

**MUSCLE MYTHS
VS
MUSCLE REALITIES**
By Thomas Griner
An Addendum to Chapter Two of
“What’s Really Wrong With You?”
A Revolutionary Look at How Muscles Affect Your Health
By Thomas Griner with Maxine Nunes

Muscle Myth #1

Sprinters use their fast-twitch muscles while marathoners use their slow-twitch muscles.

Muscle Reality #1

The above statement shows lack of attention to how fast-twitch and slow-twitch muscles relate to speed output. The slow-twitch fibers require .075 seconds to complete a twitch contraction while the fast twitch fibers do so in only .025 seconds. So, ‘fast’ means 3 times faster than ‘slow’.

For a man of average height (average length of leg levers), the slow-twitch muscles max-out at 8½ MPH, while the fast-twitch max-out at 25½ MPH (long legged sprinters *should* dominate!). The marathon is run at an average speed of just over 12 MPH, which leaves the slow-twitch muscles unable to contribute any force. This is why marathoners stoke up on carbohydrates, which are fuel for fast-twitch muscles rather than fats and oils, which are fuel for slow-twitch muscles.

The difference is not which muscles sprinters and marathoners use, but is instead which *muscle energy system* they each use. Sprinters use predominately the limited phosphagen energy storage system with a slight amount of anaerobic sugar fuel energy, while marathoners use large amounts of the anaerobic energy and aerobic hydrogen fuel energy to maintain their phosphagen system. This is why marathoners, after having run more than 26 miles, can sprint the last 200 yards or so.

The muscle energy systems are similar to a hybrid vehicle with phosphagen (creatine phosphate and adenosine triphosphate [ATP]) storage ‘batteries’ providing limited amounts of quick energy, backed up by fuel-driven re-chargers. The muscles have the two re-charging systems, each using a different fuel, while the hybrid vehicle currently has only one. (Automotive engineering is developing a supplemental steam engine which uses the waste heat from the existing fuel combustion engine in hybrids.) The muscle system cannot recover energy via dynamic braking as hybrids do.



Muscle Myth #2

The error of MM Statement #1 is further compounded by claims that marathoners have a higher number of slow-twitch over fast-twitch muscles than sprinters have.

Muscle Reality #2

Unlike lower mammals, all primates have a numerical 50/50 mix of fast and slow-twitch muscles, with little variation. (Fast-twitch muscle fibers are three or more times larger than slow-twitch muscle fibers, so fast-twitch dominates the skeletal muscles.) However, extensive marathon training excites the slow-twitch muscles to increase the number of their aerobic mitochondria as much as three-fold. Even though marathoners are not using the contractile portion of the slow-twitch muscles, they are using the metabolic portion while sprinters do not.

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**Muscle Myth #3**

The error in Muscle Myth Statement #2 is further compounded by claims that human skeletal muscle contains not two but *three* types of muscle: aerobic fast-twitch fibers.

**Muscle Reality #3**

Again, primates do NOT have the third type of muscle fibers as do lower mammals. The non-primates *need* a third type of muscle fiber because some of their muscles are far from the 50/50 mix the primates enjoy.

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Muscle Myth #4

Lactic acid is produced due to a lack of sufficient oxygen.

Muscle Reality #4

Lactic acid is produced to act as a pyruvic acid sinkhole to prevent the concentration of pyruvate from rising to the point that it stops the anaerobic sugar glycolysis (i.e. the chemical law of mass action; and pyruvate is the end-product of glycolysis.) The lactate can become over 10 times more concentrated than the pyruvate before the law of mass action stops the conversion of pyruvate to lactate, which then stops glycolysis.

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**Muscle Myth #5**

Aerobic metabolism is an oxidation reaction, while anaerobic metabolism is not.

**Muscle Reality #5**

“Aerobic” means “with oxygen”, and “anaerobic” means “without oxygen”. “Oxidation” refers to reactions that produce energy, regardless of whether oxygen is involved. (Those reactions which *require* energy are referred to as “reduction reactions”.) Both aerobic and anaerobic energy-producing metabolisms are oxidation reactions.

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Muscle Myth #6

The phosphagen system and the anaerobic sugar glycolysis system can both be easily depleted.

Muscle Reality #6

As previously said, the phosphagen system does not produce energy, but stores and releases it like batteries to drive the “electric stepping-motor” muscles. (Man-made stepping-motors are very expensive to fabricate so they are only used when very fine movement control and/or high efficiency are at a premium. These body-friendly carbon-phosphate “batteries” don’t need encasement, as do toxic lead-sulfate and lithium batteries.) This allows the phosphagen “batteries” to achieve very high-charge densities, or capacity.

The anaerobic glycolytic system is primarily responsible for metabolically producing the energy to recharge the phosphagen. But, it can only produce energy $\frac{3}{5}$ as fast as the phosphagen can discharge it. (The aerobic system produces energy $\frac{1}{4}$ as fast.) If sprinters run too far at top speed (~26 MPH), they can indeed deplete the phosphagen system and *then* they crash (ouch). This would require nearly 300 yards, however.

On the other hand, the glycolytic system is a molecular “fission” process using sugar fuel. The only way to deplete this would be to use up all of the sugar fuel stored in the muscles and liver.

(The liver can store as much as all of the muscles combined.) Again, this is why marathoners stoke up on carbohydrates the night before a race.

If a runner slows down just enough to allow the glycolytic process to keep up with the phosphagen discharge rate, the glycolysis will be generating by-products faster than they can be cleared by the blood stream (see Muscle Reality#4). This will overload the glycolysis to a stop. However, the high concentration of lactate being dumped into the blood stream will usually trigger a debilitating cramp before overload is reached. The marathoner must stay just below the speed where the lactic acid generation rate begins to exceed the clearing rate.

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### **Muscle Myth #7**

Hemoglobin is for oxygen transport, while myoglobin is for oxygen storage.

### **Muscle Reality #7**

Red cell hemoglobin is for long distance transport of oxygen, using the circulatory system to move the hemoglobin. The muscle myoglobin is for short distance oxygen transport, wherein the myoglobin does *not* move, but rather shuttles the oxygen in a bucket brigade manner. The myoglobin exists only in the slow-twitch muscles, where the aerobic mitochondria need oxygen. (And remember, lactate is produced and exits only from fast-twitch muscles, where only anaerobic activity occurs.) This presents a good segue to describe the activities in the aerobic mitochondria, which is the *second* muscle energy-producing system.

The phosphagen system has been equated to charging and discharging “batteries”, while the anaerobic glycolytic system uses a “fission” process to generate energy without discharging CO<sub>2</sub>. Engineers have developed a fuel cell with a preprocessor that allows it to use natural gas as fuel in place of hydrogen. The preprocessor strips the hydrogens from natural gas and sends them to the fuel cell while discharging CO<sub>2</sub>. This engineering system is not yet ready for prime time, but the muscles have been using a similar system, located in the aerobic mitochondria, for millennia.

The aerobic mitochondria contains three sub-systems. One, known as the “Krebs Cycle” or citric acid cycle, is the “preprocessor”, while another, known as cytochromic oxidase, is the “fuel cell”. The third subsystem, the fatty acid spiral, will be described later.

Similar to the engineering preprocessor, the Krebs Cycle starts with a catalyst (called Co-enzyme A), strips hydrogens for delivery to the cytochromic oxidase ‘fuel cell’, and discharges CO<sub>2</sub>. However, the Krebs Cycle uses H<sub>2</sub>O instead of O<sub>2</sub> to form the CO<sub>2</sub>. This allows the cycle to produce about three times as many hydrogens as are available from the carbohydrate fuel it is preprocessing. Nature is hard to beat.

The engineering fuel cell strips electrons from the hydrogen by passing it through a membrane that allows only the H<sup>+</sup> ion (also called a proton) to pass. The free electrons flow as a current in a circuit that produces energy before returning to the cell to recombine first with the H<sup>+</sup> ions, and then with air, to form H<sub>2</sub>O.

Cytochromic oxidase, the mitochondrial “fuel cell”, is where the aerobic mitochondria earn the ‘aerobic’ part of their name, as the fatty acid spiral as well as the Krebs Cycle anaerobically use H<sub>2</sub>O instead of O<sub>2</sub> for their processes. Hydrogens from the glycolytic process, the Krebs Cycle, and the fatty acid spiral are all delivered to the electron transfer chain of the cytochromic oxidase. This is why MR#1 referred to aerobic hydrogen fuel energy.

To understand the electron transfer chain, we need to start with cytochromic oxidase’s cousins: hemoglobin and myoglobin. We have already identified hemoglobin as a long-haul oxygen transporter. So naturally it is a ‘big rig’. It is made up of four sub-units linked together to enable it to carry four oxygen molecules (O<sub>2</sub>). The ferrous iron (Fe<sup>++</sup>) located in each sub-unit remains

stable while carrying O<sub>2</sub>, but becomes unstable when the O<sub>2</sub> is released. It then has a tendency to flip into the ferric (Fe<sup>+++</sup>) state. If this were to happen, it would be unable to carry O<sub>2</sub>. (This annoying instability becomes useful in the cytochromic oxidase.) The globin has a nitrogen link that is used to keep the heme iron in the ferrous (Fe<sup>++</sup>) state, while the globin is carrying CO<sub>2</sub> back to the lungs.

Each myoglobin molecule is equivalent to one of the four sub-units of hemoglobin, so they readily link together to form oxygen transfer chains. The cytochromic oxidase is similar to the myoglobin with the ferrous (Fe<sup>++</sup>) stabilizing nitrogen link replaced by a thio-ether link which allows the heme iron to safely flip back and forth between the ferric (Fe<sup>+++</sup>) and ferrous (Fe<sup>++</sup>) states. This allows the electrons to be passed along the transfer chain. As the electrons move along, their energy is tapped using a catalyst called CoEnzyme Q, which then re-charges the carbon-phosphate “batteries”. The mitochondrial substrate, which holds the cytochromic oxidase, also controls the movement of the protons (H<sup>+</sup>) so they don’t become loose-cannon free radicals before rejoining the electrons to be oxidized into H<sub>2</sub>O.

As we have said, the ingested muscle fuels provide free fatty acids and glucose for use by the Krebs Cycle. However, the Krebs Cycle can only process small molecules containing just 2 carbons, while free fatty acids contain 16 carbons, and glucose contains 6 carbons. This requires the Krebs Cycle “pre-processor” to have PRE-pre-processors for its fuel.

As has also been stated, the glucose is split into two 3-carbon (pyruvate) molecules by the glycolytic process in the fast-twitch muscle fiber. The Krebs Cycle has a side chain which can split off one carbon from pyruvate, but not from lactate. This is why lactate must be sent to the liver for processing.

This finally brings us to the fatty acid spiral, which chops the 16 carbon chain into Krebs-ready 2 carbon chunks. Unlike the anaerobic glycolytic-splitting process, the anaerobic spiral-splitting process does not produce energy, but does produce hydrogens.

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Muscle Myth #8

The prevalent and mild sub-clinical lateral scoliosis (side-to-side curvature) of the spine is a functional adaptation to imbalanced muscle use due to handedness.

Muscle Reality #8

After collecting data from approximately 3,000 cases, I did find there were two defined common patterns of mild spinal curvature with one pattern being the mirror image of the other. However, the occurrence of the two patterns did not coincide with right or left handedness but instead, appeared in the same ratio in both right and left-handed individuals. No one had the postural ideal of a straight spine.

Something this obvious must have been noticed before! A search of previous publications revealed articles from some 1970's publications by osteopathic researchers. Their method requires identifying postural distortions as a means of analyzing possible problems. They had also noted “the idea pattern is seldom if ever seen in the clinical setting”. They had also found “that approximately 80% had body patterns with the lumbar vertebrae misaligned to the left”. They named this “The Common Compensatory Pattern”. The other 20% displayed the opposite pattern. I had found the patterns to occur closer to a ratio of 85% to 15%.

They had also noted that the lumbar misalignments “can be associated with (unilateral) hypertonus of the psoas and quadratus lumborum muscles”. The left psoas muscle acts to cause the vertebral bodies to rotate clockwise as they are pulled sideways. The left quadratus lumborum muscle conversely acts to rotate them counterclockwise. By acting together, they pull

the vertebrae sideways with little or no rotation. The spinal muscles then cause compensation curves, above this primary side pull, which act to maintain the head in a centered position over the pelvis.

The osteopaths offered two possible concepts as the cause of the primary lumbar unilateral hypertonicity. First is that it “develops as a consequence of repeated minor traumas to the vulnerable lumbar spine during and after birth”. They refer to this period as “The Valley of the Shadow of Birth”. Second is that “The universal clinical nature of the pattern would tend to indicate a ‘genetic basis’”. That is to say a genetically-produced “potential anatomic vulnerability” rather than a DNA problem. The muscles which pull the lumbar vertebrae to the side appear to be acting as a guarding response to a vulnerably-placed nerve under the multifidus muscle on the opposite side of the spine.

A 1920’s publication by some chiropractors was also found. They described the same patterns in a more bone-oriented manner. The common pattern became referred to as “Category One” and the mirror image as “Category Two”. In addition, they had also noted that the inflection points for the lateral curvature patterns were the same as the inflection points for the standard anterior-posterior curvatures (i.e., the lumbar lordosis, thoracic kyphosis, and cervical lordosis). It is these inflection points that determine the pattern which the compensatory curves must follow.

Muscle Myth #9

Forcing muscles into excessive amounts of tone in order to get the “cut look” also makes them “fit”.

Muscle Reality #9

Olympic-style power lifters move such serious amounts of weight that they need to have truly healthy muscles. That is why they avoid exercise routines which elevate muscle tone. They learned decades ago that muscles burdened by excessive tone are less resilient and flexible and the elevated base level contraction subtracts from the overall power output. That is why body builders, in spite of their seemingly larger muscles, are unable to compete against the actually fit power lifters. (In other words, a six pack is a sick pack.)

The dictionary defines tone as “the normal tension of a healthy relaxed muscle”. “Cut” muscles are abnormally over-tensed, unhealthy, semi-relaxed muscle. Guyton’s Medical Physiology identifies the source of the tension: “muscle tone is a residual (low level) degree of contraction in skeletal muscles even when they are at rest”. So the heart muscles aren’t the only muscles that work 24 hours a day; skeletal muscle tone must also work 24 hours a day, mainly to prevent disjoints (ligaments only act to limit the amount of disjoints).

At times, the temperature control system temporarily elevates the muscle tone to provide heat. As this temporary tone increases, it can cause detectable muscle stiffness and even shivering or shaking from rapid spiking of the tone.

You may wonder why it is possible to create constant excess muscle tone if it is so bad. That’s the same as asking why attorneys can manipulate the legal system to perpetrate miscarriages of justice. The answer to both is vulnerabilities in the systems. Muscle tone can be manipulated by certain exercise routines to subvert its purpose.

Most excitatory nerve bodies (such as those for motor nerves to the muscles) are located in the protected central nervous system. The nerve bodies that drive muscle tone however are located out in the very muscles they are activating (oops). They are called anulospiral nerves and are located in complex neuro-mechanical organs called muscle spindles.

The cerebellum is responsible for setting the level of muscle tone, but it depends on feedback from nerves in the spindle to do so. Those feedback signal generators (called flower spray endings) can become dysfunctional from the high levels of lactic acid that certain exercise routines (such as repetitions and isometrics) cause the surrounding muscles to produce.

It is unfortunate that “Gray’s Anatomy” causes some confusion by quoting a 1955 myographic study which erroneously claimed ‘normal relaxed muscles are electrically silent’. Gray’s then uses this disputed (see previous Guyton’s quote) source as justification to claim “tone is not a basal level of contractile activity”. Since their title is “Gray’s Anatomy” and NOT “Gray’s Anatomy and Physiology”, they should mind their own business.



Muscle Myth #10

Dysfunctional muscles need not be considered as a possible cause of the vast majority of symptom patterns or of adaptive structural abnormalities.

Muscle Reality #10

Unfortunately, the current paradigm is to look for some structural pathology as the cause (etiology) of symptoms to the extent that any idea of a dysfunctional etiology is pushed-out into the cold. This leads to a tendency to think of muscles as passive connective tissue rather than functioning, reactive organs. Surgeons are often mystified when muscles react badly to having been treated as connective tissue. Alternative approaches also become overly enamored to the passive structural muscle fascia, which constitutes less than 1/2 of 1% of the muscle mass, while ignoring the 99 1/2 +% of functioning organs enclosed by the fascia.

When people are told that the muscles **are** the external organs, they respond “Oh no, the skin is the external organ”. The skin (integument) is embedded with hundreds of auxiliary, microscopic organs known as the integumental system. The integument itself is a large sheet of connective tissue with some cellular activity, but no organ activity. The arrangement of integumental collagen fibers is quite different from those in the organ (both internal and external) fascia.

The muscles are not microscopic organs. Indeed, they comprise about one-half of the body mass and so require a major portion of the activities of the internal organs to supply the needs of these external organs. Muscle Reality #9 described how muscles can be made dysfunctionally hypertonic (excessively tense). It also noted that the primary dysfunction is neurologically in the muscle spindle, but displays itself in the electro-chemical driven contraction of the muscles. Over-exercise may be one of the most common causes of hypertonicity, but there are many alternative ways to produce it. Oddly, insufficient activity can also cause it through too little circulation to provide adequate ‘flushing’ of metabolic by-products. The physical vicissitudes of life is another important factor.

Two of the results of hypertonicity were mentioned in the discussion of body builders versus power lifters. It was also noted that the **proper** muscle tone serves to hold the joints in place. Hypertonicity can elevate joint pressure to a level wherein gravity becomes minor. Hypertonic muscles become reluctant to perform, causing muscle catches (sometimes strong enough to be a muscle pull), feelings of weakness and loss of range of motion (the extreme cases being a frozen shoulder) or outright dislocation. Moderate activity improves circulation because muscles act as venous pumps. Hypertonicity leads to inefficient pumping, or even blockage. Hypertonic muscles can remain in such a contracted state of hardness that they irritate the very nerves they are supposed to cushion.