



FUNDAMENTALS OF MUSCLE GROWTH & STRENGTH

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In this module of the course, you'll learn what makes muscles grow from a physiological point of view. We'll focus on skeletal muscles, the muscles that are under voluntary control, and not the smooth muscle found in many tissues, including blood vessels, the lungs, the uterus and the gastrointestinal tract. Understanding the driving forces of skeletal muscle hypertrophy, as muscle growth is formally called in exercise science, will help lay the foundation for the coming modules on training program design.

➤ **Lecture [optional]**

[Muscle anatomy and force production](#)

Muscle fiber anatomy

A muscle cell is different from other human cells. Most types of human cells are round and contain one nucleus with all our DNA except some mitochondrial DNA, as illustrated below. [Some types of connective tissue cells, notably red blood cells, have no nucleus at all though.](#)

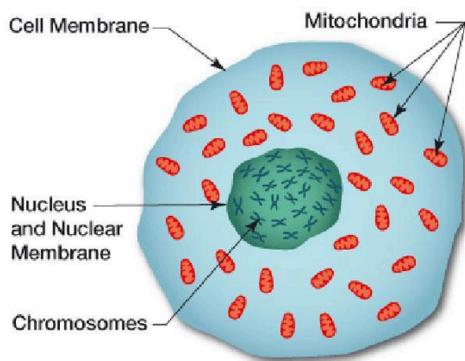


Figure 3 - Rudimentary Pictorial of DNA Containing Organelles in a Human Cell

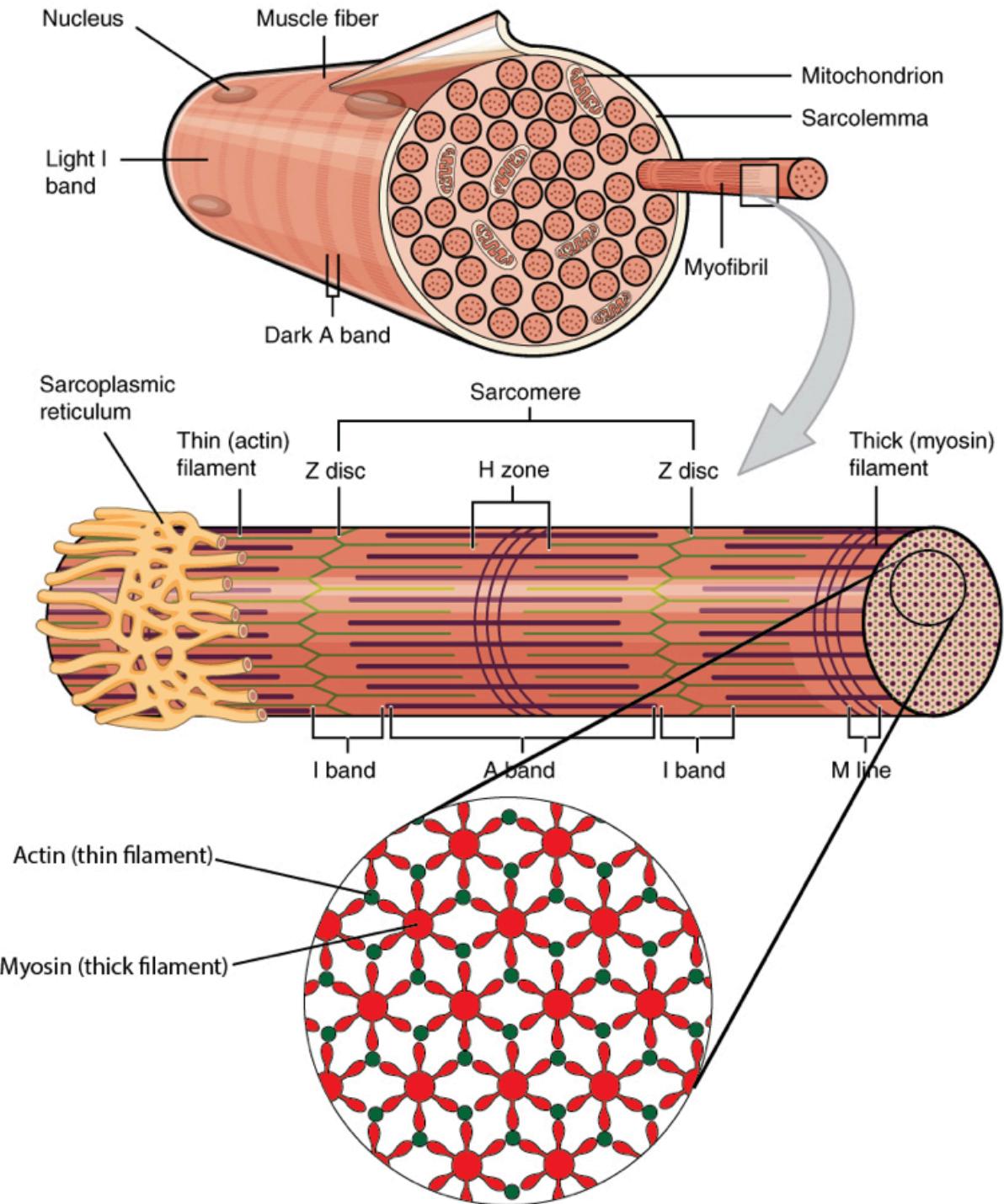
Credit: Family Tree DNA and Genetic Genealogy DNA Testing Dictionary
<http://www.ggdictionary.com>

A muscle cell is a long, cylindrical cell 50 – 100 µm in diameter, which is about the diameter of a human hair. One muscle cell can span the entire length of the muscle. Muscle cells are grouped in bundles that may contain up to 150 muscle cells. Each muscle cell possesses not just one but multiple nuclei, which are located at the outer borders of the cell. Each nucleus controls a region of the muscle protein. You can think of the muscle cell nuclei as command centers of a region of muscle tissue. Because of their unique structure, muscle cells are often referred to as muscle fibers (or myocytes).

Each muscle fiber/cell is surrounded by the muscle fiber membrane. A muscle fiber contains many components, notably the following:

- Mitochondria, which are responsible for energy (ATP) generation.
- Stored glycogen (~2%: [most glycogen is stored in between muscle fibers, not within them](#)) and triglyceride (~5%).
- The sarcoplasmic reticulum, a membrane structure that looks like ivy wrapping around the myofibrils. It contains ion channel pumps that can release calcium into the myofibrils, which starts muscle contractions. The extreme stiffness of corpses ('rigor mortis') is largely the result of the sarcoplasmic reticulum breaking down and flooding the myofibrils with calcium.
- Myofibrils, the contractile components that make your muscles flex.

Contractile components consist of protein filaments, called myofibrils. Each muscle cell contains hundreds of myofibrils. Each myofibril is about 1 µm in diameter. The components of myofibrils – the myofilaments myosin and actin – are responsible for the contraction of the muscle cell. Myosin and actin filaments are organized to form the smallest contractile unit of the muscle, the sarcomere. Sarcomeres occur over the entire length of the muscle fiber and are connected through actin filaments. At each connection between two sarcomeres, aligned actin filaments are anchored at the so-called Z-line.



The anatomy of a muscle fiber. Source unknown.

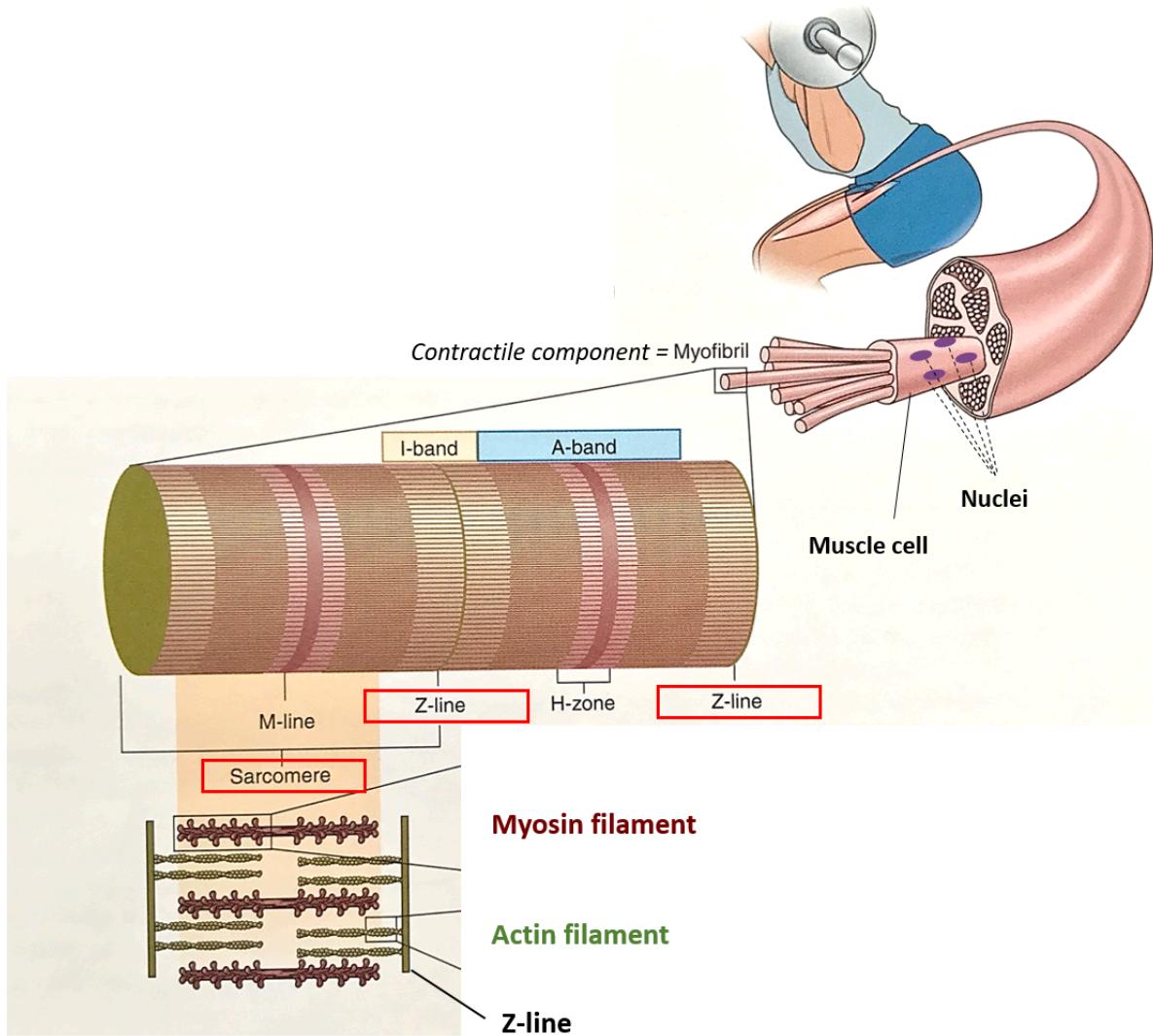
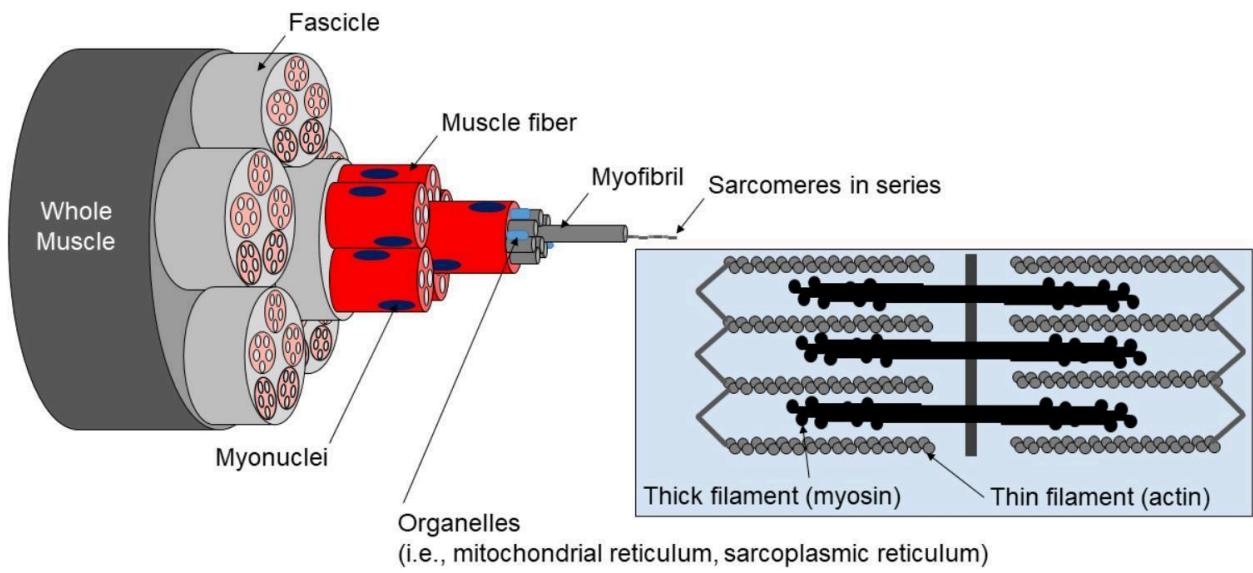
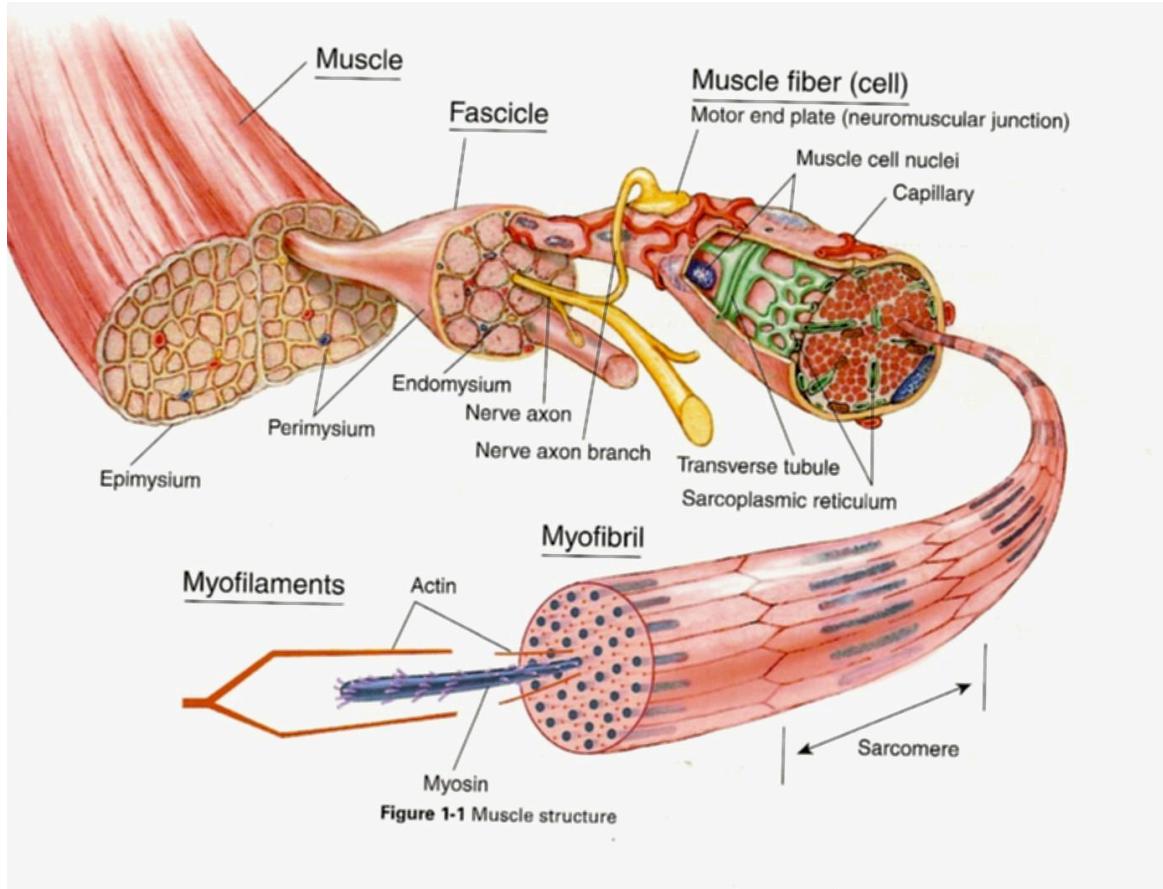
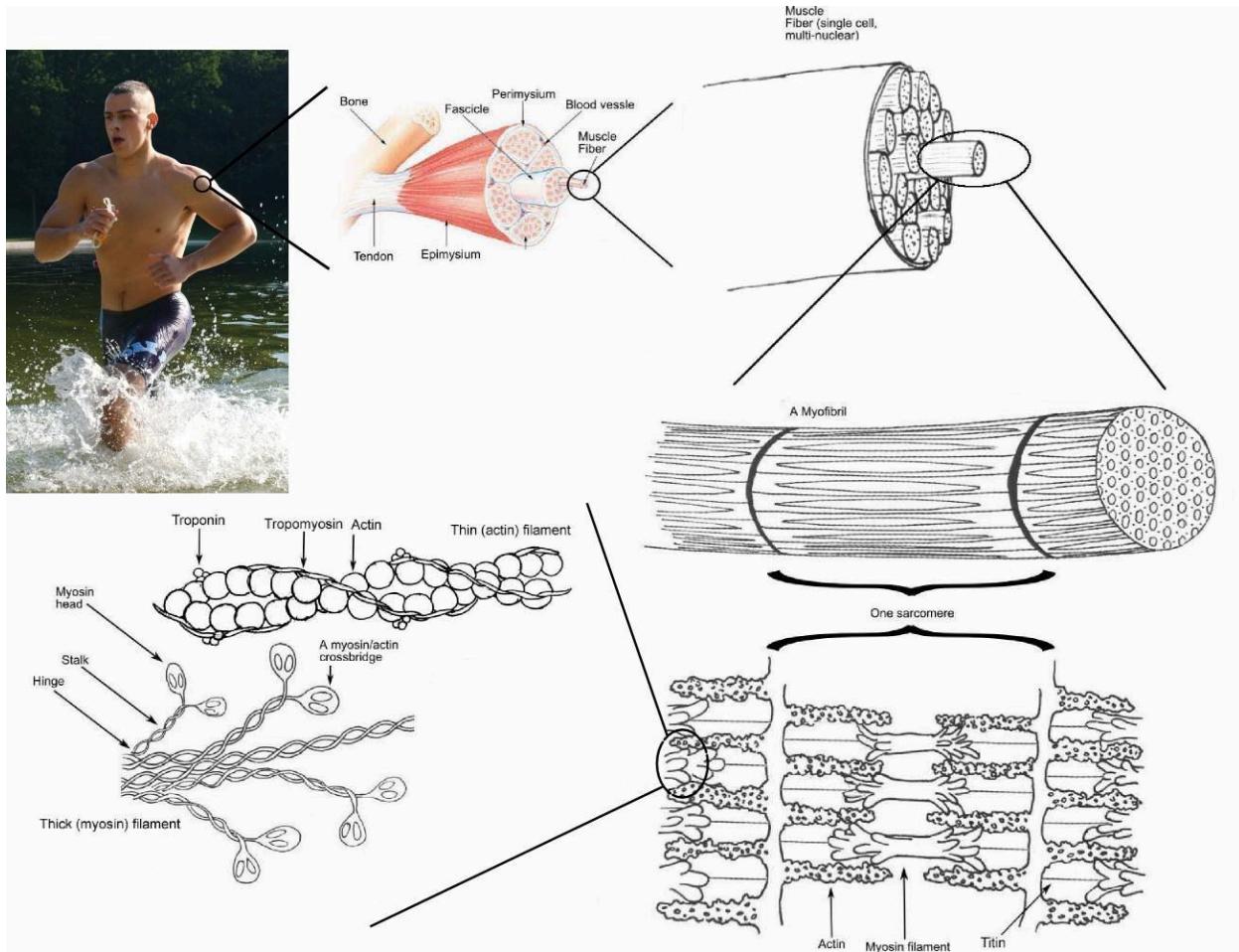


Figure adapted from Baechle TR, Earle RW. *Essentials of Strength Training and Conditioning*. 2008.





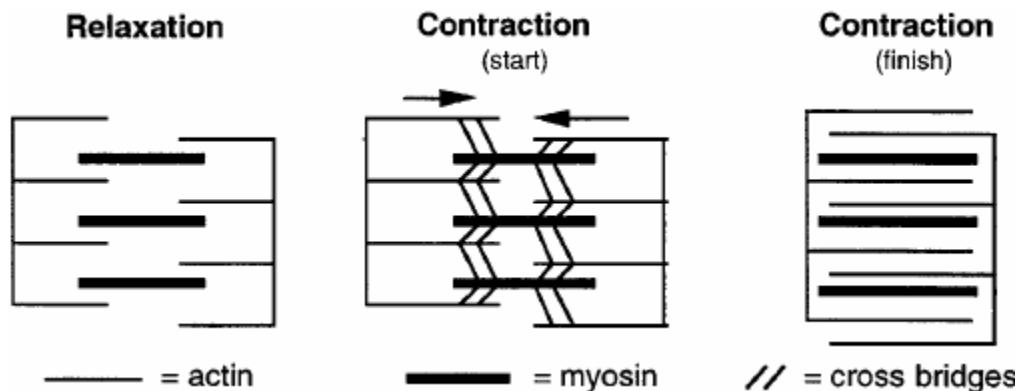
The structure of skeletal muscles. Muscles are organized into progressively smaller units of contractile tissue: fascicles > muscle fibers > myofibrils > sarcomeres > myofilaments. [Source](#)

Muscle volume outside the muscle fibers – the extracellular space – is mostly filled with fluid, connective tissue ([1-10%](#)) and vasculature. The connective tissue forms fasciae, sheets of sturdy tissue consisting mostly of collagen, that wraps up the muscle fibers into bundles.

How muscles cause movement

Sliding filament theory

Actin and myosin can form actin-myosin cross-bridges that pull the muscle tissues towards each other, causing the muscle fiber to shorten and pull on its tendons. This so-called sliding filament model is illustrated below.



The sliding filament model of muscle contraction. As the actin filament slides over the myosin filament, the distance between the Z-lines of the sarcomere decrease, cross-bridges are formed and the muscle shortens. [Source](#)

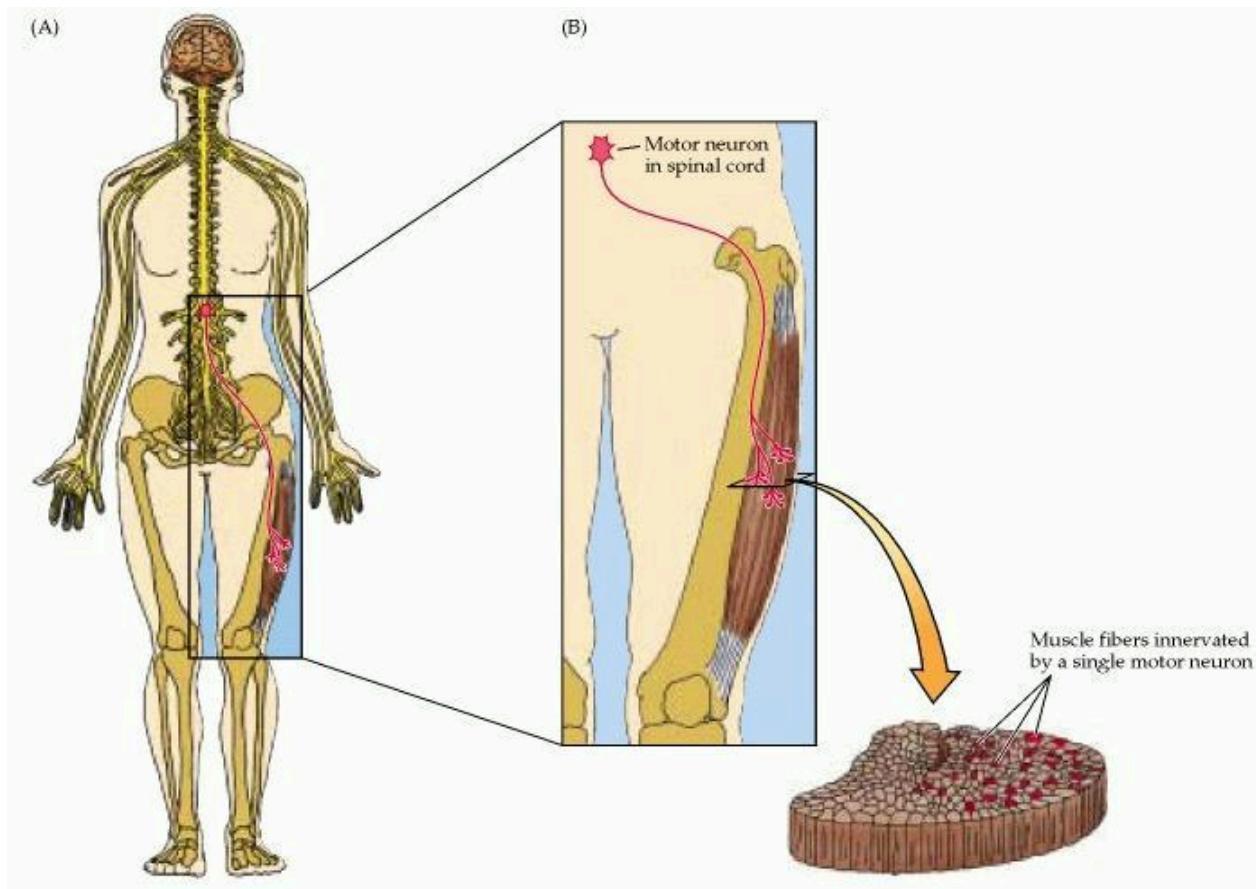
Modern research has found that the structural protein titin also plays an important role as a third filament, especially during muscle contractions where the muscle does not shorten. As opposed to the active contractions of actin and myosin, titin produces force more passively: it's effectively spring-loaded when a muscle lengthens. Titin derived its name from being the largest protein in the human body. Titin constitutes about 10% of muscle mass, making it the third most present protein in muscle after actin and myosin.

The sliding filaments allow sarcomeres and their encapsulating myofibrils and muscle fibers to contract and thereby produce force. A muscle's total force production is

directly proportional to the number of actin-myosin cross-bridges: more cross-bridges means more force production. How many cross-bridges are formed in the muscle as a whole depends on the level of motor unit recruitment.

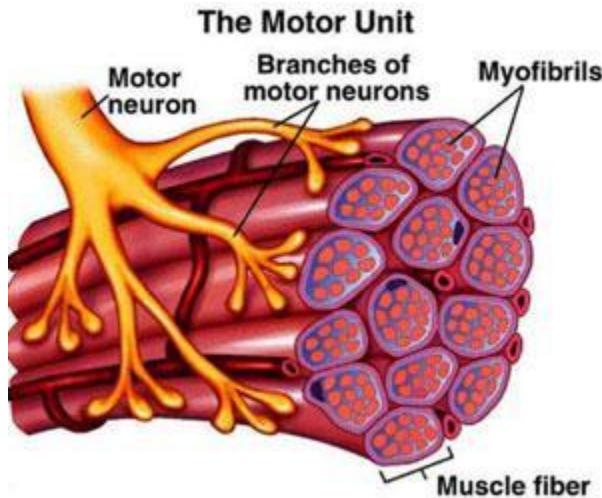
Motor unit recruitment

Our brains are in charge of voluntary muscle functioning. The specific area of our brain that controls movement is called our motor cortex. From the motor cortex, an electrical signal called an action potential is sent via our nerves down the spinal cord to motor nerves, AKA motoneurons, in our muscles to recruit them.



To contract a muscle, the motor cortex sends down an electrical signal that activates our muscle fibers. [Source](#)

Each motor neuron innervates a bundle of muscle fibers. Together, these form a motor unit, as illustrated below. Recruitment of the motor unit causes all the muscle fibers to contract maximally, which causes them to produce force. This is called a twitch.



Each motor unit consists of a motor neuron and a bundle of muscle fibers.

The total amount of force produced by the muscle is governed by 2 factors [2].

1. How many muscle fibers are recruited.
2. How frequently the muscle fibers are activated by their motor neuron (known as rate coding or motor unit firing frequency).

How many motor units are recruited depends on the amplitude of the action potential. Motor units come in various sizes, depending on how many muscle fibers they have. In general, bigger motor units are stronger but also more fatigable, whereas smaller motor units are weaker but more resistant to fatigue.

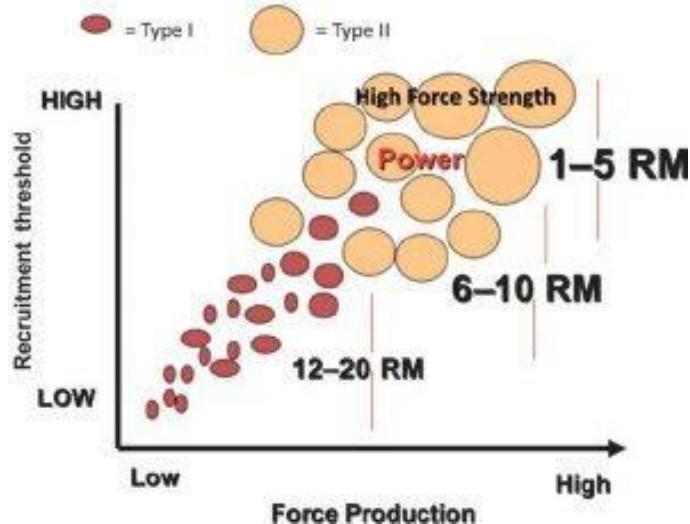
- Small motor units have a low contraction threshold and typically have more type I muscle fibers that are better at producing low amounts of force for a long period of time.

- Large motor units have a high contraction threshold and typically have more type II muscle fibers that can produce large amounts of force quickly but also tire quickly.

Efficient as it is, your brain by default only recruits low-threshold motor units by sending out low amplitude action potentials. Higher threshold motor units are only recruited for 2 reasons.

1. When a high total muscle force production is required. If the lower-threshold motor units cannot produce enough force to lift a heavy weight, higher-threshold motor units are recruited to help out.
2. The lower-threshold motor units get fatigued. [Neuromuscular fatigue of a motor unit reduces its firing rate and its force production capacity](#), resulting in a loss of total force production from that motor unit. As a result, the higher-threshold motor units have to pick up the slack.

In other words, when the going gets tough, the tough get going. Motor units are recruited in an orderly fashion from small to large. This phenomenon is called Henneman's Size Principle and is illustrated below.

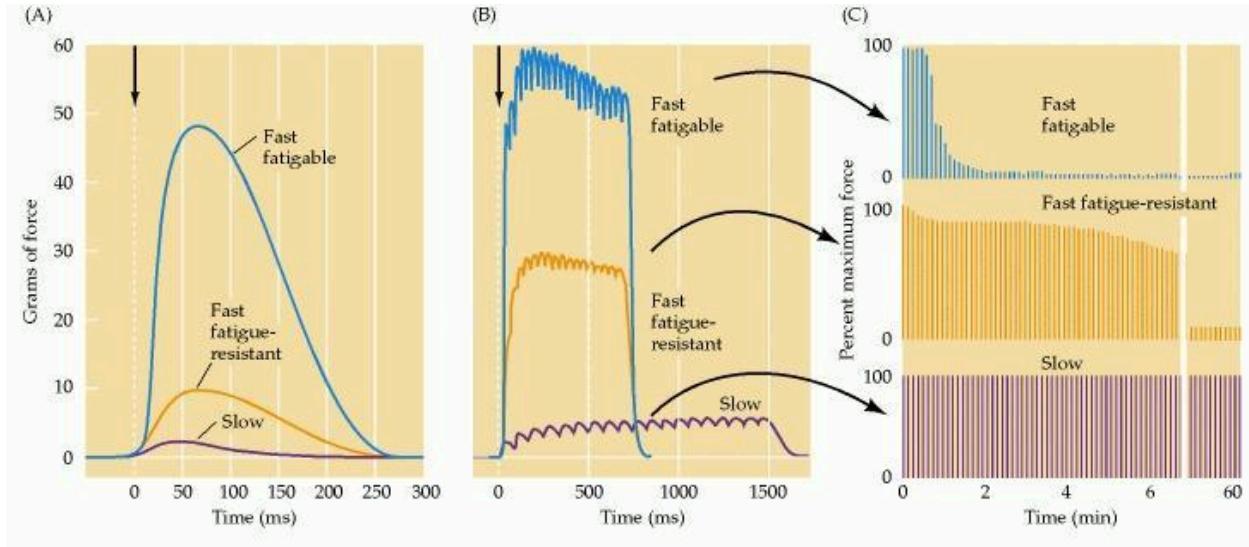


The Size Principle: “each circle represents a motor unit made up of different types and numbers of fibers. The maroon circles are Type I motor units and the light tan circles are Type II motor units, with larger circles depicting larger motor units containing more fibers. As one goes up the line of orderly recruitment, heavier and heavier resistances recruit more and more motor units and their associated muscle fibers.” [Source](#)

Muscles generally achieve full motor unit recruitment when they have to produce 75-85% of maximal force [2]. From that point onward, muscle activation levels are near their maximum, but the nervous system can ramp up muscle activation levels further by sending down more action potentials in quick succession. The frequency of stimulation of a motor unit is called rate coding and it results in an increase in the firing frequency of the motor unit.

Total muscle activation thus depends on the number of recruited motor units as well as their firing frequency. Most movements last much longer than a single muscle twitch, so they require a continuous stream of action potentials to maintain muscle tension and complete the movement. Such a continuous contraction is called a tetanic contraction.

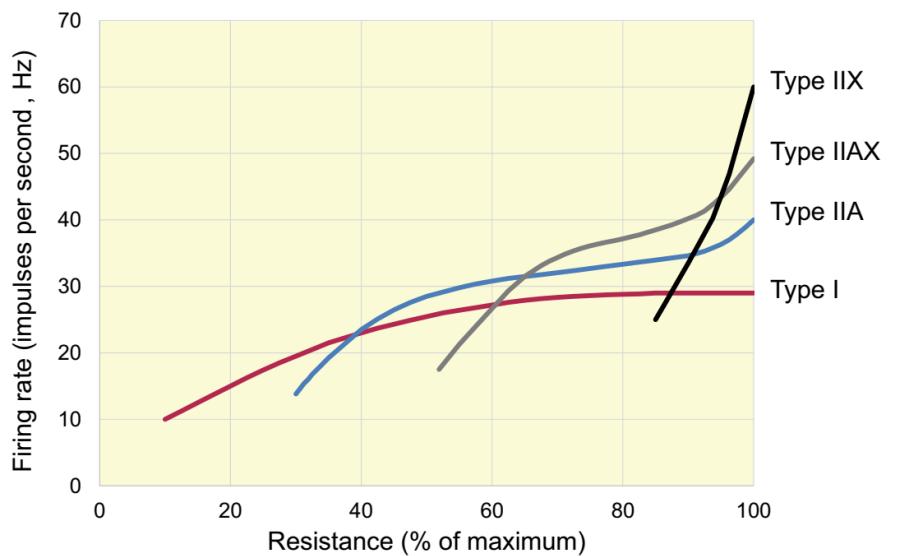
Motor unit force production is illustrated below.



Force production by 3 motor units of various sizes in response to a single motor neuron action potential (A), repeated stimulation (B) and maximum stimulation to fatigue (C).

[Source](#)

FIGURE 1 Suggested scheme of the interplay between the recruitment of motor units innervating type I, II A, II AX and II X muscle fibres and the accompanying increases in firing rates with increases in contractile force production. The scheme depicts short (~0.5–1 s) but non-ballistic contractions, as opposed to slow ramp (~10 s) contractions (see text). Modified from Sale⁴² based on data discussed in this review



The more intensive the muscle contraction, the higher the muscle activation. Up to around 85% of 1RM, motor unit recruitment increases along with rate coding. After this point, force production primarily increases through higher motor unit firing frequencies.

[Source](#)

Biomechanics 101

Now that we understand how muscles contract, we can go into how this translates into movement. Muscle contractions produce force, which drives movement. Force is equal to mass times acceleration ($F = m \times a$) and measured in Newtons (N).

1 Newton is the force to accelerate a 1 kg mass at a rate of 1 m/sec².

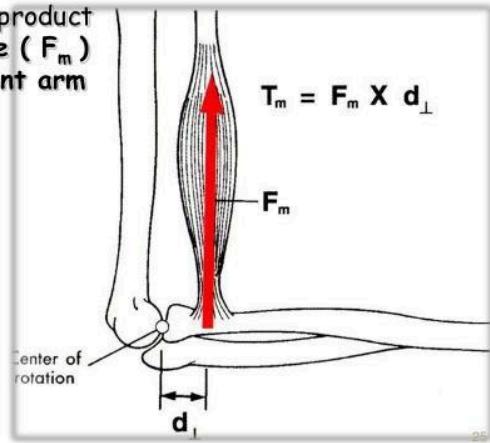
When we lift weights, the main forces acting on a weight are normally our muscles and gravity. On Earth, gravitational resistance (g) is always ~9.8 m/sec², so the force required to lift a weight is normally about 10 times its weight in kilograms. That simplifies things, as we can often ignore the acceleration component of force and pretend it's a measure of mass. For example, to lift 100 kg, we need to produce about 1,000 N of force.

That force is produced by our muscles, but producing 1,000 N of force with our muscles does not necessarily mean we can lift 100 kg, because our muscles are not directly fighting gravitational resistance. Instead, our muscles pull on our tendons, which in turn pull on our bones, and this causes our bones to move around our joints. This turning force that's applied about the joint is called a joint moment. The moment of force is also called torque, and for our purposes torque and joint moment are generally synonymous terms. Basically, torque and moment are both rotational application of force. You can think of them as turning force.

The internal joint moment is influenced not just by the forces our muscles produce but also by their leverage, specifically the distance between the joint's center of rotation (axis) and the tendon's insertion point on the bone. The further away the tendon is, the better leverage it has. The joint moment is the product of force and the moment arm, and we can measure it in Newton-Meters. Technically, we measure the internal joint moment arm as follows: take the joint's center of rotation, take the muscle's direction

of force production (normally the line along the muscle belly from origin to insertion) and draw a line perpendicular from this force production line to the axis of rotation (see images below).

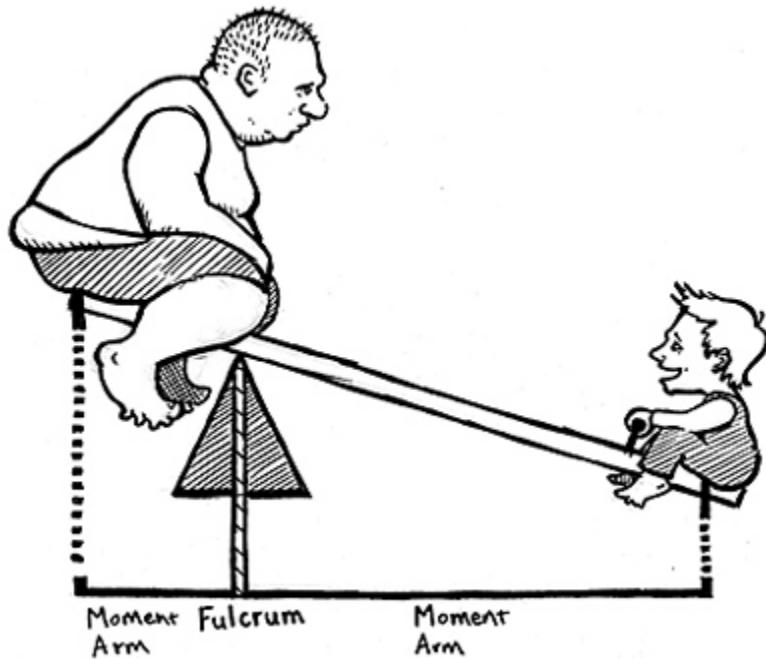
Torque (T_m) produced by a muscle at the joint center of rotation is the product of muscle force (F_m) & muscle moment arm (d_{\perp}).



Biceps force causes elbow flexion torque/moment. At a 90° angle of elbow flexion, the moment arm is exactly equal to the distance between the center of rotation and the insertion point of the biceps tendon, as that line is perpendicular to the direction of force production, which is vertical in line with the direction of the muscle fibers. As the elbow flexes or extends, the internal moment arm decreases, as [you can see in this video.](#)

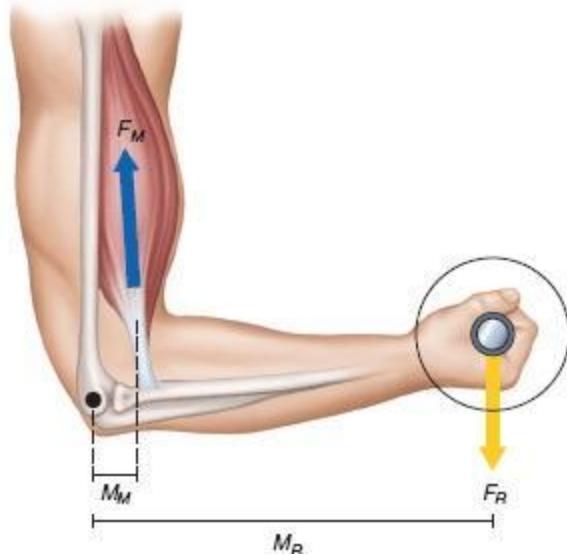
Where our muscle tendons insert on our bones is genetically determined and majorly influences how much weight someone can lift. If someone's biceps tendon inserts twice as far down their forearm as another person's, they only have to produce half the force to lift a given weight with elbow flexion. With equally strong muscles, they can lift twice as much weight. So how much weight someone can lift and how strong their muscles are, are not the same thing.

Just like our muscles have *internal* moment arms that are determined by our tendon insertion points, the gravitational resistance from weights we lift also has an *external* moment arm. Since our muscles' moment arms are typically much shorter than those of the weights we lift, which often have a moment arm that's about the length of our (fore)arms or legs, the ratio between these moment arms determines how much more force our muscles have to generate to lift a given load than just 10 times its weight (remember: gravitational downward force for any weight is roughly equal to 10 times its weight in kilograms).

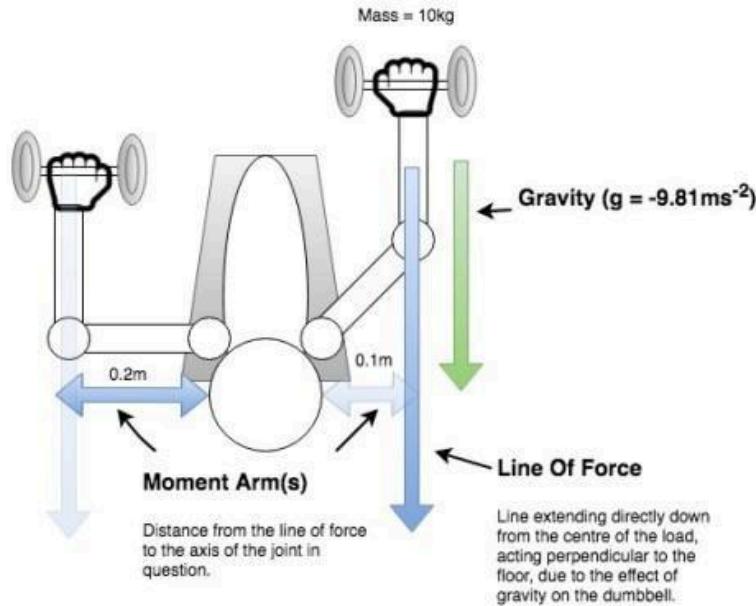


A large external moment arm can make small weights require high forces to move. Conversely, a large internal moment arm gives us good leverage and enables us to produce a large joint moment from relatively little muscular force production.

External moment arms are calculated the same way as internal ones. You take the line of force production, which is often vertical for a free weight like a dumbbell because that's gravity's direction of pull, and you draw a line perpendicular to that to the joint's axis of rotation. See the images below for examples.



The muscle's internal moment arm (M_M) vs. the resisting force's (F_R) external moment arm M_R). If the muscle's moment arm is 20x smaller than the barbell's moment arm, our muscles have to produce 20x more force than the gravitational force of the barbell to keep the barbell in place.



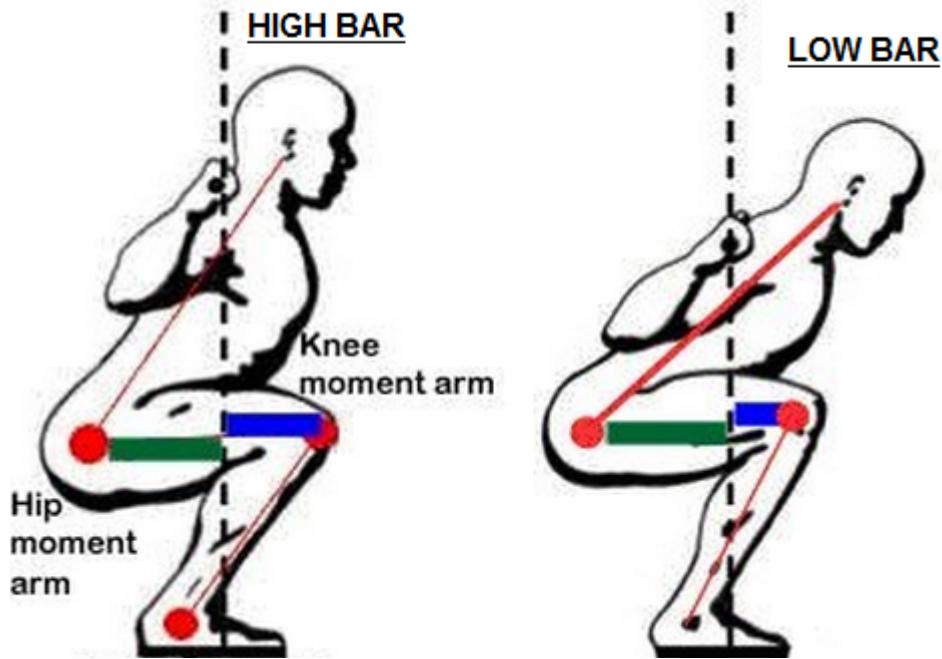
Right Shoulder Moment

Moment (Nm^{-1}) = Force (N) x Perpendicular distance from the force to the axis (m)

$$\rightarrow \text{Shoulder Moment} = (10)(9.81) \times (0.1)$$

$$\rightarrow 9.81 \text{ Nm}^{-1}$$

The external moment arm for the horizontal shoulder flexion moment during the dumbbell bench press. Notice how the moment arm is maximal when our upper arms are horizontal, so our muscles have to produce the most shoulder flexion moment there to lift the weight.



A simplified version of the external knee and hip flexion moment arms from the barbell's gravitational resistance during squat variations. Note how a low bar position makes the squat more 'hip dominant' by increasing the hip flexion moment arm and decreasing the knee flexion moment arm. You could similarly draw the ankle dorsiflexion moment arm from the ankle and the spinal flexion moment arm for each intervertebral joint in the spine. (This graphic does not take bodyweight's gravitational resistance into account, but during a good squat, the barbell should stay roughly above the total center of mass, so the moment arms would be similar.)

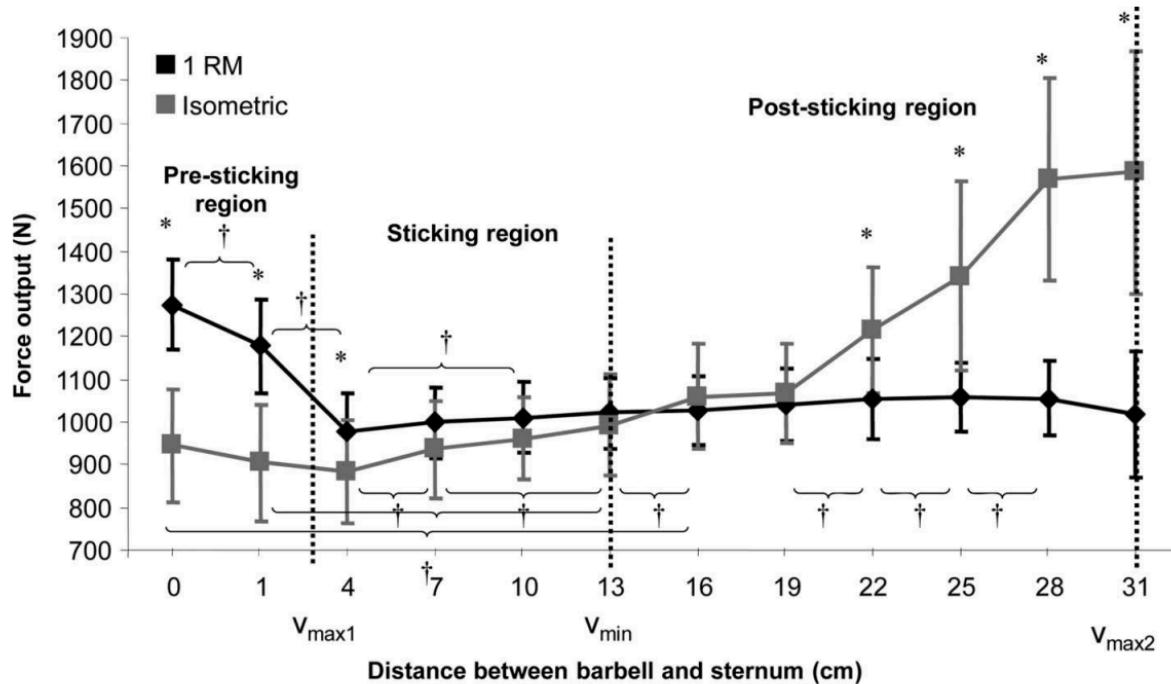
If our muscle joint moments exceed the joint moments of the weights we lift, our joints will move. For example, during a dumbbell biceps curl, we can curl up the weight if our elbow flexion joint moment exceeds the elbow extension moment from the dumbbell. You're not expected to be able to calculate any joint moments, but understanding the biomechanical principles will help you understand things like exercise selection and why you encounter sticking points during exercises.

The sticking point

You know how you usually fail exercises at the same point? That's the [sticking point](#): the point of the exercise where it's most difficult. When you fail to complete a repetition of an exercise, many people have the idea that the muscle is just too fatigued to produce further movement and it sort of happens to occur at a harder part of the movement. In fact, concentric momentary muscle failure only means that your force production was insufficient to overcome the resistance *at the sticking point*.

For example, if you end up in a squatmorning position during the ascent in the squat and you get completely stuck there to the point that there's no more movement, you have reached a position of inertia: your upward force production right there in that position is no longer equal to the gravitational pull downward. Just the tiniest bit of assistance will generally already cause further upward movement and allow you to complete the entire rep. If you lift with a spotter or have spotted others training to failure, you'll have experienced how much difference it makes if someone assists the tiniest bit.

[During the powerlifts, the sticking point is generally very noticeable](#). During the bench press, the sticking point typically occurs after the bar comes off your chest and before the midway point, about 30-50% into the rep.



Force production during a 1RM and maximal isometric contractions in the barbell bench press. Even during a 1RM, force production during the top part of the movement isn't nearly maximal (as during maximal isometric contraction), so the top half of the movement remains relatively understimulated. [Source](#)

During the squat, the sticking point typically occurs near the halfway point up at a thigh angle of about 30° to the floor and a 90-100° hip angle (squatmorning, anyone?). Interestingly, [the sticking point in the squat is not primarily the result of biomechanical weakness in that position but dissipating benefits from the stretch-shortening cycle](#). Isometric squat strength is relatively constant from the bottom position to the sticking point.

The sticking point is more variable for the deadlift than the other powerlifts. [Most strength trainees don't have a deadlift sticking point \[2, 3\]](#). Or rather, the sticking point is right on the floor, which makes sense, because [most people can produce far less force from the floor than they can produce once the bar has passed the knee](#).

Correspondingly, [mid-shin force production is significantly more correlated with 1RM deadlift strength than mid-thigh force production](#). Thus, most lifters can either deadlift the bar all the way up to lock-out or they can't get the bar off the floor at all. (Note that some studies talk about a sticking region or sticking point defined as the point where velocity decreases, but this is not a dead stop or the point where the lifters fail the rep, so it's not a true sticking point.) In contrast, [some powerlifters have a sticking point around when the bar has to pass the knee](#), at least when they could get the bar off the floor in the first place. No study has managed to find what makes some lifters have a sticking point in the deadlift. [The sticking point, if it occurs, seems to occur at a similar place during conventional and sumo deadlifts \[2\]](#) and [in weaker and stronger lifters](#). Theoretically, deadlifting with a rounded back, as powerlifters generally do, may make it more likely to fail a deadlift higher up compared to right off the floor, as it makes you get into a more favorable starting position, but then later on you have to extend the spine to stand up straight.

The sticking point can vary somewhat per individual, depending on the lifter's body structure, it can vary per intensity, because the relative load influences your ability to use momentum, and it can vary per technique due to altered biomechanics.

Strength vs. resistance curves

The sticking point occurs because of a mismatch between the resistance curve of the exercise and your strength curve.

Resistance = the amount of force required to move the weight.

Strength = the amount of force that your body can produce.

Together, these curves determine the relative muscular effort curve. Relative muscular effort is the amount of force production from a muscle relative to its maximum force

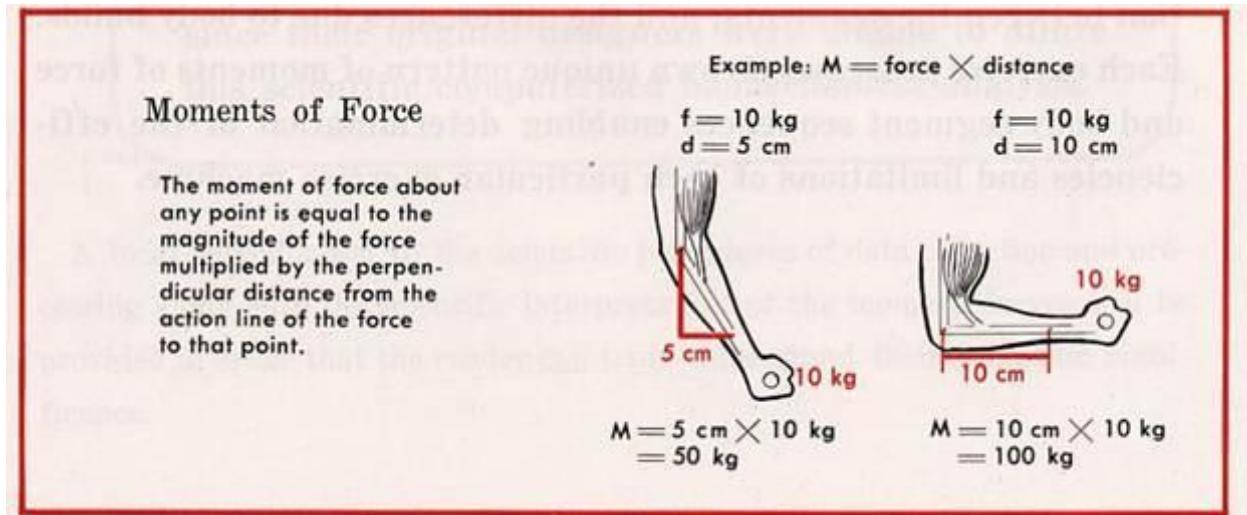
production potential. It's how hard the muscle has to work. 100% Relative muscular effort means the muscle is producing as much force as it can. 0% means it's inactive.

Ideally, exercises should maximize relative muscular effort: resistance should equal your strength throughout the entire movement. In practice though, many exercises don't even come close to maximizing relative muscular effort throughout the entire range of motion. Only isokinetic machines generally allow us to train with maximum force output throughout the entire range of motion. Isokinetic machines are theoretically a perfect form of accommodating resistance, as they change the resistance at any point during the movement to maintain a constant speed of movement (iso = 'same'; kinetic = 'movement/speed'). You can thus provide maximal effort throughout the full range of motion, during both the concentric and the eccentric phase of the movement. It's a brutal way to train.

Free-weight exercises that require the weight to move vertically (directly opposed to gravity's line of pull) generally have a flat resistance curve. That means there's a constant resistance. The weights don't change mass and gravitational acceleration is constant, assuming you're not training in a space shuttle.

Exercises in which the weight moves in a circular or rotary fashion, like dumbbell lateral raises and barbell curls, normally have a resistance curve with a maximum where the moving body parts are horizontal and a minimum where the moving body parts are vertical. The changing external moment arm alters the resistance.

For example, the biceps are under minimal resistance at the bottom of a dumbbell curl and they're handling maximal resistance at 90-degrees flexion (the midpoint).

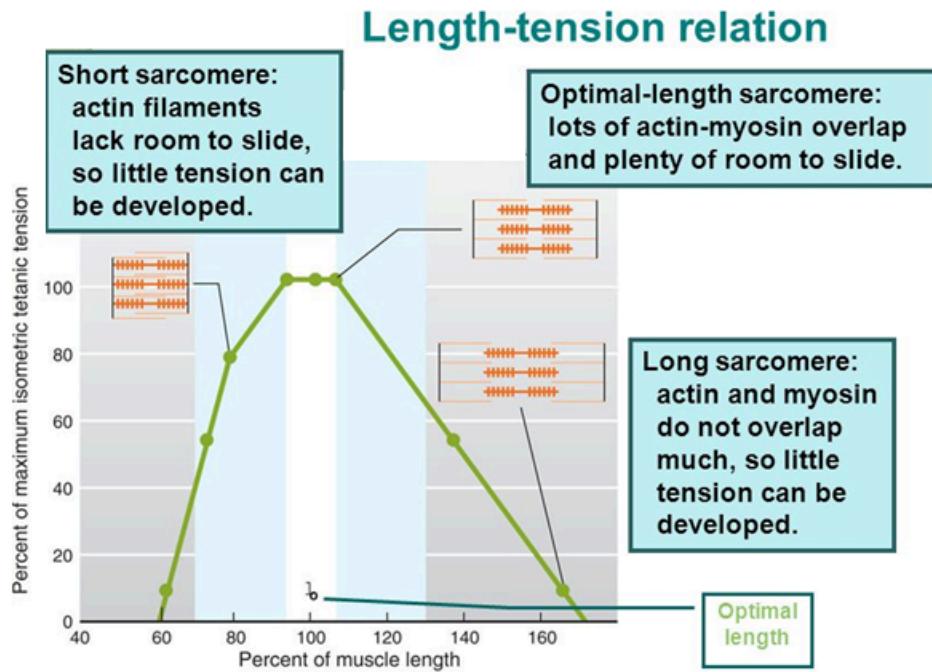


If the forearm goes all the way up to a vertical position during a biceps curl, you lose almost all biceps tension. This is why dumbbell preacher curls, where your elbow is positioned on a bench in front of you (shoulder in flexion), have a poor strength-resistance curve and as a result, [preacher curls only stimulate the biceps well in the bottom part of the movement.](#)

Determining your strength curve is generally more difficult than determining the resistance curve. Other than paying attention and simply feeling where you're strongest and where you fail during an exercise, it helps to think of a muscle's length-tension relationship. A decent rule of thumb is that muscles are strongest in their natural anatomical position (think military posture) or when in a ~20% stretched position. These positions generally achieve high total force production potential: the sum of active and passive force production.

Active force production by your muscles is generally highest around their resting muscle lengths in anatomical position (standing straight up and relaxed). This makes evolutionary sense, as this is where you use your muscles the most. So the body has become structured in such a way that your muscles are capable of high force

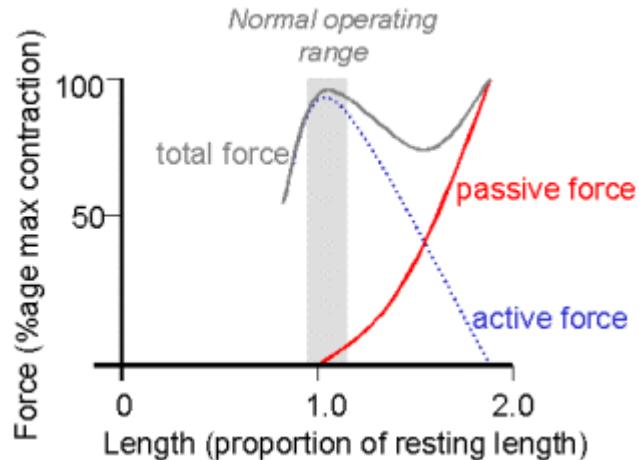
production in the corresponding range of motion. Biomechanically, at resting length, your myofilaments can achieve the most cross-bridges, giving rise to the length-tension relationship of human skeletal muscle, illustrated below.



Muscles can generally produce most active force at a certain optimum muscle length with diminished active force production capacity when they are shortened or lengthened.

Passive force production of muscles is largely the result of how much they are stretched. Since muscles are viscoelastic, the more you stretch them, the more force they produce to resist the stretch. During eccentric contractions, the myofilament titin produces a notable amount of passive elastic force by virtue of its stiffness. While this is passive force production, it's still biomechanical tension on the muscle fibers and it therefore contributes to muscle growth. Connective tissues, like your tendons, also contribute significant passive elastic force.

The graph below provides a theoretical illustration of how active and passive force and their sum change over a muscle's length.



For pushing exercises, your strength is generally lower in the bottom half of the movement than in the top half. This is why you generally fail the overhead press, bench press and squat before you're near the end of the movement.

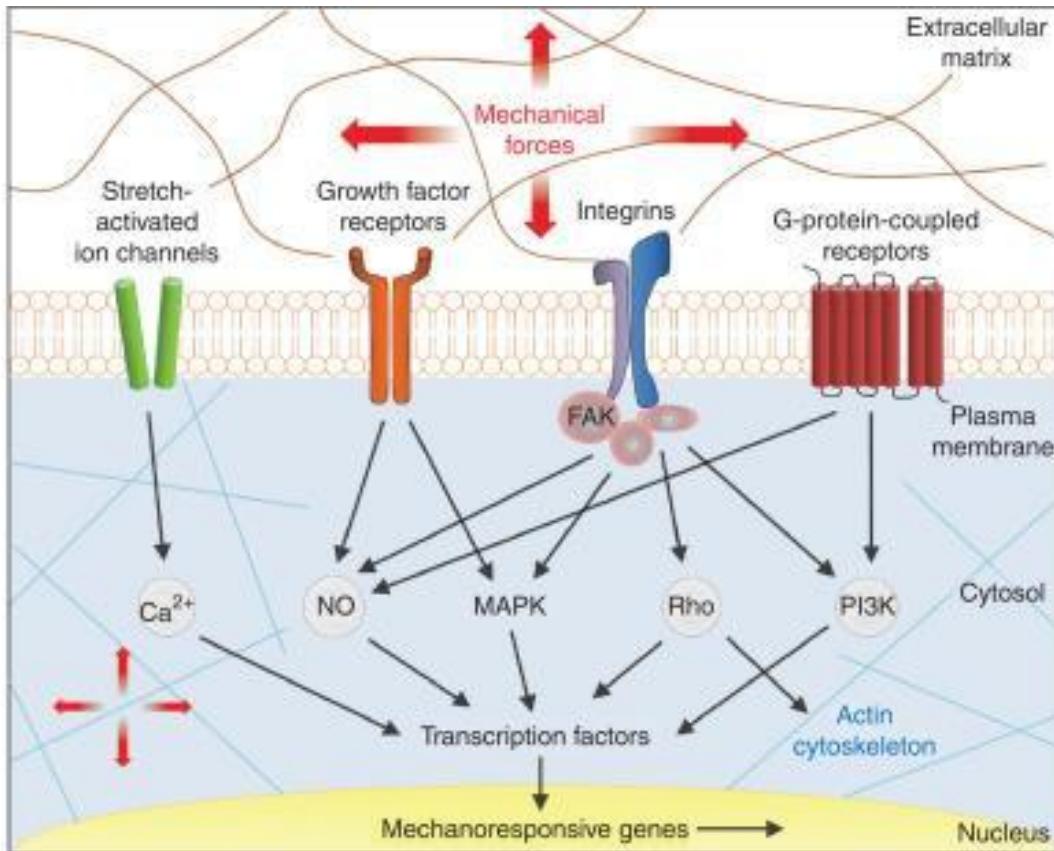
For pulling exercises, you're generally weakest at the *end* portion of the concentric. This is why, for example, so many people find it nearly impossible to actually touch their chest to the bar when doing pull-ups. (During chin-ups, it should be easy for most men and well-trained women.)

What makes muscles grow

The primary stimulus for muscle growth is sufficiently high and prolonged tension created by the actin-myosin cross-bridges of muscle fibers. In other words, muscle hypertrophy occurs when muscles produce enough biomechanical tension. As an adaptive response to this stress, the muscle fibers undergo changes in their structure, including growth of the muscle fibers to be able to produce more tension in the future. The process of muscle growth goes like this: mechanical tension → mechanotransduction → anabolic signaling → muscle protein synthesis → greater muscle size. Let's look at these processes in a bit more detail.

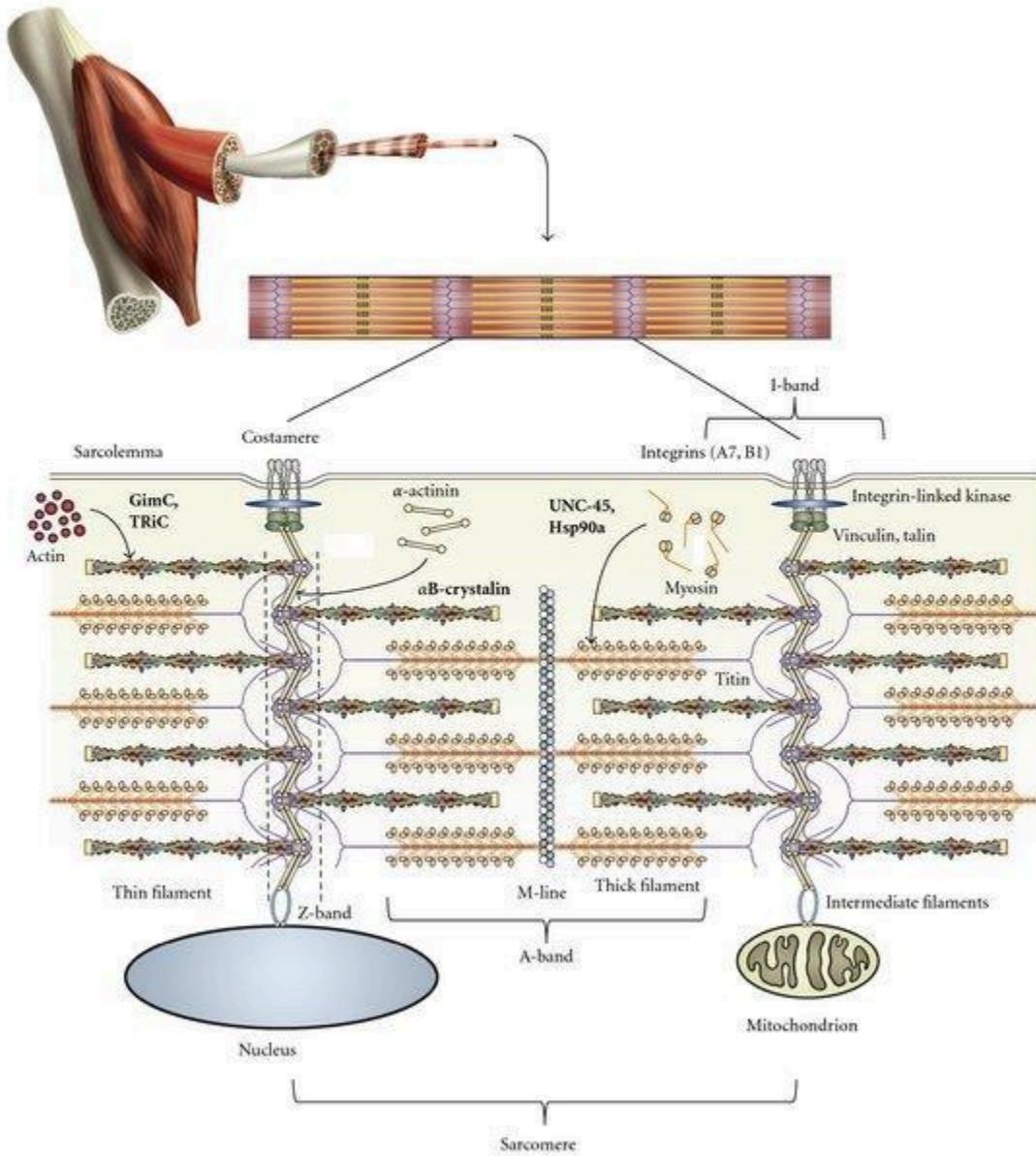
Mechanotransduction

When a muscle contracts sufficiently hard, its muscle fibers deform and trigger chemical activity. The transformation of a mechanical signal to a chemical one is called mechanotransduction and illustrated below.



Mechanotransduction is the process by which mechanical forces on muscle fibers result in chemical activity inside the muscle fiber and its cell cores (myonuclei), leading to an adaptive response to the forces the muscle experienced. [Source](#)

Mechanotransduction is initiated by mechanosensors ('tension sensors'), particularly Z-disc bound proteins and costameres. Both Z-discs and costameres serve as anchoring sites in and around sarcomeres, so they are in ideal positions to register tension, as illustrated below.



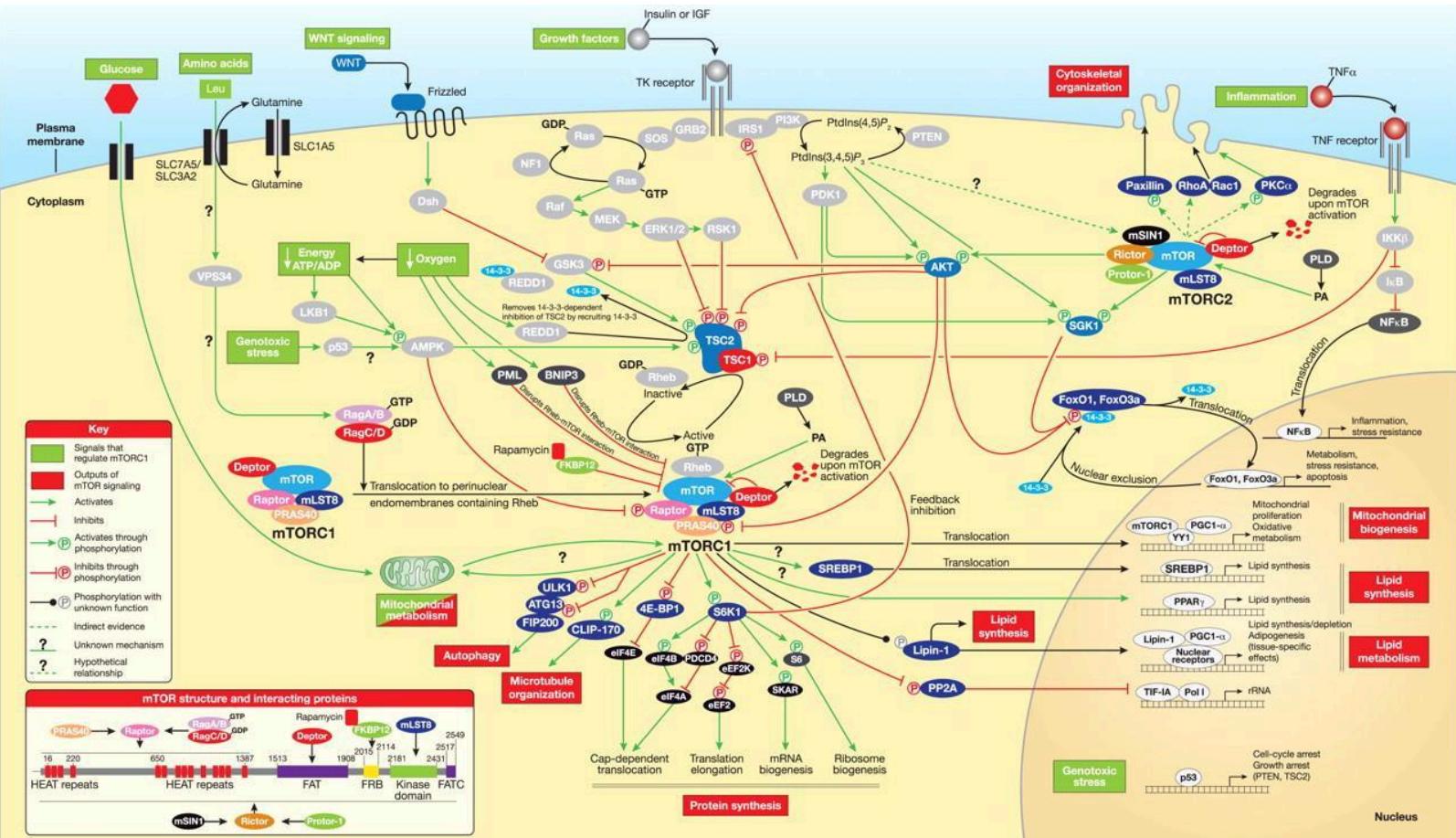
Z-discs, also called Z lines or bands, are structural zones that intersperse sarcomeres and serve as anchoring points for actin and titin. The Z stands for the German ‘zwischen’, meaning ‘in between’. Costameres are on the outside of the sarcomeres and connect it to their surrounding cell membrane (sarcolemma) and connective tissue.

Both serve as mechanosensors. [Source](#)

[Titin, a giant spring-like myofilament, is another noteworthy mechanosensor in muscle fibers.](#) Titin is effectively spring-loaded when muscle fibers are stretched, thus responding to passive muscle tension. When a sarcomere is under tension, this opens the titin kinase (TK) domain so that TK activity corresponds with the total integrated tension on the sarcomere. TK starts an anabolic signaling cascade. The muscle fibers release growth factors like insulin-like growth factor 1 (IGF-1) and myokines like IL-6 to signal the need for repair and adaptation.

Anabolic signaling

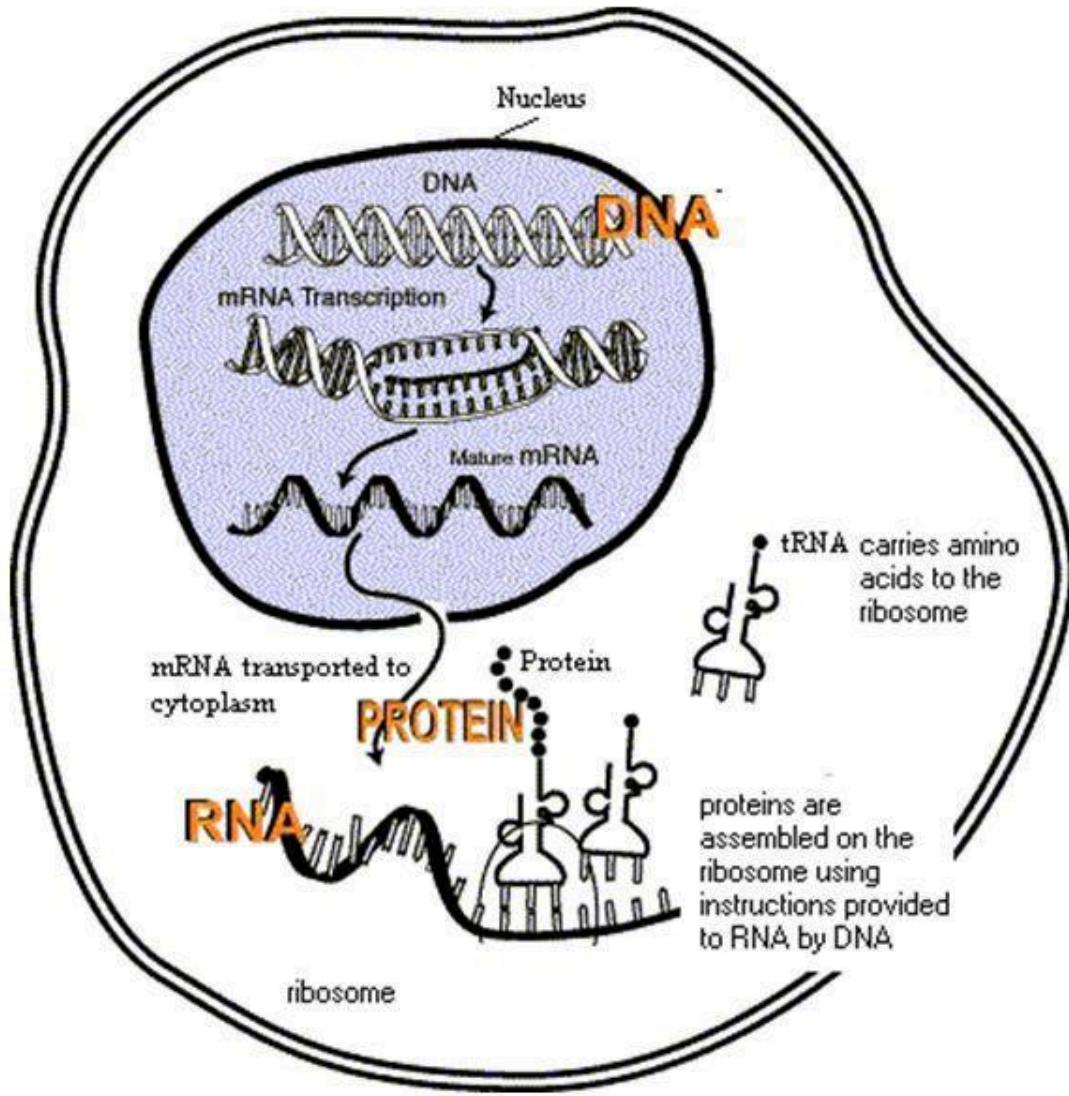
The chemical activity triggered by mechanical tension starts an anabolic signaling cascade involving multiple growth factors. A central enzyme in this process is mammalian target of rapamycin (mTOR). The mTOR master enzyme in muscle cells integrates 4 major signals – growth factors, energy status, oxygen level and amino acid concentration – to regulate muscle cell growth. It sends this information to your genes residing in the cell's nuclei, which function much like command centers. Most kinds of cells only have one nucleus, but muscle cells have many. [Not all muscle growth is mediated via mTOR](#), but [mTOR signaling strongly corresponds with muscle protein synthesis and muscle growth.](#)



Intramuscular anabolic signaling pathways are extremely complex. [Source](#)

Protein synthesis

Inside your cell's nuclei lies a molecule called deoxyribonucleic acid (DNA). DNA has 2 intertwined DNA chains that form a double helix with biological data that contain the blueprint to create your whole body. Based on the input from mTOR, parts of your DNA containing the information to create the desired proteins to remodel the cell are transcribed ('copied') into ribonucleic acid (RNA). Messenger RNA (mRNA) is then sent out to the ribosomes outside the nucleus in the cell's cytoplasm to produce the desired proteins. Ribosomes are the translational machinery than turns mRNA information into actual new proteins. These proteins are incorporated into the muscle fiber, allowing it to grow.

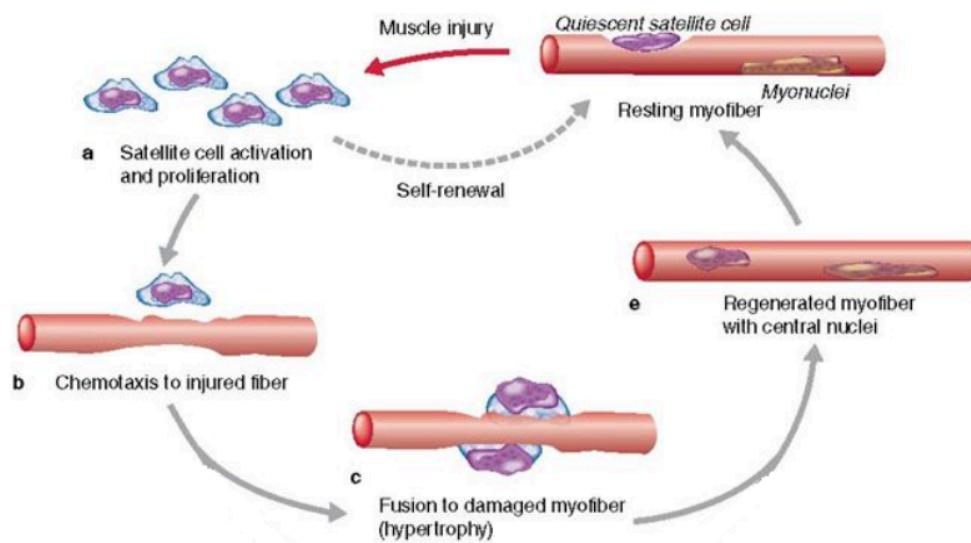


Author: Godwin Hardy

Myonuclear addition

The nuclei in the muscle fibers (myonuclei) seem to have a maximum region of the cell they can control, known as their myonuclear domain. [It was previously thought that the myonuclear domain is completely fixed, but modern research indicates it's flexible.](#) Once the extent of muscle hypertrophy starts to exceed this myonuclear domain,

[myonuclear addition gradually occurs to support further growth](#). These myonuclei cannot be created by the cell itself. Instead, they are donated by satellite cells. [Satellite cells](#) are otherwise quiescent (inactive/dormant) stem cells that are not fully differentiated into a specific role yet: ‘baby cells’. They lie just outside their parent muscle fiber. When the muscle fiber becomes damaged, the satellite cells activate, clone themselves, and fuse to the damaged site to patch it up. In this process, their cell nucleus can be incorporated into the muscle fiber, increasing its myonuclear number.



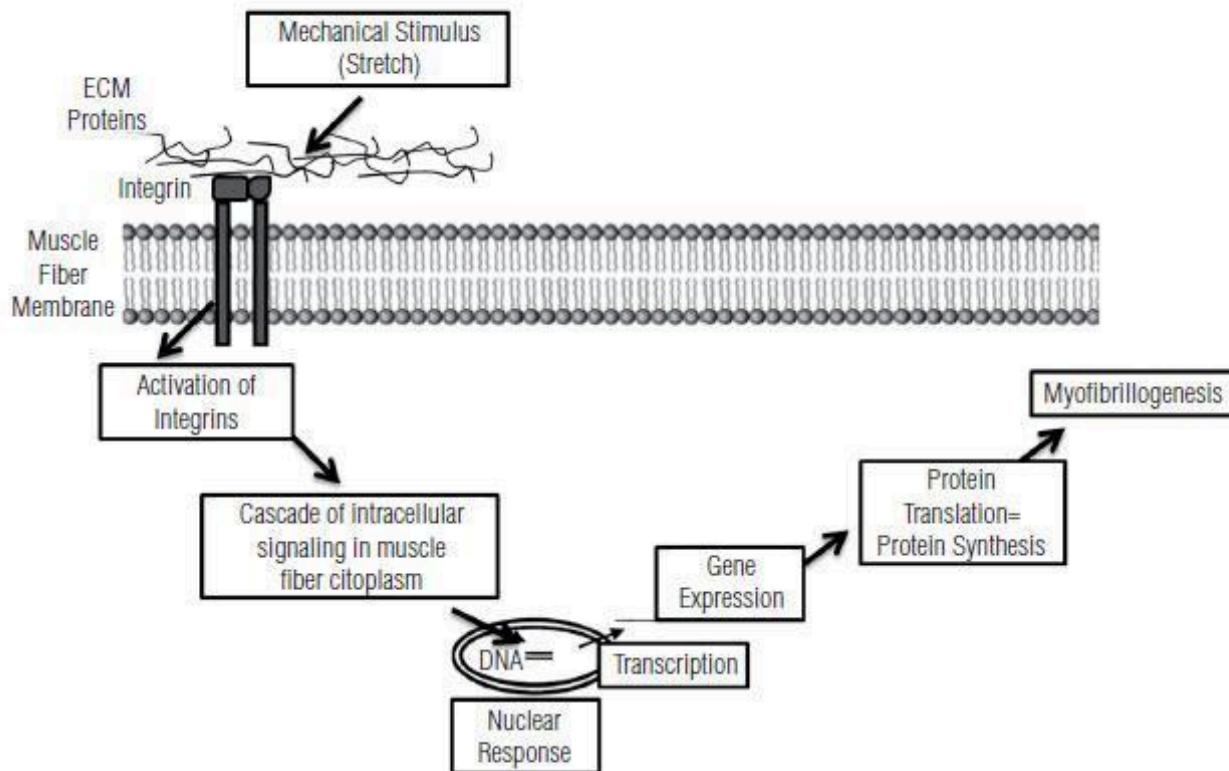
Reprinted, by permission, from T.J. Hawke and D.J. Garry, 2001, “Myogenic satellite cells: Physiology to molecular biology,” *Journal of Applied Physiology* 91: 534-551.

Muscle memory

The added myonuclei seem to persist relatively well even after muscle atrophy, so they can possibly upregulate the muscle fiber’s capacity for protein synthesis for years. This partly explains the [phenomenon of muscle memory: atrophied muscle can still have some of its former peak myonuclear number, which may facilitate rapid protein synthesis back up to its former size when someone starts training again \[2, 3\]](#), although findings are very mixed and much of the research is in rodents. Another [mechanism for](#)

[muscle memory is epigenetic](#). Strength training changes how some of our genes express themselves, making certain genes more or less active, and these changes seem to persist a considerable time during detraining, even after all muscle built from the training has been lost. As a result of the above mechanisms, it's typically considerably easier to regain lost muscle than it was to originally build it. It also generally doesn't take nearly as long to rebuild lost muscle as it took to lose it. If you stopped training for a month, it shouldn't take you a month to regain the strength and size you lost: 2 weeks is generally realistic. Even if you lose all your gains, your muscles act like they 'remember' their former size and it will be easier to get back to your top shape the second time than it was the first time.

To recap, here's an illustration of how mechanical tension leads to muscle growth.



Metabolic stress

In addition to mechanical tension, metabolic stress has been proposed as a possible mechanism of muscle growth. While the term metabolic stress has become common in the evidence-based fitness community since Brad Schoenfeld popularized it, it lacks a clear scientific definition. [Metabolic stress refers to the accumulation of metabolic byproducts in the muscle, like lactate, phosphate \(Pi\) and hydrogen ion \(H+\) along with hypoxia, i.e. the oxygen shortage, during exercise.](#)

As you've learned, lactate is a byproduct of anaerobic glycolysis, the breaking down of glucose to rapidly supply energy without a need for oxygen. [Lactate is an acid, so many people mistake it for lactic acid, but they're not the same](#): lactic acid has an extra hydrogen ion. Scientists used to think it's responsible for 'the burn' you feel during high repetition strength training. Indeed, [lactate concentration, the burn, acidosis and metabolic stress correlate very well, so well that lactate is often measured as a marker of metabolic stress and acidosis, but lactate is not the cause of acidosis](#). The acidosis is caused by hydrogen ions (H⁺). Every time ATP is broken down to ADP and P(i), a hydrogen ion is released. When there is enough oxygen available, these protons are used by the mitochondria during oxidative phosphorylation. However, when the demand for ATP is too large to sustain via aerobic pathways, ATP has to be regenerated via glycolysis and the phosphagen system, causing hydrogen ion accumulation, which increases acidity in the cell.

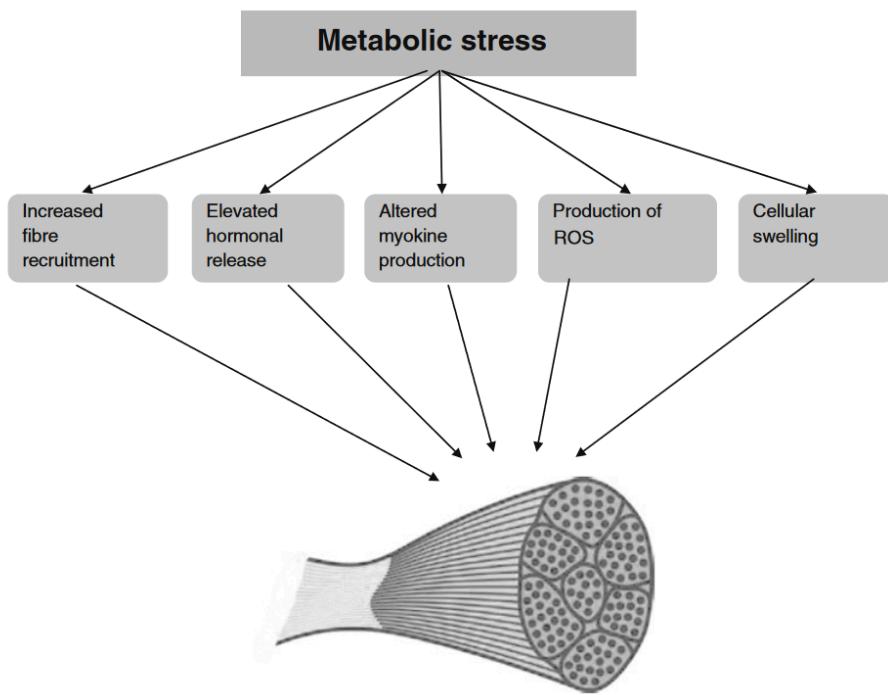
During glycolysis, lactate is also produced from pyruvate as part of the lactate dehydrogenase (LDH) reaction. The production of lactate consumes 2 protons (hydrogen ions). As such, lactate actually buffers against the burn, instead of causing it.

In short, metabolic stress is particularly high during exercise with a large demand for glucose and you feel it in part as 'the burn' in your muscles. Metabolic stress also

increases when the clearance of metabolic byproducts is hindered, such as by constant-tension exercise or blood flow restriction.

Metabolic stress may contribute to muscle growth in various ways, as shown below. However, the evidence for the importance of metabolic stress is almost entirely based on *in vitro* research on isolated muscle cells in a lab. Metabolic stress is arguably just a new rationalization for the intuition to train based on the pump and burn you feel during training. There is no direct evidence that metabolic stress itself plays an important role in muscle growth independent of mechanical tension. In fact, in many cases where metabolic stress was proposed to be beneficial for muscle growth, it even turned out to be detrimental, as you'll learn in the coming modules (in particular those on blood flow restriction training and rest intervals).

Fig. 2 Proposed mechanisms by which exercise-induced metabolic stress may mediate muscle hypertrophy. *ROS* reactive oxygen species



As such, metabolic stress is not a growth pathway you need to target with your training programs.

Muscle damage

In addition to mechanical tension and metabolic stress, muscle damage has been suggested to be the third mechanism for exercise-induced muscle hypertrophy. During heavy exertion, muscles can become damaged as a result of the mechanical tension imposed on them, especially when the muscles are lengthened, which makes them more susceptible to micro-tears. The build-up of calcium in the muscle fibers during contractions can also damage the muscle, a sort of erosion from the inside out. The extent of muscle damage can vary from just disrupting sarcomeres on a macromolecular level to large tears in muscle and connective tissue, which is generally associated with injuries.

To quote Brad Schoenfeld: “Damage can be specific to just a few macromolecules of tissue or result in large tears in the sarcolemma, basal lamina, and supportive connective tissue, and induces injury to contractile elements and the cytoskeleton. Because the weakest sarcomeres are located at different regions of each myofibril, the nonuniform lengthening causes a shearing of myofibrils. This deforms membranes, particularly T-tubules, leading to a disruption of calcium homeostasis and consequently damage because of tearing of membranes and/or opening of stretch-activated channels.

The response to myotrauma has been likened to the acute inflammatory response to infection. Once damage is perceived by the body, neutrophils migrate to the area of microtrauma and agents are then released by damaged fibers that attract macrophages and lymphocytes. Macrophages remove cellular debris to help maintain the fiber’s ultrastructure and produce cytokines that activate myoblasts, macrophages and lymphocytes. This is believed to lead to the release of various growth factors that regulate satellite cell proliferation and differentiation.” ([Source](#))

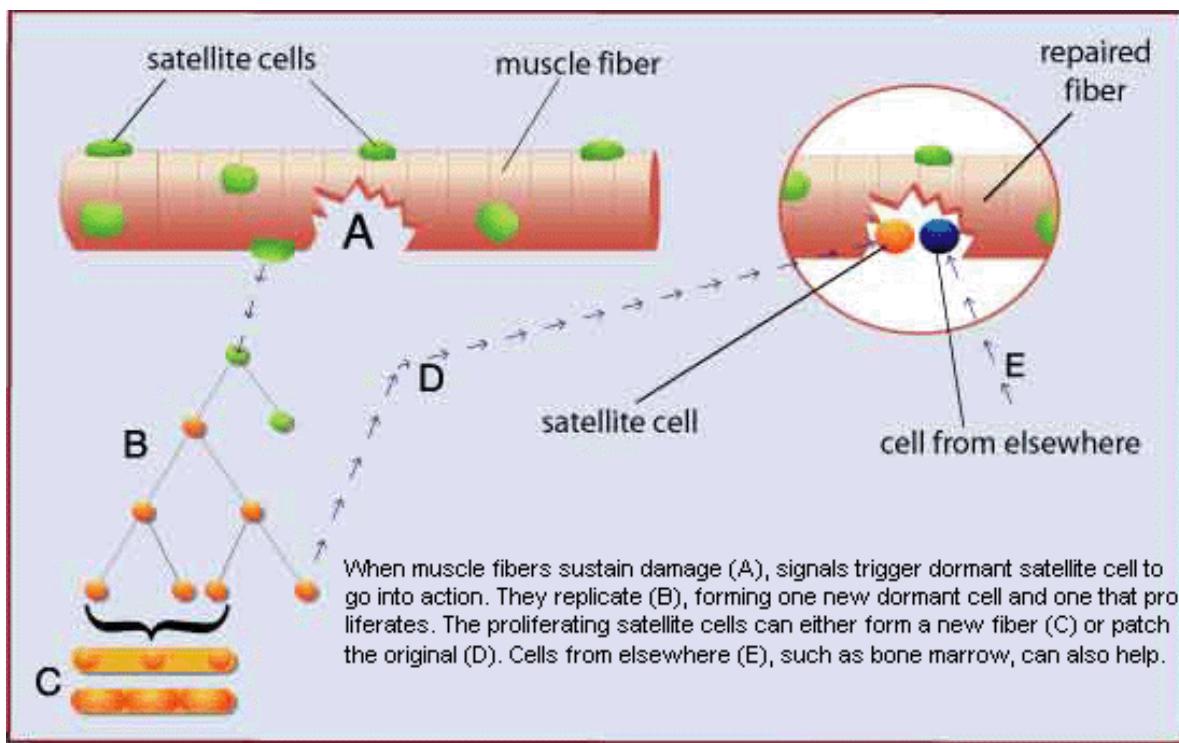
So muscle damage generates inflammation. Damaged muscle cells release pro-inflammatory molecules that attract neutrophils, a type of white blood cells. Neutrophils destroy dead muscle cells and produce molecules that attract other inflammatory cells. Macrophages, another type of white blood cells, infiltrate the damaged muscle to remove cell debris. Additionally, macrophages secrete growth factors and other molecules which play an important role in muscle repair. The neutrophils can attack healthy cells, worsening the muscle damage, so excessive inflammation may delay muscle recovery. However, [inflammation is a normal part of the muscular repair process and inhibiting this inflammation can therefore reduce muscle recovery and growth](#), as you've learned in the Energy module.

After muscle damage, fluid and plasma proteins move into the injured tissue. This can create muscle swelling, formally called edema, and is one of the reasons your muscles may appear larger after just a single workout even though minimal actual muscle growth has yet occurred.

But does muscle damage also promote actual hypertrophy? This hypothesis was based largely on 2 arguments.

First, muscle damage activates satellite cells. As you've learned, muscle hypertrophy is typically accompanied with satellite cell activation, especially when the extent of muscle fiber growth exceeds the myonuclear domains of its myonuclei. After multiplication, daughter cells diffuse to damaged muscle sites and help regenerate the muscle by fusing with damaged muscle fibers. However, [muscle hypertrophy can occur without satellite cell activation and satellite cell activation does not prove muscle growth is occurring. High intensity interval training, for example, stimulates profound satellite cell activity but minimal muscle growth](#). Satellite cell activation seems to be primarily related to muscle *repair* rather than new growth.

Excessive muscle damage may even delay muscle growth. In terms of protein balance, [muscle damage only increases protein breakdown and turn-over](#), so while muscle damage increases absolute protein synthesis, net protein synthesis doesn't increase: it's offset by the increased protein breakdown. The additional protein synthesis you gain from muscle damage is only there to replace the broken-down proteins, not to form new muscle tissue. As a result, [excessive levels of muscle damage can put new muscle growth on hold, causing 'overtraining'](#).



The second main argument for why muscle damage was thought to stimulate muscle growth is research on eccentric training. [A given number of sets of purely eccentric training tends to result in more muscle growth than purely concentric training](#), although [a 2024 meta-analysis](#) found the effect is only significant in certain study designs. Many studies find that eccentric training also stimulates much more muscle damage, so the idea was that perhaps the muscle damage was responsible for the greater growth. However, [eccentric exercise appears to primarily inflict more muscle damage than](#)

concentric exercise because it's a novel stimulus for most people, not so much because eccentric exercise is inherently that much more muscle damaging than concentric exercise: the difference in muscle damage between eccentric and concentric exercise disappears over the course of 10 weeks of training.

Moreover, the growth difference between eccentric and concentric exercise disappears when we look at studies that equated total work, indicating the hypertrophic advantage of eccentrics is likely mediated by higher mechanical tension per repetition [2], not muscle damage.

Other findings strongly dispute the role of muscle damage in promoting muscle hypertrophy. Muscle damage is not required for muscle growth and muscle damage does not correlate with muscle growth or strength development. High and low responders to exercise also don't differ in the amount of muscle damage they experience from exercise.

Indirectly, muscle damage can also impair muscle growth by decreasing strength performance. Damaged muscle fibers exhibit lower motor unit firing rates. This is a type of local fatigue, as it does not affect the contralateral limb. After intensive, novel training sessions, muscle force production can be impaired up to 72 hours in trained individuals. In untrained individuals or after extremely muscle damaging workouts, performance may suffer for 2 full weeks. Muscle damage thus reduces our ability to impose mechanical tension on our muscles, which is much more established as a primary cause of muscle growth than muscle damage.

Muscle damage also impairs motor learning. Excessive muscle damage will thus likely interfere with strength development via neural adaptations.

In conclusion, muscle damage is likely not a direct cause of muscle growth. In fact, mechanistic and empirical evidence show that high levels of muscle damage may delay recovery and muscle growth, causing overtraining and increasing injury risk, in addition to acutely impairing performance. As such, muscle damage is not something you should actively seek out but rather minimize.

DOMS

Muscle damage can be related to a feeling of muscle soreness in the days after certain types of exercise, particularly novel training stresses and eccentric (lengthening) muscle contractions. The pain normally starts 6-24 hours post-workout and peaks 48-72 hours after the exercise before gradually subsiding. The pain is typically worst when stretching the muscle. The muscle can also be sensitive to the touch. Researchers call this delayed onset muscle soreness (DOMS).

DOMS is probably the true reason many people believed muscle damage was related to muscle growth. For a lay individual, the presence of soreness in a muscle may be perceived as a sign the previous workout was effective. At least something's going in the muscle. The accompanying muscular edema (swelling and water retention) also makes the muscle bigger, further aiding the idea the workout was effective to stimulate muscle growth.

However, research finds that muscle damage is not a sign of muscle growth; moreover, DOMS isn't even a reliable measurement of muscle damage. While the presence of DOMS means there is most likely some muscle damage present, the extent of DOMS correlates very poorly or not at all with the amount of muscle damage or strength recovery [2, 3, 4, 5]. For example, one study measured muscle soreness and creatine kinase elevation, a measure of muscle damage, after a heavy leg day in weightlifters vs.

untrained individuals. While the untrained individuals experienced far more muscle damage, soreness levels were actually a bit higher in the weightlifters.

[Another study](#) measured muscle soreness vs. strength recovery after endurance vs. maximal eccentric exercise. After the endurance exercise, strength recovered rapidly with most recovery in 24 hours and nearly full recovery at 48 hours (no longer significantly different from baseline), yet this period was when soreness peaked. The eccentric exercise caused considerably more neuromuscular fatigue and strength loss than the endurance training, yet ratings of soreness weren't that different compared to after the endurance exercise. In general, the time course of soreness tends to be similar regardless of what caused it with a peak after 1-2 days.

Moreover, certain types of training, such as [electrically stimulated muscle contractions, can inflict significantly more muscle damage than conventional strength training without increasing DOMS](#). Similarly, [a high protein intake objectively speeds up neuromuscular recovery and reduces muscle damage but does not affect soreness](#). Conversely, [certain recovery techniques like massage or cryotherapy can reduce muscle soreness without speeding up actual muscular recovery](#).

Therefore, you cannot use subjective soreness as a reliable sign of whether you've recovered from your training. You can be recovered but still sore and you can not be recovered yet not sore.

[There is also major variation in the extent of muscle damage and soreness between individuals even after the exact same workout](#). Some people just get much sorer than others, regardless of how effective their programs are. Some people also keep getting sore from almost any type of training, no matter advanced they are, whereas others almost never get sore. Moreover, muscle groups within the same individual have the

same variance and this does not appear to be related to muscle growth. Many people rarely get sore delts or lats, for example, whereas many people relatively quickly get sore hamstrings or pecs after similar types of training. In conclusion, [there's no direct relation between delayed onset muscle soreness \(DOMS\) and muscle growth.](#)

DOMS is mostly experienced after novel training stresses, such as after performing a high volume of an exercise you've never done before or after trying a new sport. The first time you go snowboarding or surfing, for example, you'll probably get very sore, but neither sport is going to get you jacked. [After this initial training session, adaptations we call the repeated bout effect greatly reduce DOMS if you perform said exercise again, even if it's months later.](#) As you've probably experienced, [DOMS is already notably reduced the second time you perform a new exercise. After starting an intense, novel exercise program, DOMS and muscle damage decrease similarly over time,](#) but [the repeated bout effect's mechanism of action seems to have a significant neural component:](#) you can feel less pain even when the amount of muscle damage hasn't decreased that much yet.

There's also [emerging research](#) that muscle soreness is more closely related to our connective tissues than our contractile tissues in the muscle.

In further support that DOMS and muscle damage are distinct, training through DOMS typically does not make it worse. When you have very bad DOMS, objective measures of muscle damage are often not affected nearly as much as the perception of pain, further supporting that DOMS is more a sensory phenomenon than a reliable indication of muscle damage or recovery status.

Interestingly, [the exact mechanisms of DOMS are still unclear.](#) The most plausible explanation at this point is arguably that inflamed microscopic tears in muscular

contractile or connective tissue trigger nearby nerve cells, which trigger the sensation of pain.

In conclusion, DOMS is not a sign your program is effective and is more of a nuisance that you can't do much about. A muscle can be very sore while having relatively little damage or not sore at all while having significant damage. DOMS is neither a necessary nor sufficient condition for muscle growth, nor is it a correlate. Muscle damage in general is not something you should seek out but rather minimize, as it delays recovery and muscle growth and impairs performance. It is better thought of as a byproduct of the mechanical tension that induces muscle growth than an independent cause of the muscle growth. As Lee Haney famously said: "Stimulate, don't annihilate."

Fortunately, DOMS is generally harmless and can be trained through. Within reason of course: if after a thorough warm-up you still can't perform a certain exercise with full range of motion because you're too sore, you probably want to skip that exercise that session to avoid risking excessive muscle damage and injury.

Hormones

Mechanical tension, metabolic stress and muscle damage are all local factors. They occur within the muscle and the resulting muscle growth happens within that same muscle. In contrast, systemic factors have also been hypothesized to play a role in muscle growth. Your hormones are the most notable systemic factor for muscle growth.

Strength training newbies are often told to train their legs. When they protest that they don't really care about leg muscle growth, they're told that training their legs will make their upper body larger too. The theory is that since heavy compound leg work like squatting increases the production of anabolic hormones more than upper body work like bench pressing, this increase in circulating testosterone, growth hormone and IGF-1 then amplify the training effect in other body parts. It sounds plausible and there is no doubt that testosterone in particular is a strongly anabolic hormone in muscle tissue. But are the short-lived peaks in anabolic hormone concentrations enough to meaningfully affect muscle growth? Multiple studies suggest the answer is yes, but they all have serious methodological limitations and multiple other lines of research do not support acute systemic hormonal benefits.

1. Hansen et al. (2001) found that performing leg presses directly after biceps curls resulted in greater elevations in growth hormone and testosterone and greater isometric strength development than just performing biceps curls. However, while the subjects were untrained, the leg press + curls group had significantly lower isometric strength at baseline, so this confounder could explain the greater strength development in this group instead of hormones. This confounder would also explain why isokinetic strength, a different way to measure strength, and standing biceps curl 1RM increased similarly between the groups. As such, this study is inconclusive.

2. [Rønnestad et al. \(2011\)](#) conducted a very similar study and found that performing leg presses before performing biceps curls increased biceps CSA at the largest point along with a greater improvement in biceps curl 1RM. However, total muscle volume increased similarly between groups, suggesting the greater CSA change at that one particular point was measurement error due to misaligning the CSA slices during the MRI. And the greater 1RM biceps curl strength gain did not correspond with the similar increases in peak power and biceps training loads between the groups. It is unclear how their 1RM could have improved more while their actual training loads throughout the study did not. So while this study suggests a benefit of performing heavy lower body compound work before upper body isolation work to reap the benefits of increased anabolic hormone levels, skepticism towards these results is warranted.

3. [Mangine et al. \(2017\)](#) found that exercise induced testosterone spikes are associated with muscle growth across individuals. This was cross-sectional research, however, so it cannot demonstrate causality, just a correlation. Perhaps the high-responders to exercise also had inherently higher testosterone increases during training, or those individuals just trained harder.

4. [Bartolomei et al. \(2018\)](#) had strength-trained men perform full-body workouts in which lower body work was done after the upper body work with either higher reps (10-12) or lower reps (4-5) for the lower body work. Higher reps generally stimulate a greater elevation in anabolic hormone levels, so this could theoretically augment the upper body gains. Indeed, the higher rep group had greater improvements in bench press strength, arm size and fat loss. However, total body fat-free mass gains did not differ between groups. Major limitations of the study included a sample size of 20, a mere 6-week study duration and, worst of all, the use of skinfold calipers and arm circumferences to measure body composition.

5. [Madarame et al. \(2008\)](#) studied the effect of a leg workout with or without blood flow occlusion performed directly after biceps curls. The leg workout with blood flow occlusion increased arm size and strength compared to the one without occlusion. Oddly, the group without occlusion didn't gain significant muscle or strength at all, despite consisting of (only 7) untrained men starting to train for 10 weeks. Moreover, the difference in gains between the groups could not be interpreted to enhanced anabolic hormone levels from the occlusion, because testosterone and growth hormone levels didn't significantly differ between the groups.

[May et al. \(2018\)](#) tried to replicate these results. They again found that a leg workout with blood flow occlusion – but not without it – after biceps curls increased biceps strength gains. This time muscle growth was unaffected though. It's hard to explain how a systemic effect from training the legs would improve upper body strength but not muscle growth. This suggests a neural rather than a physiological mechanism, which is unlikely for hormones, especially since the lower body work was done *after* the biceps curls, so the hormones could not possibly affect performance acutely. The study was limited by having only 12 subjects per group and a 7-week duration.

While these data seem hard to reconcile with hormonal effects, [Cook et al. \(2014\)](#) found for the third time that blood flow restriction of the lower body increased upper body strength gains. Semi-professional rugby players that wore occlusion cuffs during their leg work not only experienced greater lower body strength gains but also greater bench press strength gains, as well as higher testosterone levels during training. However, this was again a very small study with 20 subjects and a mere 3-week duration.

Blood flow restriction training will be discussed in greater detail in the module on advanced training techniques.

In contrast to the above studies, several other studies do not support the theory that acute hormonal elevations from training can augment the gains of unrelated body parts ('the hormone hypothesis').

- [Ampomah et al. \(2019\)](#) could not replicate the cross-transfer effect of blood flow restriction training. A lower body training program with or without occlusion had similar effects on biceps curl strength, trunk strength and trunk size after 10 weeks. The overall programs were too light to stimulate robust gains in either group though.
- [Jakobbson et al. \(2021\)](#) found that lower-rep (4-6 reps) and higher-rep (10-15 reps) lower body training before upper body training resulted in similar bench press and lat pulldown strength development after 10 weeks, again failing to replicate the earlier findings from Bartolomei et al. There were also no significant effects on the total growth hormone or testosterone levels in the blood (AUCs).
- [Walker et al. \(2004\)](#) found that a biceps training program on its own was just as effective as that program as part of a more complete full-body training program. 1RM biceps curl strength and size, as measured by gold-standard magnetic resonance imaging, did not significantly differ between the groups after 10 weeks. The full-body program also had no further effect on IGF-1 or myostatin levels.
- [West et al. \(2009\)](#) found that while performing high-volume leg curls and extensions directly after biceps curls increased growth hormone, IGF-1 and testosterone levels, it did not affect cellular anabolic signaling or muscle protein synthesis in the biceps. [West et al. \(2010\)](#) found that this same protocol also did not result in superior muscle growth or strength development of the biceps after 15 weeks.
- [Spiering et al. \(2008\)](#) found that performing a high volume of bench presses, shoulder presses and rows before performing leg extensions did not affect anabolic signaling in the quadriceps.

There are also more fundamental physiological problems with ‘the hormone hypothesis’. First, anabolic hormone level differences within the physiological range are not that important for muscle growth. While a good case can be made for testosterone’s anabolic effects, which can tell your genes to start synthesizing muscle protein, reduce protein breakdown and activate satellite cells, [growth hormone, in contrast to popular belief, is not anabolic in myofibrillar tissue, only in the surrounding connective tissue like your tendons and bones](#). Most of the increase in lean body mass following growth hormone usage is merely water retention. During your workouts, growth hormone seems to be primarily active to mobilize fuel stores, such as by increasing fat burning (lipolysis), and its production is related mostly to metabolic stress, particularly blood lactate levels and hypoxia [2]. IGF-1, in turn, seems to be more strongly related to muscle anabolism in effects, particularly its splice variant mechano growth factor (MGF). However, IGF-1 is produced and used largely within the muscle, so its relevance for other muscle groups is highly dubious. Indeed, [significant muscle growth can occur without acute post-workout elevations in anabolic hormones](#), so anabolic hormone elevations are not a necessary condition for muscle growth. Higher levels also do not consistently translate into greater gains. [Several studies find no correlation within strength trainees between acute post-workout anabolic hormone levels and muscle growth or strength development](#) [2, 3, 4, 5, 6]. That said, in other research there are positive, albeit mixed and inconsistent, relations between muscle growth and [testosterone, growth hormone, mechano growth factor \(MGF\)](#) and even [cortisol](#). Given the mixed and inconsistent nature of these relations, it is very possible that they are a case of mistaking correlations for causation. For example, cortisol may positively correlate with muscle growth in some research, because people that train harder experience both greater gains and greater stress from the training.

To the extent that anabolic hormones spikes influence muscle growth in the first place, [increases in systemic hormone levels do not seem to affect their availability to other](#)

[trained muscle groups](#). So even if heavy leg training increases the anabolic hormone concentration in the legs and blood, it doesn't seem to affect how much is taken up by the biceps or other untrained muscles.

And even if we can increase anabolic hormone uptake in unrelated muscles, the timing of when we train these other muscles may not matter. [Exercise order does not affect total anabolic hormone production](#), only *when* anabolic hormone levels are highest.

Most hormonal effects are the result of the total exposure over time, the area under the curve. Rønnestad et al. believed that the key to systemic hormonal support lies in performing the heavy compound work first, like they programmed their study. However, multiple of the other studies did not perform their lower body work first and still found positive effects on upper body development somehow. If the timing is irrelevant, the take-home message may change from “train your legs before your upper body training” to simply “train your legs”.

In conclusion, the theory that immediate spikes in anabolic hormone levels influence your training has many physiological holes in it. Our anabolic hormone levels are not very consistently affected by differences in our training in the first place, their acute levels don't consistently correlate with our long-term muscular development and hormone levels in the blood don't predict local muscle uptake. There are multiple studies that found that intensive lower-body training increased upper body muscular gains, but they are contradicted by multiple other studies of higher quality. It may be tempting to conclude that full-body workouts are superior to body part splits based on systemic effects, but as we'll discuss later, that also doesn't appear to be universally true, possibly because the timing of the lower body training doesn't matter. In short, any systemic (hormonal) effect of exercise on muscle growth, if it exists at all, is far weaker than the local effect of mechanical tension.

Structural balance

Another potential systemic regulator of muscle growth is structural balance. The theory in its currently most common form is that your body tries to maintain a certain balance between muscular strength or size in every part of the body or in every movement pattern. If any body part or movement pattern becomes overdeveloped, the nervous system shuts it down to prevent further imbalance. For example, a weak rotator cuff is often said to limit bench press strength. Charles Poliquin proposed the following strength ratios for perfect structural balance in the upper body.

Structural Balance: Close-Grip Bench Press

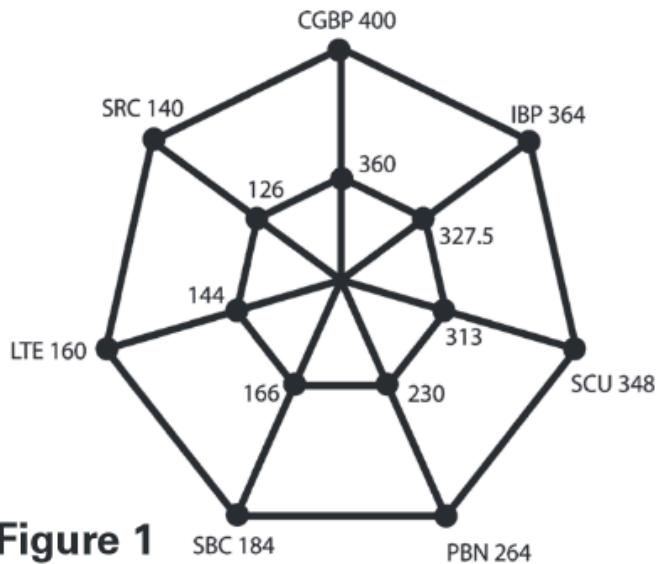


Figure 1

CGBP -- Close-Grip Bench Press	SBC -- Scott Barbell Curl
IBP -- Incline Bench Press	LTE -- Lying Triceps Extension
SCU -- Supinated Chin-up	SR -- Standing Reverse Curl
PBN -- Press Behind Neck	

If the theory sounds a bit vague, it is. There is no formal operational definition and there is no scientific research behind it. One anecdote from Charles Poliquin about how he increased some athlete's bench press strength a ton without bench pressing by

balancing out the guy's weak rotator cuff was enough to make structural balance theory spread through the fitness community like a hooker's legs.

Some refer to Olympic weightlifting practices as evidence of structural balance. Many weightlifters use ratios between the 2 Olympic lifts – the Snatch and the Clean & Jerk (C&J) – and accessory movements like the squat to assess how to improve the Olympic lifts. If a lifter doesn't progress in the C&J and her front squat is comparatively weak, getting stronger in the front squat is commonly prescribed to improve the C&J. While using strength ratios, this has nothing to do with structural balance in *other* parts of the body. It is simply a case of strategically selecting exercises with the highest carry-over to the target lifts.

In fact, Olympic weightlifters are an excellent example of athletes who are not structurally balanced. Many of them do nothing but squats and Olympic lifts. That means almost all of their training is in the frontal plane: the bar only goes up or down vertically. Imagine that, decades of Olympic weightlifters held back because they didn't know their performance in the Scott barbell curl wasn't up to par.

Examples of extreme structural imbalance abound within other sports as well.

- Most team sports athletes have significantly asymmetrical strength and performance, but this is not associated with reduced overall performance [2].
- Look at how top heavy some ring gymnasts are. Many actively avoid leg training, because muscle mass in the legs is largely dead weight for ring exercises.
- Look at the freakishly large quads of some sprint cyclists in comparison to their upper bodies.
- Look at how huge the asymmetry in arm size and strength of some arm wrestlers is.

- Look at the amazing physiques of wheelchair bodybuilders and the performance of wheelchair athletes.



Wheelchair bodybuilder Nick Scott: no structural balance, no problem.

For a scientist, the theory does not jive with the [fundamentally local regulation of muscle growth](#): each muscle's size is regulated almost entirely independently of others. Several scientific observations indirectly falsify the existence of any mechanism in the body that tries to prevent structural imbalance.

1. Antagonist inhibition for strength

Structural balance theory says that if the biceps gets stronger and the triceps doesn't, biceps activity will be restrained by the nervous system. Like one of the horses pulling a carriage being reined back by the driver because it was trying to run faster than the other horses. Sounds plausible, right?

Plausible but deceptive. A more valid analogy would be a carriage being pulled by 2 horses in opposite directions. Horse Triceps wants to move to the dumbbell rack to do overhead extensions, but horse Biceps wants to move to the mirror to look at his guns. This situation is regulated by antagonist co-activation. Antagonist co-activation is the activity of muscles with the opposite function of the prime movers (the agonists). For

example, during a biceps curl the triceps is an antagonist. Antagonist co-activation is required to stabilize movement. The triceps basically corrects for overenthusiastic actions of the biceps. Now here's where the research gets interesting. [When the prime movers become stronger, it is not their activity that is restrained by the nervous system but that of the antagonists.](#) So during a curl, the stronger your biceps gets, the weaker your triceps contracts. The motor cortex, the part of your brain that controls movement, learns to maximally contract the biceps during a curl with minimal interference from the triceps.

This makes evolutionary sense. It is an efficient adaptation. A limitation on prime mover activity would be highly unadaptive. Since nature only cares about function and adaptivity, we see extreme structural imbalance between antagonists in nature in various animals. A good example is the jaws of a crocodile. [Some crocodiles have a bite force of 3,700 pounds per square inch \(psi\).](#) That's the equivalent of 16,460 newtons, roughly the force needed to lift 3691 pounds (1677 kg). In comparison, the average guy may be able to clench his teeth into a piece of unseasoned chicken with 150 to 200 psi (890 newtons). Yet you can easily hold a croc's jaws shut because the muscles that open instead of close the jaw are extremely weak. In humans we see this in a less extreme form in the weakness of the tibialis anterior on your shins compared to the strength of the calves. Opposite function, opposite strength level.

2. The cross-training effect

[If you only train your right biceps, your left biceps will become considerably stronger too. Not only that, your triceps gets a bit stronger too, even on the untrained side.](#) This is called the cross-training effect. Is this finally proof of structural balance theory? How can cross-training occur if muscle is just dumb meat?

Cross-training has nothing to do with muscular adaptations. Researchers have used every measurement technique known to man – anthropometric measurements, imaging techniques, analysis of the muscle cross-sectional area, etc. – to see if the untrained limb gets bigger. It doesn't. [1, 2]

The mastermind behind the cross-training effect is your nervous system, mainly your brain (specifically the motor cortex). Many parts of your body are operated by some of the same parts of your nervous system. So when the nervous system learns how to recruit your muscles on one side of your body, some of these neural adaptations can also be used by the other side. That's why some researchers now call it the cross-education effect.

This is a form of structural balance, but it is in fact the complete opposite of structural balance theory. Structural balance theory says that when your right biceps gets too strong relative to your triceps or your other arm, it is shut down. What the cross-education effect and antagonist co-activation both show is that this doesn't happen. Instead, the triceps, even the other arm, adapts along with the biceps. Basically, your body automatically maintains structural balance, although putting it this way is misleading. A better way to put it is that your body automatically maintains some strength symmetry both within a limb (e.g. triceps/biceps) and across limbs (e.g. right arm/left arm). You can still develop massively asymmetrical strength across limbs, however, as [the cross-education effect is generally less than half of the strength gain of the trained limb](#). Importantly, the 50% strength transfer to the non-trained limb is based almost exclusively on research in untrained individuals. The cross-training effect may be smaller in trained individuals. Untrained individuals generally still experience significant strength development due to increased neural drive but strength-trained individuals mainly experience refinements in motor coordination, so they're unlikely to experience the same level of cross-training.

3. Removing body parts

If you're not convinced of the body's disregard for structural balance, let's look at an extreme example. We've looked at how the body responds to muscle growth of muscles with opposing functions, muscles on the other limb and muscles on the upper vs. lower half of the body. But what happens when there is extreme structural imbalance between muscles with the same function (synergists)? This scenario rarely occurs in humans, because these muscles will be activated together and therefore grow together. Fortunately (?), we can do much more extreme experiments on animals.

Researchers have looked at what happens when you surgically remove body parts (ablation) or cut them off from the nervous system (denervation) in various animals [1, 2]. When you remove a muscle with the same function or even a division (head) of the same muscle, what is left becomes stronger. Compensatory hypertrophy occurs in the remaining muscle. This again makes no sense from a structural balance perspective, which would require that the remaining body part becomes weaker to retain structural balance. But it makes perfect sense from an evolutionary perspective. The body adapts without concern for anything but function. Lost half of your calves? Better learn to walk on the other half.

Conclusion

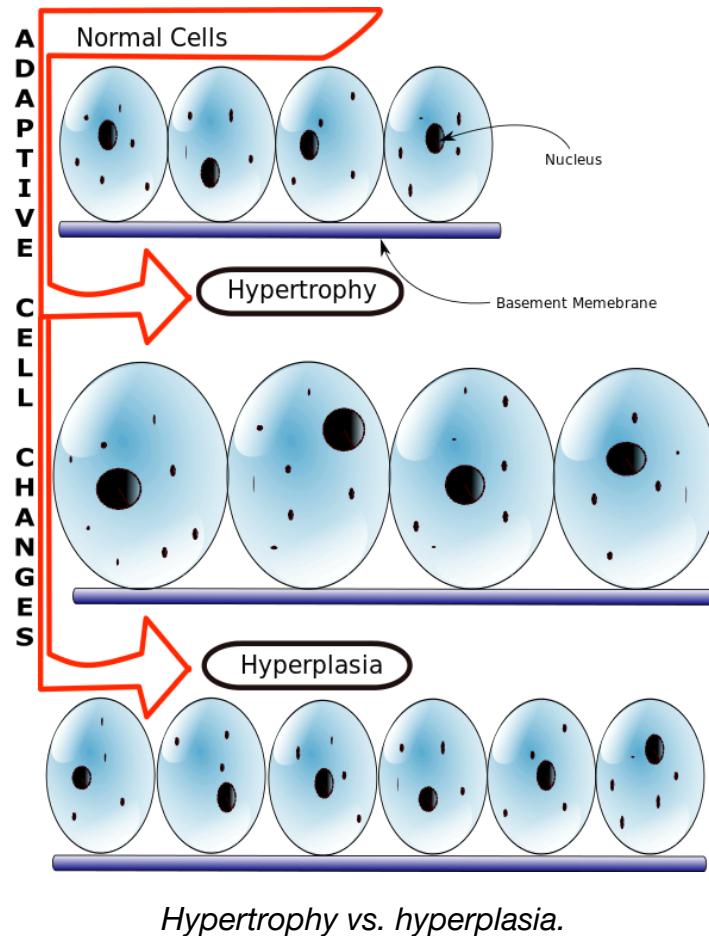
Structural balance theory is good broscience: plausible at a glance yet ill-defined and completely lacking in scientific study. Closer inspection reveals that it has no plausible neural or physiological mechanism, it makes no evolutionary sense, it cannot explain the wide display of structural imbalance in nature and sports, and it does not correspond with any empirical evidence.

How muscle grows

Muscle hypertrophy vs. hyperplasia

Muscle growth can occur via muscle hypertrophy and hyperplasia.

- Hypertrophy is *growth* of existing muscle fibers (= muscle cells), primarily via growth of the myofibrils (the contracting parts) and the addition of more myofibrils on top of each other.
- Hyperplasia is an increase in the *number* of muscle fibers.



[Whether muscle hyperplasia occurs in humans is still debated \[2, 3\]](#). It's impossible to directly count the number of muscle fibers in a human, because to study all the muscle fibers in a muscle, you'd need to take it out of the body. So we're limited to studying corpses and it's hard to assess strength training effects in a corpse, as you only have the 'after' measurement without any baseline to compare it to. Even in corpses, counting the number of muscle fibers directly is like trying to count the number of hairs on someone's head: nigh impossible. Human muscles are estimated to have [hundreds of thousands](#) of muscle fibers.

Some indirect evidence suggests muscle hyperplasia occurs in humans. A few studies from the 80s and early 90s found that bodybuilders and other trained individuals didn't have larger muscle fibers than untrained or lesser trained individuals. Obviously, they had bigger total muscles, so this suggests they must have had more muscle fibers rather than larger ones. The increased muscle fiber count could be a genetic endowment or a training effect, but either is hard to believe.

- If the bigger individuals were born with more muscle fibers, the fibers must have been unnaturally small at birth and only capable of growing to normal levels. Otherwise, their muscles would already have been their final size when they were untrained. Since the vast majority of research shows trained individuals have larger muscle fibers than untrained individuals and muscle fibers grow in response to exercise, this is an unlikely explanation.
- If it was a training effect and the bigger individuals were born with normal size muscle fibers, no muscle hypertrophy could have occurred at all. This is even more unlikely, as again the overwhelming majority of research shows muscle hypertrophy is a key driver of muscle growth. Muscle hyperplasia may play a small role, but it's outright unbelievable that it was the sole driver in these participants.

Since both explanations are incredulous, these data are most likely the result of an insufficient sample size of participants, an insufficient sample of muscle fibers, error in the estimation of average muscle fiber area or error in the estimation of total muscle cross-sectional area. The use of growth enhancing drugs could also have caused muscle hyperplasia, as many of the studies were done in competitive athletes and bodybuilders. However, [a more recent study from Maeo et al. \(2024\)](#) also found that trained individuals have more estimated muscle fibers than untrained individuals, as well as bigger fibers. These data are more credible, as they're based on a larger though still small sample size of 29 participants and they show increased fiber size in the trained individuals. Unfortunately, it's again not possible to say from this comparison across individuals if the trained individuals experienced hyperplasia from training or if there was some other confounding effect. For example, individuals with more muscle fibers may respond better to training and be more likely to engage in it and stick with it. People like to do what they're good at.

As the human data are inconclusive, scientists have sacrificed many a rodent and bird in the name of science to see if hyperplasia occurs. In animals it's still very hard to accurately count the total number of muscle fibers within a muscle. Many studies try to estimate the total from a certain area by extrapolation. However, muscle fiber size can vary across muscle depth. And the arrangement of the fibers (pennation angle) can vary along the muscle's length. Even when a complete fiber count is done, there's still great room for measurement error for both the extremely high count and the estimate of muscle (fiber) cross-sectional area. Accordingly, research findings on whether hyperplasia occurs in animals in response to 'strength training' are mixed. Strength training is in apostrophes here because scientists have found that rodents and birds are notoriously poorly motivated to perform barbell exercises and are thus often subjected to other protocols like weighted stretching to induce muscle growth, though

scientists have become inventive at forcing animals to lift weights: see the image below.

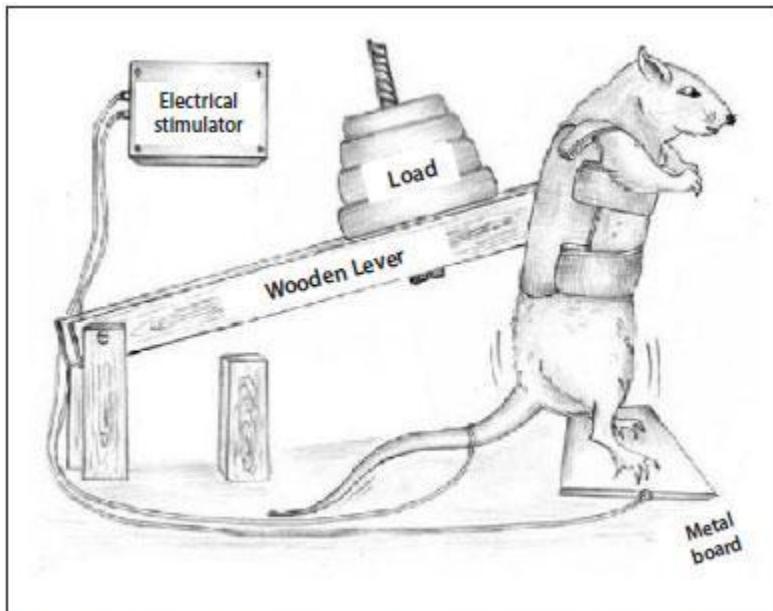


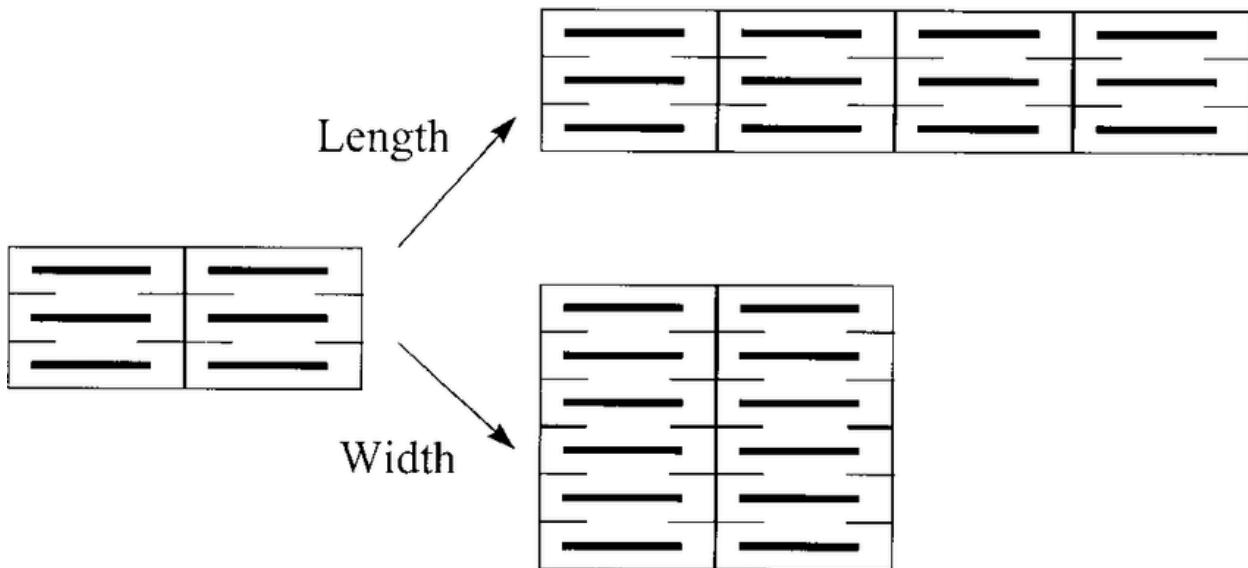
Figure 1. Strength training apparatus adapted from Tamaki et al. (1992).

All in all though, newer measurement methods to estimate or count muscle fibers tend to show no or very limited muscle fiber hyperplasia occurring along with total muscle growth in the animals.

In conclusion, there is no strong evidence that muscle hyperplasia occurs in adult humans, especially not without assistance of growth enhancing drugs. If it does, its contribution to total muscle growth is probably small and we don't know how we could target this with training. In practice, we should thus focus on maximizing muscle fiber hypertrophy. Any hyperplasia that may occur may come along for the ride naturally.

Muscle hypertrophy in series vs. in parallel

Muscles can grow by becoming longer or thicker. [Muscle length can increase by lengthening of the sarcomeres](#) and [probably via the addition of sarcomeres in series](#) [2]. Direct sarcomere addition in series has not been directly observed in humans though, only in animals. Since muscles are affixed to your bones on their ends, there is an obvious limit on how long a muscle can become while remaining functional, so muscle hypertrophy via length increases is limited. Most muscle growth occurs by an increase in sarcomeres in parallel, resulting in an increase in the muscle's thickness as more and denser myofibrils are packed together next to each other.

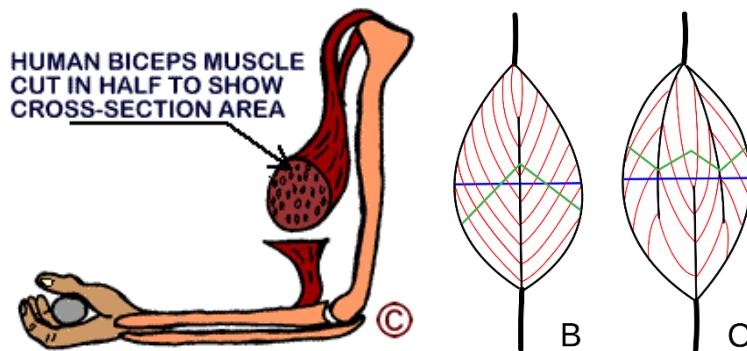


This diagram shows a cell beginning with 2 sarcomeric units of several thick and thin filaments. This cell can double in length by the addition of sarcomeres end-to-end in series, making the cell longer, or it can double in width by the addition of sarcomeres side-by-side in parallel. [Source](#)

An increase in the number of sarcomeres in parallel increases the anatomical cross-sectional area (ACSA) of the muscle: the area of the cross-section of a muscle along its longitudinal axis. If you want large peaks in your biceps, you want a large

ACSA. In research, however, muscle size is often measured by the physiological cross-sectional area (PCSA), which is more complex: it is the area of the cross-section perpendicular to the direction of the muscle fiber. [PCSA is better correlated with force production than ACSA](#), because it takes into account the leverage of the muscle fibers, though [not all research supports that PCSA is a better measure of strength than ACSA](#).

The difference between ACSA and PCSA is illustrated below.



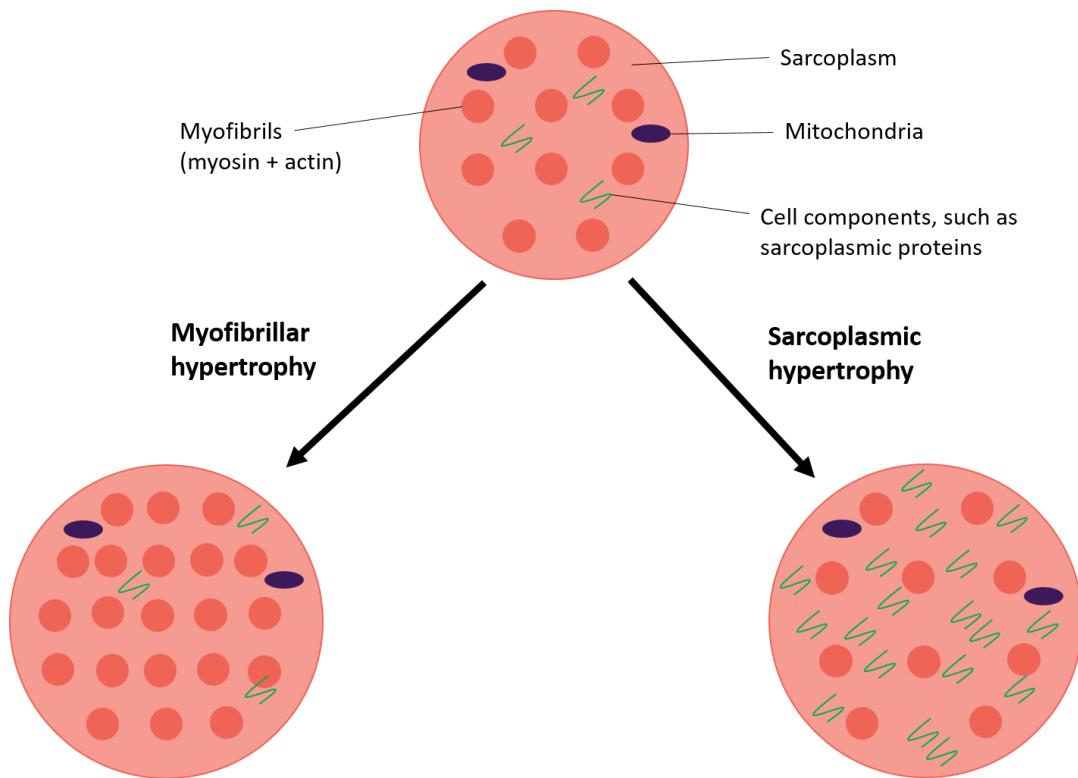
2 Measures of muscle size. Muscle anatomical cross-sectional area (blue lines) vs. physiological cross-sectional area (green lines).

Myofibrillar vs. sarcoplasmic hypertrophy

There are 2 types of muscle hypertrophy, depending on which components in the muscle fibers increase in size: myofibrillar and sarcoplasmic hypertrophy.

- Myofibrillar hypertrophy is the growth of the myofibrils (myosin and actin), the contractile components of the muscle tissue.
- Sarcoplasmic hypertrophy is the growth of any other component of the muscle fibers, the sarcoplasm and non-myofibrillar proteins.

Sarcoplasm is the cytoplasm of the muscle fiber. It's the fluid the myofibrils are located in. It contains many components, among them contractile components, other proteins, mitochondria, stored glycogen and fat particles.



There is controversy over whether sarcoplasmic hypertrophy occurs in humans, but this is in part a semantic discussion. There is no doubt in science that sarcoplasmic hypertrophy occurs. The only controversy in scientific circles is whether sarcoplasmic hypertrophy can outpace myofibrillar hypertrophy *in the long term*, leading to an increase in the *ratio* of sarcoplasm to myofibrils, as illustrated above.

Emphasis on the long term, as short-term sarcoplasmic hypertrophy is easily realized. A high carbohydrate diet will increase the amount of intramuscular glycogen, for example. Similarly, muscle damage can cause swelling with intramuscular water retention (edema). Both are technically acute sarcoplasmic hypertrophy.

But can the sarcoplasm's portion of the muscle fiber increase over the long run, more permanently?

Yes. Exercise, mostly aerobic endurance training but to a lesser extent also [strength training, increases glycogen storage capacity](#). It's not a major difference, but it's consistent. Strength trained individuals store more glycogen in their muscles than sedentary individuals and each gram of glycogen attracts ~3 grams of water into the muscle. [The peak glycogen concentration of human muscle is about 4 grams per 100 g](#), so this could entail up to 16% sarcoplasmic hypertrophy in theory.

Other osmotic (water attracting) components in the muscle fibers, such as proteins, probably increase in concentration as well, since [untrained individuals who start strength training have been found to experience an increase in intramuscular water concentration and a decrease in myofibrillary density](#). One small, controversial analysis by [Haun et al. \(2019\)](#) also found sarcoplasmic hypertrophy in trained lifters, but this may have been due to overtraining-related edema, because after a week without training after the mere 6-week training study, there was a strong trend for reduced

muscle fluid content and at that point muscle functional cross-sectional area was no longer significantly different from baseline. So we can't really say these subjects gained any muscle at all in the first place.

Cross-sectional comparison of untrained individuals, novices and elite lifters shows that the elite lifters had a lower myofibrillary density than the lesser trained lifters, but there was no difference between bodybuilders and powerlifters, suggesting that sarcoplasmic hypertrophy occurs with strength training regardless of how exactly you train. High rep training (with blood flow restriction) and low rep training also stimulate mitochondrial and myofibrillar protein synthesis similarly, suggesting they'll induce equal myofibrillar and sarcoplasmic growth. The only study suggesting it might be possible to induce sarcoplasmic hypertrophy with a specific training method is Vann et al. (2022): here a group training with a large number of very submaximal sets achieved greater accumulated sarcoplasmic protein synthesis and equal myofibrillar protein synthesis compared to a group training with fewer, heavier sets closer to failure. However, the supposed greater sarcoplasmic hypertrophy did not in fact translate into greater muscle size as measured by DXA and ultrasound, nor did it influence sarcoplasmic or contractile protein concentrations. As such, no true sarcoplasmic hypertrophy took place.

As you've seen in the course topic on nutrient timing in relation to the anabolic window, sarcoplasmic protein synthesis is normally considerably lower than myofibrillary protein synthesis, so sarcoplasmic hypertrophy is unlikely to amount to a major increase in muscle size even if we could somehow target it.

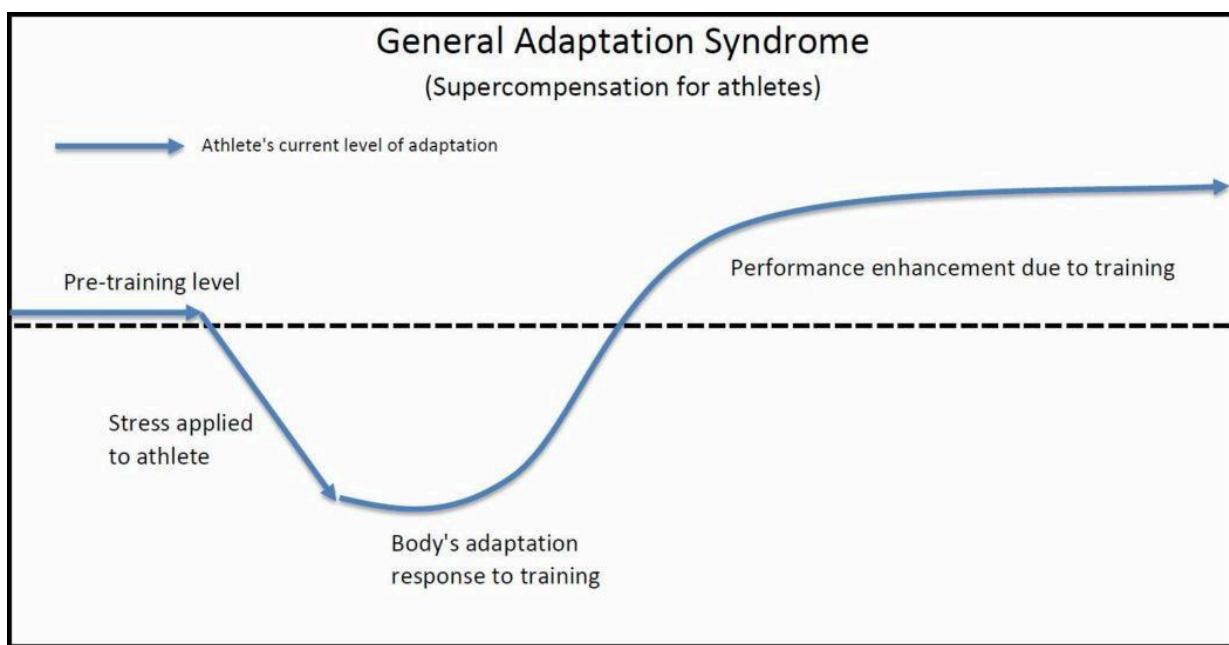
In sum, much like hyperplasia, sarcoplasmic hypertrophy is something you can largely forget about in practice. Sarcoplasmic hypertrophy probably occurs if you gain a large amount of muscle size, but its contribution to muscle growth tends to be small

compared to myofibrillar hypertrophy and it's unclear if and how you could target it in your training.

'Why' muscle grows

Sufficiently heavy muscular contractions cause enough tension to damage the muscle, in addition to metabolic stress resulting from rapid energy production, metabolic waste product formation and oxygen shortage. In evolutionary terms, this fatigue constitutes a form of stress that forms an incentive for the muscle to adapt to protect itself from this stress in the future.

This adaptation process is not unique to muscle tissue. Almost all bodily tissues adapt to stressors. Dr. Hans Selye created a framework to understand these processes called General Adaptation Syndrome (GAS). When subjected to stressors, there is an 'alarm' stage characterized by cortisol release and a state of high arousal. Afterwards, a 'resistance' phase is entered characterized by anti-catabolic and anabolic processes to bolster resistance against this type of stress. This adaptation is specific to bolster resistance against the stress imposed, a phenomenon known as Specific Adaptations to Imposed Demands (SAID) or simply the Specificity Principle.



Source: Just Fly Sports Performance

In the following lecture, we'll go into general features of the body's adaptation process after strength training.

➤ Lecture [optional]

[General Adaptation Syndrome \(GAS\)](#)

Functional training

Bodybuilding has a reputation for being the *least* functional sport there is. The sole aim of bodybuilding is improving your body composition without regard for actual performance. However, depending on your definition of ‘functional training’, bodybuilding may in fact be the most functional form of training there is.

Let’s define our terms here. Regardless of how you may specifically define it, ‘functional training’ according to many people refers to some measure of transferability of performance across activities (‘carry-over’). An activity is functional if it improves performance, defined below, in many other activities. So a leg extension is generally regarded as less functional than a squat, because leg extension strength doesn’t transfer as well to many other activities, whereas a strong squat makes you better at jumping, sprinting, etc.

Now let’s define performance.

In the broad sense, as the term is used in fitness, performance generally refers to the ability to produce force during a given movement (= F in physics). This is straightforward for Olympic Weightlifting, powerlifting and CrossFit, where someone’s score and force production are almost perfectly correlated. You move more weight, you get a better score.

However, it also applies in most sports: more force equals a faster sprint, a stronger punch, a higher jump, etc. Even an elderly person that has trouble standing up straight without shaking is often a matter of force. When we informally talk about ‘losing balance’, physically the problem is that the person cannot produce enough force during the desired movement. That’s essentially the same obstacle as not having enough strength and in both scenarios, strength training improves performance.

So functional training requires an activity to have a high degree of transferability of force production across various movements. What defines force production capacity?

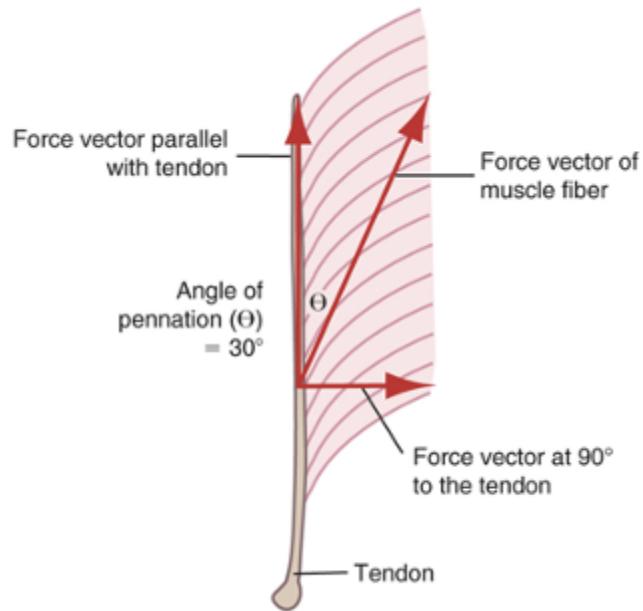
Strength vs. size

The body's ability to produce force during a given movement – strength – has 2 main components, morphological (referring to the shape of the muscle) and neurological (referring to the nervous system) [2]. We can further separate the morphological and neurological components into multiple subcomponents

Morphological components

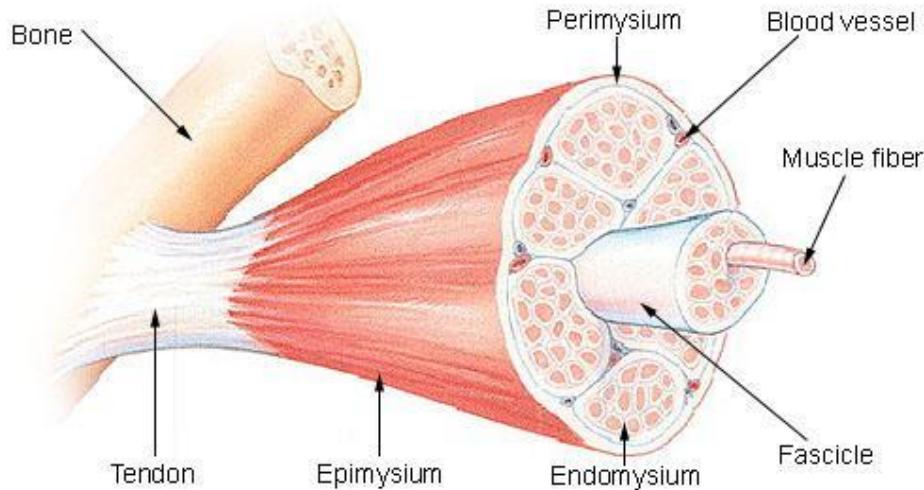
1. The first and foremost morphological component of practical interest is **muscle size**. Bigger muscle fibers produce more internal force due to having more contractile tissue. Muscle size is the engine of force production and it correlates with the other morphological components that contribute to greater strength (external force production).
2. A muscle's **internal moment arm** is a linear force multiplier of muscular force production. Greater moment arms linearly increase how our internal muscular force production is transmitted to torque around a joint. If your tendons insert twice as far away on the bone, doubling the internal moment arm of the muscle, the muscle can produce twice as much external force. So one person that can lift twice as much as another may be producing the same amount of muscular force but may lift twice as much due to having more favorable tendon insertions.
3. A muscle's **pennation angle** determines how good the leverage of its muscle fibers on the tendon are. Greater pennation angles offset the muscle fibers from the tendon's line of action along the length of the muscle, thereby reducing how much

of the muscle fibers' force production goes into the tendon. Pennate muscles cannot transmit all their force vertically into the tendon: some is 'lost' as horizontal force production.



The more pennate ('diagonal') a muscle fibers' are, the more of their force goes into horizontal force production rather than vertical force into the tendon.

4. **Lateral force transmission.** As your muscles become bigger and stronger, so does the extracellular matrix that packs the muscles (epimysium) and the muscle fascicles and the muscle fibers (perimysium) together. This fibrous connective tissue also runs through the muscle fibers (endomysium). The stronger this connective tissue is, the better it transmits the forces generated by your muscle fibers to your tendons, thereby increasing external force production.



5. **Tendon stiffness.** In contrast to the popular wisdom that stiffness is inherently bad, stiffer tendons transmit force from the muscle to the bone more effectively and thus make you stronger. Think of playing hockey with a rubber hockey stick: you can't transmit force effectively to the ball if your stick isn't hard (no pun intended).

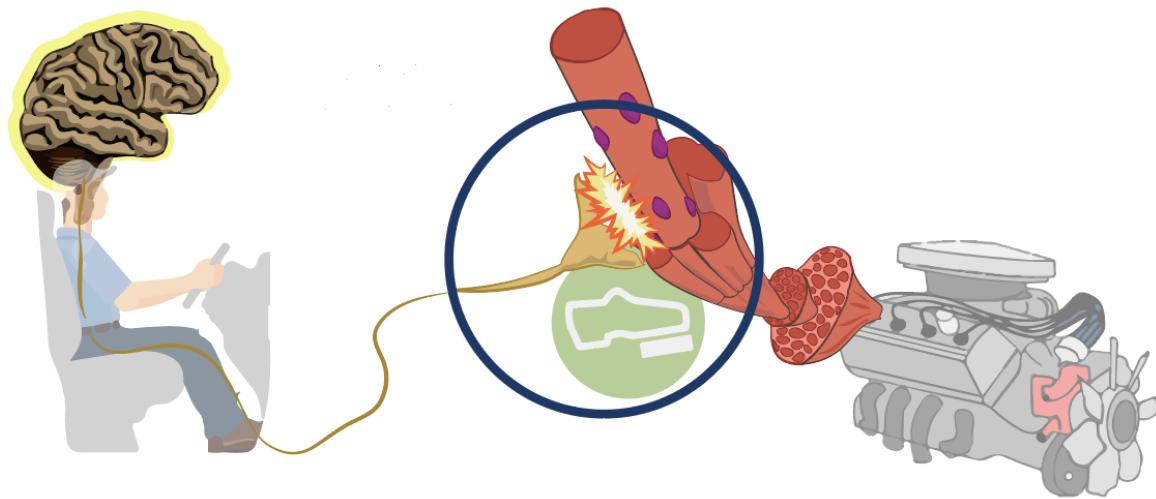
Neurological components

1. In untrained individuals, a key neural adaptation that makes you stronger is increased **voluntary muscle activation**. The increased muscle activation is primarily driven by higher **rate coding**, also called firing frequency: an increase in the firing frequency of motor units (groups of muscle fibers innervated by a motor neuron), [resulting in higher total muscle activity](#). The more often your nervous system can make your muscle fibers contract (in milliseconds), the more internal force the muscle produces. Our ability to increase voluntary activation is limited though, because [voluntary activation in untrained individuals generally already achieves over 90% of maximum force production](#) (as assessed by an electrically superimposed maximum twitch resulting in a so-called tetanic contraction).

2. **Intra- and inter-muscular coordination**, [notably a decrease in antagonist co-activation](#). During exercise, your motor cortex, the part of your brain that governs movement, figures out how to most efficiently perform the movement. As it learns which muscles to contract when, more of the muscular force production is converted to movement. For example, during a bench press, your brain figures out how to synchronize the activity of the triceps, the delts and the pecs (agonists and synergists), while generally minimizing the activity of antagonist muscles like the rear deltoids and the biceps with opposing functions. Antagonist muscles are activated for stability, but any force they produce directly counteracts the net external force output from the agonists and synergists, so their activity should be minimized for maximum strength.

Older research also suggested motor unit synchronization was a neural adaptation that contributed to increased force production, but [modern research suggests this was mostly measurement error](#) and [motor unit synchronization does not contribute considerably to higher force production](#).

You can think of muscle size as the body's engine of strength and your nervous system as the driver. Together, they determine your performance.

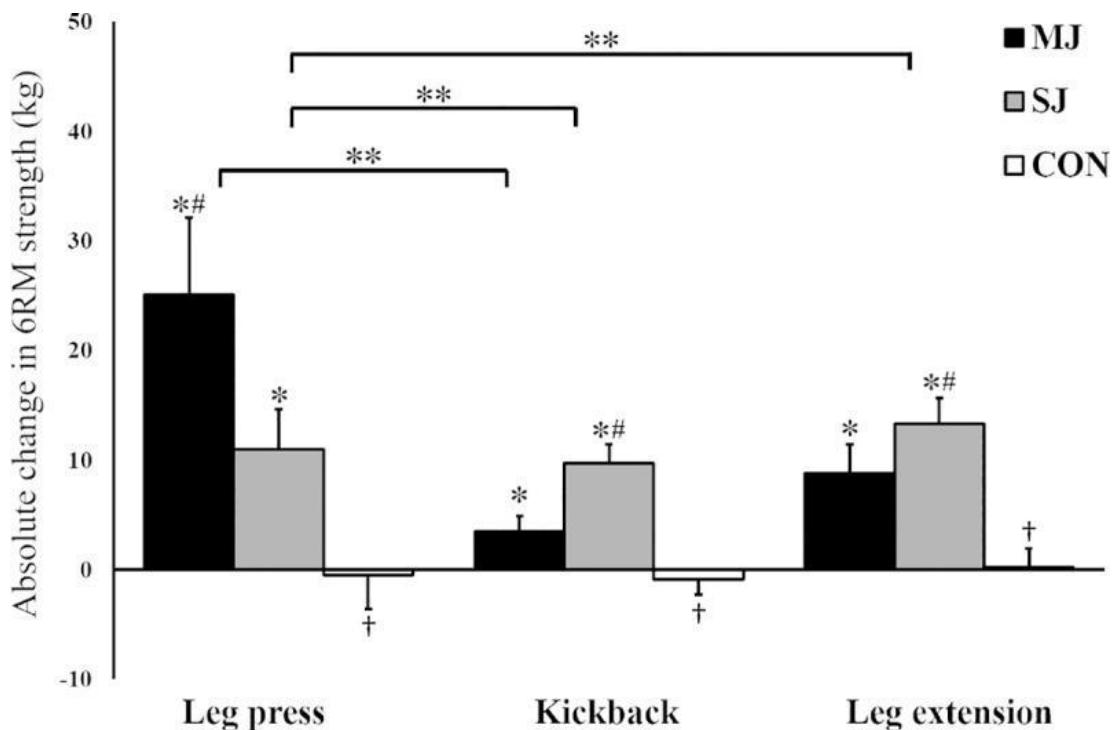


Muscle force production can be compared to the speed a car can drive at. Both the structure of the engine, particularly its size (referring to muscle mass) and the skill of the driver (referring to the efficiency of the nervous system) affect the car's top speed.

The contribution of neurological vs. morphological components to strength varies over time. During the first weeks of performing an exercise, most strength development is due to neural factors. Strength and size gains barely correlate in the short term. You can get a lot stronger on an exercise without gaining any muscle in the first weeks of performing a new exercise. Muscle growth occurs much more slowly than neurological adaptations, especially the learning of efficient exercise technique, but it continues for a very long time so that over the course of years, most strength development is attributable to muscle growth. Strength and size therefore correlate very strongly in well-trained lifters when doing exercises they're familiar with. The relationship is approximately linear, so you can expect to get 20% stronger from being 20% bigger. Getting 20% bigger takes a lot of time, which explains why in the short term, strength and size don't correlate well.

Performance carry-over

A highly important feature of neural adaptations is that they're generally highly specific. Your nervous system becomes better at performing the specific movement you're doing with limited transfer to other activities. For example, in the image below from [Stien et al. \(2020\)](#), you can see how strength increases in untrained women when they either do only leg presses (MJ group) or the exact same program of leg extensions and glute isolation kickbacks (SJ group): same muscles trained but with either 1 compound exercise or 2 isolation exercises. While compound exercises generally transfer better to isolation exercises than vice versa, most strength is developed specifically in the exercises you train.



Here are some more examples of how specific strength/performance adaptations can be.

- Intensity-specificity: [the optimal training methods for 1- to 10-meter sprints are significantly different from 30-meter sprints](#), though both are already very short distances.
- ROM-specificity: [partial range of motion strength training makes you stronger specifically in the part of motion you're training with only very limited transfer to the rest of the movement](#) [2]. That's why you see many guys in the gym that can quarter squat a ton, yet when they have to go ass-to-grass, they have to reduce the weight to less than half. [In some studies though, full ROM training is so much more effective that it tends to outshine partial ROM training even in the partial ROM strength](#) [2]. In general, full ROM training has higher carry-over to partial ROM strength than partial ROM training has to full ROM strength.
- Movement-specificity: there is very little relation between different measures of core strength, even though it's the same set of muscle groups (abs, back, etc.) performing a similar task (stabilizing the torso).

Unpublished research from *Osaka University of Health and Sport Sciences* found that there is no significant relation between trunk stability (i.e. planks) and trunk flexion (i.e. crunches).

Other unpublished research from Saeterbakken et al. in Norway found that the relations between core strength, core stability and core endurance were “non-existing to medium” (data below).

Table 1. The correlation between core strength, endurance and stability. * Indicates a significant correlation between these two parameters on a $p \leq 0.05$ level.

Tests	Strength abdom.	Strength back	Strength side	Endur. abdom.	Endur. back	Endur Side	Stability pref. leg	Stability non-pref. leg
Strength abdominal		0.51*	0.69*	0.02	-0.07	0.15	0.07	0.06
Strength back	0.51*		0.72*	0.04	-0.12	0.31*	-0.04	0.20
Strength side	0.69*	0.72*		-0.06	-0.03	0.44*	-0.01	0.22
Endurance abdominal	0.02	0.04	-0.06		0.07	0.01	0.08	0.03
Endurance back	-0.07	-0.12	-0.03	0.07		0.55*	0.16	0.02
Endurance side	0.15	0.31*	0.44*	0.01	0.55*		0.23	0.29*
Stability preferred leg	0.07	-0.04	-0.01	0.08	0.16	0.23		0.61*
Stability non-preferred leg	0.06	0.20	0.22	0.03	0.02	0.29*	0.61*	

Unsurprisingly then, the typical ‘functional training’ activities like core training and the Functional Movement Screen are “[not strong predictors of performance... Despite the emphasis fitness professionals have placed on functional movement and core training for increased performance, our results suggest otherwise.](#)” [2] It’s undoubtedly beneficial to train the core muscles to some degree, like it’s good to train every muscle involved in a sport. [A 2023 meta-analysis](#) found that core training programs in general improve physical performance that can be beneficial to sports, such as jumping, throwing/hitting power and balance. However, [a 2016 meta-analysis](#) found that the more you move away from fitness tests and the more you look at actual sports performance, the weaker the effects of core training become [2]. Core strength correlated only weakly to sports performance. The researchers concluded: “Our findings indicate that trunk muscle strength plays only a minor role for physical fitness and athletic performance in trained individuals. [While] core strength training appears to be an effective means to increase trunk muscle strength [...] it] was associated with only limited gains in physical fitness and athletic performance measures when compared with no or only regular training.” [A 2022 meta-analysis](#) also found that core training had

much stronger effects on core strength and fitness tests than sports performance, but there was still a small-to-medium effect on sports performance. There's no strong evidence that any specific type of core training, such as stability training, is superior, than training the core muscles like you'd train any other muscles. In fact, [a 2012 systematic review](#) concluded: "Targeted core stability training provides marginal benefits to athletic performance."

The lack of carry-over of many forms of exercise to other exercises may be hard to grasp for some people. Part of the reason for this is that our language is fundamentally flawed to understand biomechanics. [We talk about strength and power as traits, when they are in fact skills](#). Strictly speaking, a person cannot *be* strong or *be* powerful. A powerlifter isn't strong: a powerlifter *has* a strong bench press, deadlift and squat. Nor is an Olympic Weightlifter powerful: a weightlifter *has* a powerful Clean & Jerk and Snatch.

In contrast to core training, [there is convincing evidence that lower body strength training transfers positively to sprinting speed](#) and jumping performance. If we look at specific exercises to see which is best though, the data are highly conflicting. Although there are some data indicating full ROM free-weight exercises are relatively 'functional' in the sense of having relatively high carry-over to sports performance, most studies find that the type of exercise done does not significantly matter.

- [Wirth et al. \(2016\)](#) found that squat strength transfers over better to jumping performance than leg press strength, in line with the significantly greater similarity in movement pattern of squatting to jumping.
- [Weiss et al. \(2000\), Hartmann et al. \(2012\), Pallarés et al. \(2019\)](#) and [Bloomquist et al. \(2013\)](#) found that deeper squats improve jumping performance more than partial squats, yet [Rhea et al. \(2018\)](#) curiously found the opposite: quarter squats improved jumping performance and sprinting speed more than full

squats, with half squats performing in between. In [Pallarés et al. \(2019\)](#), the full squats also resulted in the largest improvements in sprinting and Wingate test performance.

- [Hartmann et al. \(2012\)](#) found that front and back squat training transferred over to jumping performance equally well.
- [Silvester & Bryce \(1981\)](#) found that box squats were no better than machines with accommodating resistance to improve jump height.
- [Rossi et al. \(2018\)](#) found that squats and leg presses didn't significantly differ in effectiveness to improve jump height and dynamic balance.
- [Schwarz et al. \(2019\)](#) found that the addition of neither machine-based nor regular squats significantly improved agility and sprinting performance over practicing the tested skills directly. Machine-based squats did improve jumping performance.
- [Aerenhouts & D'Hondt \(2020\)](#) found similar muscle growth, strength development and improvements in functional movement screen scores in untrained men after a 10-week full-body strength training program using either exclusively free weights (barbell squat, dumbbell presses/pulls), exclusively machines (leg press, machine presses/pulls) or starting with machines and transitioning midway to free weights.

In the same vein, when we look at studies that try to create ‘functional’ training programs, these ‘functional training’ type programs generally offer no advantage over conventional strength training.

- [Aragão-Santos et al. \(2019\)](#) found that a ‘functional training’ program was no better than a conventional strength training program to improve measures of physical functionality in the elderly.
- [Bennett et al. \(2019\)](#) found that a conventional strength training program was just as ‘functional’ as a progressive Functional Movement Screen (FMS)-based

program designed to address functional limitations and weaknesses in trained lifters. Strength and power increased similarly in both groups and even their composite MovementSCREEN score didn't improve more on the 'movement quality focused' program. All focusing on the FMS achieved was that it made them better at the FMS.

- [Ferraz et al. \(2018\)](#) found that 'functional training' was no more effective than bicycling or even exergaming (akin to Wii Fit) in elderly patients with Parkinson's disease to improve walking capacity. The 'functional training' intervention was specifically designed to improve balance and gait but evidently to no avail.
- In a similar study in older women, [de Resende-Neto et al. \(2019\)](#) found no significant benefits of a 'functional training' program compared to a traditional strength training program for the development of flexibility, cardiorespiratory fitness or strength. The 'functional training' program was similar to the traditional strength training program but with 'hipper' equipment like kettlebells and medicine balls and agility ladder work instead of walking cardio.
- [Balachandran et al. \(2016\)](#) compared cable-based exercises compared to similar exercises in machines with fixed movement patterns in elderly adults. Both programs were equally effective to develop 'functional strength', as measured by a series of tests called the Physical Performance Battery.
- [Pacheco et al. \(2013\)](#) again compared a traditional strength training program vs. a 'functional training' program, this time in elderly and middle-aged adults. Again there were no significant differences between groups in average improvements on the Functional Movement Screen and Y-Balance Test. In fact, in women the functional training groups improved less than the conventional training group.
- [Strand et al. \(2021\)](#) found no considerable difference between machine-based and 'functional' training in people with Parkinson's disease. Exchanging one weekly machine-based workout with a 'functional workout' did not affect overall functional capacity, balance or quality of life.

- [Gavanda et al. \(2022\)](#) found no significant differences between conventional strength training and a high-intensity ‘functional training’ program in adolescents for performance in jumping, sprinting, squatting and a Yoyo test.
- [Hernández-Belmonte et al. \(2023\)](#) found no significant differences in functional outcomes in strength-trained men performing a full-body workout program consisting entirely of either barbell or machine exercises that mimicked the barbell’s movement pattern. The included exercises were barbell squats vs. hack squats, barbell vs. machine chest presses, barbell seal rows vs. machine rows and seated barbell overhead presses vs. machine overhead presses. After 8 weeks, there were no significant differences between the groups' gains in muscle architecture, standing balance capacity, upper and lower body anaerobic power, sprint speed, jumping height or change of direction ability.

All in all, by far the most important training you can do to get better at any sport or movement is to practice the required skills directly. [General strength training, especially via free-weight compound exercises over a full range of motion, generally further enhances performance](#), but the specific type of training and exercise don’t seem to matter much.

Power, explosiveness and impulse

When talking about sports performance, you'll often come across the term 'power' as a measure of explosiveness. This term is often misused. [When people informally talk about 'power', that is often improper terminology for what is actually impulse.](#)

Power is formally defined as the rate of work, or work divided by time, and measured in joules per second AKA Watts. Power is thus a measure of how rapidly you can generate energy, which is related to the ability to produce momentum (impulse) and acceleration, but it's not exactly the same. Impulse is a better measure of 'explosiveness': it's how much momentum and resultant acceleration you can put in an object. If you can produce high impulse, you can make objects and your body move very fast very suddenly. Formally, it's a vector defined as the integral of force over time in a direction, measured in Newton seconds (Ns). Another useful measure of explosiveness is rate of force development (RFD), which is literally how fast you can develop force over time.

Effect of body composition on performance

Since the nervous system is highly movement specific in its function, that leaves muscle size as the main component of functional capacity. Muscle size is the only true trait that increases force production capacity without any limitation of movement specificity. If you make a muscle bigger, it will increase its ability to generate force during every movement that muscle is involved in.

A low body fat percentage is also strongly linked to performance of many movements. Given the same muscle mass, the lower your fat mass and thereby your total

bodyweight, the higher your *relative* strength. This is particularly important during weightbearing activities, which basically includes all ground sports.

Many studies show that muscle size and a low body fat percentage (= bodybuilding) determine performance in a wide range of activities (= functionality). These include:

- [Elite volleyball](#)
- [Swimming](#)
- [Competitive sprinting](#) (just look at top level sprinters)
- [Elite surfing](#)
- [Major League Baseball](#). Along with [American Football](#), these are one of the few sports that caught on to the performance benefits of being highly muscular early on. Between 1970 and 2010, the average BMI of baseball players grew by about 3 points. Linemen have increased in weight by over 50% between 1950 and 2010. Moreover, there is a clear upward hierarchy in size from the lower to the higher divisions of the sports. More muscular players perform better.
- [Basketball](#)
- [Judo](#)
- [Soccer](#)
- [Firefighting](#)
- [Track and field throwing](#)
- [Australian football](#)
- [Rugby](#)

Best of all, we have powerlifting and Olympic weightlifting, the sports that supposedly stand in stark contrast to bodybuilding because they train for performance instead of body composition. Yet [the reality is that more muscular powerlifters consistently outperform their less muscular competition \[2, 3, 4, 5, 6\]](#). [Correlations of 0.86 to 0.95 have been reported between fat-free mass and performance in the powerlifts](#). That's

close to a 100% correlation, which would mean fat-free mass and strength are essentially the very same thing! If we put powerlifters in a body composition scanner, their rankings would be almost identical to their powerlifting ranks. The correlation is generally strongest for the squat, followed by the deadlift and lastly the bench press. Indeed, [we can very accurately predict powerlifters' rankings in an upcoming competition simply based on their body composition. Changes in strength in powerlifters also correlate with changes in their lean body mass.](#)

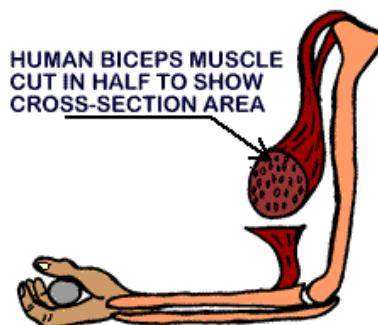
Other studies have researched what distinguishes stronger and weaker powerlifters. Anthropometric variance – differences in individual body structures – doesn't explain a lot of the variance in performance, especially at the elite level. [The greatest difference between stronger and weaker lifters is simply that the stronger ones have more lean body mass \[2, 3, 4, 5\] with few significant correlations between someone's body structure and powerlifting performance \[2, 3\].](#) As [Keogh et al. \(2009\)](#) concluded: "In terms of the segment lengths and bone breadths, the only significant difference was that stronger lifters had a significantly shorter lower leg than weaker lifters. Because the majority of the significant differences were for muscle mass and muscular girths, it would appear likely that these differences contributed to the stronger lifters' superior performance. Powerlifters may therefore need to devote some of their training to the development of greater levels of muscular hypertrophy if they wish to continue to improve their performance."

[Reya et al. \(2021\)](#) found that even exercise technique is trivial compared to the importance of muscle mass in predicting bench press performance in elite powerlifters: "structural and neuromuscular factors need to be considered in predicting and possibly improving bench press performance, while small variations in the exercise technique seem to play little or no role. Lean body mass, brachial index, and isokinetic concentric shoulder flexion torque were particularly strong predictors of 1RM BP. Muscle mass

seems to play a role in BP performance, even when the results are corrected for body mass via the Wilks coefficient.”

These findings are not specific to powerlifters. [Strength and size correlate strongly in most very well-trained individuals. In Olympic weightlifters too, there is an extremely tight relation between body mass and performance.](#) In sports in general, as competitive pressure, the celebrity status of athletes and the financial rewards increase, [athletes in many sports are becoming ever more muscular.](#)

Interestingly, there is good reason to believe the current research underestimates the correlation between muscle size and strength due to a methodological problem. A good measure of muscle size is its cross-sectional area (CSA) at its largest point: the literal thickness of a muscle, illustrated below.



Anatomical/morphological cross-sectional area. An even better measure is physiological CSA (PCSA), because this takes into account the directionality of the muscle fibers (pennation angle) and measures the cross-section perpendicular to their longitudinal axis rather than assuming all muscle fibers run straight from top to bottom.

Most research measures CSA at rest and then correlates this with a measure of strength, often maximal voluntary isometric contraction (MVIC: contracting a muscle as hard as you can without moving it) to make sure the muscle size and strength

measurements are performed with the muscle in the same position. However, testing this correlation at rest underestimates the relation between strength and size, because [the correlation between CSA and MVIC is stronger during intense contractions than at rest \[2\]](#). In short, methodological limitations mean that the relation between muscle size and strength we see in the lab is probably an underestimation of the actual relation during strength training.

So where most athletes are specialized in specific movements and *have* certain skills, bodybuilders *are* paradoxically in a sense the most functional because their size makes them good jacks-of-all-trades.

Key take-home messages

- Muscle growth is part of the adaptation process your body goes through after muscle tissue has undergone significant stress, starting with recovery (repairing damaged muscle tissue) and ending with supercompensation (building new muscle tissue). As such, adaptation is not something to avoid. It is *the* very goal of training.
- Muscle growth is primarily a local/regional/intrinsic process. Muscles grow independently of each other based on factors occurring within that very muscle.
- The primary type of stress inducing muscle growth is mechanical tension on the muscle fibers. Muscle damage, metabolic stress and exercise induced anabolic hormone elevations have been postulated to play a role as well, but their supporting evidence is much less compelling.
- Strength is influenced by muscle size but also by several other factors, chiefly:
 - biomechanical factors, such as the muscle's pennation angle and the positions of its tendons that influence the muscle's leverage;
 - neural factors, such as inter- and intramuscular coordination of muscle (fibers) and neural drive (motor unit firing frequency and motor unit recruitment) that optimize how the tension produced by your muscle fibers is translated into movement;
 - metabolic factors, such as calcium metabolism.In practice, however, within an individual, total muscle mass and strength training performance are highly correlated.