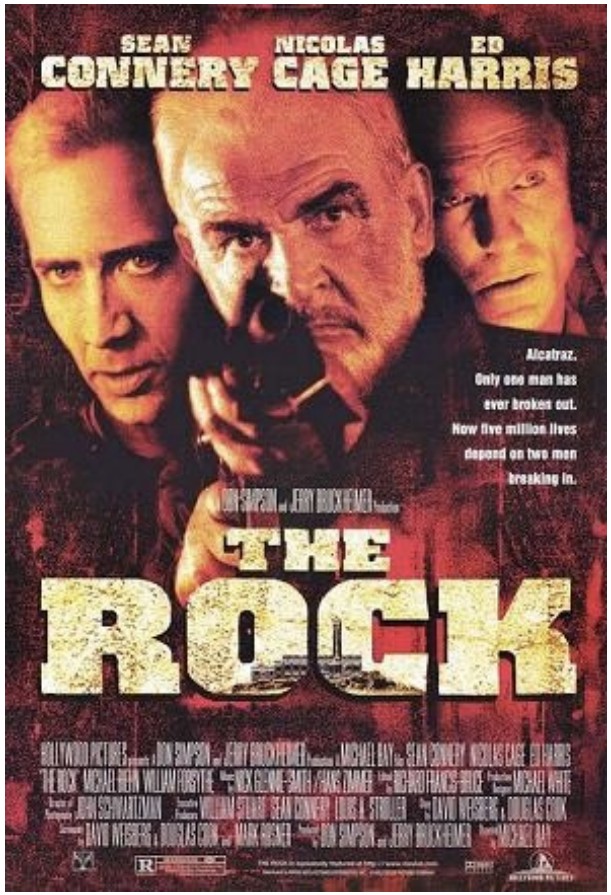
If you can't break down your acetylcholine, you can't terminate synaptic transmission.

Certain nerve gases and insecticides target (inhibit) this enzyme. That's bad.





Neuromuscular Junction:



Anticholinergic drugs (e.g., atropine) inhibit the acetylcholine receptors in the heart and smooth muscle (muscarinic). Not in skeletal muscle; those are a different type of receptor.

Consequence: Increasing of the heart rate (and other sympathetic things, like dilation of the pupils).

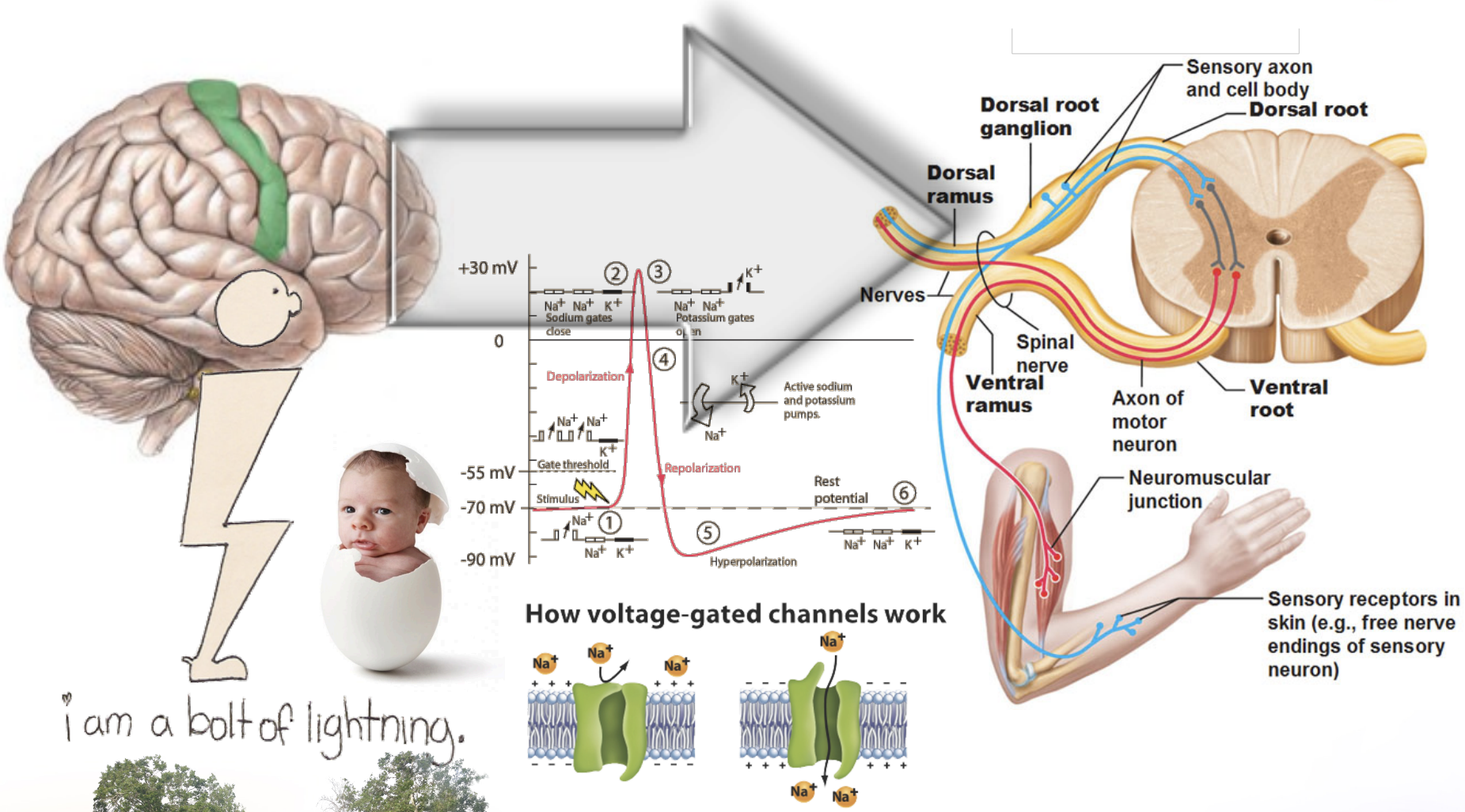
Nicolas Cage in "*The Rock*" readying to inject himself with atropine to stop the effects of nerve gas.

Excitation-Contraction Coupling:

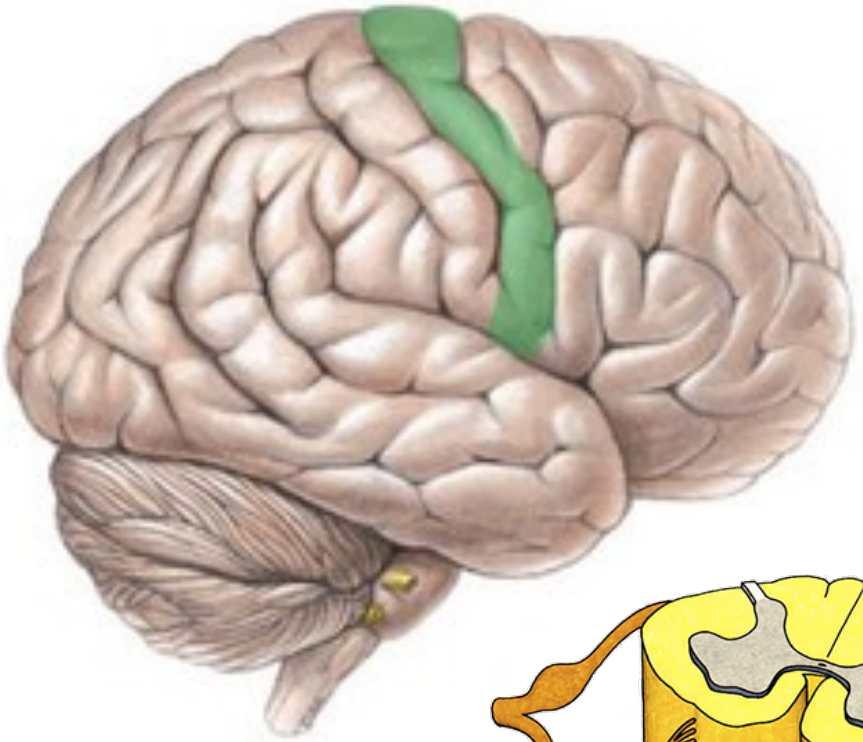
Linking nerve signals to muscle contractions

1. Alpha motor neuron is excited (depolarized)
2. Electrical signal leaves CNS through ventral horn
3. Travels to axon terminal (or "terminal bouton")
4. Calcium enters bulb, activates synaptic vesicles
5. Vesicles dock, fuse, and release their acetylcholine
6. The acetylcholine travels across the synaptic cleft; attaches to receptors on postsynaptic membrane (i.e., motor endplate on sarcolemma)
7. Depolarizes sarcolemma, signal transmitted to the sarcoplasmic reticula…

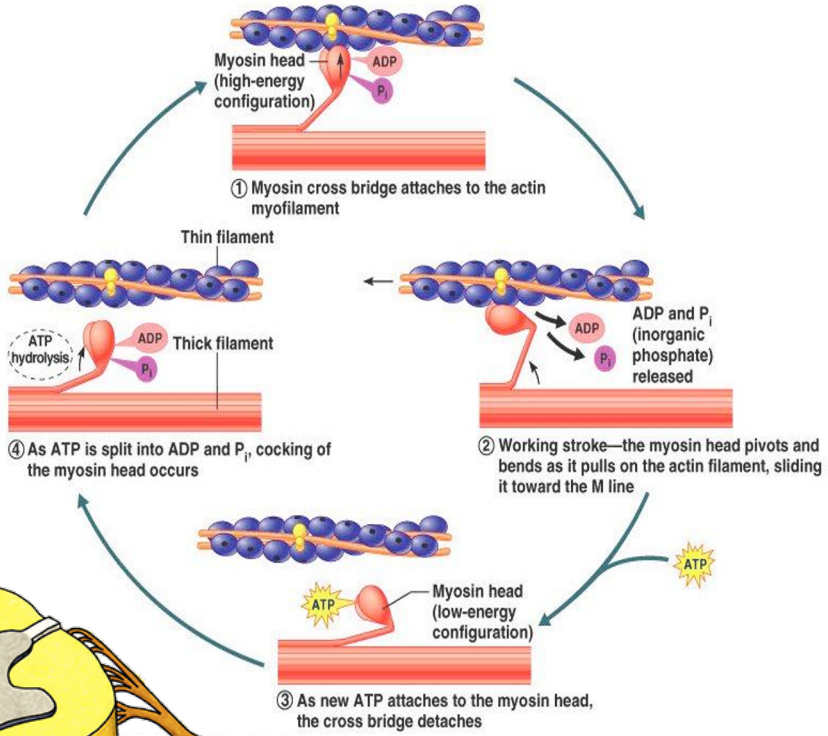
Excitation-Contraction Coupling:

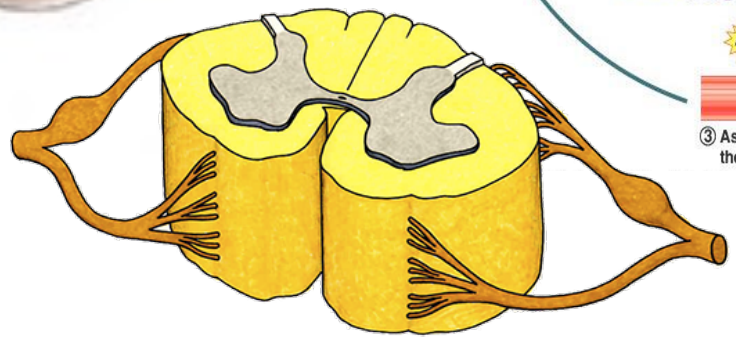


Start here:

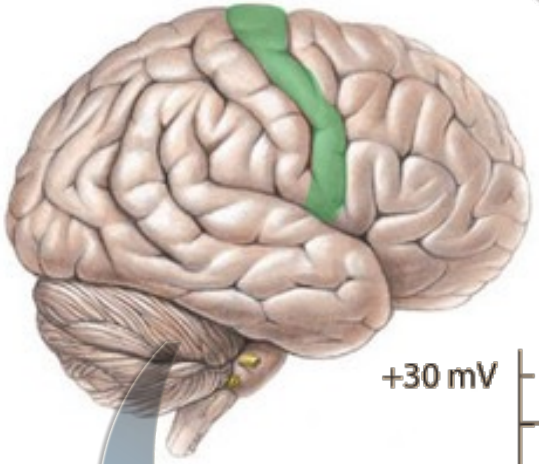


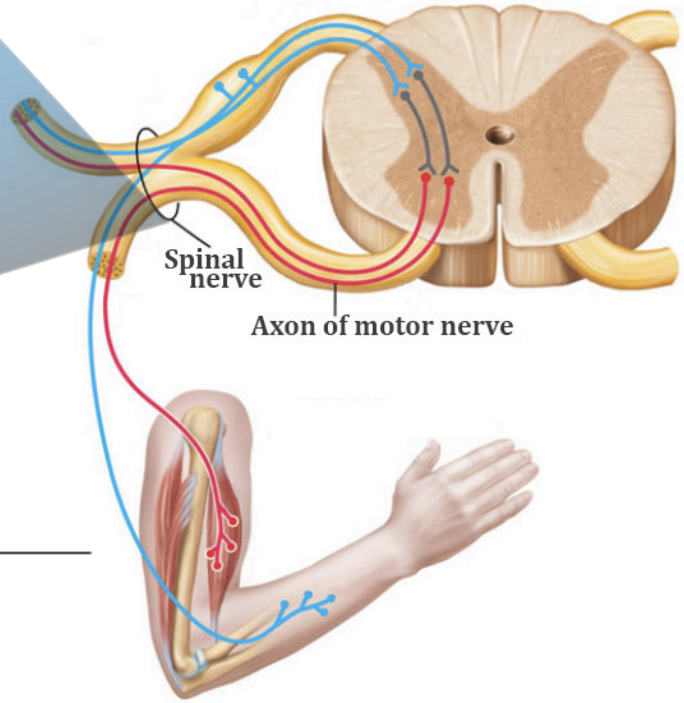
End here:

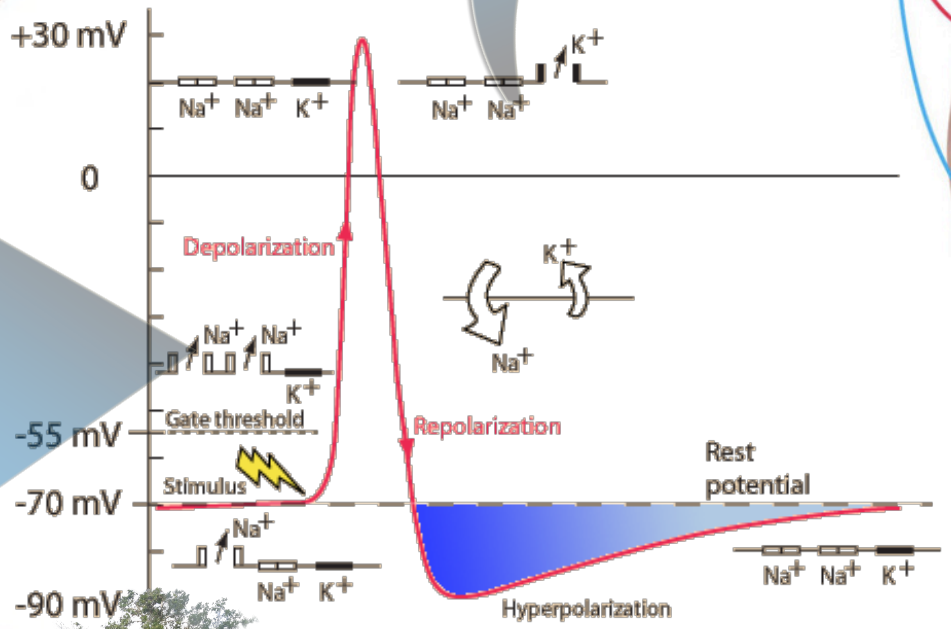


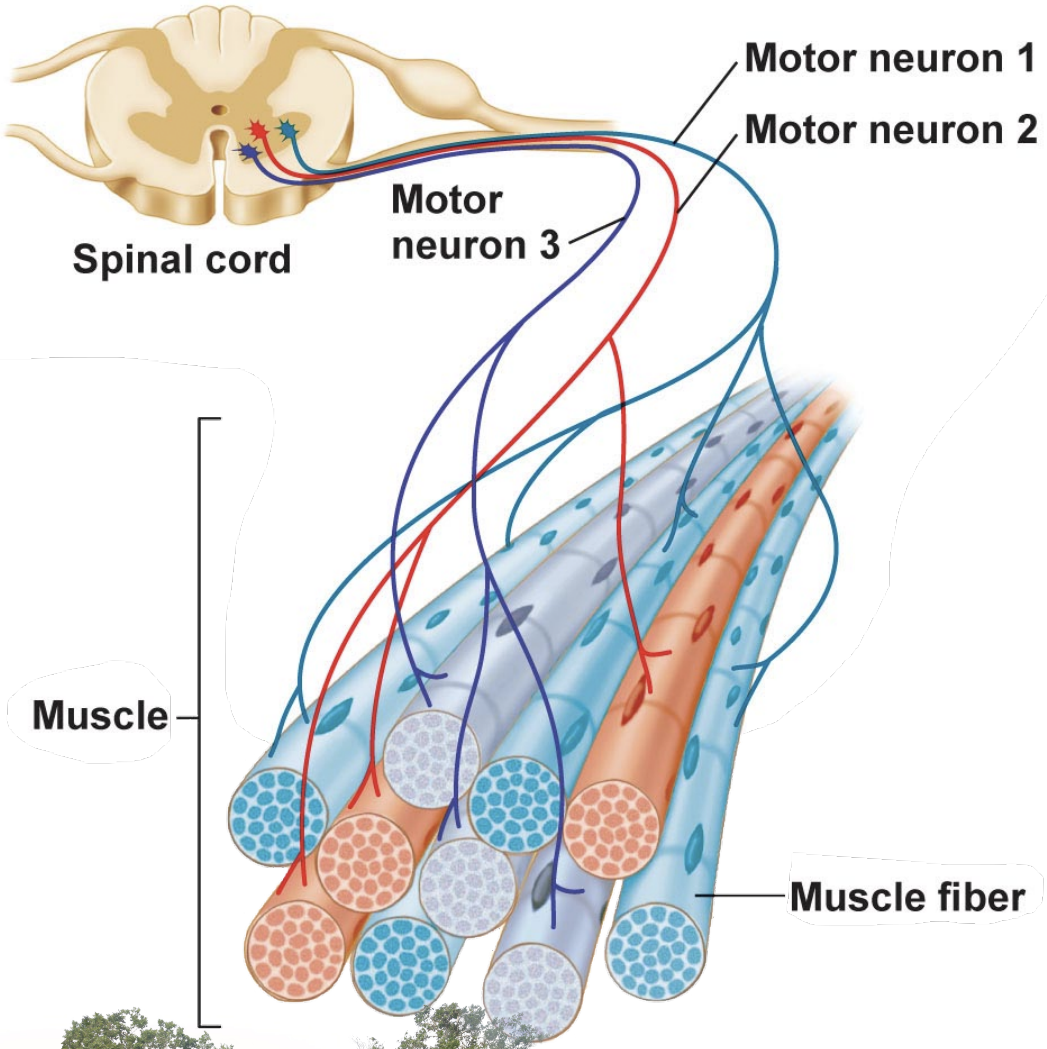


Voluntary Muscle Recruitment



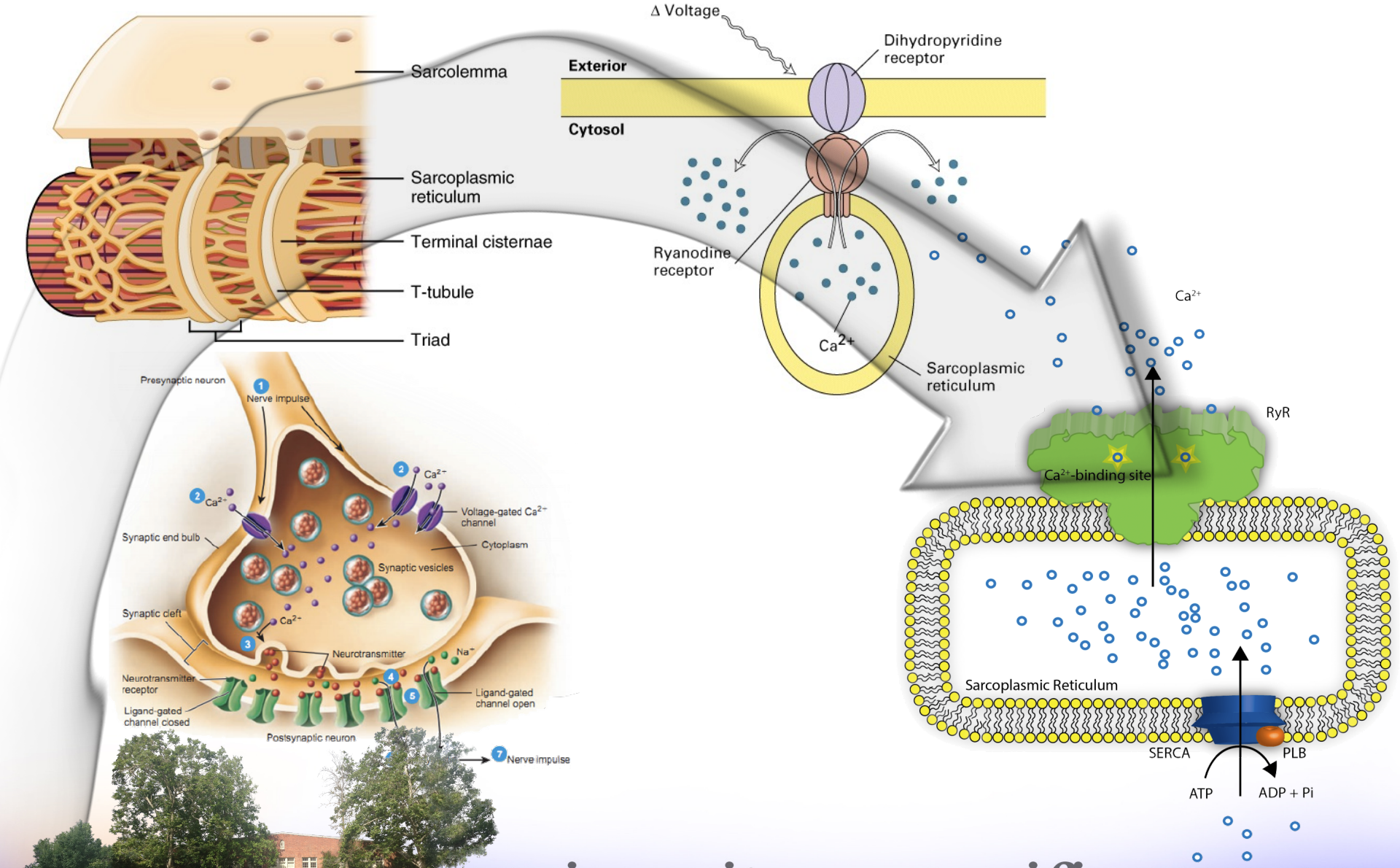


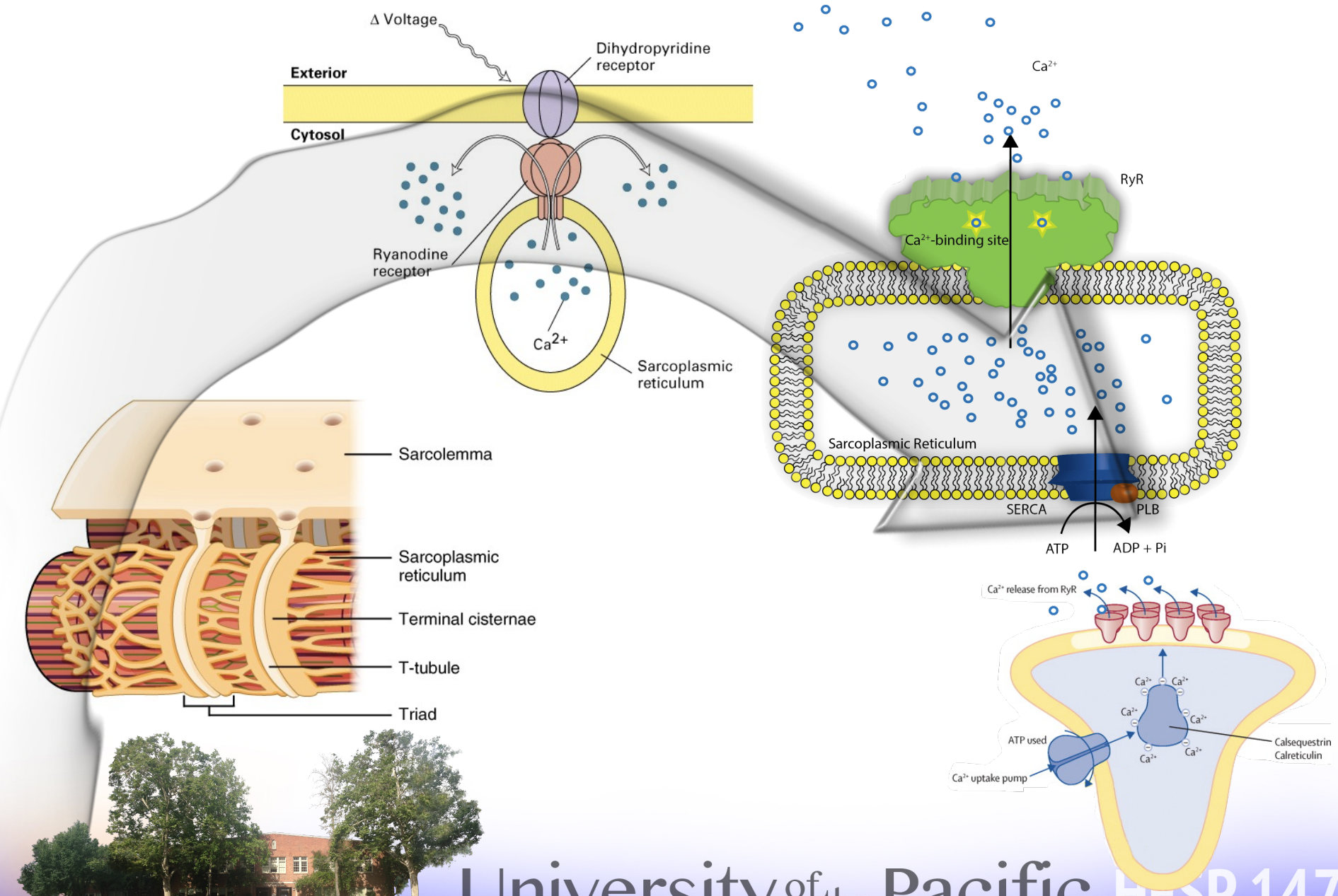


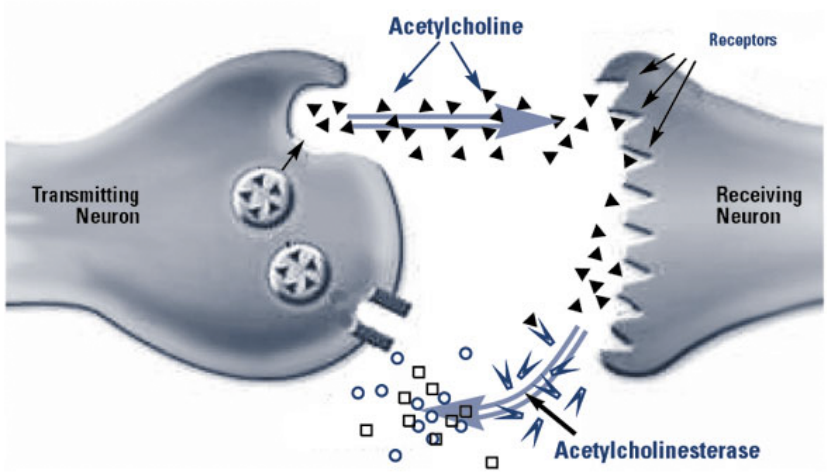


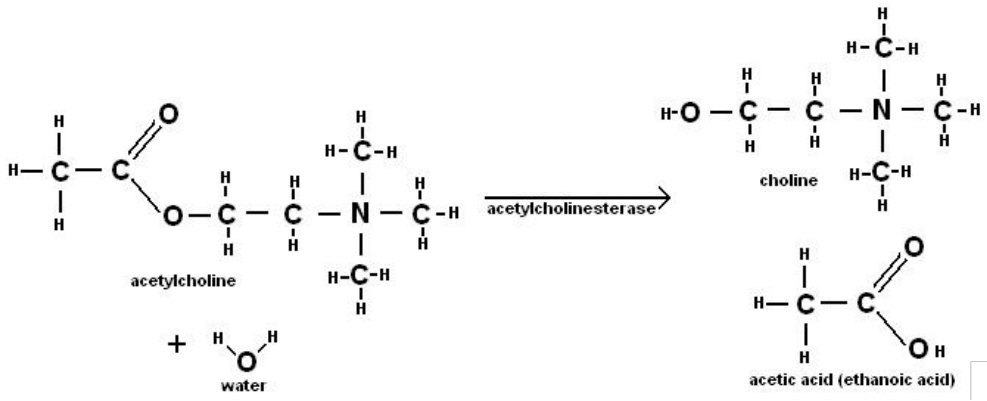
What's a motor unit?

What's the all-or-none principle?

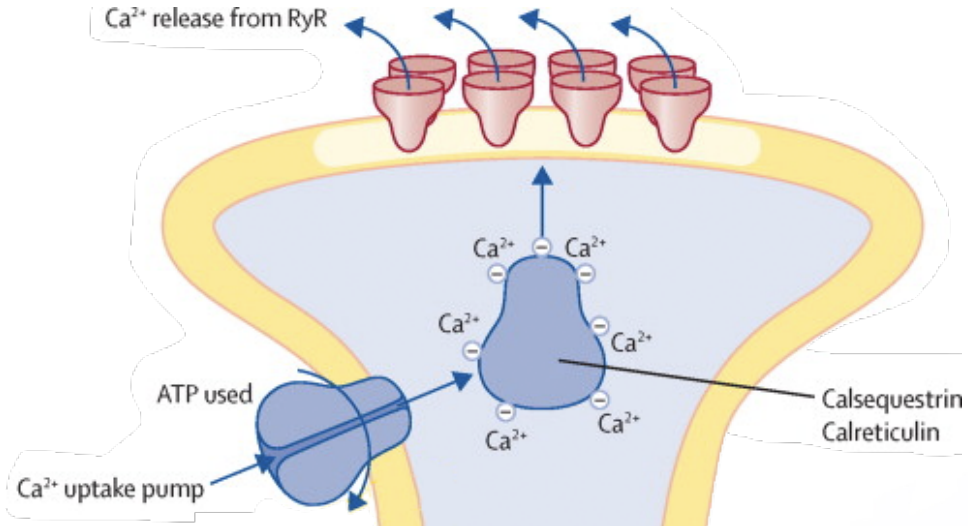

Sarcolemma
Sarcoplasmic reticulum
Terminal cisternae
T-tubule
Triad
Δ Voltage
Exterior
Cytosol
Dihydropyridine receptor
Ryanodine receptor
Ca2+
Sarcoplasmic reticulum
Ca2+
RyR
Ca2+-binding site
Sarcoplasmic Reticulum
SERCA
PLB
ATP
ADP + Pi
Presynaptic neuron
Nerve impulse
Ca2+
Voltage-gated Ca2+ channel
Synaptic end bulb
Cytoplasm
Synaptic vesicles
Synaptic cleft
Neurotransmitter
Na+
Neurotransmitter receptor
Ligand-gated channel closed
Ligand-gated channel open
Postsynaptic neuron
Nerve impulse


Δ Voltage
Dihydropyridine receptor
Exterior
Cytosol
Ryanodine receptor
Ca2+
Sarcoplasmic reticulum
Ca2+
RyR
Ca2+-binding site
Sarcoplasmic Reticulum
SERCA
PLB
ATP
ADP + Pi
Sarcolemma
Sarcoplasmic reticulum
Terminal cisternae
T-tubule
Triad
Ca2+ release from RyR
ATP used
Ca2+ uptake pump
Calsequestrin
Calreticulin


Acetylcholine
Receptors
Transmitting Neuron
Receiving Neuron
Acetylcholinesterase


acetylcholine
acetylcholinesterase
choline
water
acetic acid (ethanoic acid)

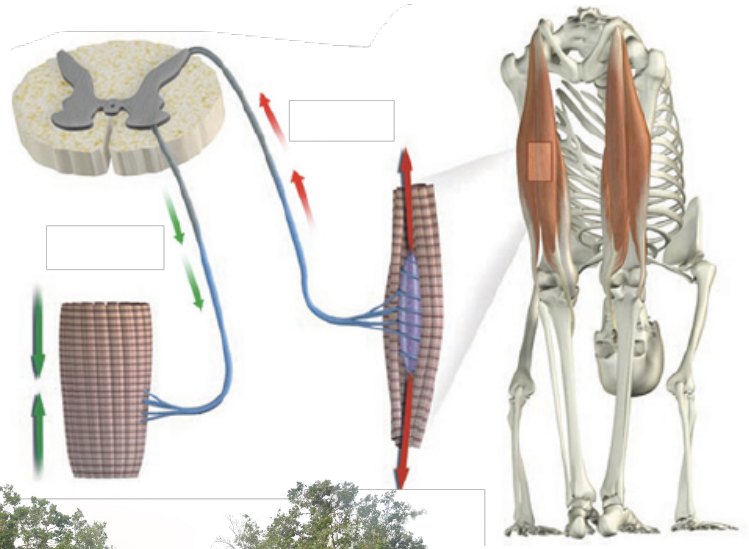

NERVE GAS IS NOT A TOY
NDC 10019-250-37
Atropine
Sulfate Injection, USP
0.4 mg/mL
FOR IM, IV OR SC USE

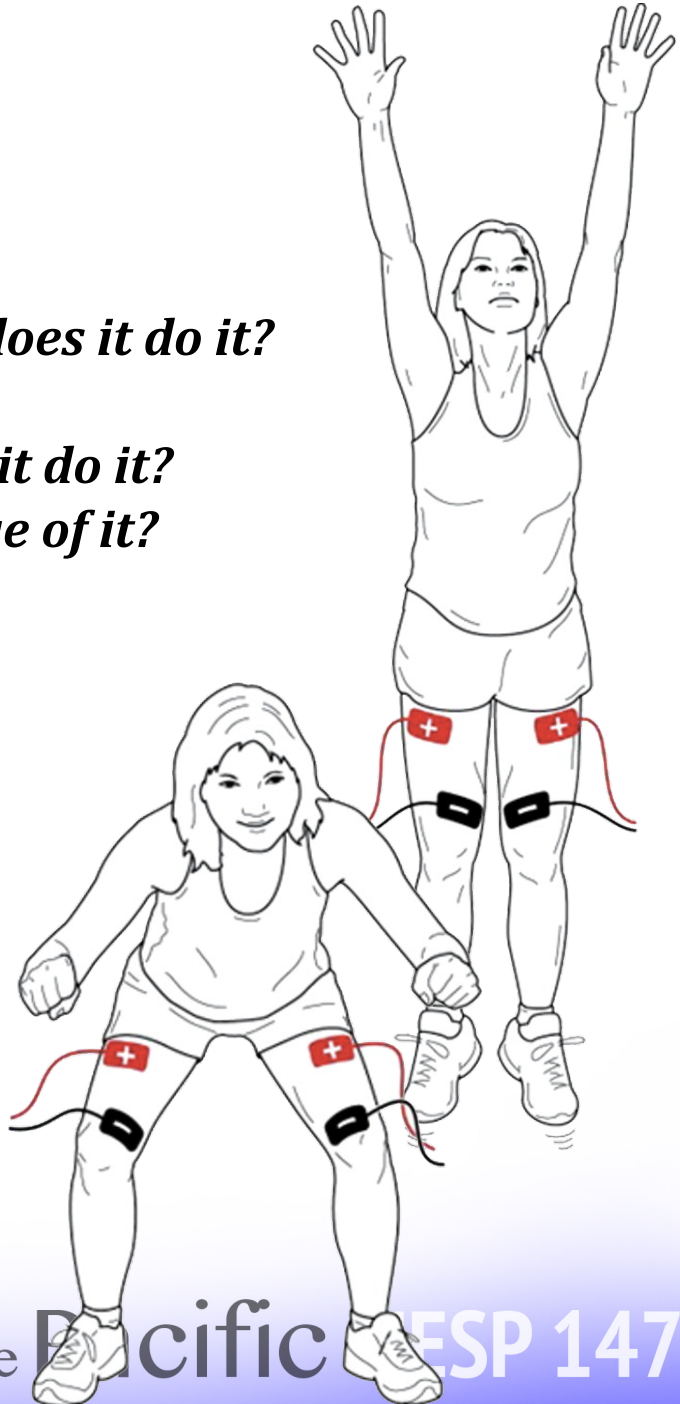

Ca2+ release from RyR
Ca2+
ATP used
Ca2+ uptake pump
Calsequestrin
Calreticulin

Reflex Arc: What is it?

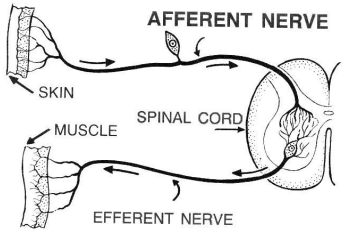
Golgi tendon organ: ***What does it do and why does it do it?***

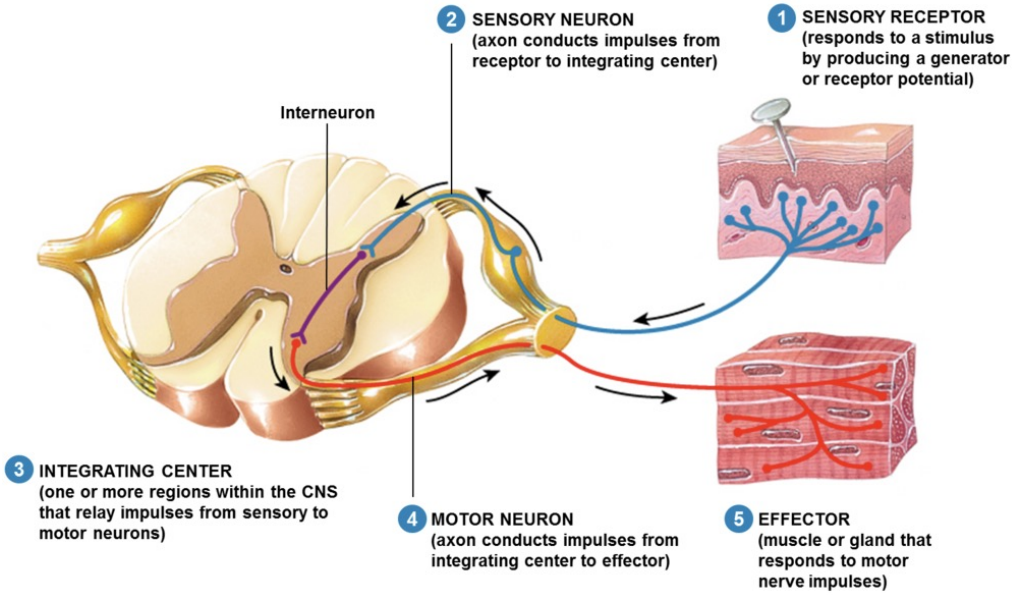
Muscle spindle: ***What does it do and why does it do it?***
How would you take advantage of it?

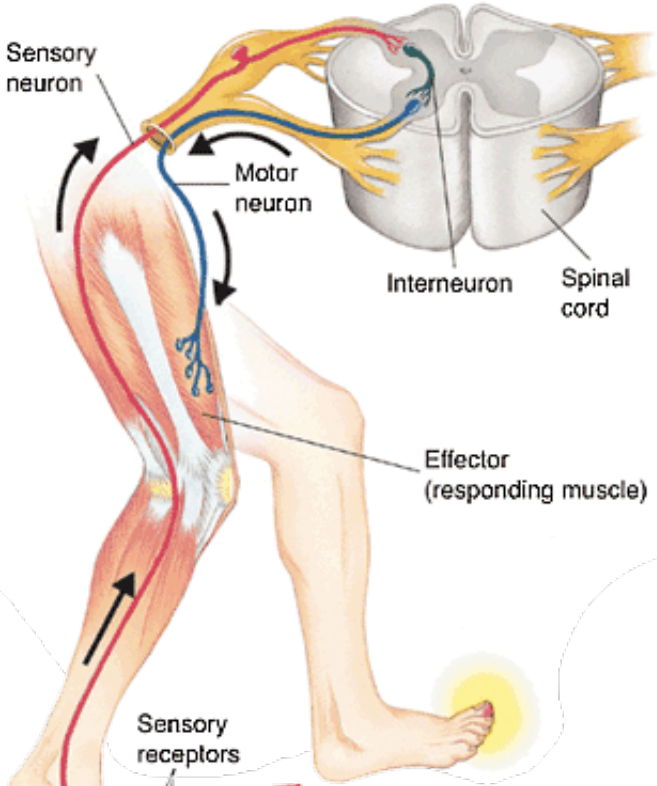


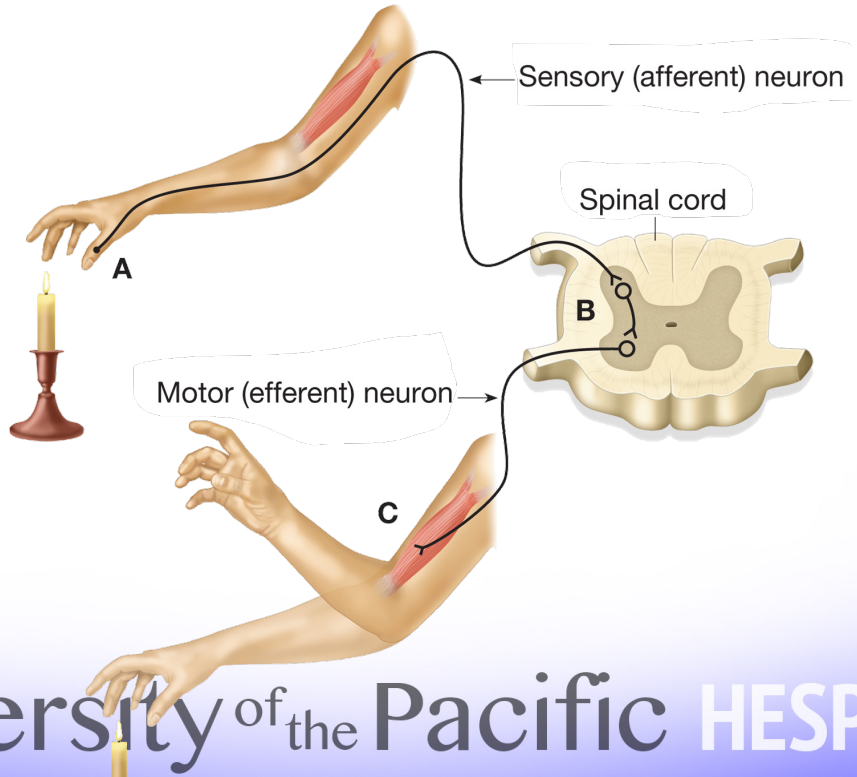


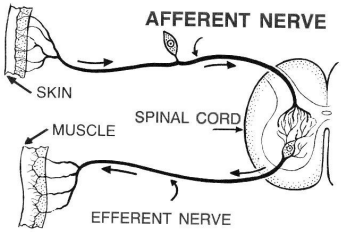
Reflex Arcs


AFFERENT NERVE
SKIN
MUSCLE
SPINAL CORD
EFFERENT NERVE

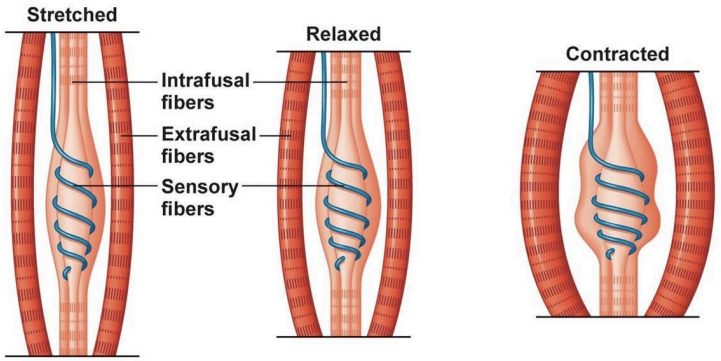

2 SENSORY NEURON
(axon conducts impulses from receptor to integrating center)
1 SENSORY RECEPTOR
(responds to a stimulus by producing a generator or receptor potential)
Interneuron
3 INTEGRATING CENTER
(one or more regions within the CNS that relay impulses from sensory to motor neurons)
4 MOTOR NEURON
(axon conducts impulses from integrating center to effector)
5 EFFECTOR
(muscle or gland that responds to motor nerve impulses)

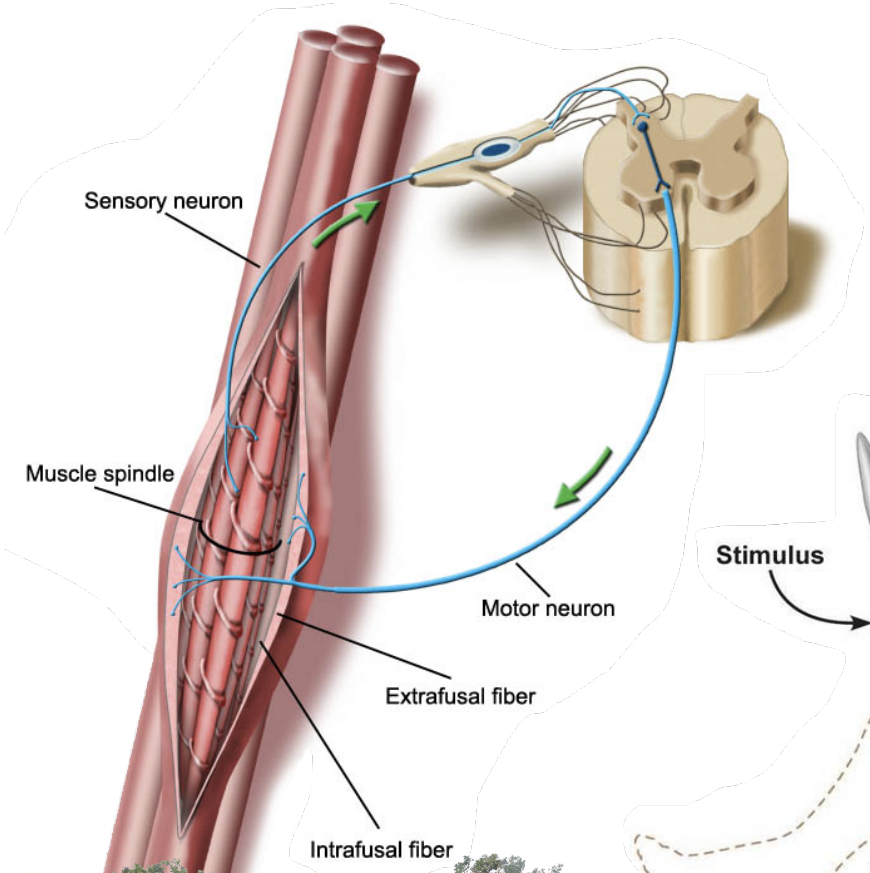

Sensory neuron
Motor neuron
Interneuron
Spinal cord
Effector (responding muscle)
Sensory receptors

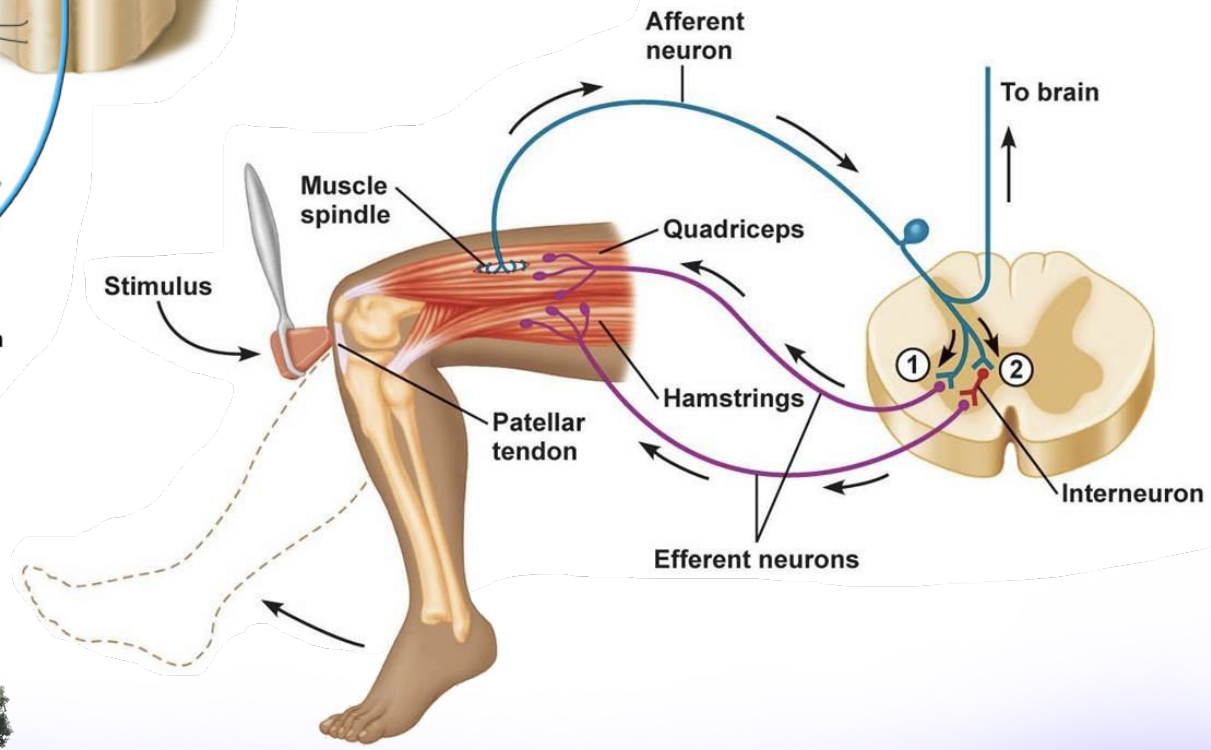

Sensory (afferent) neuron
Spinal cord
A
B
Motor (efferent) neuron
C

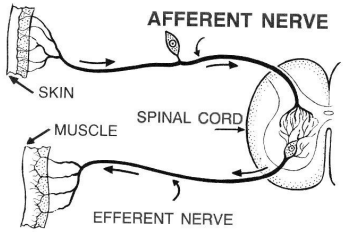


Reflex Arcs: Muscle Spindle

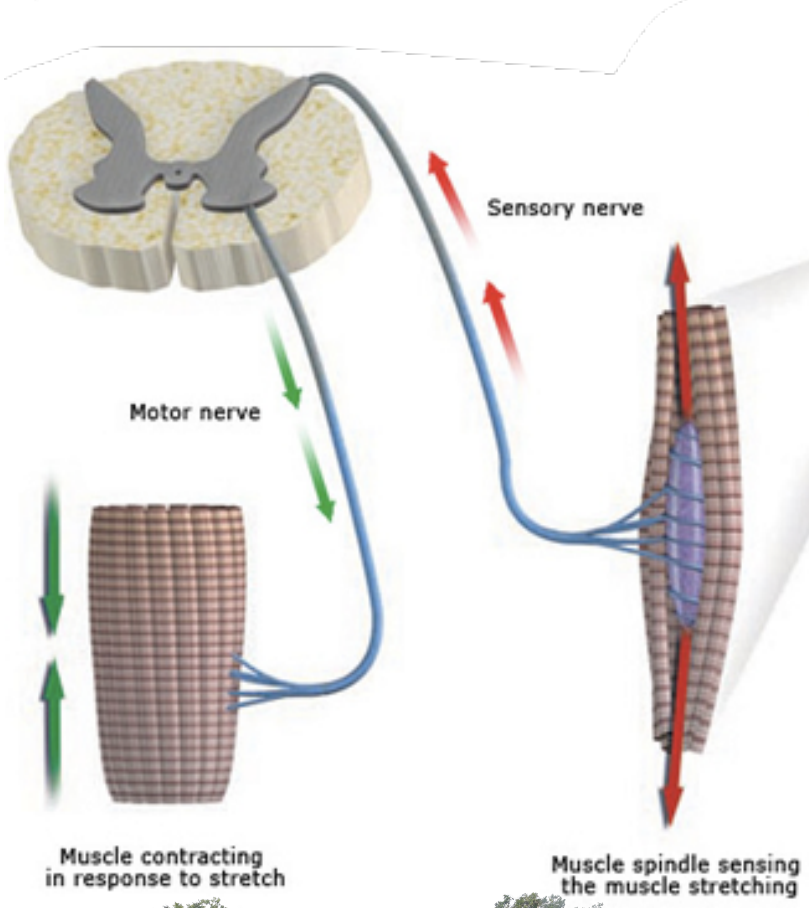


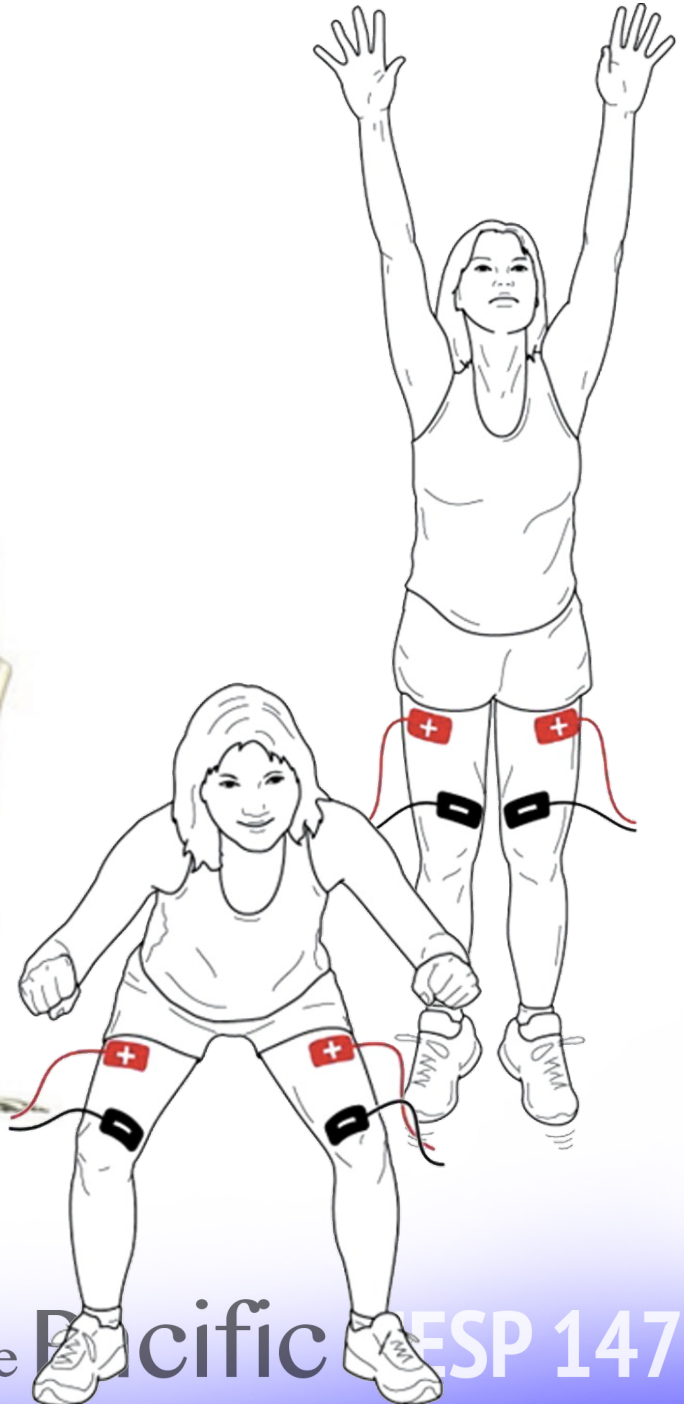


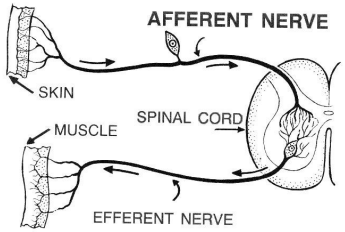




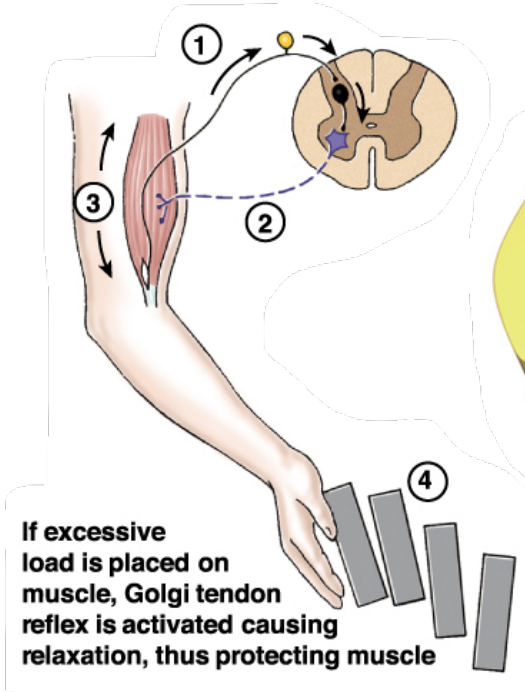
Reflex Arcs: Muscle Spindle



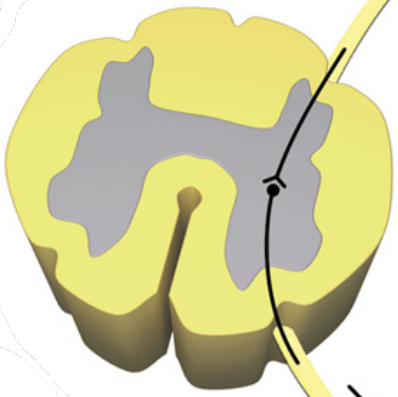


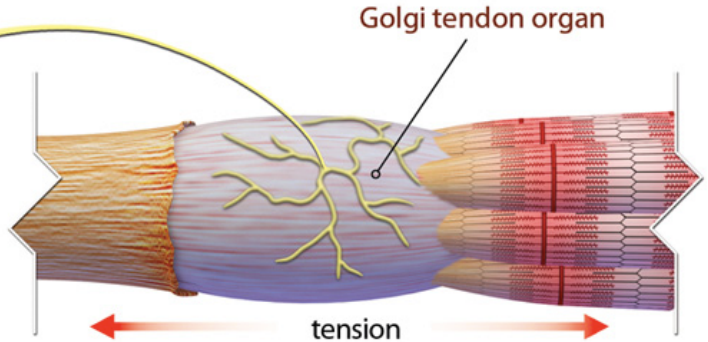


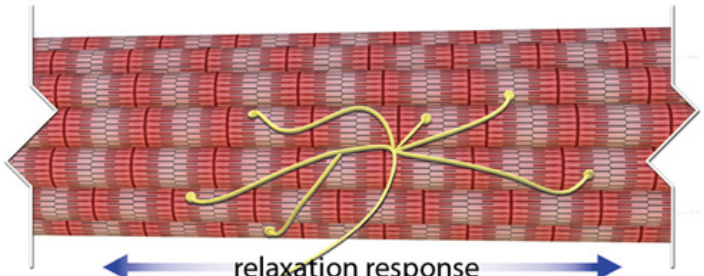
Reflex Arcs: Golgi Tendon Organ



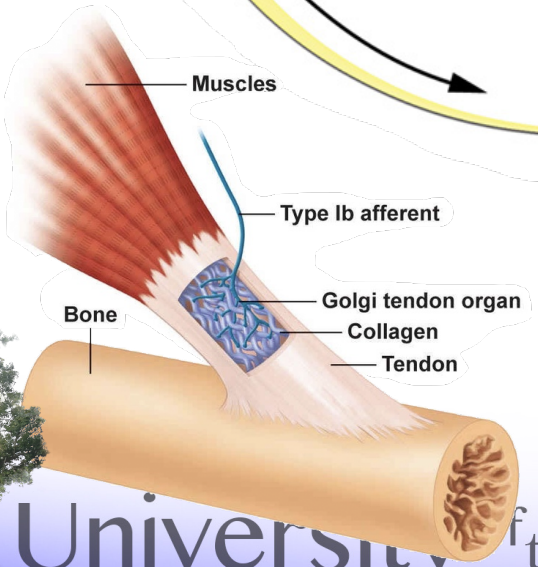
If excessive load is placed on muscle, Golgi tendon reflex is activated causing relaxation, thus protecting muscle







1. Neuron from Golgi tendon organ fires.
2. Motor neuron is inhibited.
3. Muscle relaxes.
4. [illegible] released.



Muscle Fiber Type

TYPE I	TYPE 2
SLOW TWITCH	FAST TWITCH
OXIDATIVE	GLYCOLYTIC
RED	WHITE

Muscle Fiber Type

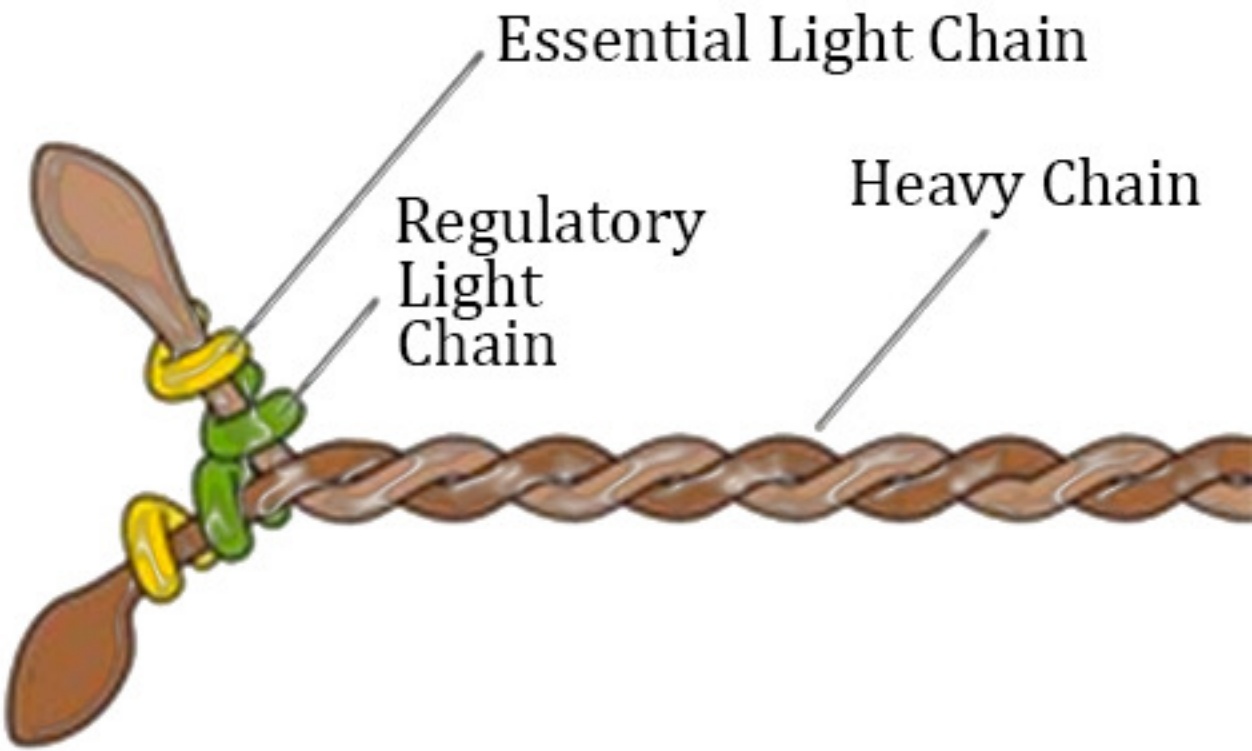
	FIBER TYPES		
Characteristic	**Type I**	**Type IIa**	**Type IIx***
Motor neuron size	Small	Large	Large
Nerve conduction velocity	Slow	Fast	Fast
Contraction speed	Slow	Fast	Fast
Relaxation speed	Slow	Fast	Fast
Fatigue resistance	High	Intermediate/Low	Low
Force production	Low	Intermediate	High
Power output	Low	Intermediate/High	High
Endurance	High	Intermediate/Low	Low
Aerobic enzyme content	High	Intermediate/Low	Low
Anaerobic enzyme content	Low	High	High
Capillary density	High	Intermediate	Low
Myoglobin content	High	Low	Low
Mitochondria size/density	High	Intermediate	Low
Fiber diameter	Small	Intermediate	Large
Color	Red	White/Red	White

Muscle Fiber Type

I ➔ Ic ➔ IIc ➔ IIac ➔ IIa ➔ IIax ➔ IIx

<u>Myosin heavy chain:</u>
Head and tail of myosin.

<u>Myosin light chain:</u>
Two per head, they bind heavy chains in neck to the tail.



Muscle Fiber Type

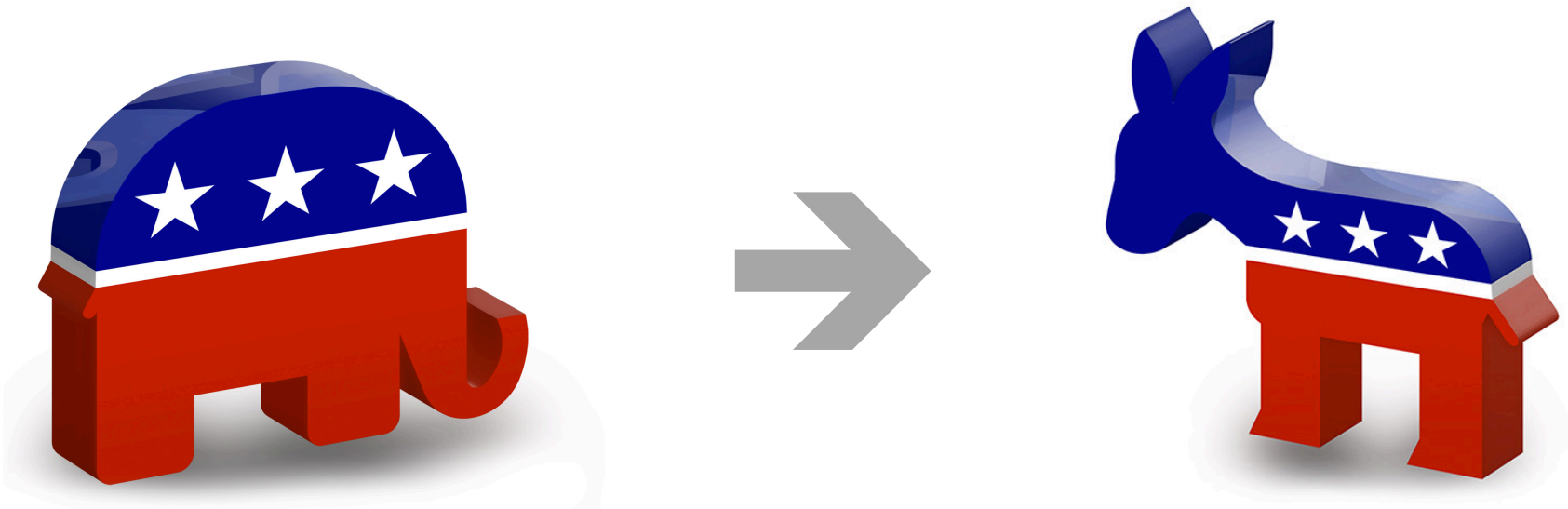
I → Ic → IIc → IIac → IIa → IIax → IIx

You want your muscle to contract all hard and fast. Stuff that matters:

- α-motor nerve characteristics
- Myosin heavy chain isoform
- Myosin regulatory light chain isoform
- Myosin ATPase activity
- Sarcoplasmic reticulum concentration and enzymes for calcium release and reuptake

Muscle Fiber Type

I → Ic → IIc → IIac → IIa ← IIax ← IIx



Fiber type switching as an exercise adaptation?

Muscle Fiber Type

I ➔ Ic ➔ IIc ➔ IIac ➔ IIa ➔ IIax ➔ IIx

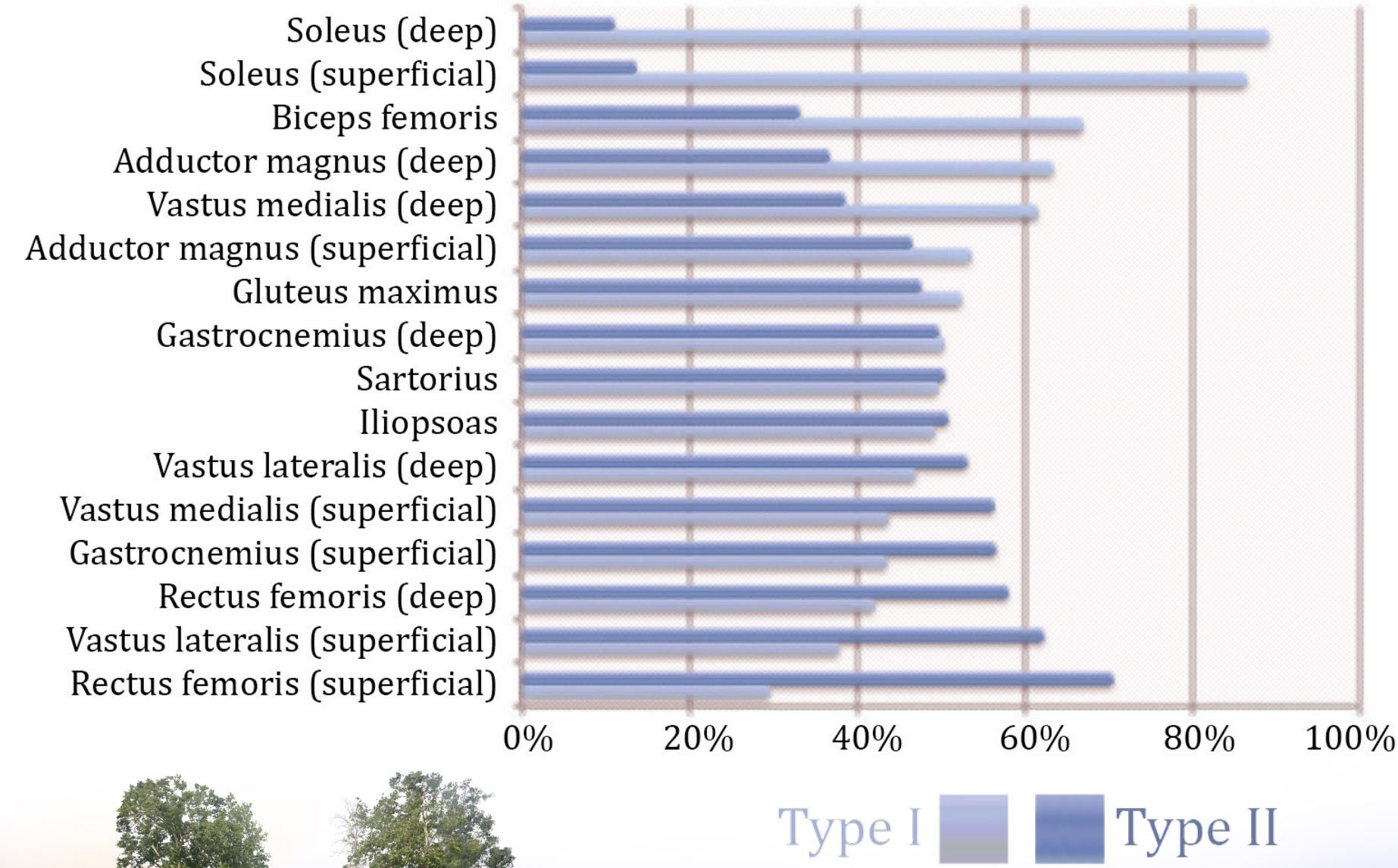
Journal of Neurocytology **20**, 903–913 (1991)

Axonal sprouting and changes in fibre types after running-induced muscle damage

A. WERNIG*, T. F. SALVINI[‡] and A. IRINTCHEV

Fiber type switching as an exercise adaptation?

Muscle Fiber Type Composition



Muscle Fiber Type

Cycling efficiency is related to the percentage of Type I muscle fibers

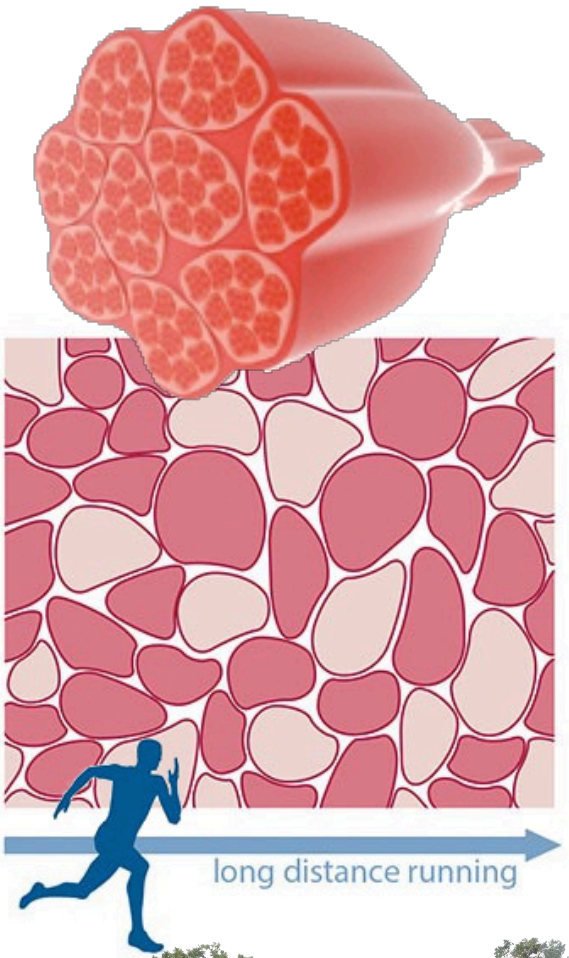
EDWARD F. COYLE, LABROS S. SIDOSSIS, JEFFREY F. HOROWITZ, and JOHN D. BELTZ

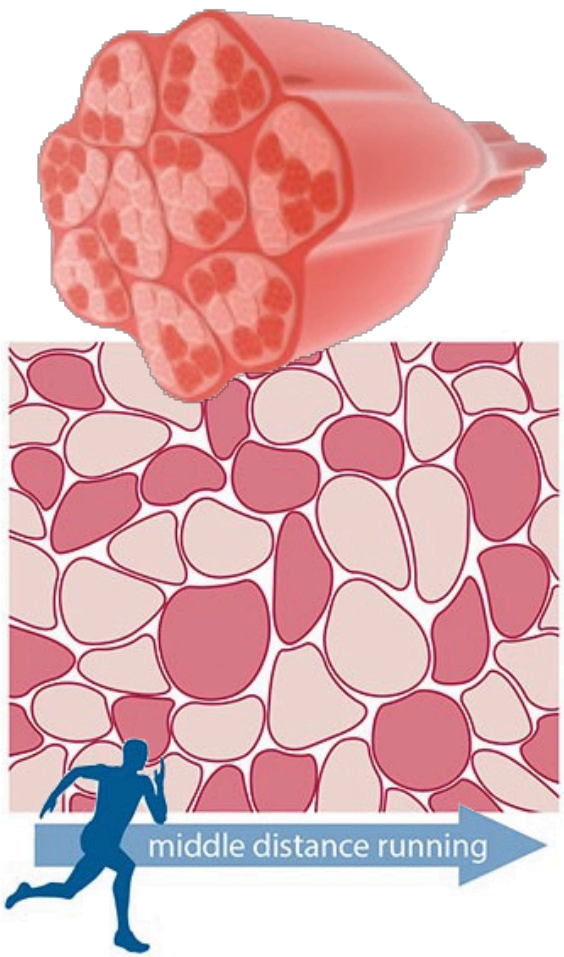
0195-9131/92/2407-0782$3.00/0
MEDICINE AND SCIENCE IN SPORTS AND EXERCISE

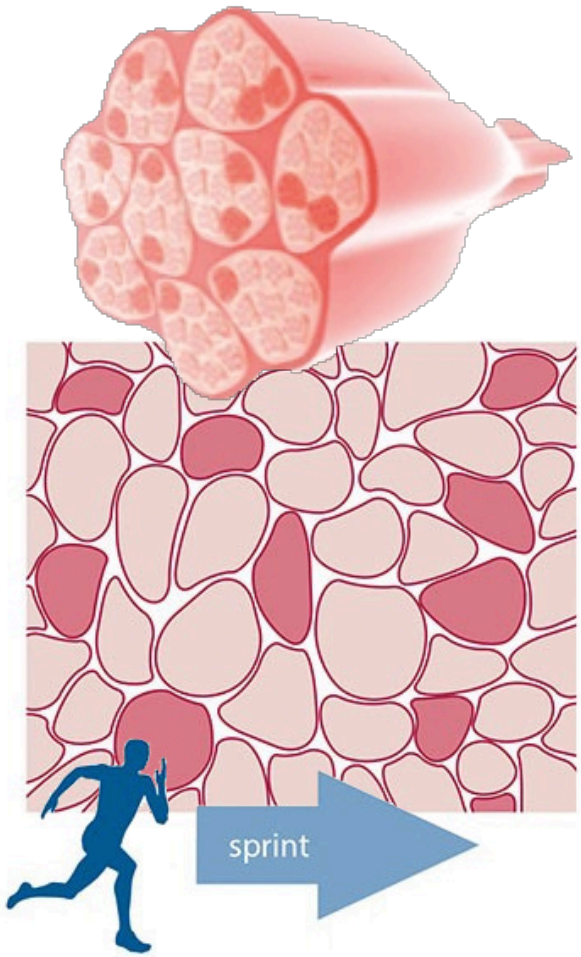
Subject Number	% Type I (*N* = 19)
1	32
2	37
3	38
4	40
5	45
6	50
7	50
8	54
9	54
10	55
11	61
12	63
13	63
14	64
15	64
16	70
17	70
18	75
19	76

% Type I fibers in vastus lateralis of male twenty-somethings (n=19).

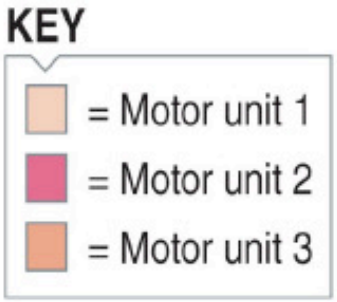
What sports require this makeup?

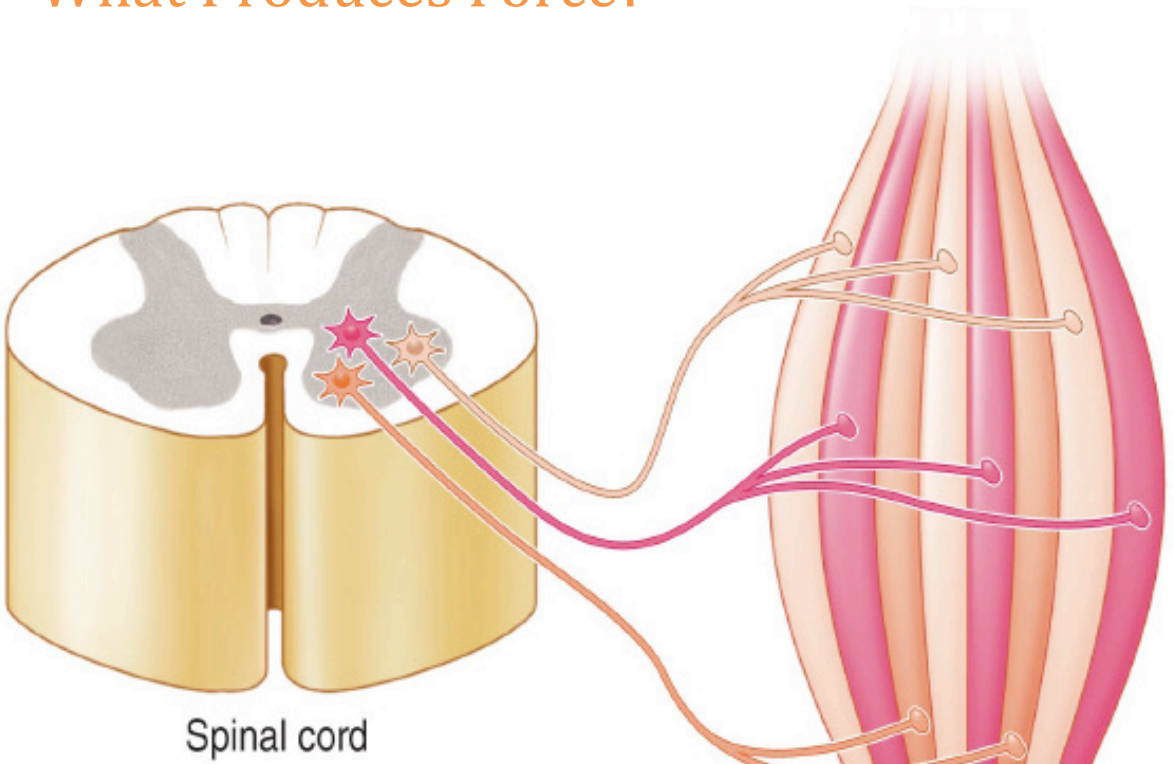


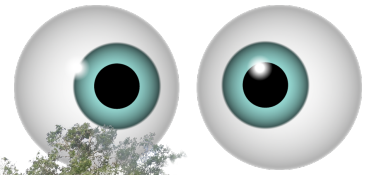




What Produces Force?







Vs.

What Produces Force?

From the *neural* perspective, where does strength come from?

The total number of motor units recruited

The size of those units (size principle)

Rate coding.

What Produces Force?

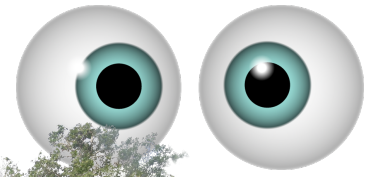
Muscle	Number of motor axons	Number of muscle fibers	Innervation ratio
Biceps	774	580 000	750
Brachioradialis	315	129 000	410
First dorsal interosseous	119	40 500	340
Medial gastrocnemius	579	1 120 000	1934
Tibialis anterior	445	250 200	562

Disorders of Voluntary Muscle, Eighth Edition

Section 1 The scientific basis of muscle disease

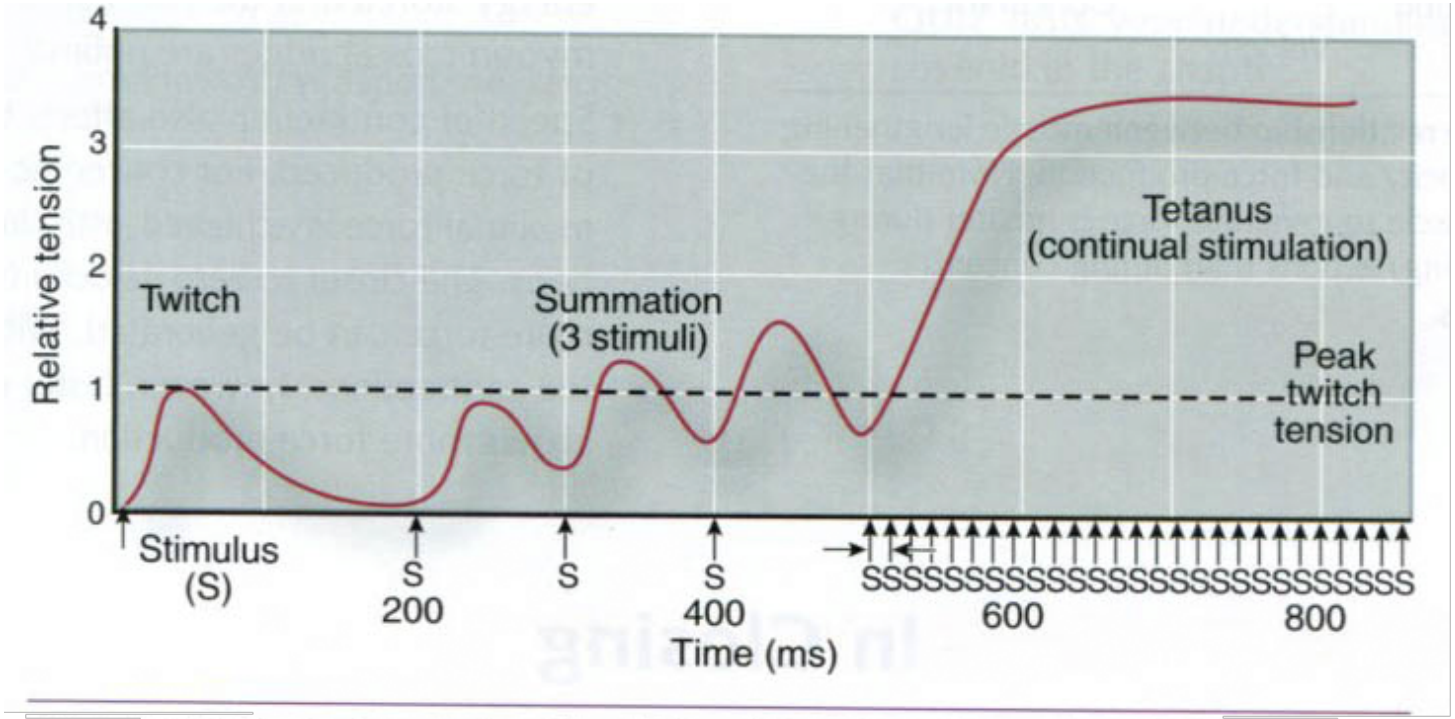
Cambridge University Press

Mary Kay Floeter



Vs.

At the neural level…



Variation in force or tension produced based on electrical stimulation frequency, illustrating the concept of a twitch, summation, and tetanus.

Adapted, by permission, from G.A. Brooks, et al., 2005, *Exercise Physiology: human bioenergetics and its applications*, 4th ed. (New York: McGraw-Hill), 388. With permission of the McGraw-Hill Companies.

Rate coding is the frequency of achieving an action potential (duration of interspike intervals); measured as firing rate over a given duration

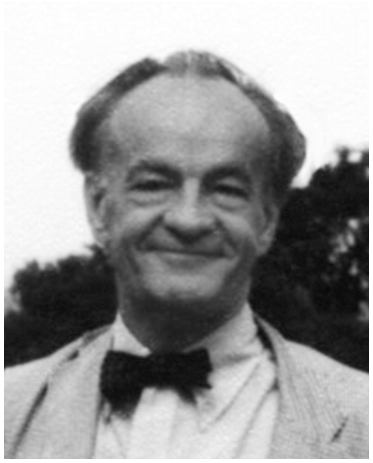
Henneman's Size Principle

First to discover the phenomenon:

Denny-Brown D, Pennybacker J. (1938). Fibrillation and fasciculation in voluntary muscle. *Brain,* 61: 311-334

"Orderly Recruitment"

Henneman's Size Principle



Second to discover it:

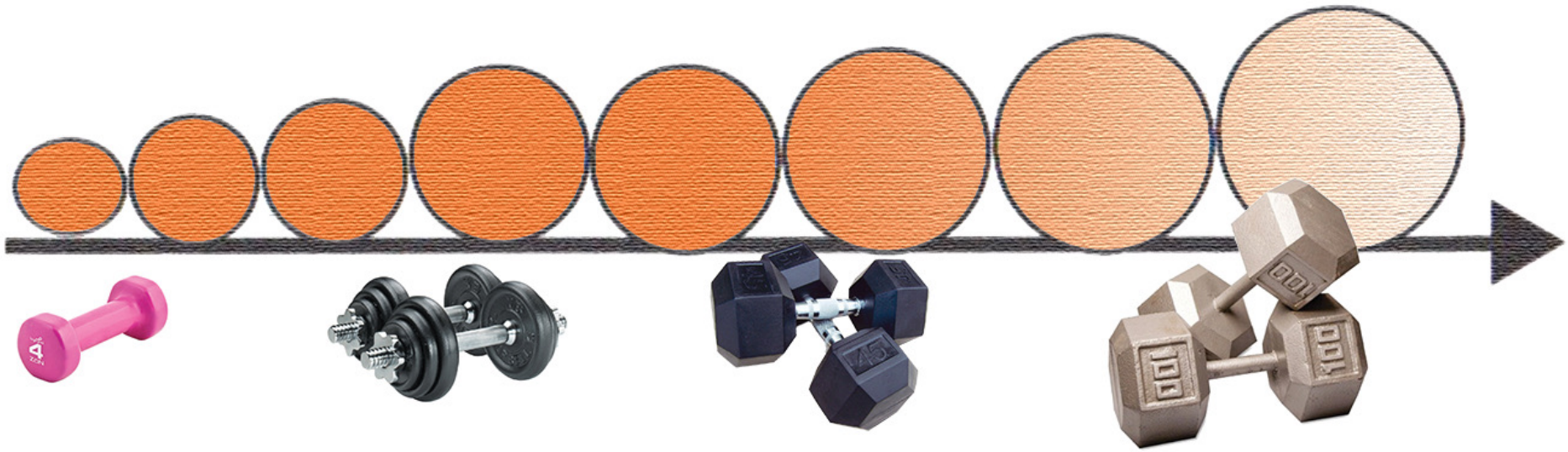
Science
AAAS

Relation between Size of Neurons and Their Susceptibility to Discharge
Author(s): Elwood Henneman
Source: *Science*, New Series, Vol. 126, No. 3287 (Dec. 27, 1957), pp. 1345-1347

VE RI TAS
HARVARD

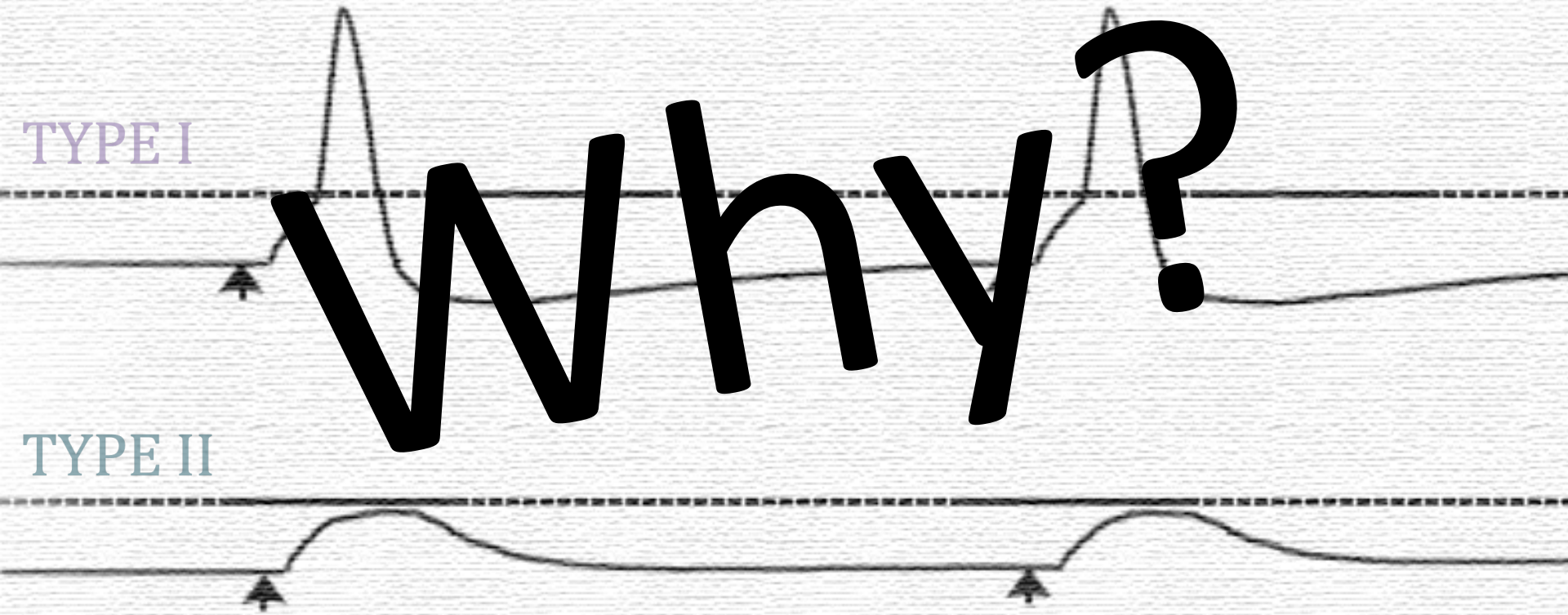
"Size Principle"

How Are Motor Units Controlled?



How Are Motor Units Controlled?

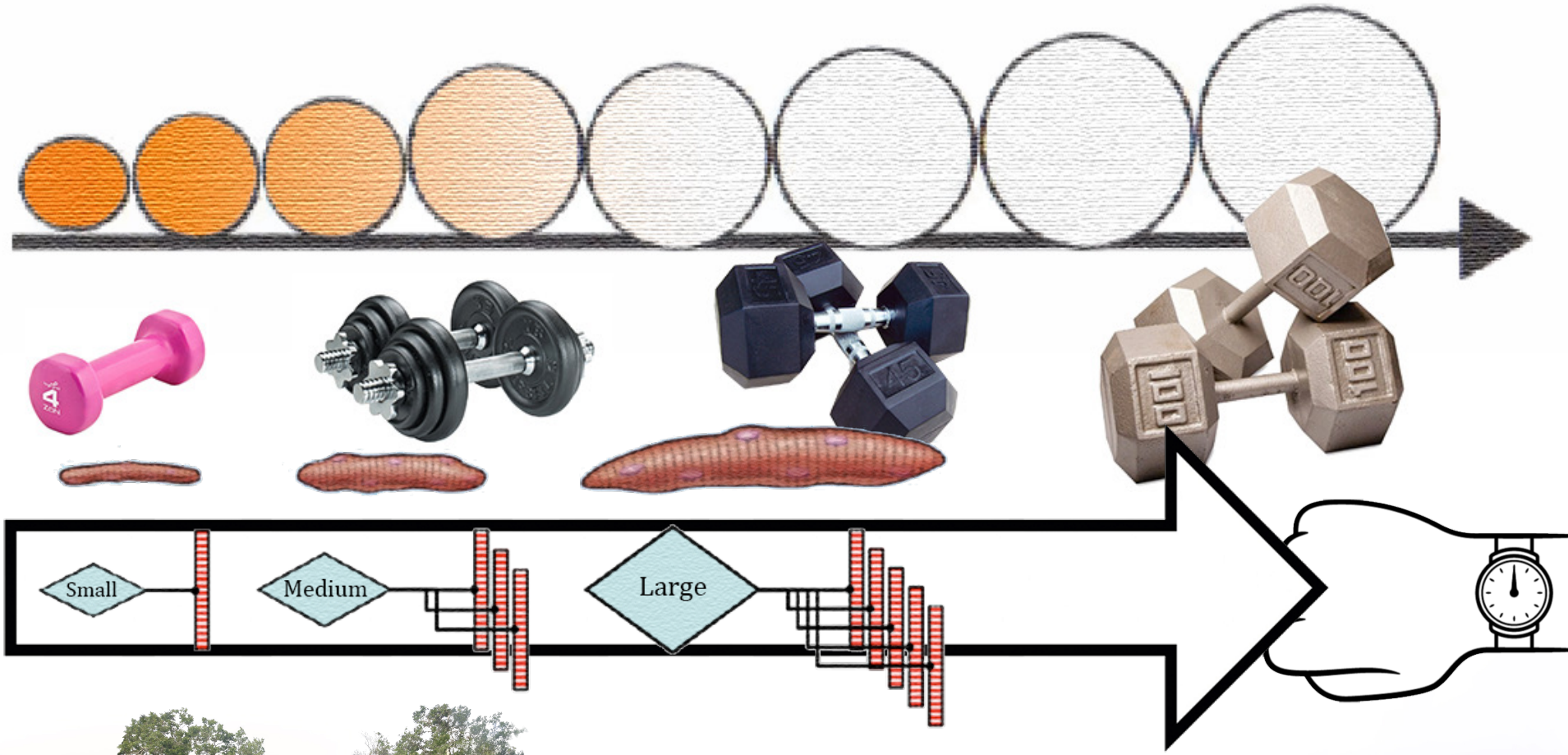
How Are Motor Units Controlled?



Why?

How Are Motor Units Controlled?

I → Ic → IIc → IIac → IIa → IIax → IIx

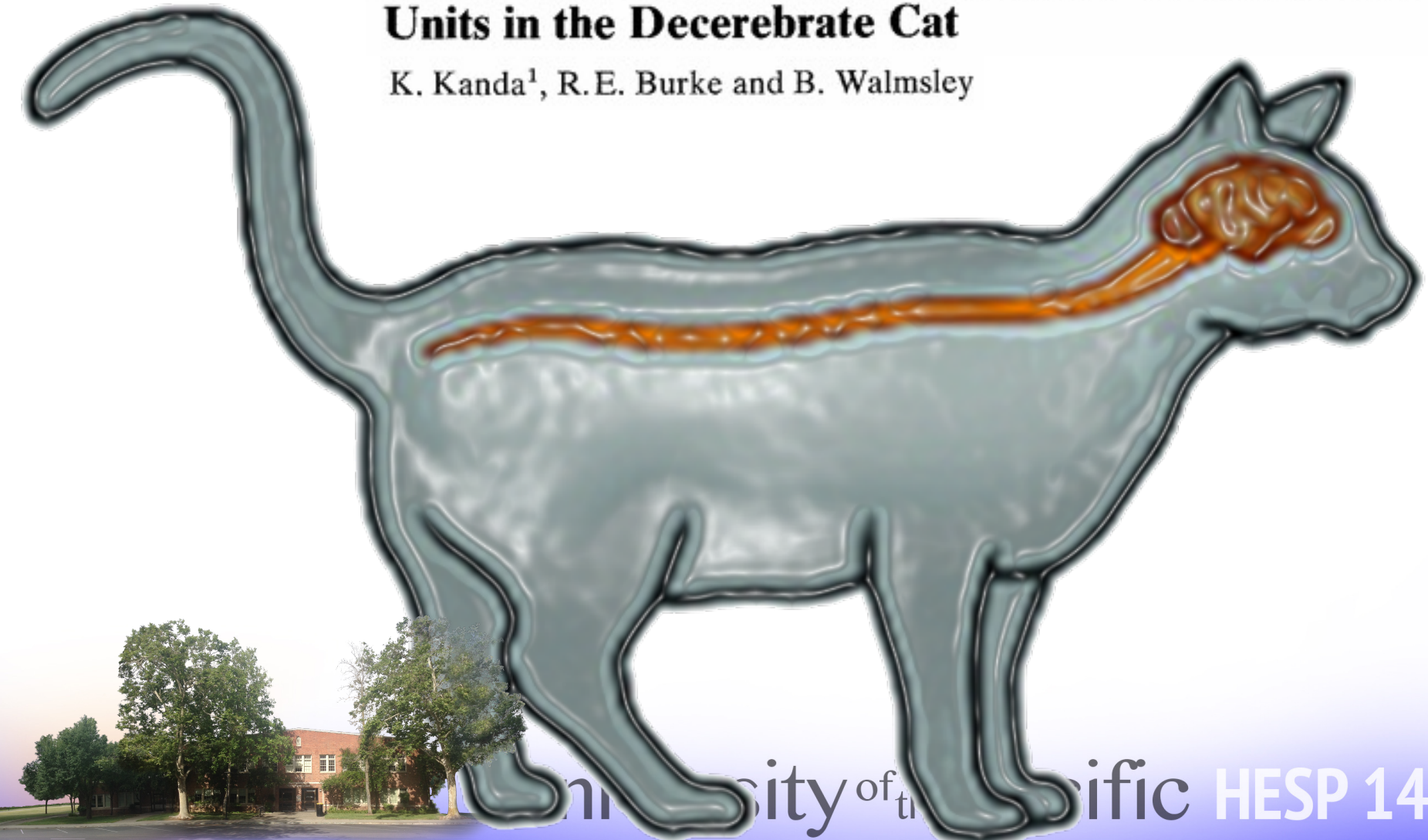


Are there exceptions to this rule?

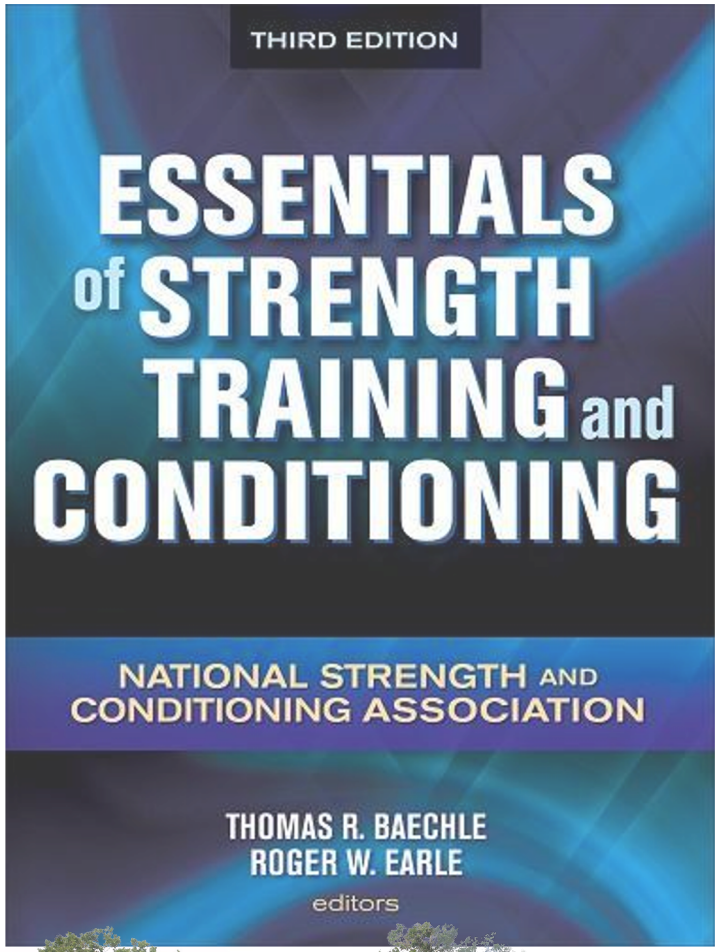
Exp. Brain Res. 29, 57–74 (1977)

Differential Control of Fast and Slow Twitch Motor Units in the Decerebrate Cat

K. Kanda[1], R.E. Burke and B. Walmsley



Are there exceptions to this rule?



A few exceptions to the size principle exist. That is, **selective recruitment** of fast-twitch motor units may occur under certain circumstances that allow an athlete to inhibit lower-threshold motor units and in their place activate the higher-threshold motor units critical to optimal speed and power performance. Rapid changes of direction in force production and ballistic muscular contractions (97, 164, 206), comparable to movements in plyometric, speed, power, and agility training, have been shown to lead to preferential recruitment of fast-twitch motor units. These variations in recruitment order may benefit high-velocity power training in which time of force production is vital to success. For example, it would be very difficult for an athlete to generate enough angular velocity and power to attain maximal height for the vertical jump test if that athlete had to recruit the entire slow-twitch motor unit pool prior to activation of the fast-twitch units. Because the time between the countermovement and the subsequent jump takeoff is often less than 0.4 second, there simply is not enough time to recruit all of the motor units in order and still be able to perform an explosive jump. Thus, selective recruitment appears to be a beneficial intrinsic neural mechanism favoring explosive exercise. In addition, specific training may enhance selective recruitment, which may improve sport performance.

Practical Application

The International Journal of Sports Physical Therapy

CLINICAL COMMENTARY

POSTACTIVATION POTENTIATION: AN INTRODUCTION

Daniel Lorenz, DPT, PT, ATC/L, CSCS
Kansas City, Kansas, USA

Volume 6, Number 3 | September 2011

The most important muscle characteristic affecting the magnitude of PAP is fiber type, with the greatest potential for enhanced PAP in muscles with the highest proportion of Type II fibers.[15,19,20] Further, PAP is greater in muscles with the shortest twitch contraction time.[17,19,21,22] Based on muscle fiber type, athletes who perform in maximal intensity activities that depend on Type II muscle fibers (i.e. sprinting, weightlifting, throwing, jumping) would also show the greatest PAP in muscles involved in their sports performance.[23]

Practical Application

The International Journal of Sports Physical Therapy

CLINICAL COMMENTARY

POSTACTIVATION POTENTIATION: AN INTRODUCTION

Daniel Lorenz, DPT, PT, ATC/L, CSCS
Kansas City, Kansas, USA

Volume 6, Number 3 | September 2011

Sort of practical application

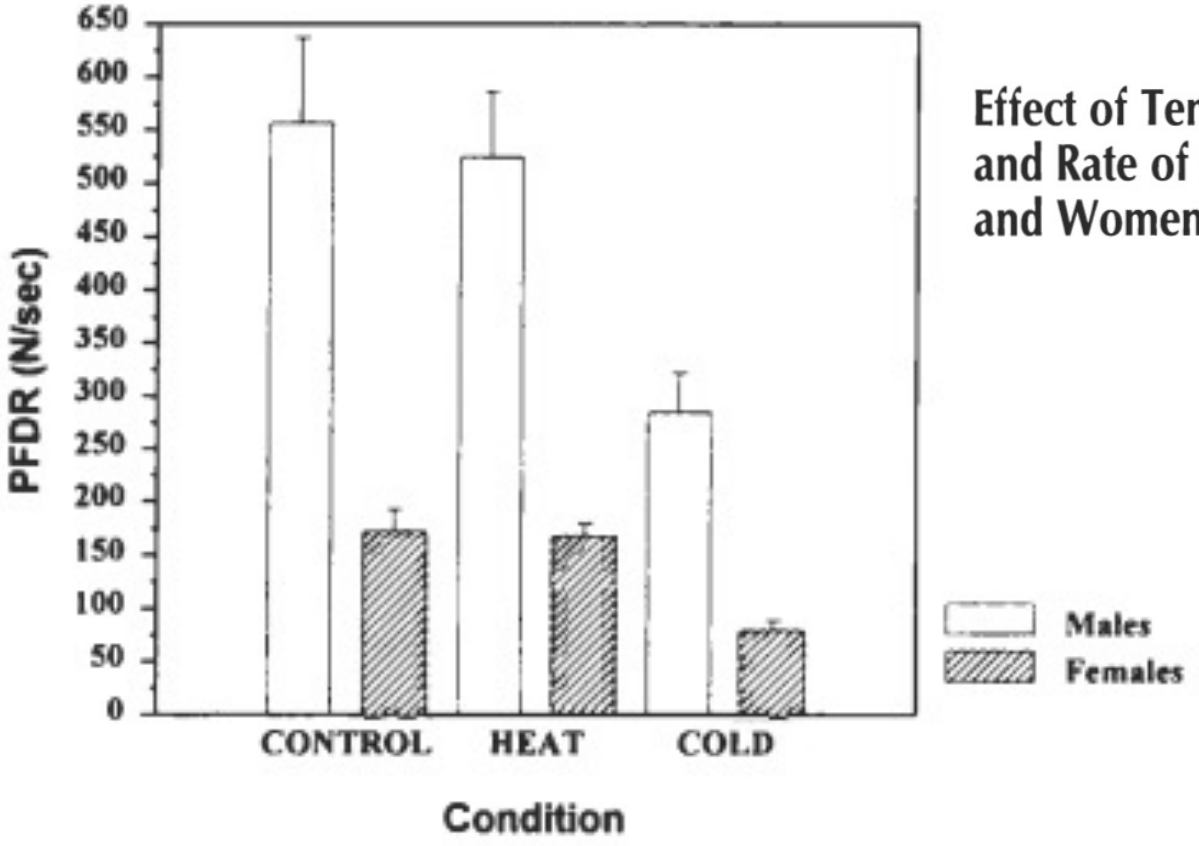
FIG. 153

J Appl Physiol 94: 1410–1420, 2003.
First published December 13, 2002; 10.1152/japplphysiol.01183.2001.

Activation imbalances in lumbar spine muscles in the presence of chronic low back pain

Lars I. E. Oddsson[1] and Carlo J. De Luca[1,2]
[1]NeuroMuscular Research Center and [2]Department of Biomedical Engineering, Boston University, Boston, Massachusetts 02215

Sort of practical application



Volume 20 • Number 2 • August 1994 • JOSPT

Effect of Temperature on Muscle Force and Rate of Muscle Force Production in Men and Women

Mark W. Cornwall, PhD, PT[1]

Sort of practical application

Journal of Sports Sciences, 2004, **22**, 898–916

Exercise in the cold

MYRA NIMMO*

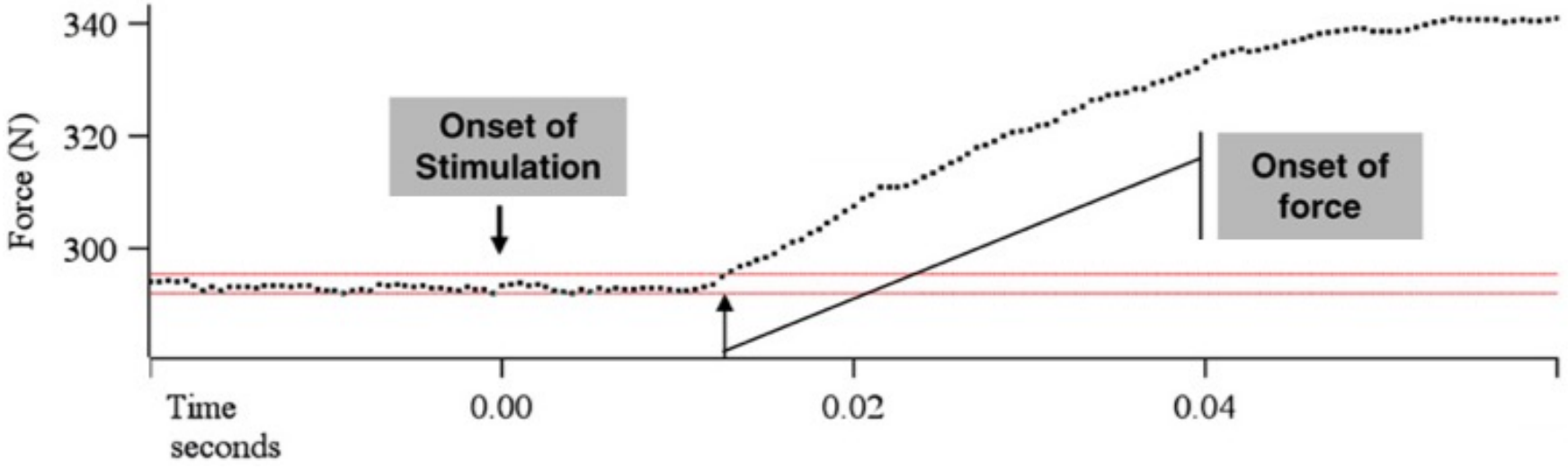
Exercising in the cold is not an attractive option for many athletes; however, defining what represents cold is difficult and is not standard for all events. If the exercise is prolonged and undertaken at a moderate intensity, environmental temperatures around 11°C can be an advantage. If the intensity is lower than this value and the individual does not generate sufficient metabolic heat to offset the effects imposed by the cold environment, then temperatures of 11°C can be detrimental to performance. Similarly, when the performance involves dynamic explosive contractions, then a cold ambient temperature can have a negative influence. Additional factors such as the exercising medium, air or water, and the anthropometric characteristics of the athlete will also make a difference to the strategies that can be adopted to offset any negative impact of a cold environment on performance. To plan for a performance in the cold requires an understanding of the mechanisms underpinning the physiological response. This review attempts to outline these mechanisms and how they can be manipulated to optimize performance.

Sort of practical application

Clinical Neurophysiology 121 (2010) 1700–1706

Effect of gender, age, fatigue and contraction level on electromechanical delay

Ş. Utku Yavuz [a,b], Aylin Şendemir-Ürkmez [b,c], Kemal S. Türker [b,*]



Sort of practical application

Effects of Dynamic and Static Stretching on Vertical Jump Performance and Electromyographic Activity

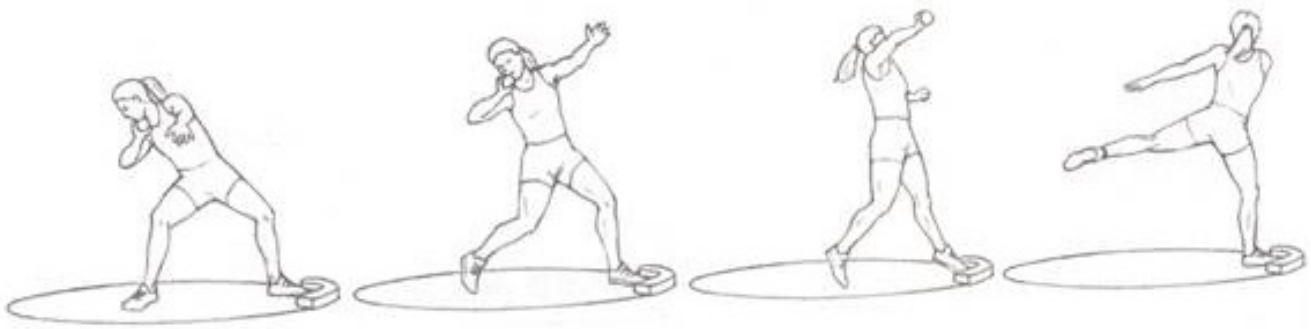
Paul A. Hough,[1] Emma Z. Ross,[2] and Glyn Howatson[1]

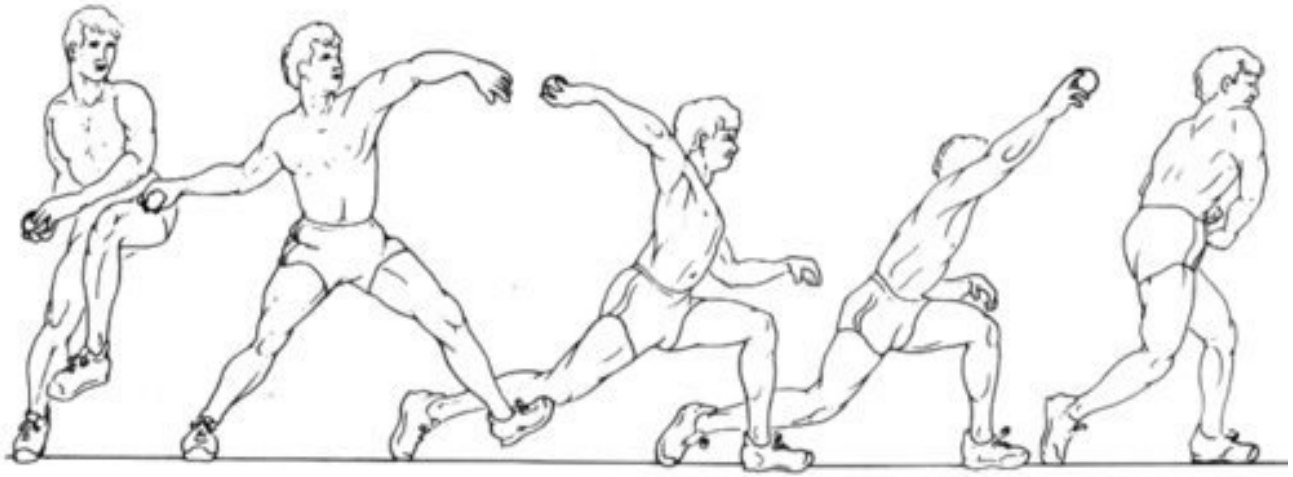
[1]School of Human Sciences, St. Mary's University College, Twickenham, United Kingdom; and [2]Centre for Sports Medicine and Human Performance, Brunel University, Uxbridge, United Kingdom

Abstract

Hough, PA, Ross, EZ, and Howatson, G. Effects of dynamic and static stretching on vertical jump performance and electromyographic activity. *J Strength Cond Res* 23(2): 507–512, 2009–The results of previous research have demonstrated that static stretching (SS) can reduce muscular performance and that dynamic stretching (DS) can enhance muscular performance. The purpose of this study was to assess the effects of SS and DS on vertical jump (VJ) performance and electromyographic (EMG) activity of the m. vastus medialis. Eleven healthy men (age 21 ± 2 years) took part in 3 conditions (no stretching [NS], SS, and DS), on separate occasions in a randomized, crossover design. During each condition, measurements of VJ height and EMG activity during the VJ were recorded. A repeated-measures analysis of variance and post hoc analysis indicated that VJ height was significantly less (4.19 ± 4.47%) after SS than NS ($p < 0.05$) and significantly greater (9.44 ± 4.25%) in DS than SS ($p < 0.05$). There was significantly greater EMG amplitude in the DS compared with the SS ($p < 0.05$). The results demonstrated that SS has a negative influence on VJ performance, whereas DS has a positive impact. Increased VJ performance after DS may be attributed to post-activation potentiation, whereas the reduction in VJ performance after SS may be attributable to neurological impairment and a possible alteration in the viscoelastic properties of the muscular tendon unit (MTU). This investigation provides some physiological basis for the inclusion of DS and exclusion of SS in preparation for activities requiring jumping performance.

Practical Application





Motor Unit Conduction Velocity

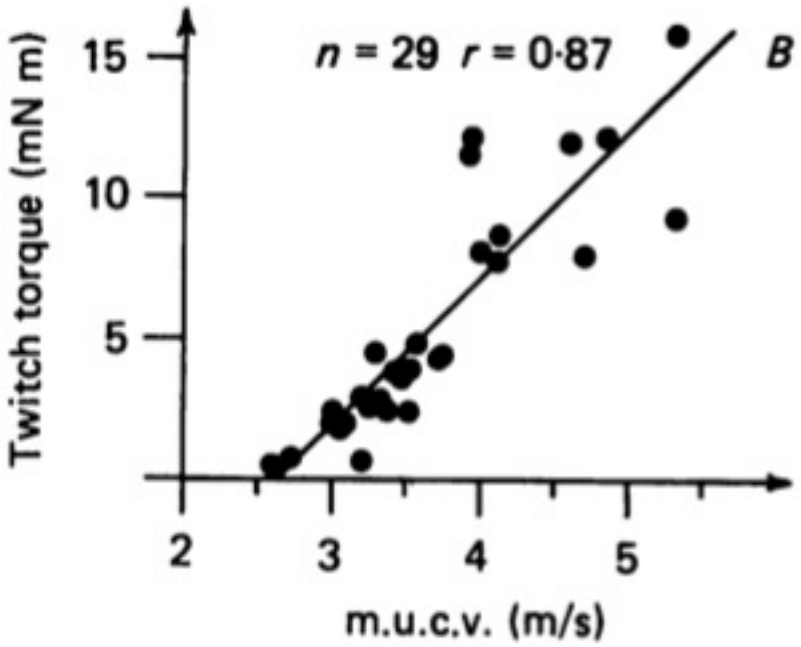
Higher threshold motor units have faster conduction velocities

J. Physiol. (1987), **391**, *pp.* 561–571

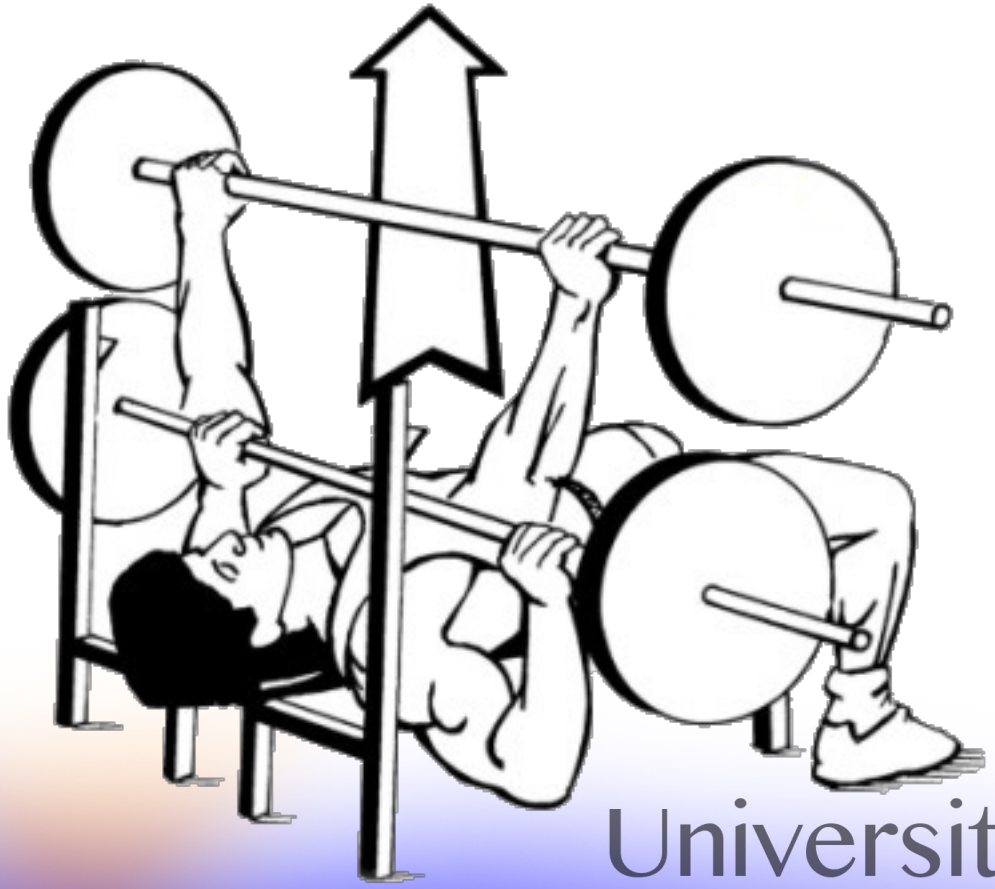
MUSCLE FIBRE CONDUCTION VELOCITY IN MOTOR UNITS OF THE HUMAN ANTERIOR TIBIAL MUSCLE: A NEW SIZE PRINCIPLE PARAMETER

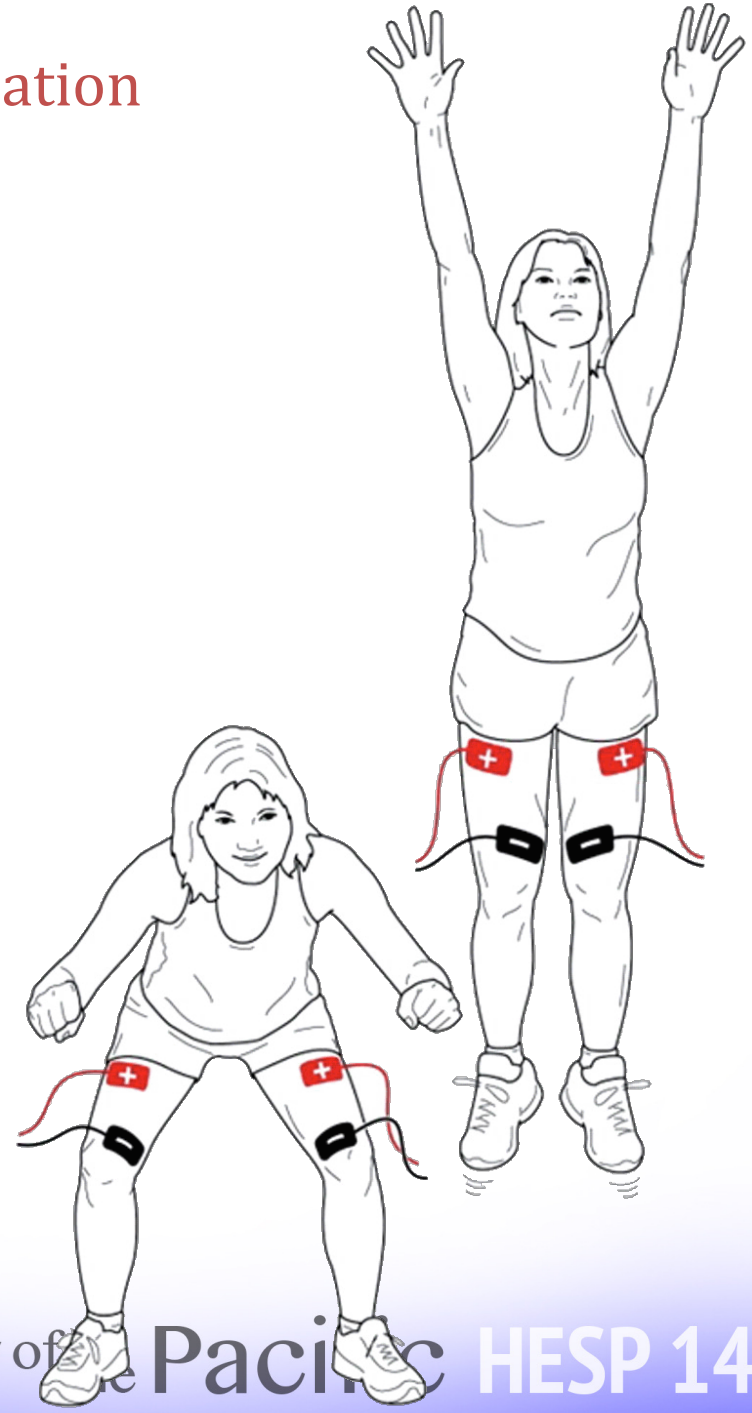
BY STEEN ANDREASSEN AND LARS ARENDT-NIELSEN

From the Institute of Electronic Systems, Aalborg University, Strandvejen 19, DK 9000, Denmark



Practical Application





Climbing Henneman's Ladder

What is the most dependable way to recruit every motor unit in a particular tissue?

Most studies rely on MVC to achieve max motor unit activation:

1: De Serres SJ & Enoka RM. (1998). Older adults can maximally activate the biceps brachii muscle by voluntary command. *Journal of Applied Physiology*, 84: 284-291. **2:** Jakobi JM, Rice CL. (2002). Voluntary muscle activation varies with age and muscle group. *Journal of Applied Physiology*, 93:457–62. **3:** Kent-Braun JA & Ng AV. (1999). Specific strength and voluntary muscle activation in young and elderly women and men. *Journal of Applied Physiology*, 87: 22-29. **4:** Klass M, Baudry S, Duchateau J. (2005). Aging does not affect voluntary activation of the ankle dorsiflexors during isometric, concentric, and eccentric contractions. *Journal of Applied Physiology*, 99: 31-38. **5:** Klein CS, Rice CL, Marsh GD. (2001). Normalized force, activation, and coactivation in the arm muscles of young and old men. *Journal of Applied Physiology*, 91: 1341-1349. **6:** Knight CA & Kamen G. (2001). Adaptations in muscular activation of the knee extensor muscles with strength training in young and older adults. *Journal of Electromyography and Kinesiology*, 11: 405-412. **7:** Roos MR, Rice CL, Connelly DM, Vandervoort AA. (1999). Quadriceps muscle strength, contractile properties, and motor unit firing rates in young and old men. *Muscle Nerve* 22: 1094–1103. **8:** Vandervoort AA, McComas AJ. (1986). Contractile changes in opposing muscles of the human ankle joint with aging. Journal of Applied Physiology, 61: 361-367.

Climbing Henneman's Ladder

Size Principle is not based *strictly* on load

It's based *mostly* on load, but those mechanoreceptors crunch a lot of data:

- Amount of force
- Duration of tension
- Speed of contraction
- Angles, muscle length, etc.

"Run the numbers, then crunch them just to be sure."

Depending on the muscle and the characteristics of the load, it may be possible to recruit nearly all of its motor units using a load that's just 30% of one's maximum strength; in other muscles (and other contexts), it might require as much as 90% of one's maximum strength to achieve full recruitment.

Practical Application

Should you do this:

Or this:

What happens with age?

Why is the human form adaptable?

Biological purposes of life:

1. Self preservation, and **2.** Replication of the gene.

On some level, these are the same thing. How else would the gene replicate if the host was always dying?

Some responses are relatively permanent;
Other responses are relatively impermanent.

A lot of the immediate environment-tolerating changes can be accounted for by accommodation:

All very different phenomena

- Habituation
- Sensitization
- Accommodation
- Adaptation
- Genetic Adaptation

Some responses are relatively permanent;
Other responses are relatively impermanent.

All living cells, organisms, creatures (etc.) spend their lives making changes to better tolerate their environments. Types of changes:

Habituation: Decrease in sensitivity to a stimulus.

Sensitization: Increase in sensitivity to a stimulus.

Some responses are relatively permanent;
Other responses are relatively impermanent.

A lot of the immediate environment-tolerating changes can be accounted for by accommodation:

Adaptations are relatively permanent. Other alterations aimed at tolerating biological stresses are relatively impermanent. If one *adapts* to a stressor, that adaptation takes time to undo. If one merely ***accommodates*** *it*, it's readily reversible.

1: Cevik MÖ. (2014). Habituation, sensitization, and Pavlovian conditioning. *Frontiers in Integrative Neuroscience*, 8: 13. **2:** Harris JD. (1943). Habituatory response decrement in the intact organism. *Psychological Bulletin*, 40: 385-422. **3:** Rankin CH, Abrams T, Barry RJ, Bhatnagar S, Clayton DF, Colombo J, Coppola G, Geyer MA, Glanzman DL, Marsland S, McSweeney FK, Wilson DA, Wu CF, Thompson RF. (2009). Habituation revisited: an updated and revised description of the behavioral characteristics of habituation. *Neurobiology of Learning and Memory*, 92(2): 135-138. **4:** Thompson RF & Spencer WA. (1966). Habituation: A Model Phenomenon for the Study of Neuronal Substrates of Behavior. *Psychological Review*, 73(1): 16-43.

Some responses are relatively permanent;
Other responses are relatively impermanent.

A lot of the immediate environment-tolerating changes can be accounted for by accommodation:

Accommodation: Immediate, relatively impermanent environment-tolerating changes. *(Acute, immediate form of habituation.)*

Adaptation: Delayed, relatively permanent environment-tolerating changes.

Accommodation is readily reversible; adaptation takes time to undo.

Accommodation vs. Adaptation

In accommodation, you get used to stuff (pretty quickly).

You develop a tolerance to the immediate stress.

If it's bright out, your pupils accommodate (acutely change) so you can see things without squinting so hard.

Notice the dilation.

Adaptation

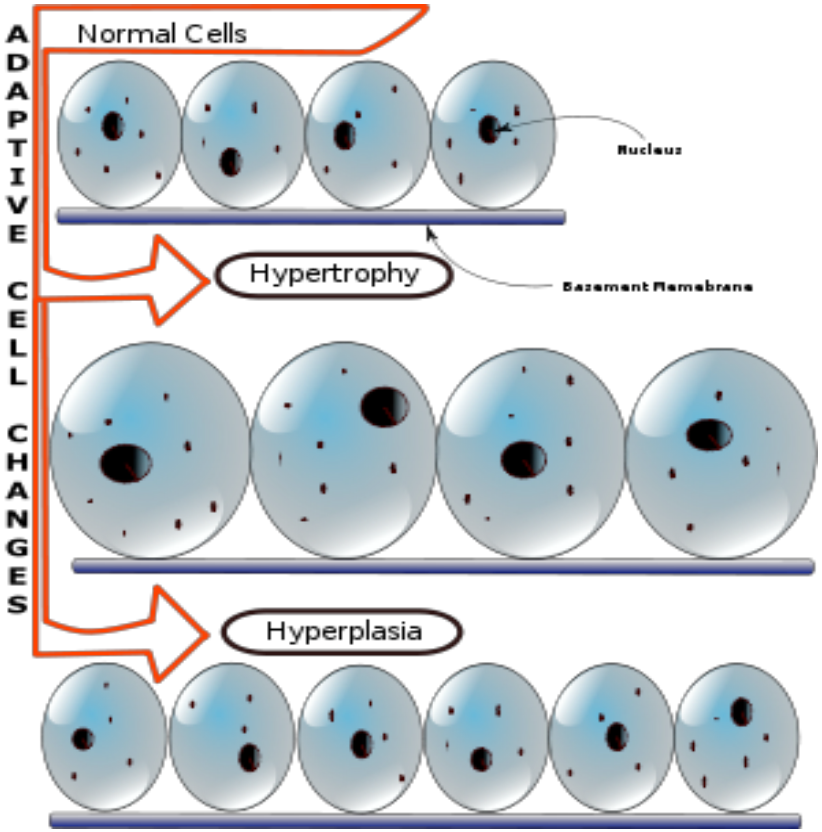
What is it?

A chronic change:

If bright lights caused the cells in your eyes t
multiply (hyperplasia) or grow (hypertrophy)
or shrink (atrophy) or change their cell type
(metaplasia), and the changes were relatively
permanent, that would be adaptation.

Accommodation vs. Adaptation

Hypertrophy
vs.
hyperplasia



We adapt to tolerate our environment, but no two adaptations are the same.

1892: Julius Wolff publishes *The Law of Bone Remodeling.*

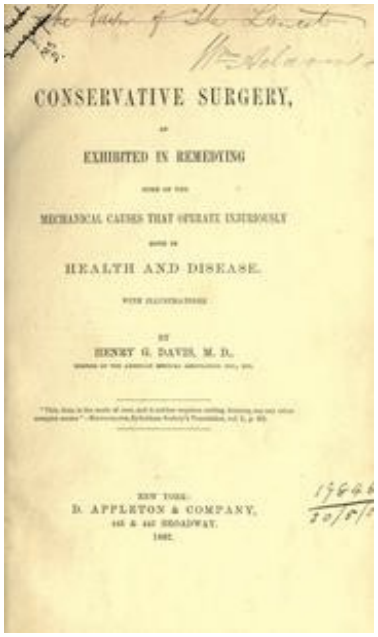
Discusses the metabolism of bones.

Architecture of bones can be altered with mechanical loading.

And the nature of the load matters.

Came to be known as “Wolff’s Law”.

1867: Henry Gassett Davis publishes his (slightly less famous) book, *Conservative Surgery*.



CONSERVATIVE SURGERY,

EXHIBITED IN REMEDYING

MECHANICAL CAUSES THAT OPERATE INJURIOUSLY

HEALTH AND DISEASE.

HENRY G. DAVIS, M. D.

D. APPLETON & COMPANY,

1867.

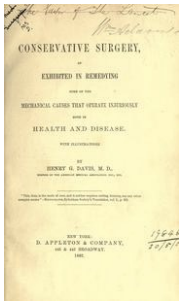
Ligaments ("or any other soft tissue") adapt to the forces of stretching with a lengthening response.

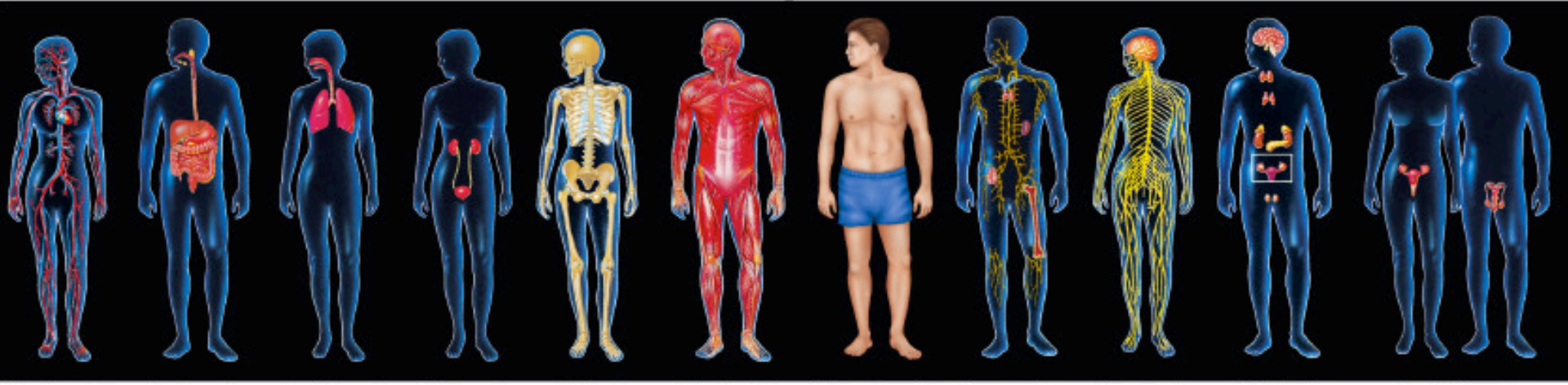
"Davis's Law".

Regarded as a corollary to Wolff's.

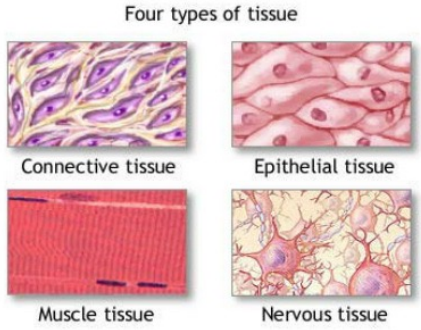
Take-home message:

Practically everything in your body is in a constant state of change. And given consistent exercise (or a consistent absence of exercise), it will be in a constant state of adaptation. Not just your muscle or your heart, but your bones and your glands and organs all of your energy systems, etc. Everything in your body adapts according to the stresses imposed upon it… just as Wolff and Davis discovered in the 19th century.

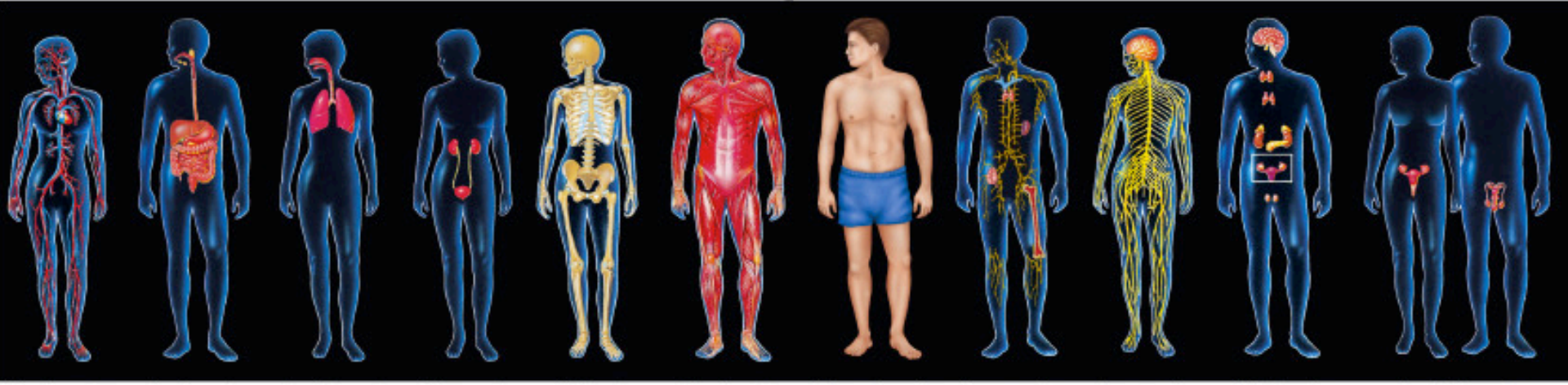




Circulatory system	Digestive system	Respiratory system	Urinary system	Skeletal system	Muscular system	Integumentary system	Immune system	Nervous system	Endocrine system	Reproductive system
heart, blood vessels, blood	mouth, pharynx, esophagus, stomach, small intestine, large intestine, salivary glands, exocrine pancreas, liver, gallbladder	nose, pharynx, larynx, trachea, bronchi, lungs	kidneys, ureters, urinary bladder, urethra	bones, cartilage, joints	skeletal muscles	skin, hair, nails	lymph nodes, thymus, bone marrow, tonsils, adenoids, spleen, appendix, and, not shown, white blood cells, gut-associated lymphoid tissue, and skin-associated lymphoid tissue	brain, spinal cord, peripheral nerves, and, not shown, special sense organs	all hormone-secreting tissues, including hypothalamus, pituitary, thyroid, adrenals, endocrine pancreas, gonads, kidneys, pineal, thymus, and, not shown, parathyroids, intestine, heart, skin, and adipose tissue	*Male:* testes, penis, prostate gland, seminal vesicles, bulbourethral glands, and associated ducts *Female:* ovaries, oviducts, uterus, vagina, breasts



Today, we know that all living cells and tissues (not just bones and ligaments) adapt according to the specificity of the stresses and demands placed upon them.



Circulatory system
heart, blood vessels, blood

Digestive system
mouth, pharynx, esophagus, stomach, small intestine, large intestine, salivary glands, exocrine pancreas, liver, gallbladder

Respiratory system
nose, pharynx, larynx, trachea, bronchi, lungs

Urinary system
kidneys, ureters, urinary bladder, urethra

Skeletal system
bones, cartilage, joints

Muscular system
skeletal muscles

Integumentary system
skin, hair, nails

Immune system
lymph nodes, thymus, bone marrow, tonsils, adenoids, spleen, appendix, and, not shown, white blood cells, gut-associated lymphoid tissue, and skin-associated lymphoid tissue

Nervous system
brain, spinal cord, peripheral nerves, and, not shown, special sense organs

Endocrine system
all hormone-secreting tissues, including hypothalamus, pituitary, thyroid, adrenals, endocrine pancreas, gonads, kidneys, pineal, thymus, and, not shown, parathyroids, intestine, heart, skin, and adipose tissue

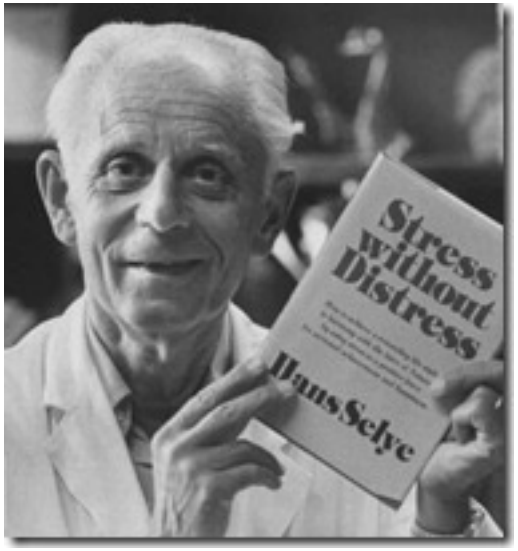
Reproductive system
Male: testes, penis, prostate gland, seminal vesicles, bulbourethral glands, and associated ducts

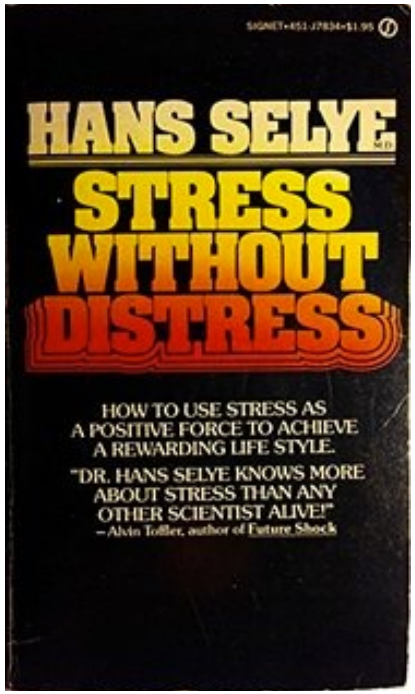
Female: ovaries, oviducts, uterus, vagina, breasts

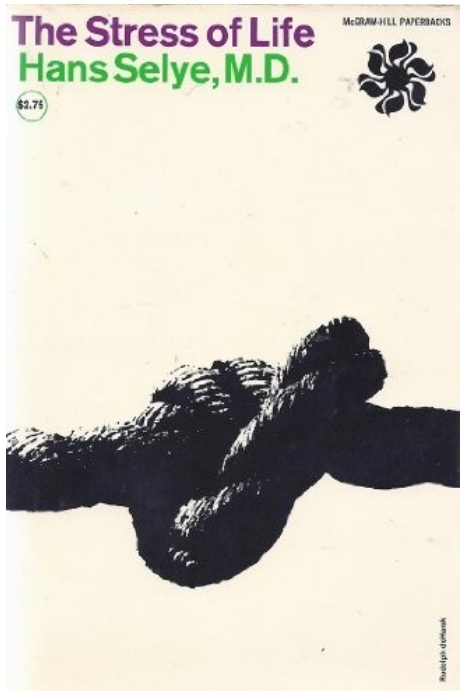
Name 5 exercise adaptations other than skeletal muscle and bone.

Name Some.

One of the most famous (and misleading) principles in performance physiology was proposed by the Austrian-Canadian endocrinologist, Hans Selye.



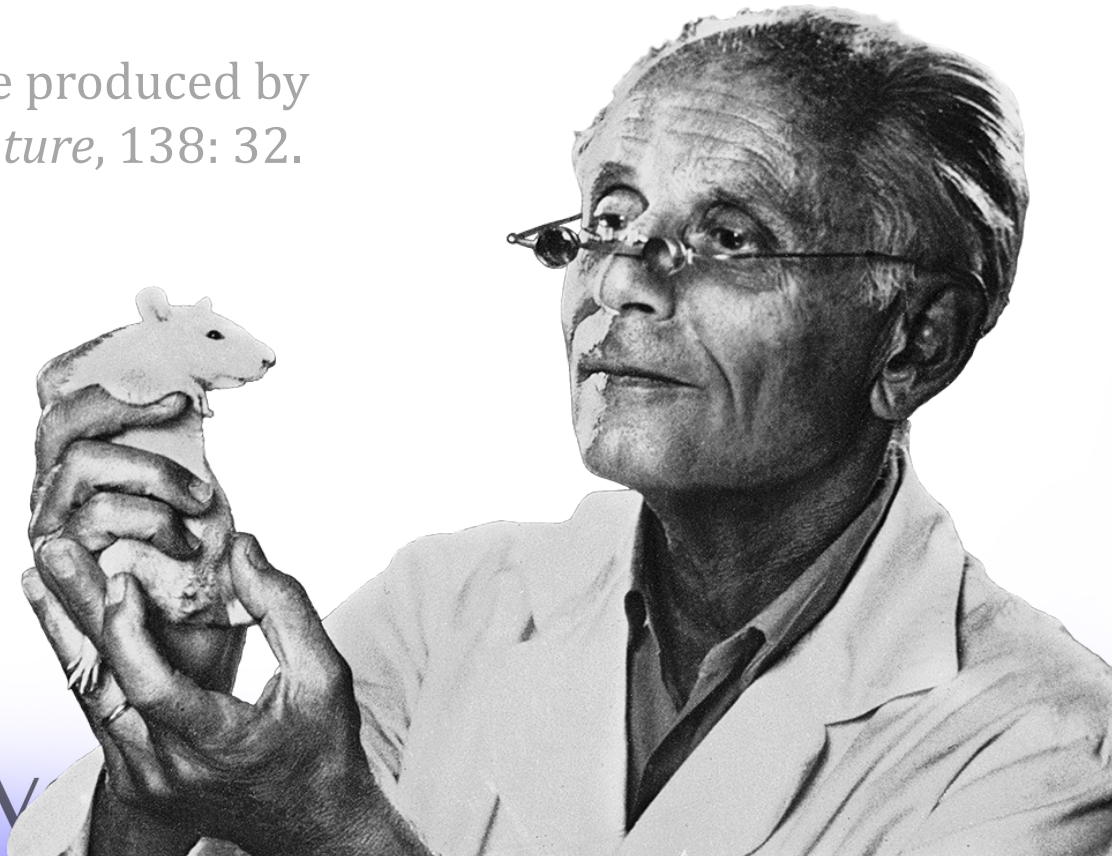




One of the most famous (and misleading) principles in performance physiology was proposed by the Austrian-Canadian endocrinologist, Hans Selye.

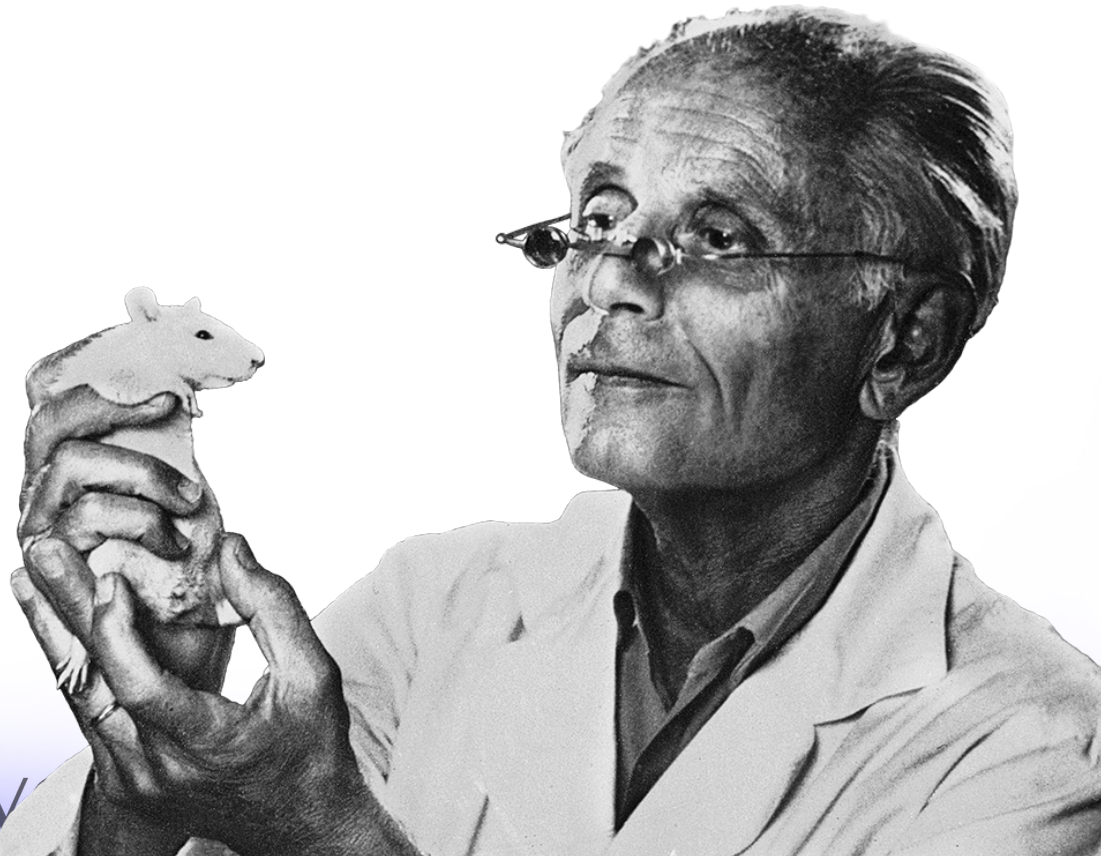
July 4th, 1936: Selye published his thoughts on "stress" responses in a letter to the editor in *Nature*.

Selye H. (1936). A Syndrome produced by Diverse Nocuous Agents. *Nature*, 138: 32.



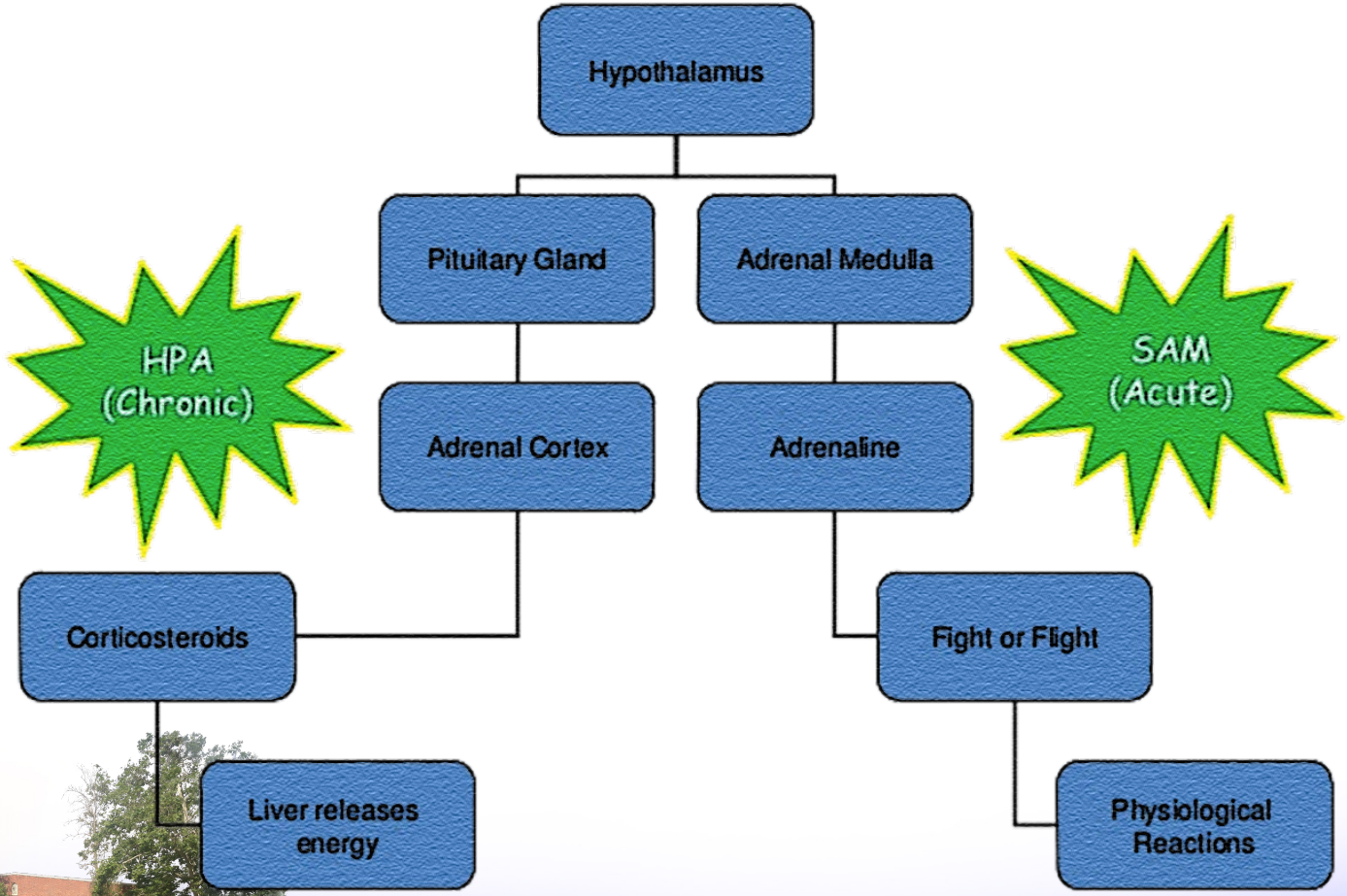
Instead of there being a variety of responses, depending on the particulars of the stress, Selye proposed a single one, which he called *General Adaptation Syndrome* (GAS).

GAS: The body reacts to every source of stress (physical and emotional) in the same way.



The end of the article:

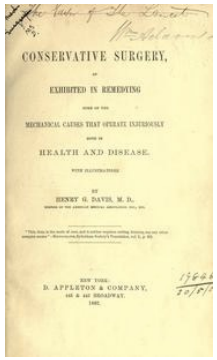
We consider the first stage to be the expression of a general alarm of the organism when suddenly confronted with a critical situation, and therefore term it the "general alarm reaction."



The end of the article:

We consider the first stage to be the expression of a general alarm of the organism when suddenly confronted with a critical situation, and therefore term it the "general alarm reaction." Since the syndrome as a whole seems to represent a generalized effort of the organism to adapt itself to new conditions, it might be termed the "general adaptation syndrome." It might be compared to other general defense reactions such as inflammation or the formation of immune bodies. The symptoms of the alarm reaction are very similar to those of histamine toxicosis or of surgical or anaphylactic shock; it is therefore not unlikely that an essential part in the initiation of the syndrome is the liberation of large quantities of histamine or some similar substance, which may be released from the tissues either mechanically in surgical injury, or by other means in other cases. It seems to us that more or less pronounced forms of this three-stage reaction represent the usual response of the organism to stimuli such as temperature changes, drugs, muscular exercise, etc., to which habituation or inurement can occur.

Despite ultimately giving specificity a rather wide berth, Selye never abandoned his thesis that the body exhibited nonspecific responses to most stressful exposures. Following this "landmark" discovery, it was several decades before researchers began to realize that Selye's GAS had rather missed the mark. And that the truth was a bit closer to work being done in the 19th century by Wolff and Davis.



That doesn't mean there's no overlap in stress responses. The biological response to one stressor may be highly similar to that of another. There is no shortage of redundancies in human physiology.

1950s: Environmental physiologist Edward F. Adolph brought these redundancies to attention, pointing out that the adaptive responses of the body are more sophisticated than GAS, but redundant nonetheless. While our biology reacts to every threat and source of stress very specifically, there *is* plenty of overlap in those reactions.

General and Specific Characteristics of Physiological Adaptations

Received for publication November, 1954.

E. F. ADOLPH

From the Department of Physiology, University of Rochester School of Medicine and Dentistry, Rochester, New York

Adolph EF. (1956). General and specific characteristics of physiological adaptations. *American Journal of Physiology,* 184(1): 18-28.

1950s: Environmental physiologist Edward F. Adolph brought these redundancies to attention, pointing out that the adaptive responses of the body are more sophisticated than GAS, but redundant nonetheless. While our biology reacts to every threat and source of stress very specifically, there *is* plenty of overlap in those reactions.

> Adaptations to altitude, or to cold air, or to other circumstance (stressor) have each been studied separately. The aim of this investigation is to compare adaptations to several diverse stressors in order to see whether any two of them arouse the same modifications. It has been widely supposed that adaptations to various stressors have much in common. I ask, how much? The conclusion will be reached that combinations of modifications differ markedly from one stressor to another.

Adolph EF. (1956). General and specific characteristics of physiological adaptations. *American Journal of Physiology*, 184(1): 18-28.

1950s: Environmental physiologist Edward F. Adolph brought these redundancies to attention, pointing out that the adaptive responses of the body are more sophisticated than GAS, but redundant nonetheless. While our biology reacts to every threat and source of stress very specifically, there *is* plenty of overlap in those reactions.

End of the article →

Adaptates overlap; for instance, adaptation to high altitude is not wholly separate from adaptation to cold air. Nevertheless, the combination of manifestations found is specific to the stressor. Although this possibility has been heretofore recognized (8), it has been neglected in the belief that the general syndrome predominates. The tally of specific instances now shows that adaptates are not the same for several stressors.

Adolph EF. (1956). General and specific characteristics of physiological adaptations. *American Journal of Physiology*, 184(1): 18-28.

What we know toady:

Every source of stress we experience has a unique fingerprint. And the ways that we respond to those stresses are specific to those fingerprints. Every cell and tissue in your body adapts according to its *exact* functional need.

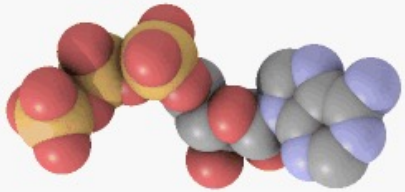
1: Bain CC & Mowat AM. (2011). Intestinal macrophages – specialised adaptation to a unique environment. *European Journal of Immunology*, 41(9): 2494-2498. **2:** Fulda S, Gorman AM, Hori O, Samali A. (2010). Cellular stress responses: cell survival and cell death. *International Journal of Cell Biology*, Article ID 214074, 23 pages. **3:** López-Maury L, Marguerat S, Bähler J. (2008). Tuning gene expression to changing environments: from rapid responses to evolutionary adaptation. *Nature Reviews Genetics*, 9: 583-593. **4:** Sanders JE, Goldstein BS, Leotta DF. (1995). Skin response to mechanical stress: adaptation rather than breakdown—a review of the literature. *Journal of Rehabilitation Research and Development*, 32(3): 214-226.

If a cell or tissue were to adapt *shy* of its needs, that would threaten its survival (given the likelihood that it would be exposed to more of that stress in the future). If it were to adapt *beyond* its needs, that would at least be inefficient, if not (potentially) life threatening as well. Unnecessary adaptation uses valuable energy that could contribute to survival.

Before

After

Adaptation isn't free. At the cellular level, it often requires a considerable investment of energy, which is a limited resource. And this initial energy cost might not be the *total* energy cost. It's possible that the adaptations being made would result in a higher basal metabolic rate (i.e., a higher *ongoing* cost of living).



1: Dolezal BA & Potteiger JA. (1998). Concurrent resistance and endurance training influence basal metabolic rate in nondieting individuals. *Journal of Applied Physiology*, 85(2): 695-700. **2:** Sjödin AM, Forslund AH, Westerterp KR, Andersson AB, Forslund JM, Hambraeus LM. (1996). The influence of physical activity on BMR. *Medicine and Science in Sports and Exercise*, 28(1): 85-91.

"Angular specificity."

If you subject your tissues to the same anatomical position over and over and over – experiencing isometric stress at a specific angle or training through a narrow range of motion – the cellular adaptations induced by that stress will allow you to tolerate that stress… and only that stress. Your tissues will make changes to accommodate that exact, limited region, giving you advantageous (mechanical, metabolic) function in that anatomical position.

It's not ***no*** gains at any other point in the ROM.

With elbow flexors, hold it at 90° and you can expect some improvement from 70 – 110°, but the most at exactly 90° (less at 95°, etc.).

Similar ranges exist in most muscle groups (quads, plantar flexors, etc.).

1: Kitai TA & Sale DG. (1989). Specificity of joint angle in isometric training. *European Journal of Applied Physiology and Occupational Physiology*, 58(7): 744-748. **2:** Knapik JA, Mawdsley RH, Ramos MU. (1983). Angular specificity and test mode specificity of isometric and isokinetic strength training. *Journal of Orthopaedic and Sports Physical Therapy*, 5: 58-65. **3:** Weir JP, Housh TJ, Weir LL, Johnson GO. (1995). Effects of unilateral isometric strength training on joint angle specificity and cross-training. *European Journal of Applied Physiology*, 70: 337-343.

More lengthened states = a *little bit* better range of improvement beyond isometric position (improvements stretch into more contracted states).

More contracted states = pretty pointless.

1: Bandy WD & Hanten WP. (1993). Changes in Torque and Electromyographic Activity of the Quadriceps Femoris Muscles Following Isometric Training. *Physical Therapy*, 73: 455-465. **2:** Meyers CR. (1967). Effects of two isometric routines on strength size and endurance in exercised and nonexercised arms. *Research Quarterly*, 1967: 38: 430-440. **3:** Thepaut-Mathieu C, Van Hoecke J, Maton B. (1988). Myoelectrical and mechanical changes linked to length specificity during isometric training. *Journal of Applied Physiology*, 64: 1500-1505.

Isometric exercise begets isometric capacity; dynamic begets dynamic.

A specific muscle action will elicit improvements in the capacity for the muscle to perform that type of action. There's a *little* overlap (but only a little).

1: Folland JP, Hawker K, Leach B, Little T, Jones DA. (2005). Strength training isometric training at a range of joint angles versus dynamic training. *Journal of Sports Sciences*, 23(8): 817-824. **2:** Jones DA & Parker. (1989). Development of a portable strain gauge to measure human muscle isometric strength. *Journal of Physiology*, 145(11): 3.

The same rule applies to concentric and eccentric activations:

If you want to increase eccentric muscle strength, train with eccentric muscle stresses; if you want to increase concentric muscle strength, train with concentric muscle stresses.

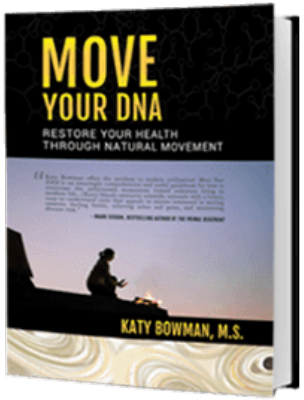
1: Duncan PW, Chandler JM, Cavanaugh DK, Johnson KR, Buehler AG. (1989). Mode and speed specificity of eccentric and concentric exercise training. *Journal of Orthopedic and Sports Physical Therapy*, 11(2): 70-75. **2:** Higbie EJ, Cureton KJ, Warren GL 3rd, Prior BM. (1996). Effects of concentric and eccentric training on muscle strength, cross-sectional area, and neural activation. *Journal of Applied Physiology*, 81(5): 2173-2181. **3:** Hortobagyi T, Barrier J, Beard D, Braspennicx J, Koens P, Devita P, Dempsey L, Lambert J. (1996). Greater initial adaptations to submaximal muscle lengthening than maximal shortening. *Journal of Applied Physiology*, 81(4): 1677-1682. **4:** Hortobagyi T, Hill JP, Houmard JA, Fraser DD, Lambert NJ, Israel RG. (1996). Adaptive responses to muscle lengthening and shortening in humans. *Journal of Applied Physiology*, 80(3): 765-772. **5:** Miller LE, Pierson LM, Nickols-Richardson SM, Wootten DF, Selmon SE, Ramp WK, Herbert WG. (2006). Knee extensor and flexor torque development with concentric and eccentric isokinetic training. *Research Quarterly for Exercise and Sport*, 77(1): 58-63. **6:** Seger JY, Arvidsson B, Thorstensson A. (1998). Specific effects of eccentric and concentric training on muscle strength and morphology in humans. *European Journal of Applied Physiology*, 79(1): 49-57. **7:** Seger JY, Thorstensson A. (2005). Effects of eccentric versus concentric training on thigh muscle strength and EMG. *International Journal of Sports Medicine*, 26(1): 45-52 **8:** Tomberlin JP, Basford JR, Schwen EE, Orte PA, Scott SC, Laughman RK, Ilstrup DM. (1991). Comparative study of isokinetic eccentric and concentric quadriceps training. *Journal of Orthopaedic and Sports Physical Therapy*, 14(1): 31-36.

This same rule applies to cognitive tasks and motor skills:

Football is not foosball;
π is not pie…

Linking movement and eating: *Physical activity as "nutrition"*

I *think* Katy is the one who came up with this concept. Very good description of it in this book ⟶



Katy Bowman:
MS in biomechanics,
very talented writer.

Linking movement and eating: *Physical activity as "nutrition"*

Nutrition Facts

Serving Size 1/2 muffins (57g)
Servings Per Container 8

Amount Per Serving

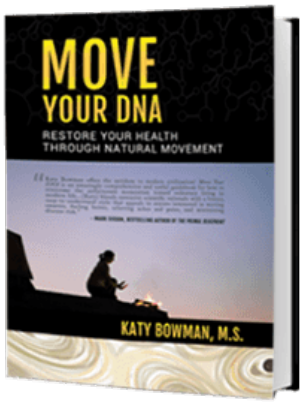
Calories 220	Calories from Fat 110
	% Daily Value*
Total Fat 12g	**19%**
Saturated Fat 2g	**9%**
Trans Fat 0g	
Cholesterol 35mg	**12%**
Sodium 170mg	**7%**
Total Carbohydrate 24g	**8%**
Dietary Fiber 1g	**4%**
Sugars 13g	
Protein 3g	

Vitamin A 2%	• Vitamin C 15%
Calcium 2%	• Iron 2%
Thiamin 6%	• Riboflavin 4%
Niacin 2%	• Folate 0%

*Percent Daily Values are based on a 2,000 calorie diet. Your daily values may be higher or lower depending on your calorie needs:

	Calories	2,000	2,500
Total Fat	Less Than	65g	80g
Saturated Fat	Less Than	20g	25g
Cholesterol	Less Than	300mg	300 mg
Sodium	Less Than	2,400mg	2,400mg
Total Carbohydrate		300g	375g
Dietary Fiber		25g	30g

Calories per gram:
Fat 9 • Carbohydrate 4 • Protein 4



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Sugars 13g	
Protein 3g	

Vitamin A 2%	•	Vitamin C 15%	
Calcium 2%	•	Iron 2%	
Thiamin 6%	•	Riboflavin 4%	
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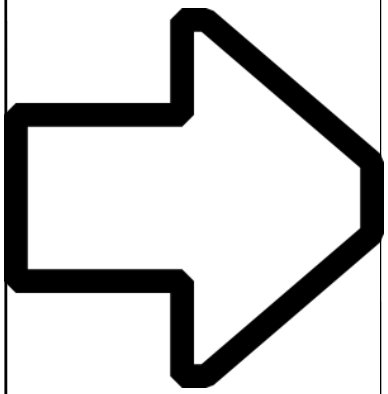

Nutrition Facts

Serving Size 1 Workout (57 min)
Servings Per Container 8

Amount Per Serving

Calories 220	Calories from Fat 110
	% Daily Value*
Type of Load	**19%**
Dynamic (Con/Ecc)	**9%**
Isometric	
Magnitude of Load	**12%**
Rate of Application	**7%**
Direction of Load	**8%**
Compression or Stretching	**4%**
Torsion or Shear	
Duration of Load	

Frequency	•	Variability
Location of Load	•	Coordination
Range of Motion	•	Number of Tissues
Temperature	•	Time of Day

*Percent Daily Values are based on a 2,000 calorie diet. Your daily values may be higher or lower depending on your calorie needs:

	Calories	2,000	2,500
Total Fat	Less Than	65g	80g
Saturated Fat	Less Than	20g	25g
Cholesterol	Less Than	300mg	300 mg
Sodium	Less Than	2,400mg	2,400mg
Total Carbohydrate		300g	375g
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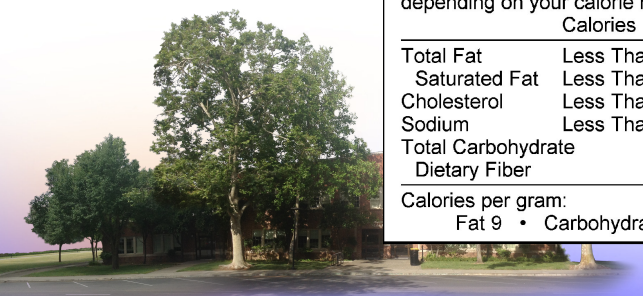
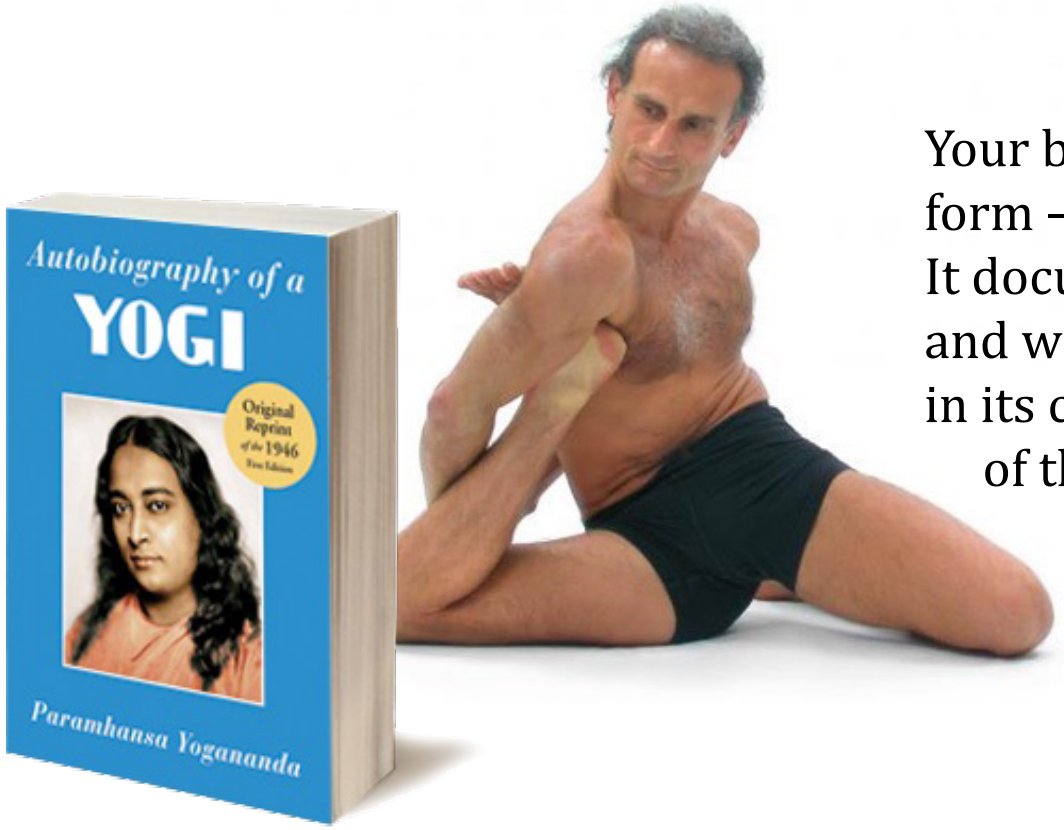
Calories per gram:
Fat 9 • Carbohydrate 4 • Protein 4





Your body – your physical form – is autobiographical. It documents every stress and writes their passages in its cells. The summation of those passages is what you see in the mirror.

To write a good biography,
we need stress.

Dear autobiography

Remember the broccoli

Health benefits and possible risks of broccoli – An overview

Klaus Peter Latté, Klaus-Erich Appel, Alfonso Lampen *

Food and Chemical Toxicology 49 (2011) 3287–3309

Good vs. Bad Stress

Int. J. Low Radiation, Vol. 1, No. 4, 2005

Nuclear shipyard worker study (1980–1988): a large cohort exposed to low-dose-rate gamma radiation

Ruth Sponsler*

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*Corresponding author

John R. Cameron#

Departments of Medical Physics,
Radiology and Physics,
University of Wisconsin-Madison
#Deceased 2005

1980s: U.S. Department of Energy tracked nuclear shipyard workers in Baltimore, Maryland from 1980 to 1988.

~28,000 workers were consistently exposed to a "high dose" of radiation from the materials they handled.

~32,000 employees (with similar jobs) were not exposed to radiation.

What differences were found between groups?

Good vs. Bad Stress

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Abstract: This paper is a summary of the 1991 Final Report of the Nuclear Shipyard Worker Study (NSWS), a very comprehensive study of occupational radiation exposure in the US. The NSWS compared three cohorts: a high-dose cohort of 27,872 nuclear workers, a low dose cohort of 10,348 workers, and a control cohort of 32,510 unexposed shipyard workers. The cohorts were matched by ages and job categories. Although the NSWS was designed to search for adverse effects of occupational low dose-rate gamma radiation, few risks were found. The high-dose workers demonstrated significantly lower circulatory, respiratory, and all-cause mortality than did unexposed workers.

Good vs. Bad Stress

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Departments of Medical Physics,
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Radiation was good; mortality was 24% lower in the radiation group.
"Toxins" were not ruining health, but bolstering it.

Physiological relationship between stress and protection.

That's basically how broccoli and vaccines work.

Good vs. Bad Stress

SPORTSCIENCE · sportsci.org

Reviews / Pathology

SUDDEN DEATH AND EXERCISE

Timothy D Noakes MBChB MD

Physiology, University of Cape Town Medical School, Observatory 7925, South Africa. Email: noakes@iafrica.com

Sportscience 2(4), sportsci.org/jour/9804/tdn.html, 1998 (6281 words)

Reviewed by George D Swanson, Physical Education and Exercise Science, California State University, Chico, California

Sudden death in athletes will always be an emotive topic, for it suggests that athleticism may not prevent the development of heart disease and may actually increase the likelihood that the athlete will die suddenly during exercise. Persons who die suddenly during exercise have advanced heart disease of which they are frequently unaware. The commonest forms of heart disease associated with sudden death during exercise are coronary artery disease and hypertrophic cardiomyopathy. Less common cardiac conditions linked to sudden death in athletes include anomalous origin of the coronary arteries, aortic rupture associated with Marfan's syndrome, myocarditis, mitral valve prolapse and various arrhythmias. The incidence of these predisposing diseases in the athletic population is extremely low, possibly of the order of 1 per 10,000 to 1 per 200,000 athletes. Detection of some of these conditions in asymptomatic athletes may be difficult, if not impossible. Regular exercise reduces the overall risk of sudden death in persons with latent coronary artery disease, yet acutely increases the risk of sudden death during exercise for those with heart disease that predisposes to sudden d… practical terms, only athletes with symptoms or clinical signs of, … factors for coronary or other forms of heart disease should und… routine maximal exercise testing when they commence an e… training program. However, once symptoms suggestive of … are present in athletes, detailed cardiological testing is m…

KEYWORDS: cardiomyopathy, coronary heart disease

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HEART DISEASE

Infrequent Sex or Exercise Can Trigger Heart Attacks

By Alice Park @aliceparkny | March 22, 2011 | Add a Comment

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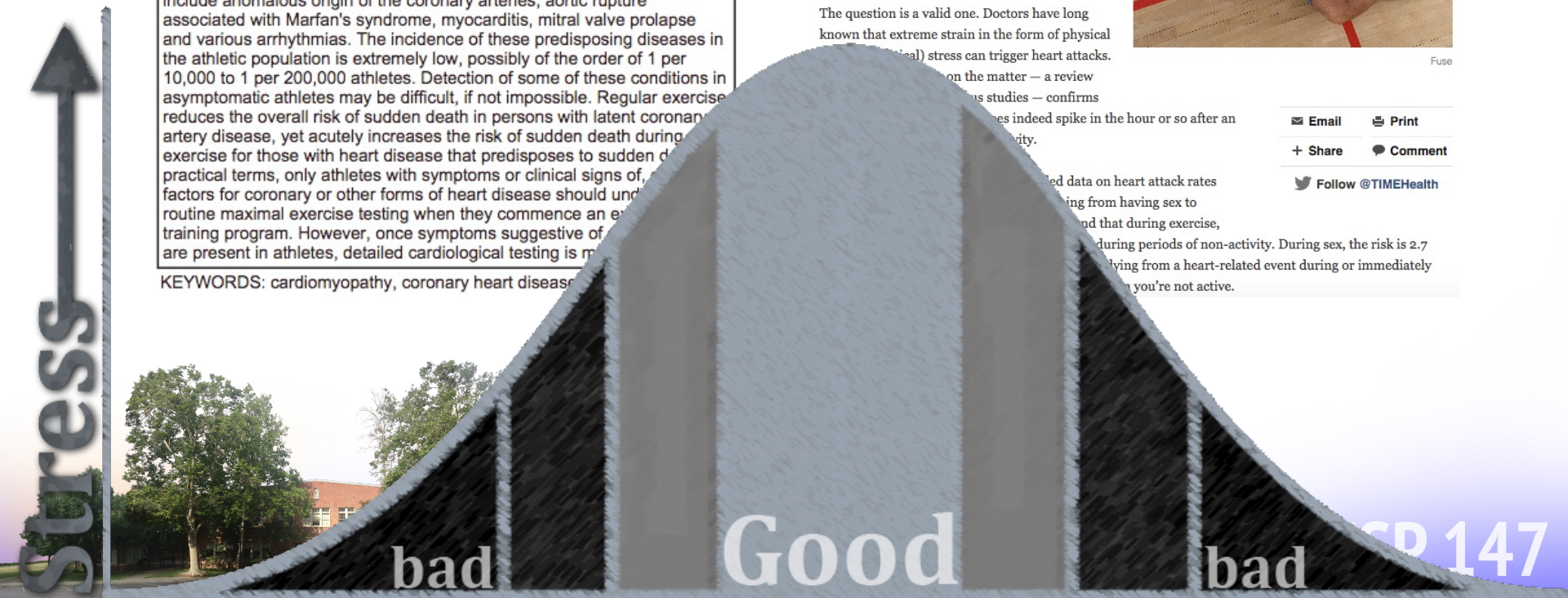
Fuse

There's no denying that regular physical activity and a healthy sex life are good for your heart. But if you've ever huffed and puffed your way through a jog or a particularly strenuous bout of lovemaking, you might have wondered whether the exertion was actually stressing, rather than strengthening, your heart.

The question is a valid one. Doctors have long known that extreme strain in the form of physical …ical) stress can trigger heart attacks. …on the matter — a review …s studies — confirms …es indeed spike in the hour or so after an …rity.

Email Print Share Comment Follow @TIMEHealth

…ed data on heart attack rates …ing from having sex to …nd that during exercise, …during periods of non-activity. During sex, the risk is 2.7 …lying from a heart-related event during or immediately … you're not active.



Specificity of Adaptation

Your physical form becomes the embodiment of what it endures.

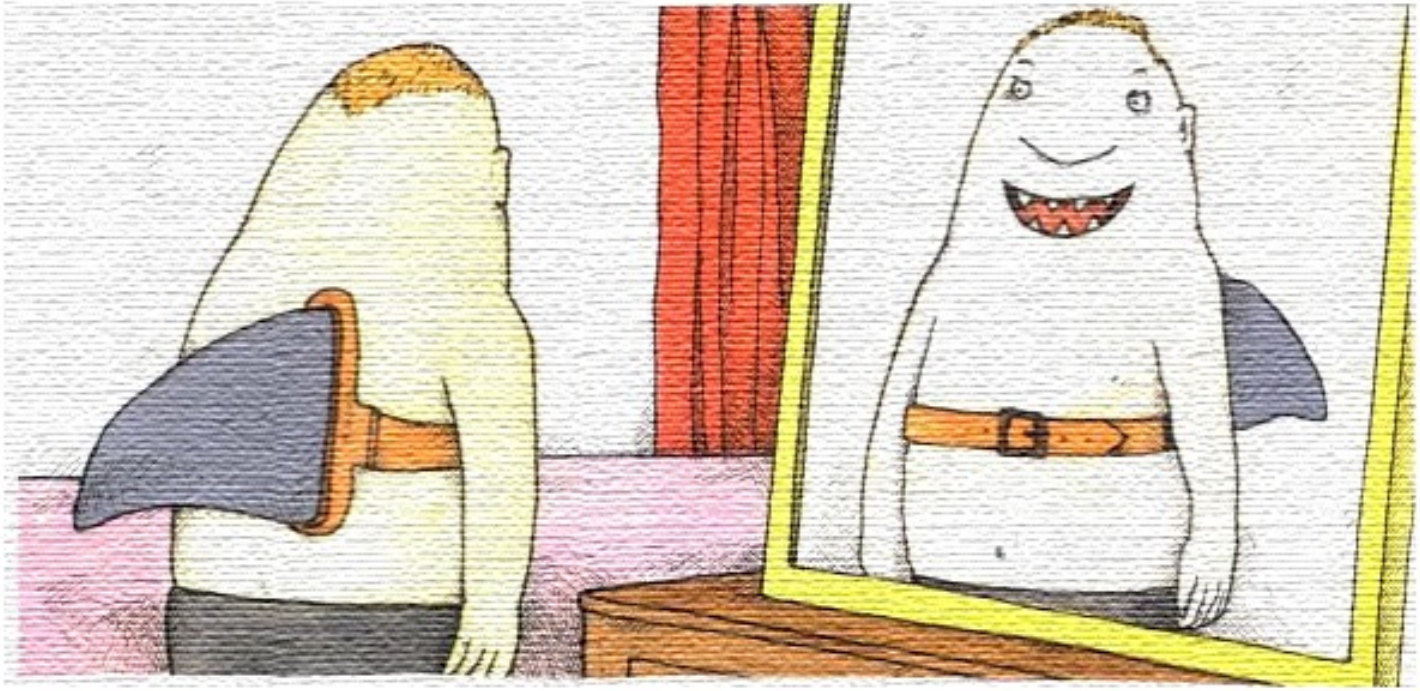
Keep moving: body attempts to retain the ability of movement.

Stop moving: future moving privileges are (slowly) revoked.

***Weird* moving:** the cause of most musculoskeletal pathology.

Specificity of Adaptation

People don't have dorsal fins…



But we do have:

Environments that are not "natural."

And we adapt *specifically* to those environments.
Whether that's good or bad depends on the specificity of the loads and stresses.

Sit too much. Bench press too much. Wear high heels. Carry all your stuff with one arm. Wear a sports bra.

Specificity of Adaptation

Run at 4.0 mph. What happens?
Run at 5.0 mph. What happens?
Run at 6.0 mph. What happens?

Run at a 5-minute mile pace, 6-minute mile pace, etc.

MPH	km/hr	Min/Mi	Min/Km	3 mi	5 km	8 km	10 km	1/2 mar.	Marathon
3.0	**4.8**	**0:20:00**	**0:12:26**	**1:00:00**	**1:02:08**	**1:39:25**	**2:04:16**	**4:22:13**	**8:44:26**
3.2	5.1	0:18:45	0:11:39	0:56:15	0:58:15	1:33:12	1:56:30	4:05:50	8:11:40
3.4	5.5	0:17:39	0:10:58	0:52:56	0:54:50	1:27:43	1:49:39	3:51:22	7:42:44
3.6	5.8	0:16:40	0:10:21	0:50:00	0:51:47	1:22:51	1:43:34	3:38:31	7:17:02
3.8	6.1	0:15:47	0:09:49	0:47:22	0:49:03	1:18:29	1:38:07	3:27:01	6:54:02
4.0	**6.4**	**0:15:00**	**0:09:19**	**0:45:00**	**0:46:36**	**1:14:34**	**1:33:12**	**3:16:40**	**6:33:20**
4.2	6.8	0:14:17	0:08:53	0:42:51	0:44:23	1:11:01	1:28:46	3:07:18	6:14:36
4.4	7.1	0:13:38	0:08:28	0:40:55	0:42:22	1:07:47	1:24:44	2:58:47	5:57:34
4.6	7.4	0:13:03	0:08:06	0:39:08	0:40:31	1:04:50	1:21:03	2:51:01	5:42:01
4.8	7.7	0:12:30	0:07:46	0:37:30	0:38:50	1:02:08	1:17:40	2:43:53	5:27:46
5.0	**8.0**	**0:12:00**	**0:07:27**	**0:36:00**	**0:37:17**	**0:59:39**	**1:14:34**	**2:37:20**	**5:14:40**
5.2	8.4	0:11:32	0:07:10	0:34:37	0:35:51	0:57:21	1:11:42	2:31:17	5:02:34
5.4	8.7	0:11:07	0:06:54	0:33:20	0:34:31	0:55:14	1:09:02	2:25:41	4:51:21
5.6	9.0	0:10:43	0:06:39	0:32:09	0:33:17	0:53:16	1:06:35	2:20:28	4:40:57
5.8	9.3	0:10:21	0:06:26	0:31:02	0:32:08	0:51:25	1:04:17	2:15:38	4:31:16
6.0	9.7	0:10:00	0:06:13	0:30:00	0:31:04	0:49:43	1:02:08	2:11:07	4:22:13

Specificity of Adaptation

Consider the sit-up and the curl-up (and the crunch, etc.). Form matters here. If you train by crunching with a helper (someone anchoring your feet), but the test has no anchor, there's a difference in the mechanics of your training and the test. You will do very badly.

Specificity of Adaptation

If you get hit a bunch, the body adapts to better tolerate getting hit a bunch. "Contact adaptation." It accounts for differences in pain, injuries, and recovery in football players at the end of their season compared to the beginning.[1] Nobody knows exactly how it works (perhaps there are changes in the matrix of the muscle) but whatever the alteration, athletes can better tolerate contact stresses.

1: Hoffman JR, Kang J, Ratamess NA, Faigenbaum AD. (2005). Biochemical and hormonal responses during an intercollegiate football season. *Medicine and Science in Sports and Exercise*, 37(7): 1237-1241. **2:** Kraemer WJ, Spiering BA, Volek JS, Martin GJ, Howard RL, Ratamess NA, Hatfield DL, Vingren JL, Ho JY, Fragala MS, Thomas GA, French DN, Anderson JM, Häkkinen K, Maresh CM. (2009). Recovery from a national collegiate athletic association division I football game: muscle damage and hormonal status. *Journal of Strength and Conditioning Research*, 23(1): 2-10.

Specificity of Adaptation
Take-Home Message

There is no source of stress (isometric exercise, running at a five-minute mile pace, taking a punch, etc.) that doesn't have a unique fingerprint.

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Those adaptations are aimed at self-preservation.

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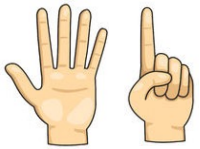


Corrective exercise (i.e., physical therapy) **is** the manipulation of biological stresses and mechanical loads placed upon tissues to restore proper cell signaling.

Toleration?

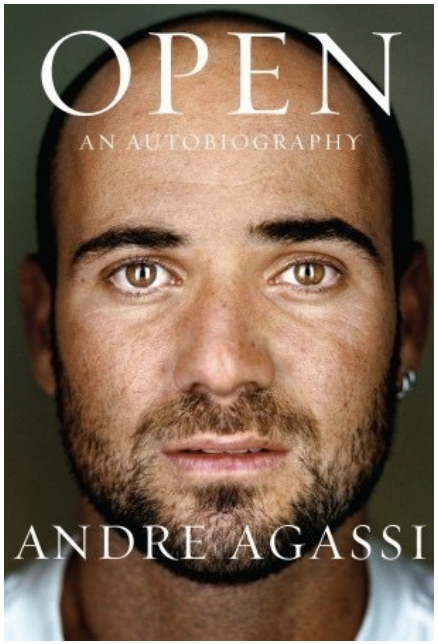
Acclimation vs. Acclimatization?

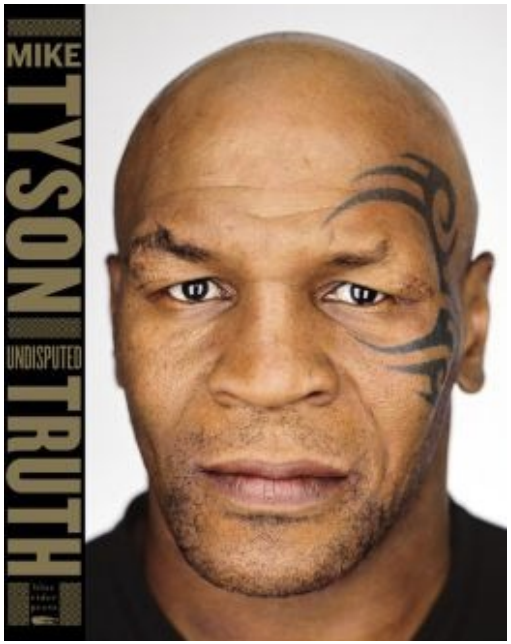
Habituation vs. Sensitization?

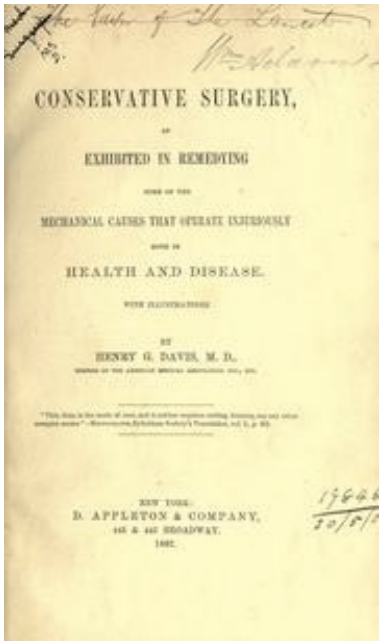
Accommodation vs. Adaptation?

Individual vs. Genetic Adaptation?

Specificity of Adaptation






CONSERVATIVE SURGERY,

EXHIBITED IN REMEDYING

MECHANICAL CAUSES THAT OPERATE INJURIOUSLY

HEALTH AND DISEASE.

HENRY G. DAVIS, M. D.

D. APPLETON & COMPANY,


Das Gesetz

Transformation der Knochen

Julius Wolff

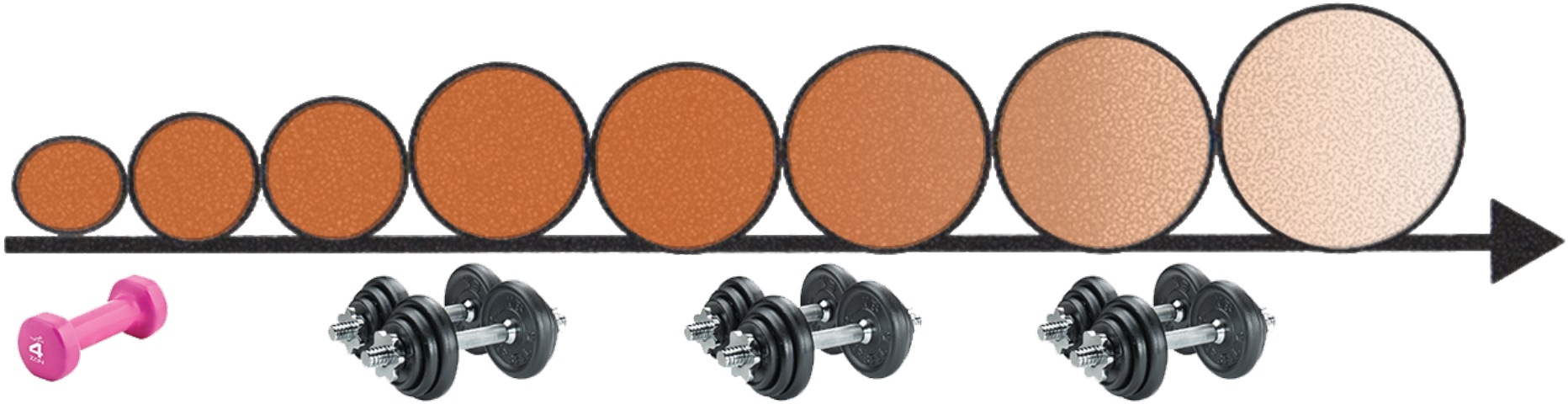
Specificity of Adaptation
Take-Home Message

Accommodate, Accommodate, Accommodate, Accommodate, Adapt.

How long does it take before resistance training begins to cause increases in muscle cross sectional area?

At the onset of an exercise prescription, what accounts for improvements in performance?

Specificity of Adaptation
Take-Home Message



Just because you can activate high threshold fibers (in some tissues) with a low load doesn't mean adaptations for size, strength, and power are being signaled.

That’s everything.