

Download this page in Adobe Acrobat Format: click on the acrobat logo on the right



Download the definitions page in Adobe Acrobat Format: click on the acrobat logo on the right



Version française

if frames do not appear [click here](#)



[Introduction](#)

[Simplified muscle structure](#)

[Sarcomere structure](#)

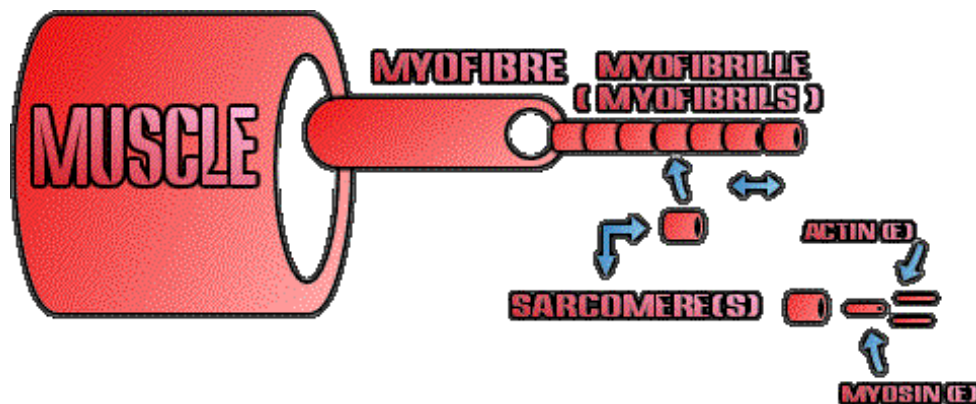
[Summary of events](#)

INTRODUCTION [back](#)

Muscle contraction mechanics are totally different from what we usually think it to be. Understanding this mechanism makes a better type of training possible as well as teaching how to avoid many errors (such as trying to "shorten" muscles, which is anatomically impossible).

Before plunging into the inner mechanisms at work in a muscle, one has to understand its structure first. A muscle is made up of a set of muscle fibres ([myofibres](#)) held together by a girdle ([conjunctive tissue](#)). This muscle fibres exist by the thousand (we have more than a million in pectoral muscles), and delimit a walled-in environment thanks to a girdle which is proper to them. Each [myofibre](#) is itself made up of smaller fibres ([myofibrils](#)). Each [myofibrille](#) is in turn made of a series of longitudinal contraction units ([sarcomeres](#)) separated by elastic zones and themselves sheltering filaments. These filaments are made up of "thick" ([myosin](#) molecule) filaments surrounded by "thin" ([actin](#) molecules) filaments. It is by connecting to these thin filaments and drawing them inwards so that the thick ones shorten the whole contraction unit ([sarcomere](#)). Because this phenomenon occurs for every contraction unit, the whole muscle contracts.

MUSCLE STRUCTURE OVERVIEW [back](#)

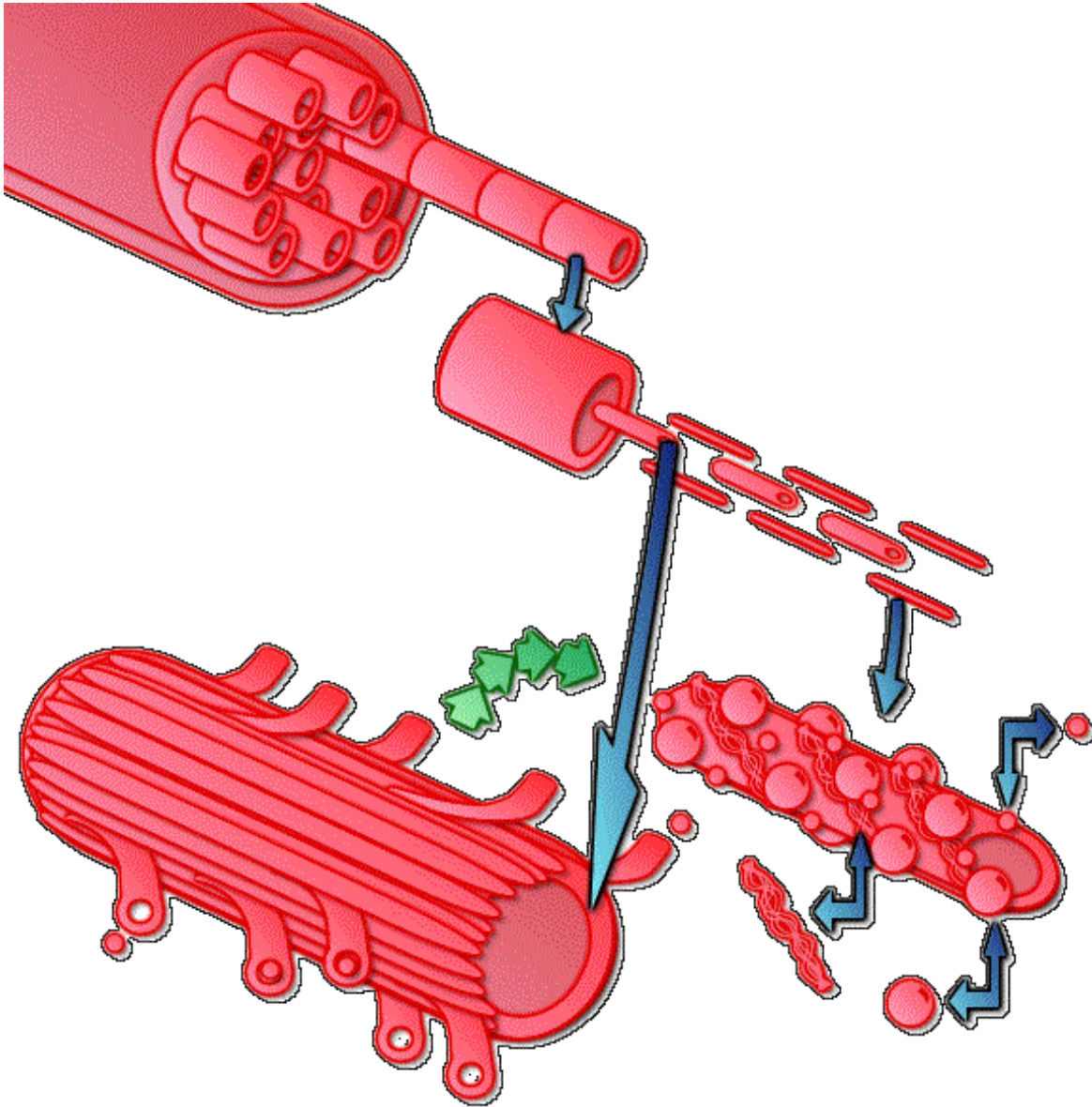


This simplified drawing above shows the inner muscle organisation: what we will be mainly dwelling upon for the rest of this long (... and passionating) plunge into muscle contraction is the unit called [sarcomere](#), because this is the level at which occur all the events leading to our own movements. [Sarcomere](#) has a structure akin to that of a telescopic aerial, at least in the main lines : its own elements ([actin-myosin](#) filament-like molecules) are going to get closer to one another in an inward longitudinal gliding motion. The "elements" themselves won't see their length being altered, for they will only glide along one another, pretty much like trains which get closer to one another, while still remaining on their respective rails.

SARCOMERE STRUCTURE [back](#)

The following drawing derives from the first one, in a more detailed way: we find the starting muscle fibre again (myofibre), and the now getting more famous [sarcomeres](#). One of them is isolated apart to show what it is made of: [myosin](#) and [actin](#) molecules. [Myosin](#) is the main inner molecule, also referred to as the "thick" filament, and [actin](#) is a group of molecules set together to form a thin filament, which are all both laid forward and also rearward from [myosin](#). It is the inward gliding of [actin](#) molecules threads, drawn inward to the center of the contractile unit ([sarcomere](#)) by the [myosin](#) molecule, which shortens the [sarcomere](#), hence shortening the whole [myofibril](#) (to which they belong), which collectively shorten the [myofibre](#), eventually shortening the whole muscle.

(click on the drawing to get definitions)



One can see that the [myosin](#) molecule is much bigger than the [actin](#) molecules filament surrounding them. We will see below that the [actin](#) filament is in fact made of more than one molecule (thousands in fact) of globular shape (it will be shown below - the drawing above is not "deep" enough yet). The [myosin](#) molecule also ends with outgrowths. These are referred to as "[crossbridging sites](#)" which will literally stretch forward to bind to [actin](#) molecules of the "thin filament", and draw it inward to make it glide along the sides toward the centre of the [myosin](#). We can also see on the drawing that the thin [actin](#) filament is itself surrounded by an even smaller molecule coiled around it: [tropomyosin](#). It is a molecule coiled around the actin filament (like the blades of a screw) and which blocks by its very position the particular area on which [myosin](#) will try to bind. That molecule's change of position will make muscle contraction possible, because [myosin](#) will then eventually be able to bind to [the filament of actin molecules](#). This change of position is due to another molecule "stuck" to both [actin](#) and [tropomyosin](#): it is [troponin](#). This one reacts in presence of [calcium](#) (we will see how below) and moves, thus dragging along [tropomyosin](#) out of

the [actin binding site](#) it previously blocked (inhibited).

Muscle functioning general principle is thus the following:

Describing muscle structure by gradually going deeper.

- Let us imagine a huge mains cable: it is made of thousand of tiny cables :this is the muscle.
- This is a telescopic cable: it is able to either shorten or widen.
- The inner little cables are all laid in the same direction as its neighbors:these are muscular fibres ([myofibre](#)).
- Each inner cable is itself made of dozens of tiny and much smaller cables ([myofibrils](#)).
- Each one of these tiny cables is then longitudinally divided into "logs", set in rows: these are [sarcomeres](#).
- Each [sarcomere](#) is made of two types of molecules filaments: large ones ([myosin](#)) / smaller ones ([actin](#)).
- At the end of each thick filament are six thin ones, all bound in the same longitudinal direction.

Describing muscle contraction from the most hidden to the most obvious levels.

- The thick filament (made of [myosin](#) molecules) "binds" to the thin ones (made of [actin](#) molecules) and draw them inward along its sides.
- By way of consequence, the whole "log" ([sarcomeres](#))shrinks.
- If each contraction unit (log) shortens, then the whole little cable does ([myofibrils](#)).
- If all little cables shorten simultaneously, then the bigger one ([myofibre](#)) does the same.
- If all big cables do the same, then the whole muscle contracts.
- ... All this because, at the lowest level, a big molecule bound to a filament of smaller molecules and drew it inward.

MUSCLE STRUCTURE [back](#)

To sum it all, the whole contractile system structure can be boiled down to the following "organisation chart": the color code legend is detailed on the right:

IMPORTANT ELEMENTS
INTERNAL RELATIONSHIPS
MEMBRANES AND RELATED ELEMENTS
NEURO-MUSCULAR LEVEL

(Click on the names below to get definitions on the right frame)



So much for the overall principles, now, let's get down to details: Let us consider the order of the phenomena which makes up muscle contraction. Each of these is explained in details in the sections below.

01 Reception of brain stimulation: Everything begins with an order coming from the brain. This is dispatched via a particular type of neuron ([motor neuron](#)) the end parts of which ([axons](#)) are in contact with the muscle fibre itself ([myofibre](#)). The [motor neuron](#) connects to many [myofibrils](#), and transmits a substance (a neurotransmitter called [ACh](#)) which is in charge of producing an electric impulse ([action potential](#)) which is going to reach the protective mantles of [myofibrils](#) contracting units ([sarcomeres](#)).

02 The role played by calcium: The mantle of [sarcomeres](#) ([sarcoplasmic reticulum](#)) is fitted with stores of [calcium](#) (terminal tanks) which will release their contents into the void within [myofibres](#) (referred to as [cytosol](#)) thanks to an [action potential](#). [Cytosol](#) operates as a combustion chamber. [Calcium](#) thus released will indirectly make muscle contraction possible, which is inhibited by default. [Calcium](#) will bind to [troponin](#) and by doing so will alter its position on [actin](#). Because [troponin](#) is connected to [tropomyosin](#) as well, the latter will be dragged along and this will uninhibit the [actin binding site](#) on which it rested, and [myosin](#) will try to connect. The door is thus unlocked.

03 The locking system: Thin filaments are made of globular molecules ([actin](#)) arranged in pairs side by side, and these pairs are associate in a linear pattern making a filament (see drawing below). Each molecule has its own binding site. The problem is that [actin's binding sites](#) are blocked by the presence of another molecule, even smaller, coiled around the filament, mostly right over them. This coiled molecule ([tropomyosin](#)) operates as a lock's latch, or the plug-blocker that parents having

toddlers know well. muscle contraction is impossible as long as these binding sites on [actin](#) have not been unblocked (uninhibited), as long as [tropomyosin](#) is still "above" them.



The locking operation: For the [tropomyosin](#) to move and thus enables [myosin](#) molecules to operate, it must be drawn away by another molecule. It is at that time that the third molecule of thin filaments ([troponin](#)) gets onto the stage. It is the one on which [calcium](#) ions (which have just been released) will bind. When [calcium](#) ions binds on it, they alter its general shape (one refers to this phenomenon as a "conformation alteration"), which triggers a necessary re-positioning of [troponin](#) on the surface of the thin filament. This [troponin](#) being both connected to [actin](#) (the door) and to [tropomyosin](#) (the latch) it thus operates as a lock which, by moving, opens the door.



Contraction thanks to the inward gliding of molecule filaments: [Myosin](#) is a complex molecule filament endowed with "arms" ([crossbridging sites](#)) which connect to the [actin binding site](#). When connection occurs, [myosin](#) works as someone who would stretch the arm to draw an object back towards him. Let us imagine a timberman having felled a little tree who grabs a little branch to draw it towards him. When the [myosin](#)'s "arm" is completely bent rearward, it unhooks and fetches another binding site forward, gets connected to it again, and draws it rearward again, thus having the whole filament of molecules gradually glide inward along its sides, at [sarcomere](#) level. When the whole process has reached its maximum length reduction, muscle contraction is considered maximum.

This phenomenon occurs in each "log-shaped" contractile unit ([sarcomere](#)) making up the whole [myofibril](#), and when all these see their length reduced, they in turn shorten the [myofibril](#) to which they belong, and so each muscle fibre, therefore the muscle involved as a whole. Contraction ends voluntarily when contraction orders cease to be sent and when [calcium](#) is pumped out of the [cytosol](#) back into the [sarcoplasmic reticulum](#) "terminal tanks", or unvoluntarily in case of muscle exhaustion (which will be discussed below).

The general principle is thus that of a fake "contraction" (because no molecule alters its size) thanks to a closing in of molecules which glide along one another. This phenomenon occurs on a very small scale but thousands of times simultaneously everywhere in the muscle structure. It occurs in every independent part of little muscle fibres, which collectively shorten the main muscle fibre to which they belong. When little fibres draw in together, the whole muscle fibre does so (at a larger scale), and so does the muscle as a whole.



[Sequence of events](#)

[Contraction intensity](#)

SEQUENCE OF EVENTS [back](#)

What is called "nerve impulse" is responsible for the release of [calcium](#) into [cytosol](#), and these [calcium](#) ions will bind to [troponin](#), thus enabling muscle contraction cycles.



Creation of nerve impulse. This occurs within the type of neuron in charge of movements ([motor neuron](#)). When the muscle is in a relaxed state, there is an electric potential difference between the external and internal sides of the [motor neuron](#) membrane. This potential difference is due to the presence of an ion current going through the neuron's membrane, and also to the fact that there is also a difference in the number of ions between the internal zone of the neuron and the rest of the environment. Nerve impulse is created when there is a movement from ions passing through the neuron's membrane: sodium gets into the cell, which alters the whole membrane's potential (this is a [depolarisation](#) : shifting from + to -). Then, potassium gets out of the cell, which gives it a positive potential back again. Hence, it is an in-and-out between sodium and potassium ions which, for a very short time, creates an electric polarity change in the whole neuron (from [depolarisation](#) to [polarisation](#) back again). It is such a change that is referred to as "[action potential](#)", or nerve impulse.



Spreading out of action potential. This is the role of the nerve fibres (called "[axon](#)") which can be more than a meter long. The action potential is created and spreads towards the end of the [motor neuron](#). After a series of [depolarisation-re-polarisation](#) at the very core of [axone](#), the [action potential](#) reaches the tip of the [axon](#) without loss of energy because it does not really "move", but is transmitted by a series of successive regeneration (linear duplication).



Release of neurotransmitter on the motor end plate. The successive [action potentials](#) have reached the tip of the

"electric cable" ([axon](#)). For them to reach and spread into muscles, there must be a connecting link between the tip of the [axone](#) and the [motor end plate](#). This relay in charge of transmitting nerve impulses is called a "synapse". It is the connection link between the [axone](#) and its objective. It is at that level that the neurotransmitter ([ACh](#)) will be created, thanks to an [action potential](#). Because an [action potential](#) can not directly go from the [axone](#) to the muscle, there needs to be a "shuttle", and it is the role of the neurotransmitter. It will then spread over the [myofibre motor end plate](#).



The action potential reaches the calcium tanks of sarcomeres. The neuro-transmitter ([ACh](#)) reaches the [motor end plate](#) receptors: the membrane is thus depolarised and a new action potential is created again - hence ensuring the continuity of potential actions. It spreads over the muscle fibre and down into the transverse tubules located at the tip of [sarcomeres](#) and going between its calcium tanks. The action potential thus reaches, via transverse tubules, the calcium store sites of [sarcomeres](#) which then release [calcium ions](#). [Calcium](#) will then flow into the [cytosol](#), and the contraction cycle will begin ([calcium ions](#) will bind onto [troponin](#) molecules, which will change their position on the thin filament to which they are connected, thus drawing along [tropomyosin](#) molecules, which now no longer inhibit the [actin's binding site](#) so that [myosin](#) can in turn connect to it and begin the shortening of [sarcomeres](#)).

What happens after the release of [calcium](#) will be further investigated below ([muscle contraction chapter](#)).

CONTRACTION INTENSITY [back](#)

When an [action potential](#) reaches a [sarcomere](#) contraction does not happen instantaneously; there is a "time lag". This lag is due to the momentary increase in power within muscles before its effort reaches the level of the weight to be lifted: this lag period is thus longer for a heavier weight than for a lighter one, which is quite obvious. This corresponds to the moment when one is producing a gradual effort yet the weight is not moving because the effort is not powerful enough. In that case [crossbridging sites](#) develop power but there is no [sarcomeres](#) shortening yet because the weight has not yet been "defeated". We are still at the level of isometric contraction. Then, the amount of power created by [crossbridging sites](#) goes beyond weight resistance and molecular filaments shortening can begin. Meanwhile, [action potentials](#) have never ceased being created and added one to the other.

There is also a huge difference in terms of duration between an [action potential](#) (1-2ms) and that of a muscle contraction (10-100ms): this means that many [action potentials](#) combine during a single muscle contraction. This adding-up of [action potentials](#) will increase muscle tension: it is referred to as "Sommatation". A contraction thus held on by a series of [action potentials](#) is called "tetanus". There exist two types of tetanus : the first one is called "merged tetanus" (when the frequency of [action potentials](#) is very high - and muscle contraction maximum), and non-merged tetanus (when there are short slackenings during contraction). Merged tetanus contraction develops more power because the succession of [action potentials](#) brings about successive and cumulative releases of [calcium ions](#), thus overcoming the sheer fact that [calcium](#) is generally pumped back faster than the time required by [crossbridging sites](#) to bind to [actin](#). With successive [action potentials](#) all [crossbridging sites](#) get enough time to bind to [actin](#) and the effort is thus bigger.



[Myosin-Actin binding](#)

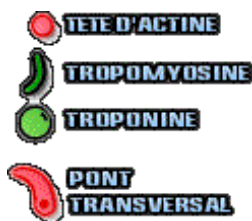
[Close up on Actin](#)

[Chemical elements at work](#)

Everything occurs at the level of the plasma membrane, which is sensible to [action potentials](#), and is in charge of transmitting them. This membrane shrouds [myofibrils](#) (which collectively make up [myofibres](#)). The electric activity of the plasma membrane leads to the release of [calcium](#) into the [cytosol](#) (let us remember that the [cytosol](#) is the "free" space between each [myofibril](#) and the plasma membrane). As long as [calcium](#) is mainly located into the [cytosol](#) muscle activity will occur. [Calcium](#) will literally fill in the [cytosol](#) void, coming from the sarcoplasmic reticulum shrouding each [myofibril](#). This mantle is fitted with tanks storing [calcium](#). The effort will end when the sarcoplasmic reticulum will pump [calcium](#) back. Yet, muscle contraction will continue for a short while after the "stop" signal (back-pumping of [calcium](#)) was given, because pumping [calcium](#) back takes a little longer than releasing it. The [sarcoplasmic reticulum](#) pump consumes [ATP](#).

ACTIN - MYOSIN BINDING [back](#)

(click on drawings to get definitions)



Mechanically speaking, two molecular elements (green) and two molecular elements (red) are at work in the shortening of [sarcomere](#): the [actin](#) molecule is endowed with a binding site for the [myosin](#) molecules: this binding site is called a "head", and the part of [myosin](#) which will bind on it is called the [crossbridging site](#). [Tropomyosin](#) is close to the [actin binding site](#) while muscles are relaxed, and it is the binding of [calcium ions](#) on [troponin](#) which will help it move, in turn dragging [tropomyosin](#) away, and thus uninhibit the binding site and make muscle contraction possible.

1) ACTIN BINDING SITES INHIBITED BY TROPOMYOSIN



In [sarcomeres](#), [myosin](#) has a central position, and is surrounded at both ends by thin molecule filaments, made of [actin](#) molecules set in pairs and connected one pair behind the other to form a sinusoidal chain. Each [myosin](#) molecule is surrounded at both ends by six thin [actin](#) filaments (only one is shown here for clarity reasons). [Myosin's](#) [crossbridging sites](#) will bind to each [actin's](#) [binding site](#) (only 3 such sites are shown here instead of thousands) to then draw [actin](#) rearward, thus shortening [sarcomeres](#) (and muscle by way of consequence).

At that level, contraction is impossible because a [tropomyosin](#) molecule (coiled around a thin filament) inhibits the access to [actin binding sites](#). [Calcium](#) will have to alter the shape of this [troponin](#) molecule for it to change its position and by doing so drag [tropomyosin](#) along with it, thus uninhibiting the [actin binding site](#).

2) ACTIN BINDING SITES FREED BY TROPONIN DRAGGING



[Calcium](#) (not yet shown here - look below) has just been released in the vicinity and connected itself to each [troponin](#) molecule. These got their general shape altered by this connection and have thus re-positioned themselves onto the thin actin molecules filament, dragging [tropomyosin](#) molecules along, which uninhibits [actin binding sites](#).

3) CROSSBRIDGING OF MYOSIN ON ACTIN BINDING SITE

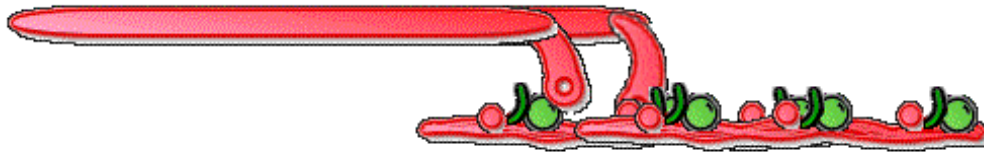


Thanks to the energy provided by [ATP hydrolysis](#) (not shown here) [myosin's crossbridging sites](#) will move forward to connect to the [actin's binding sites](#). When connection occurs contraction can begin. One single connection is shown here to make the drawing more intelligible, but there are thousands of these in fact.

4) MYOSIN GLIDING FORWARD BY DRAGGING ACTIN REARWARD



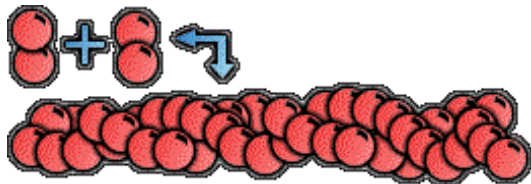
[Myosin](#) connects to the [actin's binding site](#) and makes it glide inward in its direction, thus shortening the [sarcomere](#) and leading to muscle contraction (at a larger scale because this phenomenon occurs for thousands of [crossbridging sites](#) into thousands of [sarcomeres](#)). Another [ATP](#) molecule will bind onto the [crossbridging site](#) and thus unhook it for another cycle to begin.



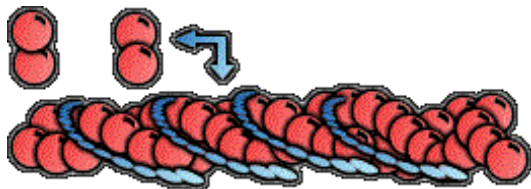
There is a superb animation showing all this in a much better way than mine, and comes directly from the French national scientific institute ([here](#)).

ACTIN CLOSE - UP [back](#)

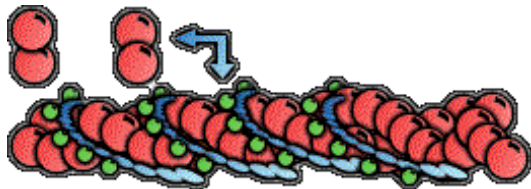
Let's have an even closer look and let's cut the thin filament to see how the lock opens: the thin filament, as we saw above, is made up of three different molecule types: a big one ([actin](#)), and two others connected to it ([troponin](#)) and ([tropomyosin](#)).



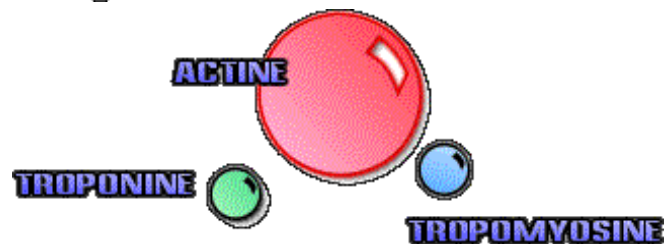
This drawing shows the minimum unit of a thin filament. 2 globular [actin](#) molecules side by side, connected to other duos of the same type on both sides to eventually form a filament of [actin](#) molecules.



Coiled around this filament is another molecule (of linear shape) called [tropomyosin](#) which is in charge of inhibiting and uninhibiting [actin binding sites](#) and thus prevent contraction. On this drawing, the [actin binding sites](#) are shown as a tiny black surface on each [actin](#) molecule, and one clearly sees that [tropomyosin](#) does not "block" them all.

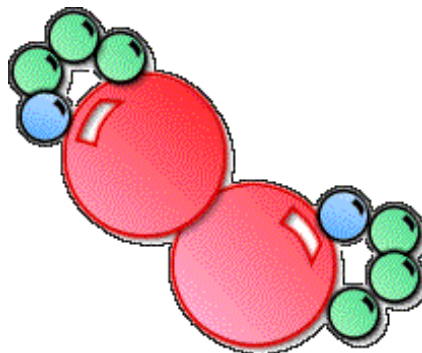


Now we have the complete set, with [troponin](#) (tiny green globule) acting as a connecting link between [actin](#) and [tropomyosin](#), and operating as a latch the movement of which will drag away the lock (i. e. [tropomyosin](#) inhibiting any connection to the actin filament by blocking the [actin binding sites](#)).



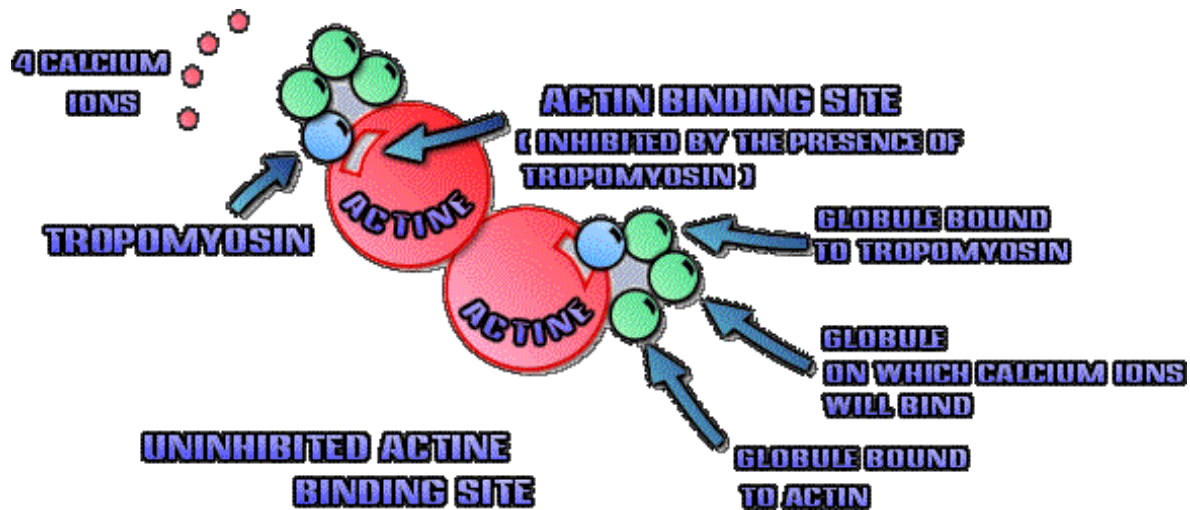
Thin filaments are thus made up of three molecules: [actin](#) (with its binding site shown as a white plate symbol), [tropomyosin](#) uninhibiting or not this binding site, and [troponin](#) which moves [tropomyosin](#) away to open or close the binding site.

(click on the drawing to get definitions)



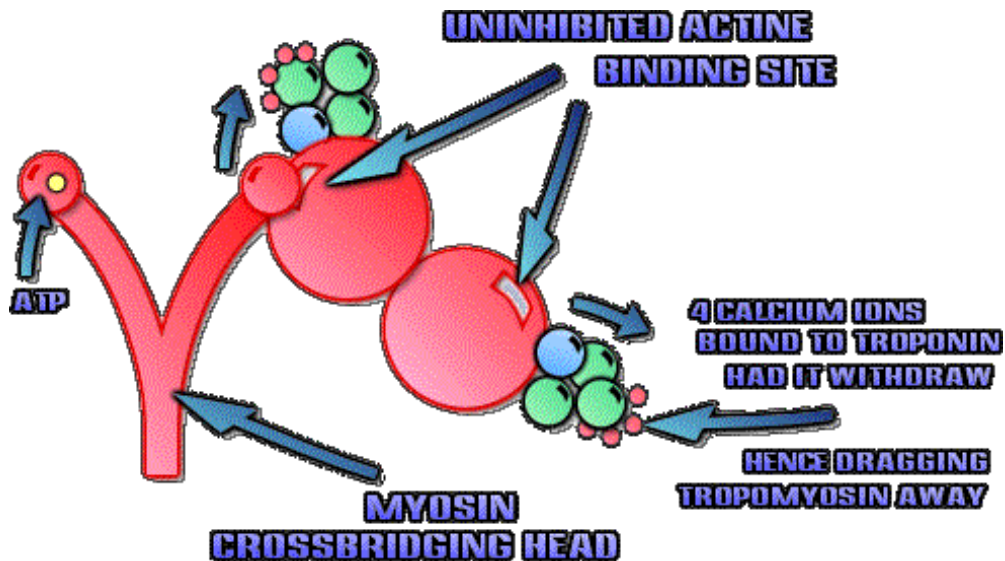
In reality, the overall pattern is more complex because a thin filament is in fact made up of a series of [actin](#) molecules set in pairs forming a synusoidal line. [Troponin](#) is in fact a set of three globular molecules with each a very definite function. One is directly connected to [actin](#), another to [troponin](#), and the one in the middle is the one which will host [calcium ions](#), which will change its shape and draw the set of 3 globular molecules closer to one another, thus dragging [tropomyosin](#) along.

(click on the drawing to get definitions)



All the elements are thus ready on the thin filament stage for muscle contraction to begin: one can see 4 [calcium ions](#) entering the stage on the left, and about to bind to the central [troponin](#) molecule. At that moment, [tropomyosin](#) is set right above the [actin binding site](#), and within a split second the whole process will begin..

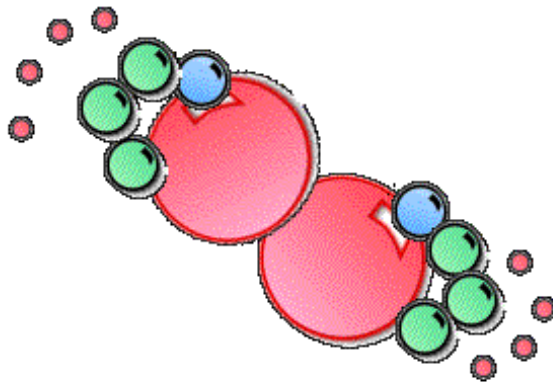
(click on the drawing to get definitions)



[Calcium ions](#) have just bound to the central globular [troponin](#) molecule. Their presence modified its general disposition and balance, leading to a shrinking of the [troponin molecules 3 set](#). because of them is connected to [tropomyosin](#), this one will also be dragged away along the other 3 [troponin](#) molecules. The [actin binding site](#) is thus no longer inhibited.

A crossbridging head from [myosin](#) appeared, and one of its tip came to connect on the now uninhibited [actin binding site](#), and within micro-seconds this [crossbridging site](#) is going to bend (as a finger would do) and lean on [actin](#) to move it rearward, hence dragging [myosin](#) forward, and by doing so shortening the whole [sarcomere](#).

(click on the drawing to get definitions)



This little animation shows the arrival of [calcium ions](#), their binding onto the central [troponin](#) molecule, its movement (because its shape has been altered) and the dragging of [tropomyosin](#) away from the [actin binding site](#).

CHEMICAL ELEMENTS [back](#)

Action potentials have just reached the muscle and helped release [calcium](#), muscle contraction can now begin...

We are now getting back at the level of the [sarcomere](#) (the "logs" set in line, making up the whole [myofibril](#), the level at which muscle contraction occurs) many electro-chemical reactions occur at that level: two elements play a key role in these. They are [calcium](#) and [ATP](#). [Calcium](#) will be used to move the "key" molecules, and [ATP](#) will provide energy for molecule connections-deconnections.

Let us explain what happens in two steps: let us first consider the role of [calcium](#) and then we will get deeper into the process to have a look at what happens at the tiny level of [ATP](#) "combustion". To put it bluntly, [Calcium](#) will help "unlock" the gears of muscle contraction, while [ATP](#) will provide fuel.

CALCIUM'S ROLE

General Principle: [Calcium](#) operates as a key which is the only one to open the binding site enabling [myosin](#) to bind to [actin](#). As soon as the key ([calcium ions](#)) gets into the lock (in that case it gets onto the [troponin](#) molecule) and makes it move, the bolt ([tropomyosin](#)) withdraws, thus uninhibiting the whole mortise ([actin binding site](#)) and letting [myosin's crossbridging site](#) in. The hooking-unhooking cycle can now begin.

ATP'S ROLE

General Principle: [ATP](#) makes the unhooking-hooking cycle possible (that of [myosin's crossbridging sites](#)). The main function of [ATP](#) is to modify the end shape of [myosin](#). It will thus enable this "[crossbridging site](#)" to bend forward and catch the [actin binding site](#) (the socket if one wants to draw a comparison), bend inward to draw [actin](#), and then unhook from it to begin another cycle. Like [calcium](#) binding on [troponin](#) and making it move, [ATP](#) binds to the [crossbridging site of](#) (the plug if one wants to draw a comparison) of [myosin](#) for a similar result. One [ATP](#) molecule is required to provide the energy to bend [myosin's](#) end, then another one is required to make it unhook from the binding site. The presence of [ATP](#) decreases the connectivity properties of the link between [actin](#) and the tip of [myosin](#). Once this link has been separated, [ATP](#) is degraded thanks to its [hydrolysis](#), which creates ADP.

**Release of [calcium ions](#) into the [cytosol](#):**

[Calcium](#) is stored around each [sarcomere](#) in the "terminal" tanks, and these are linked to the exterior by transverse tubules. When the tanks release their contents into the transverse tubules these release [calcium](#) into a closed-in "void" (the [cytosol](#)) walled-in by the membrane shrouding each [myofibre](#) (plasma membrane). When [calcium](#) « fills up » the [cytosol](#) it is going to be attracted by molecules at the surface of thin molecular filaments: i.e. [troponin](#).

- Let us remember that the « [thin molecular filaments](#) » are filaments made up of three molecules: [actin](#) set in pairs and then in line forming a long sinusoidal filament. There is also another type of molecule ([tropomyosin](#)) coiled around this filament of [actin](#), and covering up the binding sites for [myosin](#). Finally, there are [troponin](#) molecules connected both to [actin](#) and to [tropomyosin](#).



Binding of [calcium](#): [Calcium](#) binds to [troponin](#) and alters its shape: it moves and drags [tropomyosin](#) which previously inhibited the bindings sites on which [myosin](#)'s [crossbridges](#) will bind before drawing [actin](#) in. In reality, [troponin](#) is made of three globular molecules: one is connected to [actin](#), another is connected to [tropomyosin](#), and the one holding a central position and hosting [calcium ions](#) (four of them can connect to it). It is the central globular molecule of the [troponin](#) trio which will draw the other two [troponin](#) molecules inward and by doing so, while still connected to [actin](#), will drag [tropomyosin](#) away from the bindings sites ready for [myosin](#): It is now up to [myosin](#) to get on the stage.



Connection between [myosin-actin](#): For the "arms" of [myosin](#) ([crossbridges](#)) to move, some energy is required: this is provided by the [hydrolysis](#) of [ATP](#) molecule once connected to [myosin](#)'s end. These [crossbridges](#) operate an angular movement (bending) toward the [actin](#) binding sites. At that moment [myosin](#) is said to be in a "high energy" position ("arm" stretched forward). [ATP](#) hence eroded remains on [myosin](#) as ADP+P.



Pull from [myosin](#): After this binding phase, the "head" of [myosin](#) bends inwards (as a finger that one would fold in), drawing along the [actin](#) molecule. The [crossbridging sites](#) « draw » [actin](#) in their direction, thus shortening the whole [sarcomere](#) and creating power. This movement consumes energy stored in the "head" which now gets back to a "low" energy level. Meanwhile, ADP molecules and phosphate are released and leave behind an empty place for other [ATP](#) molecules. When connection occurs between [actin](#) and [myosin](#) the latter is said to be in a "low energy" level.

As long as [ATP](#) molecules are present in this environment, [myosin](#)'s heads will find the energy necessary to bend and pull the filament of [actin](#) molecules towards [myosin](#). "Head" movements do not occur at the same time, and during a muscle contraction there are always [actin](#) molecules connected to [myosin](#) by a few sites to prevent any backward gliding.



Un-binding for another cycle: another [ATP](#) molecule binds on [myosin](#) to separate it from [actin](#) and unhook [crossbridges](#) for another cycle.

And this cycle will take place as long as [calcium ions](#) will be found into the [cytosol](#) and bound to [troponin](#) molecules (which, by drawing [tropomyosin](#) away indirectly "opens" the [actin binding sites](#)).

We've seen that while [calcium](#) operates on thin molecule filaments to "open" them for molecular connection, it is [ATP](#) which provides the energy necessary for thick molecule filaments to hook-unhook to/from [actin](#) filaments.

The [ATP](#) molecule's role is threefold:

- Its very presence on [myosin's crossbridges](#) triggers the unhooking of the [myosin-actin](#) connection.
- Its degrading thanks to [hydrolysis](#) provide the necessary energy to the drawing of [actin](#) by [myosin](#) (shortening [sarcomeres](#)).
- Its [hydrolysis](#) also provides the energy used to carry [calcium](#) from the [cytosol](#) back to the [sarcoplasmic reticulum](#), and thus put an end to muscle contraction (we will get back to this role in the [exhaustion](#) chapter).

The problem of its regeneration will be dealt with in the [conclusion](#) chapter, for it has many consequences on workouts.



[Definition](#)

[when contraction ends](#)
[voluntarily](#)

[concentric failure](#)
[\(unvoluntary\)](#)

DEFINITION [back](#)

"Exhaustion" as we consider it in this chapter is to be understood as the phenomenon leading to an unintentional stop of muscular contraction. Muscle contraction ends and becomes impossible despite our efforts and our will-power. We generally refer to this a "concentric failure" (when one makes an effort but the weight no longer moves), or "eccentric failure" (when one is no longer able to withhold the weight).

VOLUNTARY STOP [back](#)

The end of muscle contraction occurs when [calcium](#) is pumped from the [cytosol](#) back to the sarcoplasmic membrane in the "terminal" tanks. When the [calcium](#) rate has fallen down to the lowest possible level there isn't enough left to bind to [troponin](#) which then finds its original shape back and its original position [actin](#) as well: This movement pushes [tropomyosin](#) back in its original position (inhibiting the [actin binding sites](#)) and [myosin crossbridges](#) can't bind to them any more: contraction ends. It is the [hydrolysis](#) of [ATP](#) which will provide the energy required by the pump to suck [calcium](#) from the [cytosol](#) back into the plasma membrane "terminal" tanks.

CONCENTRIC FAILURE [back](#)

Contrary to what could be thought, in the case of very intense effort such as those usually met in powerlifting, exhaustion is not always related to a decrease in [ATP](#) stores (it nevertheless happens sometimes) because [ATP](#) levels in a relaxed muscle and in a muscle developing its full power are rather similar because [ATP](#) stores are continuously re-filled (pretty much like a CD burner buffer). Exhaustion is rather considered as a defense mechanism to prevent a complete muscle rigidity : if contractions continued endlessly the [ATP](#) stores would eventually reach a level so low that they could not unhook [myosin](#) from the [actin](#) (2nd [ATP](#) role) : we would end with completely blocked muscles (it is what happens in a corpse: there is no longer any [ATP](#) available to unhook [myosin's crossbridges](#), which blocks muscles beyond recovery : it is referred to as "rigor mortis").

Muscle exhaustion during a short and intense effort is the by-product of 4 phenomena : it is always related to the disturbance of muscle structure operation or to the jamming of the whole system by the piling up of contraction's waste.

	Potassium Ions piling up	Hydrogene Ions piling up	ADP Piling up	Lactic Acid Piling up
At which level ?	Transverse tubules terminal tanks (from sarcoplasmic reticulum).	Transverse tubules terminal tanks (from sarcoplasmic reticulum).	Cytosol	Cytosol
Which type of molecular effect ?	Troponin is freed of calcium , and thus gets back to its initial position and pushes tropomyosin back in place on the actin binding site .	Crossbridges cycles are blocked because there is too much calcium . All actin binding sites get hooked.	Increased delay in myosin unhooking from actin binding sites .	Crossbridges which no longer hook ? no longer move forward ? (to be checked out).
Which type of effect at muscle level ?	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !
Why ?	Depolarisation of plasma membrane, leading to a brutal stop of calcium release into the cytosol .	Too much calcium into the cytosol	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !

How can that be possible ?	The plasma membrane is no longer conductive, and nerve impulse can't travel any more, so calcium isn't released anymore. Each time a nerve impulse depolarises-repolarises the plasma membrane potassium ions (waste-deposits) are created and eventually depolarise it completely (because they gradually jam the plasma membrane).	Hydrogene ions disrupt the functioning of the sarcoplasmic reticulum pumps which can't pump calcium back from the cytosol . Contractions continue until total tetanus.	When ATP is created ADP (by-product of that creation) gradually piles up until the moment this slows down crossbridging cycles.	When ATP is created in an environment lacking oxygene lactic acid is not eliminated and piles up until hindering the release of calcium and the role of ATP in crossbridgings .
In short...	Calcium no longer reaches cytosol.	Calcium is no longer released into cytosol.	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !
Mechanical analogy.	Fouling of sparking plugs.	Pistons and gearings foulings.	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !
What type of effort ?	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !
Muscle recovery	Fast	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !	knowing more ? please contact the webmaster !



ATP regeneration modes	The relevance of different fibre types	Isometric and Excentric work relevance
Deliberately slow movements relevance	General adaptation response	One just can't "shorten" a muscle

USEFULNESS OF KNOWING THE # ATP REGENERATION MODES [back](#)

Introduction: the muscle system as compared to an internal combustion engine:

If one compares a muscle to an internal combustion engine we find ourselves facing a particular system which in fact produces its own fuel before using it. Depending on the emergency and the amount of power required, this particular engine will first choose one type of refinery rather than an other. When starting the engine, it will use the tiny amount of fuel immediately available, then, if the effort continues it is going to create its own using oxygene. If the effort is of a moderate type (a little hill to climb) the oxygene will be used to recycle toxic waste coming from combustion and the engine will keep functioning as long as it will have nutriments to make fuel, it won't be clogged by waste deposits inside. If, on the other side, the power required is important (a steep road for instance) and lasts for a while, the waste created by the engine will be more important in volume than its capacity to recycle it thanks to the use of oxygene, and waste deposits will be beyond any recycling / filtering capability because oxygene used to do so is not provided in sufficient volumes : the engine will eventually "stall", bogged inside by its own waste. this is what happens with "concentric failure".

- If the engine must deliver the whole of its power capacity within seconds, it will use the directly available "left-over" fuel to do so at once (and not only to start), so fast that the engine will then be clogged quickly and will stall.
- If the engine is used for a medium-distance run, but on a rough terrain, it will begin to create its own fuel, but will "heat-up" and will eventually stop by want of having been able to filter all waste and combustion residues.
- If the engine is used for a moderate-speed run on a straight road, it will be able to filter its own waste thanks to a sufficient amount of oxygene and will keep running as long as nutriments will enable it to create its own fuel.

The "fuel" that we have been referring to in this simplistic analogy is a very energetic molecule called [ATP](#). There is always a very low amount of it already present in muscles and ready for action, but this quantity can only lead to a few muscle contractions. Muscle metabolism must thus create and regenerate more to sustain a longer contraction. As [ATP](#) is used (hydrolysed), it must be regenerated out of substances which are creatin and ADP. [ATP](#) is synthesised by a [phosphorylation](#) process (i.e energy will be used to assemble molecules - whereas [hydrolysis](#) uses energy to separate them). [ATP](#) is thus built by assembling molecules, and this can be done in three different ways (according to the duration and intensity of required effort).

[ATP](#) is created with or without oxygene. When oxygene is not provided in sufficient amounts we are in the field of very intense efforts which use the already available [ATP](#) stores, and when oxygene is provided in sufficient amounts we in the field of longer and more moderate types of efforts. The general rule is that the more violent the effort, the more glucose will account for [ATP](#) synthesis, and the longer the effort, the more [ATP](#) will be synthesised out of fat.

NON-AEROBIC (LACTIC ACID-FREE)	NON-AEROBIC (WITH LACTIC ACID)	AEROBIC
PHOSPHOCREATINE PHOSPHORYLATION	ADP PHOSPHORYLATION IN MITOCHONDRIONS	ADP PHOSPHORYLATION IN CYTOSOL
Creatine phosphate.	Glycolysis: glucose degradation.	glucose, fat acids, lactic acid , amino acids, lipids.
<p>This is the quickest way to answer to ATP needs. Creatine phosphate is a very energetic molecule formed by muscle fibres during periods of rest. This synthesis of ATP is very fast because all the elements are already present and ready to be processed before the need for it, they are to be found in the immediate environment of myofibrils, but the stores of creatine phosphate naturally present inside muscles are very limited indeed. It can only last for 25-30 seconds maximum: this is the time-scale of benchpress. The increase in power is quick because its synthesis is not the result of long and complex enzymes inter-reactions. The two other ways of synthesizing ATP aren't as fast but last longer.</p>	<p>ADP oxydative phosphorylation in mitochondrions is used when the effort is less violent and longer (this is "resistance", a thing medium-distance runners know well). This covers up the first 5 to 10 minutes of effort. One transforms muscle glycogene into glucose to obtain the necessary "fuel" to then create ATP. The intensity is inferior to that of the first case yet high enough for oxygene supply to be nevertheless insufficient to filter muscle contraction waste.</p> <p>Because it lasts more than 10 secondes one needs oxygene, but because the effort in question is intense this oxygene supply is not sufficient. Glucose is mainly used, but it is incompletely degraded because of the lack of oxygene and creates lactic acid that oxygene cannot eliminate. It is not quite right to talk about an "anaerobic process" in that case because breathing nevertheless occurs but unsufficiently.</p> <p>In these conditions glucose cannot give more than 2 ATP molecules. This is a complex process divided into no less than 12 steps and occurs within the sarcoplasmic reticulum.</p>	<p>ADP Phosphorylation by using glycogene in the cytosol is used for long efforts. This procedure has the highest yield and efficiency, as well as the ability to use many different substances successively to produce ATP. It uses them in a pre-determined order according to their successive shortages: oxydating glucose phosphorylation, followed by that of fat acids, that of lactic acid itself (!), and amino acids if need be.</p> <p>The interest of that process lies in its ability to recycle lactic acid thanks to the Krebs cycle. Lipids can even be used to provide ATP, and stores are huge.</p> <p>The overall energetic yield and efficiency of that method is very high and this is the least consuming method, because the destruction of a single glucose atom in this process helps producing 38 ATP. As long as oxygene is present the effort remains possible because it recycles lactic acid (muscle toxic waste is recycled by</p>

		oxygene).
Maximum effort, very intense but very short: a "max-out" in powerlifting for example. The intensity is very high, immediate, but decreases very rapidly.	Intense effort of moderate duration: between 10seconds to 1mn: typically the case in 15 to 20rep sets, or a series of long sets (8x10). Intensity reached at first is high, yet inferior to that found in anaerobic alactic processes, but the decrease in intensity is progressive.	Moderate intensity and long duration endurance exercises. The increase in power is longer than in the first two examples. the maximum intensity reached is reduced, but its duration longer.
Max-outs and short rep sets (2-4reps).	A set of 5 sets of moderate length (5 to 7 reps for example) or "endurance" benchpress (such as the "100kg dash" benchpress competition).	A long "pecs" workout, with many different exercises.
The 10 to 30 first seconds: as long as creatine phosphate limited stores (built up during periods of rest) are available in muscles.	Beyond ten seconds, and up to a minute.	Beyond nine minutes.
Transforming intermediate-twitch fibres into fast-twitch fibres using creatine phosphate, increasing the ability to store more of it during periods of rest. Concentrating on neuromuscular ability improvements to increase motor neurons reactivity.	Training muscles in an acid environment via pre-fatigue methods, methods aiming at training muscles to work in a state of exhaustion such as the Bulgarian methods and the like.	Use fast movement workout exercises with moderate weights to increase muscle endurance and the number of mytochondrions set around myofibrils .
Aircraft carrier catapult launch. Hill start.	After-burner. Second and Third shifts.	Cruise speed. Fifth and sixth shifts.

ATP synthesis particular characteristics as related to Weightlifting (the same applies to Benchpress)

Muscle biopsies carried out after violent efforts in among weightlifters have given amazing results: a decrease in [ATP](#) concentration was detected, whereas its amount is supposed to remain constant and stable both before and after an effort in the great majority of cases. This shows that [ATP](#) consumption ([hydrolysis](#)) during a very violent effort is faster than the muscle capacity to constantly regenerate its level (by [Phosphorylasing](#) ADP - see table above). This proves that the type of intensity met in weightlifting is such that the [ATP](#) regeneration cycle rate just can't "follow". This shows that in order to maintain a constant level of [ATP](#) stores, weightlifters very quickly give up the Alactic Anaerobic procedure to create [ATP](#) thanks to glycolysis. A very important decrease in the triglyceride rate has been discovered after such an effort, which betrays the use of fat as energy supply, while this type of fat usage is usually the very last resort to produce energy, it comes last in the list of substances used to produce [ATP](#) (what's more, fat is usually used in aerobic procedure).

Powerlifting and Weightlifting share a common characteristic: these imply very intense and concentrated efforts that muscles are forced to shift more quickly than usual from a procedure to another to try to maintain an [ATP](#) buffer level stable, despite the fact that it is being consumed at a very high rate, faster in fact that the muscle's ability to constantly regenerate this supposedly "stable" directly-available store.

USEFULNESS OF KNOWING THE # FIBRE TYPES [back](#)

It is the relative shortening speed of [sarcomeres](#) and [ATP](#)'s synthesis modes (immediate [oxydation](#) or creation out of glycogene) that determines fibres classification between the "fast-twitch" and the "slow-twitch" types. The difference between these is partly related to the contraction time of each: fast-twitch fibres have a contraction time of roughly 10ms, instead of 100ms for slow-twitch fibres. This contraction time depends up the duration of [calcium](#) presence into the [cytosol](#). Hence, the release of it is much more important and sudden in fast-twitch [myofibrils](#).

Another great difference is to be found at the level of [myosin](#) which does not degrade [ATP](#) at the same speed, and does not move its [crossbridges](#) at the same speed neither.

Fast-twitch fibres move [crossbridges](#) roughly four times as quickly as slow-twitch ones. But, on the contrary to a pre-

conceived idea, the amount of power thus created by both fibre types is the same, it is only the molecule movements speed that changes.

Some fibres are endowed with much more numerous [mitochondrions](#) and can thus produce more [ATP](#) using [phosphorylation](#) («endurance» types of workouts) : these are referred to as «oxydating fibres» (or "red" fibres). These are irrigated by blood and thus receive a lot of nutrients.

Other have much less [mitochondrions](#) but big glycogene stores instead: these are referred to as "glycolitic fibres" or ("white" fibres). These use but very few oxygen and have a larger diameter as compared to the oxydating fibres. Glycolitic fibres develop more power than oxydating ones because they are endowed with many more thin and thick threads than the others..

Fibre Type	ATP synthesis mode	Contraction speed	Color	Particularities	Type of sport to which it is best adapted
Type I	oxydation	slow-twitch	Red (many mitochondrions)	small-sized, enduring but not very powerful	long-distance running
Type IIa	oxydation	fast-twitch	White (big glycogene store)	intermediate size, but easily exhausted.	powerlifting, weightlifting, heavy-weight sets.
Type IIab	knowing more about it ? write to the webmaster	fast-twitch	White (big glycogene store).	thought to be able to get close to IIa and IIb fibre type functioning modes.	knowing more about it ? write to the webmaster
Type IIb	glycolitic	fast-twitch	White (big glycogene store).	large-sized, very powerful but not very enduring.	weight-lifting and powerlifting competition 1rep max.

Motor unit triggering sequence follows a single and pre-determined order: it always begins by triggering slow-twitch oxydation fibres ([motor unit 1](#)), then fast-twitch oxydation ones ([motor unit 2](#)) then eventually fast-twitch glycolitic fibres ([motor unit 3](#)). In short, the weakest and most enduring fibres are called upon first, then follow more powerful ones (which are less enduring) when the first are exhausted.

It is the size of neurons which determines this order: the diameter of neurons decides everything: the intensity of nerve impulse gradually increases if the effort continues: the first type of nerve influx is of a low intensity type, and thus goes through small neurons first, and these small neurons are connected to small motor units: so, slow-twitch oxydation fibres are called upon first. When nerve impulse increases in intensity it uses larger neurons, which are connected to larger motor units, hence fast-twitch glycolitic are called upon in last resort.

Generally speaking, the effort has to reach around 40% of the maximum potential contraction intensity before fast-twitch glycolitic fibres (the most powerful ones) are triggered.

Consequences for benchers :

- to reach a given type of fibres one must previously have exhausted all other ones. One can only reach glycolitic fast-twitch (powerful) fibres after having previously used and exhausted the other ones (from this derives the usefulness of using pre-fatigue workout techniques).
- use the specially-designed software available on this website ([download page](#)) to determine your personal fibre-types proportion (genetically determined at birth). If it so happens that the average results you'll get out of it are above what you are able to do at the gym, especially in short rep sets (from 2 to 4 reps), this means that you have a natural proportion of slow-twitch fibres higher than average. If, on the other hand, you are able to perform more, and lift heavier weights than what the software calculates, this means that in your case you have a higher proportion of fast-twitch fibres than the average. Having an idea of this enables one to understand one's performances, and to concentrate on the type of reps with which you are the least successful.

ISOMETRIC AND EXCENTRIC METHODS ARE USEFUL [back](#)

During a **concentric** contraction [crossbridging sites](#) develop a **more** powerful force than that of the weight used.

During an **excentric** contraction [crossbridging sites](#) develop a **less** powerful force than that of the weight used and muscle nevertheless continue stretching out.

During an isometric contraction we have a situation of equilibrium between forces, but [crossbridging sites](#) are not unmoving; they nevertheless perform the connection-deconnection cycle; the only difference is that binding occurs exactly on the same

[actin](#) molecule.

A MUSCLE CANNOT BE SHORTENED [back](#)

It is impossible to "shorten" a muscle by doing only half of the complete movement: this technique, once dear among some bodybuilders, consisted in only doing half of workout exercises movements, usually the final part, but this proved to be an illusion: Because the length of [sarcomeres](#) is only shortened during a contraction, only to get back to its original length at rest, one can not really shorten it. If the muscle seems to have "shortened", it is only because of an optical illusion due to its hypertrophy which made it get "higher". Let us also remember that muscle fibres are not added, they only get thicker by increasing the number of [myofibrils](#) inside.

SLOW MOVEMENTS ARE USEFUL [back](#)

muscle contraction speed obviously depends on weight, on the number of motor units involved and above all on the motor end plate capability. Working slowly makes two improvements possible:

- Suppressing the ill-effects of impetus in movements: one thus forces muscles to keep the same level of workload and intensity during the whole duration of movements, which is beneficial in terms of general adaptation response triggering, and also because it reduces the risks of joint injuries due to shocks and brutal impulses.
- An increase in intensity as far as [action potential](#) sommation is concerned, which is beneficial in terms of the muscle's ability to develop, and after workout, further and more deeply inter-mingled nerve connections during the general adaptation response phase. A slower movement leaves more time to [calcium](#) to connect to a maximum of [troponin](#) molecules and thus indirectly allow a maximum of [crossbridgin sites](#) to operate: muscle contraction is maximum, and so will be the adaptation response.

GENERAL ADAPTATIVE RESPONSE [back](#)

The General Adaptation Response cycle is as follows:

- after each workout we witness a decrease in muscular abilities: it is what we refer to as "exhaustion".
- then occurs a recovery period.
- if workouts follow a regular pattern we witness an increase in muscle power: this is "overcompensation".
- if it is not the case we will witness a loss of power instead and a progressive return to the initial potential.
- if workouts follow one another in a succession leaving no place for sufficient rest, overcompensation will not have enough time to operate and we fall into the danger of over-training, because energetic stores are depleted without having enough time to be replaced, muscles are used without enough time to adapt and regenerate (hence leading to both potential exhaustion and injuries).

General Muscular Adaptation Response : This notion is the locus of pre-conceived ideas. In fact, training modifies muscle fibre properties, but it never alters their number. One does not build up muscle, properly speaking, it is more complex than that: everything occurs at the level of [myofibrils](#). Muscles can improve by increasing their abilities in three possible domains: Hypertrophy, Metabolic alteration, Neuro-muscular efficiency (see below).

- **Muscular hypertrophy:** Muscles grow not thanks to an increase in the number of muscle fibres ([myofibre](#)) but to a thickening of these, by adding ([myofibril](#)) within. It can be compared to enlarging a big cable by increasing the number of invisible filaments that each wire is made of. If the muscle remains unstimulated (when the arm is in a cast for example) we witness a fibre atrophy : their diameter of muscle fibres decreases because of a decrease in the number of internal [myofibrils](#) (the little filaments making up each ([myofibre](#))). When you hear someone saying that muscles grow by increasing the number of fibres, you can tell him it is both right and wrong: it is not the number of muscle fibres that is increased, but the number of sub-fibres within each one of them.

Muscle fibres can increase in diametre (hypertrophy), and in their ATP production abilities enhanced. But in no way can their number change. Usually, improvement occurs at the level of metabolic capability.

- **Metabolical fibre alterations:** muscle fibres increase thus in terms of the number of [myofibrils](#) they host within, but an increase in power is also the by-product of an alteration of [ATP](#) production method.

- « aerobic» exercises (in which breathing is important - such as running), lead to an increase in the number of [mitochondrions](#) and tiny vessels: the same fibres witness an improvement in their capacity to host food and

oxygene. Their diametre even tends to get slightly smaller, and stregh consequently decreases.

- « anaerobic» exercices (in which air is not brought in sufficient amounts) triggers glycolitic fibres quickly. These fibres also see their structure (and not their number) modified: their diametre increases (thanks to an increase in the number of [myofibrils](#) inside), and their ability to store glycogene is improved.

The innate proportion between slow-twitch and fast-twitch fibres won't be altered by training, we rather face a change in fibres internal strucutre according to their specialised modes of "fuel" storage capabilities: we either increase the number of [mytochondrions](#) (endurance) or the number of glycogene stores (powerlifting) in each fibre. What is altered is the way [ATP](#) is being produced. These are reversable alterations if training is stopped for a long period of time. With training, one can sometimes double the initial number of [mytochondrions](#), hence doubling up the muscle's ability to produce [ATP](#).

- **Neuro-muscular efficiency improvements :**

Hypertrophia is brought about by a moderately long practice of weight lifting (with sets above an average of 7-8 reps) and with a moderately high intensity training. On the other hand, when benchers keep practicing 2rep-sets, or 3-4reps sets, their general strength will improve not thanks to hypertrophia but thanks to another type of adaptation response involving the creation of a more efficient neuromuscular network. By doing so, the athlete will improve his capacity to trigger a maximum of muscle fibres thanks to a more efficient [motor end plate](#) rather than developping them more.. Nobody can trigger all his/her myofibres, even during a very intense work. Among untrained people, around 30% of muscle fibres are but harly ever used. Among trained athletes, there still remain around 10% which are not (which is still a lot - just imagine what you could do if you were able to trigger these 10% extra myofibrils !). Only doped athletes nearly manage to trigger them all (but these people are not worth talking about).

Early beginnings and quick improvements :

When a beginner is lifting his very first weights, he improves very quickly. In reality, this has nothing to do with hypertrophia, because this will only be triggered much later, but this is only to be related to an improvement in neuro-muscular ability and the development of a more efficient nerve network. That is the reason why improvements seem to happen so fast. But here also lies the explanation of the difficulty to go beyond the first "plateau", the first moment of stagnation: this "plateau" happens when neuro-muscular improvement slows down and muscular growth work begins, which is a much more unthankful matter. It is at that very moment when those who can be considered "up to it" and who are patient enough (let us remember that powerlifting is a sport for patient people) step out of the whole lot, while others take their leave.

