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The Lactic Acid Myths

By Matt Fitzgerald

There are many myths about lactic acid. Perhaps the greatest of all is the notion that there is lactic acid in the human body. There is not. The body actually produces lactate, which is lactic acid minus one proton.

The difference between lactic acid and lactate is, for all practical purposes, semantic. But other popular beliefs about lactic acid (or, as I will properly call it from this point forward, lactate) are about as wrong as can be. Most triathletes believe that lactate is an end product of anaerobic muscle metabolism that causes local muscle fatigue by increasing the acidity of the tissues to the point where they no longer can function effectively. In fact, we now know that lactate is an intermediate link between anaerobic and aerobic muscle metabolism that serves as both a direct and indirect fuel for muscle contraction and *delays* fatigue in a couple of different ways.

Our new understanding of the nature and function of lactate is interesting to all athletes who are curious about how the human body works. But does it make any practical difference? Does the new understanding of lactate suggest a different approach to training than the old science did? I would suggest that it does call for a subtle tweaking of the standard approach to endurance training, but it

warrants no major overhaul. Before we get to that, however, let's take a closer look at how the classic beliefs about lactate were exposed as myths and replaced by an almost opposite explanation.

The classic explanation of lactate in exercise dates back to the 1920s, when researchers showed that the exposure of frog legs to high levels of lactic acid (not lactate) interfered with the ability of the muscles to contract in response to electrical stimulation. Later research determined that lactate was produced through anaerobic glycolysis, or the breakdown of glucose or glycogen molecules for energy without the help of oxygen. It was then concluded that fatigue occurred at high exercise intensities because the cardiovascular system could no longer supply the muscles with enough oxygen to keep pace with muscular energy demands, resulting in increased reliance on anaerobic glycolysis, hence lactate buildup.

How exactly did lactate buildup cause the muscles to fatigue? Biochemists believed that lactate was formed in the body by the removal of a proton from lactic acid. When protons accumulate in living tissues, these tissues become more acidic. And when muscles become too acidic, they lose their ability to contract.

This tidy little explanation began to unravel in 1977, when South African biochemist Wieland Gevers showed that the reaction producing lactate actually *consumes* a pair of free protons, thus retarding muscular acidosis rather than promoting it. Much more recently, scientists have observed that, while protons do indeed accumulate in the muscles during high-intensity exercise, increasing muscle acidity, these protons are produced through a reaction that is completely separate from that which produces lactate.

To make matters even worse for supporters of the classic lactate hypothesis, we now know not only that lactate does not cause muscular

acidosis, but also that the muscles never reach a level of acidity that would directly cause dysfunction (or fatigue) of the muscle fibers anyway. The body's normal pH at rest is approximately 7.4. During intense exercise, as the muscles become more acidic, pH may drop as low as 7.0 at the point of exhaustion. However, when muscle cells are electrically stimulated outside the body, mechanical failure only occurs when the pH drops all the way down to 6.8. This observation suggests that fatigue always occurs before a catastrophic loss of acid-base homeostasis in the muscles takes place.

What's more, research conducted within the past decade has shown that lactate counteracts another cause of muscle fatigue at high exercise intensities: namely, depolarization. Muscle contractions are stimulated by electrical currents that flow throughout the body via minerals including sodium and potassium. Each muscle cell contraction involves a lightning-fast exchange in which potassium molecules inside the muscle cell and sodium molecules outside the muscle cell switch places. These exchanges are most efficient when there is a high degree of polarization (a difference in the strength of the electrical charge) between the spaces inside and outside the cells. At the beginning of high-intensity exercise, the inside of the muscle cell has a much stronger positive charge than the area outside the muscle cell. This difference in charge strength makes it easy for sodium and potassium to cross the cell membrane. During sustained high-intensity activity, potassium is released from the muscle cells faster than it can be channeled back in through special potassium pumps in the cell membrane. The resulting buildup of potassium outside the muscle cells causes a progressive lessening of the difference in charge strength between the intracellular and intercellular spaces, hence weaker and less efficient muscle contractions (i.e. fatigue).

It is now widely recognized by researchers in this area that muscle cell depolarization is a much more significant cause of muscle fatigue than muscular acidosis. Where does lactate fit in? In a series of studies beginning in 2001, Ole Nielsen of the University of Aarhus in Denmark, has shown that high levels of lactate partially restore muscle cell function in a depolarized state. Hence, if your muscles did not produce large amounts of lactate during high-intensity exercise, your muscles would actually fatigue a lot sooner.

The story does not end there. In the new scientific understanding of lactate, arguably the most important role of lactate during exercise

is not to delay fatigue caused by muscular acidosis or muscle cell depolarization but rather to serve as a direct and indirect fuel for muscle contractions. That's right: the substance that was once thought to be a worse-than-useless byproduct of anaerobic glycolysis turns out to be one of the most important energy sources for high-intensity muscle activity.

Our knowledge of lactate as a muscle fuel is largely the product of the work of one man: George Brooks of the University of California-Berkeley. Brooks became interested in lactate in the 1960s, when his track coach at Queens College told him that lactic acid was the cause of the burning sensation and loss of performance he experienced when running hard. Brooks went on to earn a doctoral degree in exercise physiology and made the study of lactate his life's work.

Brooks began to suspect that the classical lactate theory was dead wrong when, in one early experiment, he gave radioactive lactic acid to rats (so he could trace it) and found that their bodies used it faster than any other energy source. He then set about figuring out how lactate was used. The result of this process was the discovery of the lactate shuttle (now known as the extracellular lactate shuttle). Lactate is a highly mobile compound that easily leaks through the walls of the muscle cells that produce it into the bloodstream. From there the lactate flows to other muscles (especially resting muscles and muscles working at lower intensities) and organs—especially the heart, liver, and brain—and used as a fuel. Lactate that reaches the liver is even converted back into glucose and sent back to the hardest-working muscles to replenish declining fuel stores.

When Brooks published his first research on the lactate shuttle in the mid-1980s, he did not propose that any organ used lactate as a direct energy source. While his proposal that widespread use of lactate as an indirect energy source during exercise was radically new, Brooks did not initially challenge the notion that the human body is incapable of directly oxidizing lactate to release energy. Instead he hewed to the universally held conviction that lactate had to be converted to pyruvate before oxygen could do anything useful with it. But secretly Brooks suspected that some types of cells, including muscle cells, could break down lactate aerobically, and in the past few years he has definitively proven that this is indeed the case.

First Brooks showed that endurance training reduces the amount of lactate that enters the bloodstream without affecting the amount

LACTIC ACID-RELATED SUPPLEMENTS

There are various sports nutrition supplements that claim to enhance exercise performance by reducing lactic acid levels or by buffering the lactic acid that the muscles produce during exercise. But we now know that lactate does not cause muscle fatigue and is actually used as a fuel. Does this mean that all such supplements can't possibly work? Not necessarily.

Whenever any supplement is definitively proven to enhance exercise performance, it doesn't matter whether the proposed explanation for the effect is right or wrong. For example, sodium phosphate is an acid buffer that was shown to increase cycling time trial power by 9.8 percent in athletes who took the compound supplementally for six days. It almost certainly achieved this effect by absorbing hydrogen ions produced by the working muscles. It's just that these hydrogen ions were not produced through the same process that produces lactate, as was previously believed.

Supplements that enhance exercise performance and appear to reduce lactate production may achieve their effect not by preventing

lactate-induced muscle fatigue, but instead by increasing fat-burning capacity, as increased fat-burning capacity is associated with increased endurance and fat oxidation produces no lactate. For example, green tea extract has been shown to increase swimming endurance and fat oxidation and to reduce lactate production in mice. (These results have not been duplicated in humans, except that green tea extract has been shown to boost fat burning during low-intensity exercise in humans.)

Other supplements that enhance exercise performance and appear to reduce lactate production may achieve their effect not by preventing lactate-induced muscle fatigue and, indeed, not by reducing lactate production at all, but instead by enhancing lactate oxidation. For example, in a human study from Beijing Medical University, a supplement containing mushroom extracts was shown to reduce blood lactate levels during treadmill running. In a separate animal study, the same supplement was shown to increase swimming endurance and reduce blood lactate levels in mice. The authors of these studies speculated that the supplement worked by accelerating the lactate shuttling process.



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of lactate that the muscle cells produce—a strong piece of circumstantial evidence that lactate is somehow used within the cell. In fact, as much as 75 percent of the lactate produced by any given muscle cell never leaves it. Then, in 2006, Brooks was able to peer through a confocal microscope and all but see aerobic lactate metabolism in the mitochondria, the intracellular site of aerobic metabolism. Gathered together there he saw the transporter proteins that deliver lactate to the mitochondria, the enzymes that catalyze the first step of lactate breakdown and the protein complex where oxygen is used to complete the process of energy release. A smoking gun if there ever was one!

It would be difficult to overstate the magnitude of this discovery. George Brooks showed that there is a direct link between aerobic and anaerobic metabolism. In fact, what was previously thought to be anaerobic metabolism is actually just incomplete aerobic metabolism. During moderate-intensity exercise, most of the carbohydrate that is broken down for energy is processed aerobically and produces no lactate. But at high intensities, a second pathway—the lactate pathway—ramps up, giving the muscle two parallel pathways to release energy aerobically at very high rates to keep up with the muscle's energy demands. In this second pathway, glycogen or glucose is broken down to lactate without oxygen, and then lactate is broken down to carbon dioxide and water with oxygen.

Brooks is not done yet. His most recent research has examined the role of lactate in cell signaling. It suggests that the high levels of intracellular lactate that arise during intense exercise stimulate some of the beneficial fitness adaptations that occur in response to such training. Specifically, high lactate concentrations trigger the production of free radicals that “upregulate” a variety of genes. Some of these genes govern mitochondrial biogenesis. So it appears that intracellular lactate accumulation during intense exercise stimulates the muscle cell to produce more mitochondria, thus enhancing its ability to burn lactate (and other fuels) in future workouts.

If I had to package all of the forgoing science into a single upshot, it would be this: According to the classical theory of lactate, one of the highest priorities of training was to reduce the amount of lactate the body produces at higher exercise intensities so that the athlete can race faster without fatiguing due to high lactate levels. According to the new theory of lactate, one of the highest priorities of training is to increase the body's capacity to use lactate during high-intensity exercise so that the athlete can race faster.

So what practical difference does this shift make in terms of how we train? In truth, not much, because the advanced training methods that today's best-informed triathletes use were developed through blind trial and error, not fashioned consciously to conform to now-discredited ideas about lactate.

That said, for many years lactate-conscious coaches have counseled athletes to strictly limit the amount of training they do above the lactate threshold because the large amounts of lactate produced in such workouts are very stressful to the body. The rationale for this widely heeded caution has disappeared. It certainly remains true that the physiological stressfulness of exercise increases exponentially as the intensity does, such that the amount of training the body can handle is inversely related to its intensity. But lactate is not the reason. And lactate threshold intensity is not particularly high. In the typical trained triathlete it corresponds to the fastest swimming, cycling or running speed that can be sustained for one hour. There's plenty of room to go faster in your training without wearing yourself down.

Furthermore, as we have seen, far from stressing the body, high lactate levels trigger some of the most important performance-boosting muscle adaptations. You might not be able to handle a high volume of training above the lactate threshold (again, for reasons that have nothing to do with lactate), but the new science of lactate suggests that you should nonetheless go there frequently. Many triathletes wait until the race phase of training to introduce supra-threshold training into their bike and run regimens (swimming, as always, is another matter. Training in this discipline is entirely based on high-intensity interval work). It would be better to do a small amount of supra-threshold training throughout the training cycle, with the greatest volume of such training immediately preceding races, for those who compete in short-course events (because lactate threshold pace is close to race pace at these distances) and falling somewhat earlier for those who compete in long-course races.

How much supra-threshold training is enough? A Spanish study involving cross-country runners found that a mix of 81 percent moderate-intensity training, 10.5 percent lactate threshold training and 8.5 percent supra-threshold training produced optimal results. That 8.5 percentage is a sensible median target. All triathletes should do 5 percent of their bike and run training at supra-threshold intensities as a baseline. Short-course specialists can peak at roughly 12 percent and long-course triathletes at 8-10 percent.

Research has shown that the greatest lactate exposures occur during workouts consisting of 3- to 5-minute intervals at VO_2 max velocity separated by 2- to 3-minute active recoveries and in 30- or 60-second intervals at the same intensity separated by active recoveries of equal duration. VO_2 max velocity is approximately the fastest speed you can sustain for 10 minutes in swimming, cycling or running. Lactate interval workouts featuring shorter intervals are a bit more manageable and should therefore come earlier in the training process. Never try to do more than 20 total minutes of VO_2 max-intensity swimming, cycling or running during a single session. If you do, you will boil alive in toxic lactic acid.

Just kidding. ▲