It is not lactic acid's fault

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ABSTRACT

Lactic acid and lactate are widely believed to be the cause of fatigue, cramps and soreness in athletes. The authors take issue with this orthodoxy, citing a number of recent studies to support their view. They point out that it is possible to observe muscle fatigue while the lactic acid concentration in the muscle remains low and observe an absence of fatigue when the lactic acid concentration in the muscle is high. They argue that in many situations performance does not depend on the ability of the runner to produce less lactic acid, as many people think, but in the ability to produce more. They also question the existence of the anaerobic threshold – the point in exercise intensity beyond which the source energy moves from an aerobic metabolism to a combination of aerobic and anaerobic metabolisms - arguing that current scientific knowledge does not support its existence. If an anaerobic threshold really does exist, they say, it does not have all the uses people currently ascribe to it.

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'There's nothing so useless as a bad theory'.

Leonid Brezhnev

Introduction

You feel the pain as soon as you push yourself hard. Your muscles and your stomach hurt, not to mention your ego. And it stands to reason that if you feel pain, something has to take the blame for it. In the world of endurance sport, including running, it is claimed that lactic acid is the cause of all pain. You have cramps or stiffness? Lactic acid is the culprit. And while we're there, why not blame lactic acid for overuse injuries, overtraining, baldness, the decline in moral standards and continental drift?

The widely accepted belief that lactic acid is the source of all ills is still supported by some of those sports scientists who are not up to date or who have not had the courage to confess the truth to athletes, simply because they fear going against popular opinion.

Let's start with a fact: in high intensity exercise muscles produce lactic acid, which appears in the blood in the form of a salt that we call 'lactate'. If, however, we study the details of how energy is produced in the muscle during efforts of varying intensities, we find that lactate is not responsible for the ills that some think it is.

A simple but ill-defined theory

To blame lactic acid for muscular fatigue satisfies the simplistic logic that commands us to find a reason for every problem. And when we've gone as far as blaming something for a *particular* problem, why not accuse it of being liable for as many problems as possible? This approach offers many advantages. For one, we can avoid having to figure out the cause of each problem. For those who do not want to bother pondering on the process of fatigue, lactic acid is the perfect scapegoat. Nevertheless, reality is more complex than that.

In truth, the concept of a muscular fatigue does not exist; we should rather speak of muscular *fatigues*. Despite the fact that the outward signs of these fatigues may be the same (they make continued effort impossible) the fatigue felt by a 400m runner is not the same as that felt by a marathoner, which in turn is not the same as that felt by a body builder or a mountain-bike specialist, and so on. It would be naïve to think that lactic acid is the only villain responsible for these varied forms of muscular fatigue.

We find that between the brain's neurons (which send out the motor commands) and the muscles' myofilaments (which carry out these commands) there are several links in the information transmission chain that allows for power output. Any of these links can fall short in its task and so block the continuance of muscle contraction and exercise. Thus, these links appear on the long list of possible suspects in the development of the different types of fatigue. This list might, in some cases, include lactic acid. However, it is undoubtedly not the sole or even the main culprit.

Many research studies have shown that lactic acid is, in the end, only marginally responsible for muscular fatigue. As some of these arguments are quite complex, they will not be discussed here; we will only highlight that the most recent reviews on the subject all point to the conclusion that "The disturbance of the balance of the skeletal muscle acid base is not as critical a factor as is sometimes suggested" (FITTS, 1996; JONES et al., 2003; PÉRONNET and MORTON, 1994; PÉRONNET and THIBAULT, 2005, ROGBERGS et al., 2004; SCHWANE et al., 1983).

Myth 1: muscular cramps are caused by the presence of lactic acid in the muscle

A cramp is not the result of the accumulation of lactic acid. Of course, one can observe the occurrence of cramps at high lactate concentrations, but muscle lactate can be elevated, even towering, without cramps occurring. This is the case with the 400m race, where all the runners finish with a blood lactate concentration that is 20 to 25 times higher than that of the resting level, but where cramps are rare. On the contrary, some people suffer from cramps while sleeping, when the blood lactate concentration, as well as the effort level, is low.

In most cases, cramps occur during strenuous efforts of long duration, as in a very long training session. In such conditions, the lactate concentration is perhaps clearly higher than at rest, but far below the maximal levels observed during very intense but brief efforts. Therefore, one cannot blame the accumulation of lactic acid for the occurrence of cramps, which is unquestionably due to a hyper-excitability of muscular tissue or of the nerves that innervate it (SCHWELLNUSS et al., 2004).

One can also take into account the case of people with McArdle's disease. They cannot produce or accumulate lactic acid. However, they still suffer from cramps, a further argument confirming that lactic acid is not related to the occurrence of cramps.

Table 1: Effects of isometric exercise

	Immediately after an isometric exercise	Two minutes after an isometric exercise
Degree of muscle acidity	Very high	High
Fatigue	Very high	Very low
Strength	Weak	Normal

The most convincing argument in this debate is that on the one hand it is possible to observe muscle fatigue while the lactic acid concentration in the muscle *remains low* and, on the other hand, observe an absence of fatigue when the lactic acid concentration in the muscle is *high*. For example, at the end of a 100km race, a particularly demanding event, the fatigue level is quite high but the blood lactate concentration is not much higher than in the resting state. Moreover, people who suffer from McArdle's disease are incapable of producing (and thus of accumulating) lactic acid and are very prone to suffering from muscular fatigue. Thus, muscular fatigue can be accompanied by a *very low* lactic acid level, or even with *no* lactic acid at all.

On the other hand, if one performs an exhausting isometric effort with the quadriceps (e.g. the 'chair exercise', with the back leaning against the wall), fatigue will tend to reduce strength temporarily. However, this fatigue rapidly fades and goes away almost entirely after a two-minute period of recuperation: after this period, the muscle can once again produce the initial power. When observing the degree of acidity in the muscles, we see that it has increased considerably during the isometric contraction, which might support the hypothesis that asserts lactic acid is responsible for fatigue. However, during the recuperation period, the degree of acidity in the muscles only returns to normal rather slowly. Hence, two minutes after completion of the exercise, the degree of acidity remains very high but since

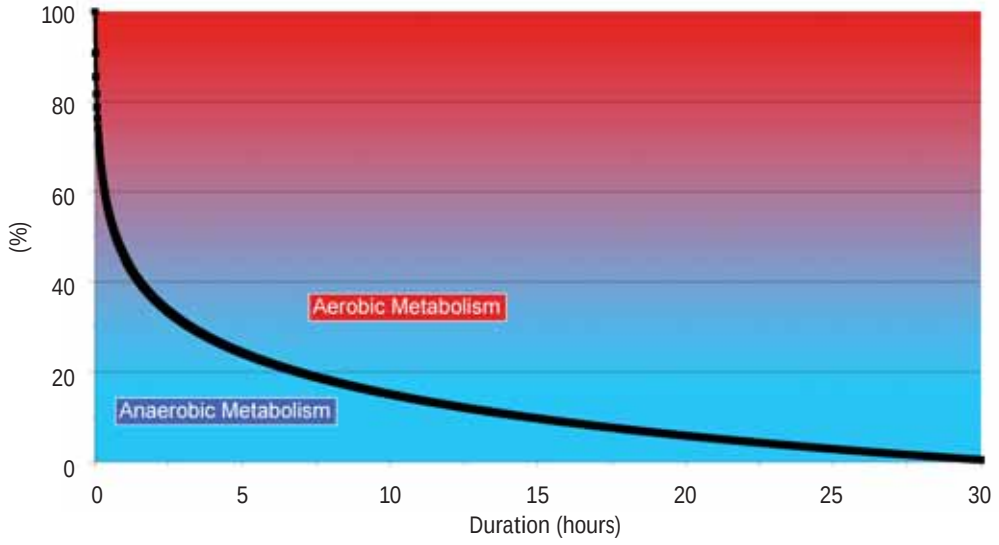

Figure 1: Percentage of energy generated by aerobic metabolism and by anaerobic metabolism, in relation to the duration of maximal efforts, supposing that each test is carried out from start to finish at a constant intensity (as is generally the case in athletics races run on a track or flat course).

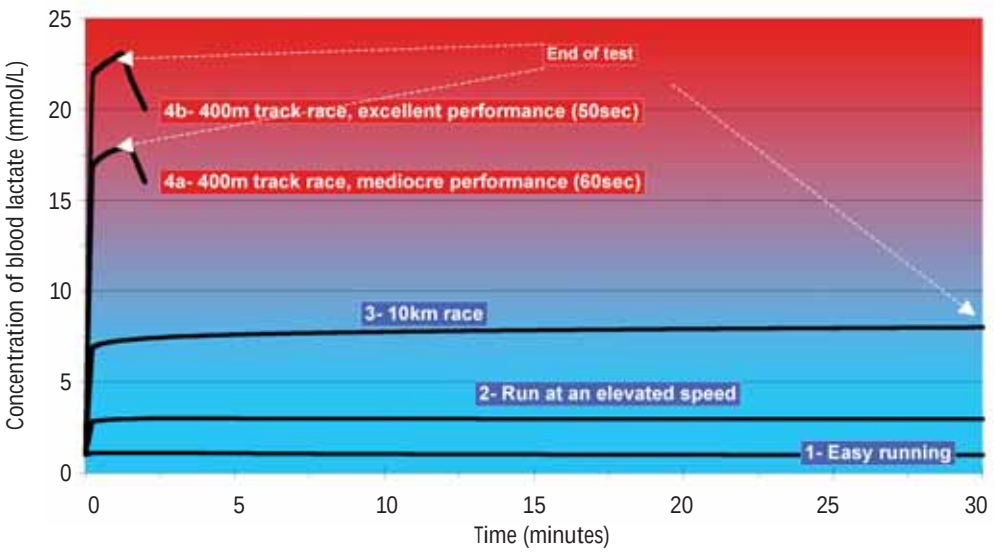

Figure 2: Schematic representation of the production of the blood lactate concentration during efforts of varying relative intensities.

the muscle can once again produce its initial force, the fatigue is obviously gone (see Table 1). For this reason, it is difficult to embrace the idea that the increase in lactic acid in the muscle causes fatigue, since a high degree of acidity *without* fatigue can be observed.

Myth 2: the presence of lactic acid in the muscle causes muscle stiffness and soreness

Delayed onset muscle soreness (DOMS) is the pain that appears a day or two after an unfamiliar intense effort. This type of pain occurs mainly when the exercise entails eccentric muscle contractions, namely contractions during which the muscles contract while lengthening themselves (e.g. absorbing the shock of a falling weight). These muscular pains have nothing to do with the presence of lactic acid in the muscles.

Sometimes lactic acid is accompanied by soreness, but it is also possible to get lactic acid without soreness and vice versa. Laboratory studies provide evidence to confirm this. In one study, the subjects had to run two interval tests (9 x 5 minutes at 7.5mph, with 2 minute recovery periods), first on the flat and then on a 10% descent. The flat run (higher concentration of lactate) did not generate soreness. In contrast, the day after the downhill run (lower concentration of lactate) the subjects suffered severe soreness (SCHWANE et al., 1983).

This is well known to people who run on hilly courses: it is neither the flat stretches nor the climbs that cause stiffness, but the downhill stretches, which call for a much larger number of eccentric contractions. These cause more damage to the musculature because the number of muscle fibres solicited to produce a contraction of a specific tension is 4 to 8 times as great for an eccentric contraction as opposed to a concentric contraction. The tension to which each fibre is subjected is therefore far greater, and this is what causes the microtraumas and the ensuing inflammation. This is a smart demonstration that lactic acid has nothing to do with muscle soreness.

To summarise: the diverse types of fatigue experienced by runners depend on a mixture of causes, depending on the type of effort; but nothing proves that lactic acid or lactate is the sole cause, nor even one of the major causes of any of these forms of fatigue.

Table 2: Advantages and disadvantages of anaerobic and aerobic energy production

Process	Advantages	Disadvantages
Anaerobic glycolysis	Energy produced at an elevated rhythm	Limited amount of total energy produced
Aerobic	High volume of total energy produced	Energy produced at a low rhythm (depends on VO_2max)

Which is better: producing more or less lactate?

Some believe that the more lactate you produce, the less effective you are. In reality, it is exactly the opposite. In numerous track and field events, if you produce more lactic acid, it is a sign that you are working at a higher intensity and therefore running at a higher speed.

In relatively violent efforts, like the 400m, 800m or 1500m, an important part of the energy used in muscular contraction comes from 'anaerobic glycolysis'. In this process, muscle cells produce the energy necessary for contraction through the degradation (-lysis) of glucose (glyco-), without using oxygen (anaerobic: *without* oxygen). If we compare it to aerobic (*with* oxygen) processes of energy production, anaerobic glycolysis, which always accompanies the release of lactic acid, has both advantages and drawbacks (see Table 2). Hence, in sprint and middle-distance events, where the quantity of energy produced during each second is very high, an especially large part of the energy is supplied by anaerobic glycolysis. In longer events, where the total quantity of energy deployed is increased, an extremely important part of the energy comes from the aerobic process (Figure 1).

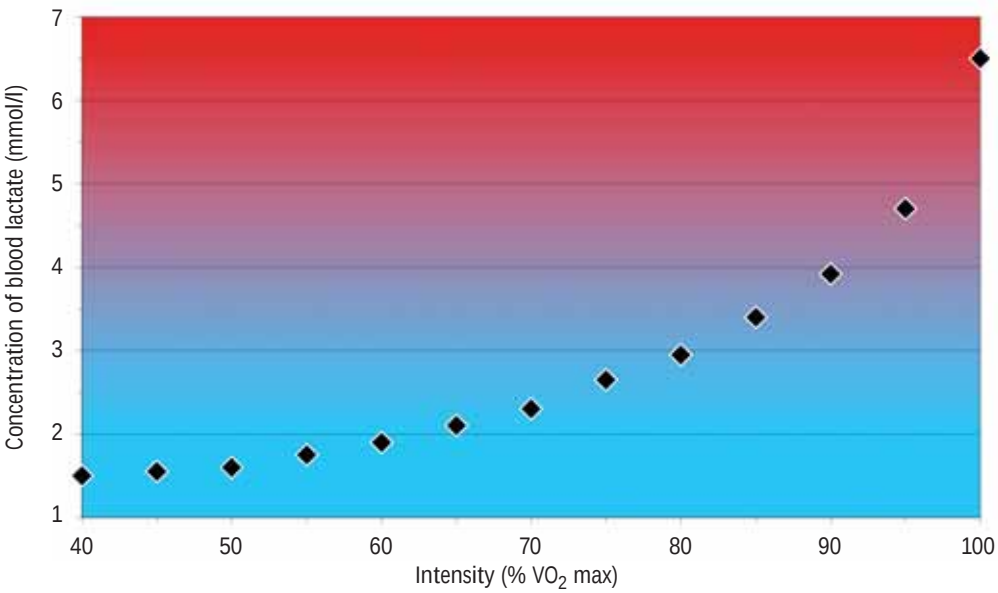

Figure 3: Lactate variations during the course of a maximal progressive treadmill test. Believers will see here a threshold, which a thorough examination does not confirm: it is simply a parabola without a deflection point (PÉRONNET and MORTON, 1994).

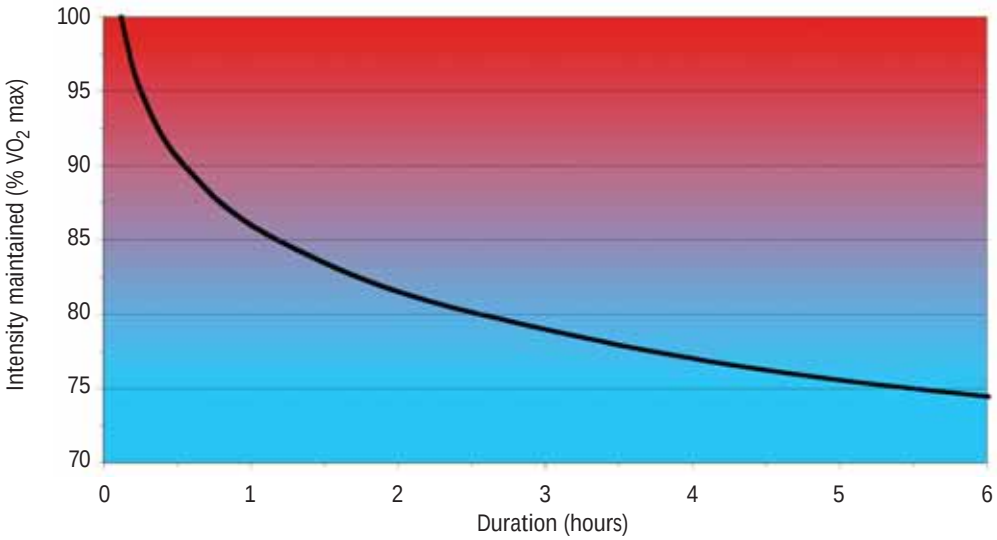

Figure 4: If runners are subjected to a series of maximal tests of different lengths, you find that the intensity attained reduces in proportion to the duration of the test: 100% of VO_2max for 7 minutes, 90% for 30 minutes, 80% for 2 1/2 hours, and so on. If the anaerobic threshold had the significance it is granted, this reduction would show a deflection with regard to the intensity corresponding to this famous threshold (PÉRONNET and THIBAULT, 1989).

Consequently, one should not be surprised that athletes engaging in brief, intense exercises produce a lot of lactate and that, as a result, the more they produce the better they perform. Thus (and as shown in Figure 2), the blood lactate concentration, which is about 1 mmol/l in the resting state, increases to about 18 mmol/l at the end of a 400m race for average runners, and up to 23 mmol/l for elite athletes. Therefore, in short efforts (10 seconds to 10 minutes), athletes who produce a lot of lactate, and thereby supply their muscles with much anaerobic energy, will tend to be those that succeed.

To believe or not to believe ... in the anaerobic threshold

There are those who have no doubt in the existence of the anaerobic threshold; that somewhere between the intensity of a leisurely jog and the most frantic sprint there is a point beyond which you go from aerobic metabolism to a combination of aerobic and anaerobic metabolisms. This convenient and attractive theory has many devotees at present. Popular magazines frequently cite it, implying that its existence is something that is generally agreed upon. Indeed, during a high intensity run for several minutes, you sometimes feel that it would require great courage to increase your speed by even the smallest amount.

However, current scientific knowledge refutes the anaerobic threshold theory. Presenting the details here would be tedious, but we highlight the following points:

- There is no power threshold below which a muscle does not produce lactate. A muscle constantly produces lactate, even from the lowest work level, and a muscle produces lactate even when the supply of oxygen is adequate.
- During a ramp test (such as the ones carried out in the laboratory in which the runner must run at a regularly increasing intensity until exhaustion), the blood lactate concentration never appears as a threshold, as some people argue. The curve obtained shows no deflection (Figure 3). To see one, a very fertile imagination is required. It is true that many sports scientists (whose fame is somewhat inferior to the revenues they obtain from the tests they conduct) unscrupulously possess such

an imagination but, in reality, the shape of this curve is most likely the result of a delay in the appearance of the lactate in the blood (PÉRONNET and MORTON, 1994).

For a given running intensity (for example, at 150 heart beats per minute), the lactate concentration decreases from the effect of following a good training programme. But this is not related to the anaerobic threshold: the reason behind this is definitely the fact that training improves the precision of metabolic control.

If an anaerobic threshold existed and if it had the physiological importance we now give it in sporting groups (transition aerobic-anaerobic), the relation between running intensity and critical time (the period during which a runner can sustain a given intensity) would not produce such an even curve (Figure 4) and there would be a deflection (PÉRONNET and THIBAULT, 1989).

If it were as demanding as believers think it is to exercise at an intensity above the anaerobic threshold, athletes could not perform at an intensity above that corresponding to the anaerobic threshold – which is what virtually all runners with the least motivation do over the classic distances such as 5km and 10km.

Measuring something that does not exist: what a challenge!

Moreover, finding the best way to identify this famous anaerobic threshold triggers some puzzlement. We have gathered a list of twenty or so ways: complex, subjective, dubious and harebrained. In this last category sits the famous Conconi test, valued by athletes but sternly criticised by scientists. These varied methods, simply because they are so different from one another, generate measures of the anaerobic threshold that are far too widely spread to be convincing. Indeed, one can see a relatively good correlation between, on the one hand, speed at the so called 'anaerobic threshold', and on the other hand, performance, for instance, in a 10,000m race. However, that can be explained: the speed that the runner can sustain at the anaerobic threshold (established by one means or another) depends on the maximal oxygen consumption (VO_2max) more than on any other factor. The higher the VO_2max, the better the performance, regardless of the event, as long as it lasts more than a few minutes.

Conclusion: not guilty

Lactic acid and lactate are not the cause of fatigue, cramps or muscle soreness. Performance in sprint and middle distance races depends on the ability of the runner to produce more, not less lactic acid. And if an anaerobic threshold exists, it definitely does not have all the uses people apply to it.

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