

# The contemporary role of lactate in exercise physiology and exercise prescription – a review of the literature

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## Abstract

Lactate is a key molecule in exercise metabolism. In the last two decades, there has been a revolution in the understanding of its role – from a byproduct of hypoxic muscles to a major energy resource and a signaling molecule – ‘lactormone’. The aim of this review is to compile all the up-to-date information that is available on the general metabolism of lactate, but also the specific use that exercise physiologists, coaches, and athletes can get out of lactate measurements, lactate-based training zones, and periodization of training sessions. Since the revolution in the understanding of lactate’s role in normal metabolism and disease in the late 20th century, more evidence has been assembled regarding the specific part it plays in exercise. From the vast body of knowledge, the researchers developed the concept of training intensity distribution into zones according to the level of blood lactate. The end goal of training to a specific lactate level or ‘threshold’ is the increased adaptation to specific training stimuli and ultimately better performance, which has been backed up by the achievements of numerous athletes training according to this concept.

## Keywords

endurance, lactic acid, metabolism, threshold

## Introduction

Lactate, also known as lactic acid, is an organic molecule that is produced in the process of glucose breakdown, named glycolysis. This catabolic process is widespread in all cellular types, from prokaryotes to complex organisms. In human cells, glycolysis terminates in one main product – pyruvate, which can be further metabolized in mitochondria and the Krebs cycle to yield ATP molecules or can be transformed into lactate by lactate dehydrogenase (LDH) in the cytosol.<sup>[1]</sup> Lactate was first discovered as a molecule in 1780 by Carl Wilhelm Scheele in milk, who named it ‘Mjölksyra’ which was later translated to lactic acid. In the next two centuries, as a result of numerous studies, namely by Pasteur, Warburg, Cori, and Claude Bernard, to name a

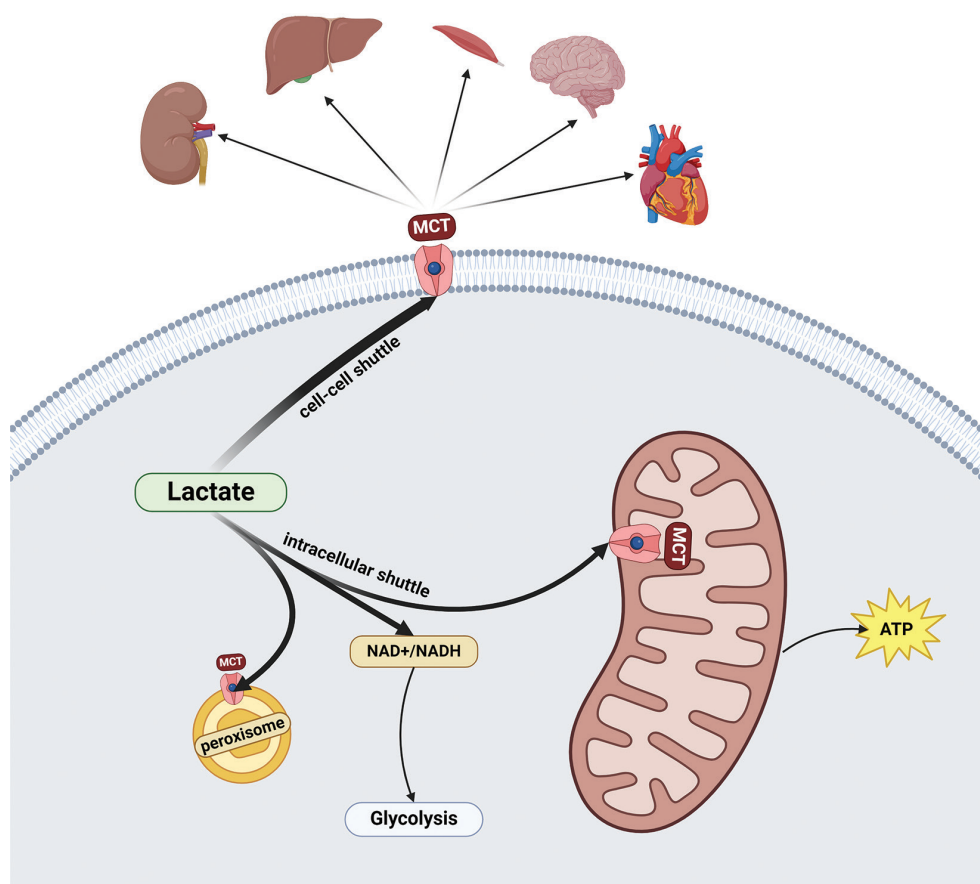
few, a correlative relationship was established between the production and accumulation of lactate and the amount of oxygen that is available to cells for their metabolic processes. This ‘classical view’ of lactate metabolism led to inception of the dogma that its production is solely related to hypoxia, which was later extrapolated to exercise physiology and muscle metabolism during exercise, despite the fact that extensive research conceived results that contradicted this but never challenged it.<sup>[2]</sup> Still, that view persisted through the majority of the twentieth century, and it made its way firmly into exercise physiology, where the term ‘anaerobic threshold’ was coined in the 1960s by Wasserman et al.<sup>[3]</sup> They defined it as the metabolic point during exercise where blood lactate concentration starts to increase and blood bicarbonate concentration starts to decrease. It

was not until the early 1980s that this concept was put to the test by Brooks et al.<sup>[4]</sup>, who demonstrated that lactate is not only not a metabolic waste and a marker for anaerobiosis but is produced under fully aerobic conditions and has the potential of a universal cellular fuel. While detailed lactate metabolism was still not clear, other researchers in the late 70s were starting to put their findings into practice. Kindermann et al.<sup>[5]</sup> were one of the pioneers of using lactate levels measured during exercise as a marker of intensity, which could be quantified and used to prescribe training at specific exercise intensities for enhancing performance capacity. Although using lactate as an effective training tool has been around for more than four decades, it has only gained traction recently thanks to the achievements of the Ingebrigtsen brothers in running<sup>[6]</sup>, Kristian Blummenfelt in triathlon<sup>[7]</sup>, Tadej Pogačar in cycling<sup>[8]</sup>, and many other elite athletes who have demonstrated that lactate-based training can bring tremendous performance results.

## Newer perspectives of lactate metabolism

Since the 1980s, a heap of new research has thwarted the traditional thought of what lactate actually is. The proposal of the lactate shuttle theory by George Brooks<sup>[9]</sup> has

significantly shifted the paradigm of lactate understanding – from a waste product and fatigue-inducer to a universal cellular fuel and a key metabolic ‘cog’ (Fig. 1). The gist of this hypothesis is that lactate is not produced in challenging conditions through only anaerobic glycolysis but rather is a major product during rest in normoxic conditions and even more so during exercise. This lactate is then ‘shuttled’ intracellularly but also to numerous organs and tissues which can directly oxidize it and use it for ATP production, as well as in the process of gluconeogenesis, all of that depending on the specific cellular needs. Examples of the cell-cell shuttles include the exchange of lactate between working muscles and the heart, brain and liver, red and white oxidative muscle fibers, astrocytes and neurons, etc. The intracellular shuttles on the other hand include the exchange between the cytosolic lactate and the mitochondria and peroxisomes.<sup>[10]</sup> Apart from its strictly energetic role, lactate can also serve as a signaling molecule – it has been appreciated that lactate supposedly stimulates and binds a myriad of cytosol targets which regulate cellular metabolism and immunity (PGC-1 $\alpha$ , HCAR-1, SIRT-1, SIRT-3, etc.). The downstream physiological effects of these interactions include mitochondrial biogenesis, lipid metabolism control, immune system control, etc.<sup>[11]</sup> These pathways, however, can be maladaptive when activated in pathologically transformed cells, such as in cancer, where



**Figure 1.** The lactate shuttle hypothesis. MCT: monocarboxylate transporter. Created with Biorender.

lactate metabolism is of gross importance to their survival and malignant behavior like metastasis.<sup>[12]</sup>

One of the basic tenets of the cell-cell lactate shuttle hypothesis is the discovery of the monocarboxylate transporter (MCT) system<sup>[13]</sup> – the proteins which transport lactate between different cells and the blood, but also between different organelles in the same cell by facilitated diffusion. These MCTs are ubiquitously found in cell membranes and are further subdivided into more than 14 types, which transport not only lactate, but also pyruvate, short-chain fatty acids, and monocarboxylate drugs.<sup>[14]</sup>

## Lactate in exercise

As a product of glycolysis, lactate's concentration rises steadily with the increasing rates of carbohydrate oxidation during physical activity and it can reach high values of approximately 20 mmol/l. Early research by Lundin and Strom<sup>[15]</sup> designated a correlation between the changes in blood lactate levels during exercise and the intensity of exercise or the amount of work that was being done. Although that relation was based on the assumption of a dysoxia-lactate dependence, it correlated well with other physiological determinants of physical exertion – mainly gas exchange, and that assumption stuck in the literature. These conclusions on the role of lactate in muscle were based on the research of prominent physiologists like Meyerhof, Fletcher, Hopkins, Hill, Dill, and many others, and it is important to note that current understanding does not dismiss but builds upon that knowledge.<sup>[16]</sup> We now understand the basic metabolism of lactate and its kinetics during exercise, and appreciate that glycolysis and increased carbohydrate oxidation is the driver of lactate accumulation in the blood, while hypoxia may play a secondary, if any, role. On the other hand, even during physical activity, lactate can be cleared via shuttling and oxidation – a quality that improves with higher levels of aerobic capacity, thus it is often used as a marker of improved endurance performance.<sup>[17,18]</sup> This adds to debunking the 'myth' of lactate's role in fatigue, since it is cleared rapidly after cessation of exercise. Contemporary research<sup>[19]</sup> identifies ATP depletion, oxygen insufficiency, impaired calcium kinetics, phosphate accumulation and neural mechanisms as main contributors to muscle fatigue, rather than lactate.

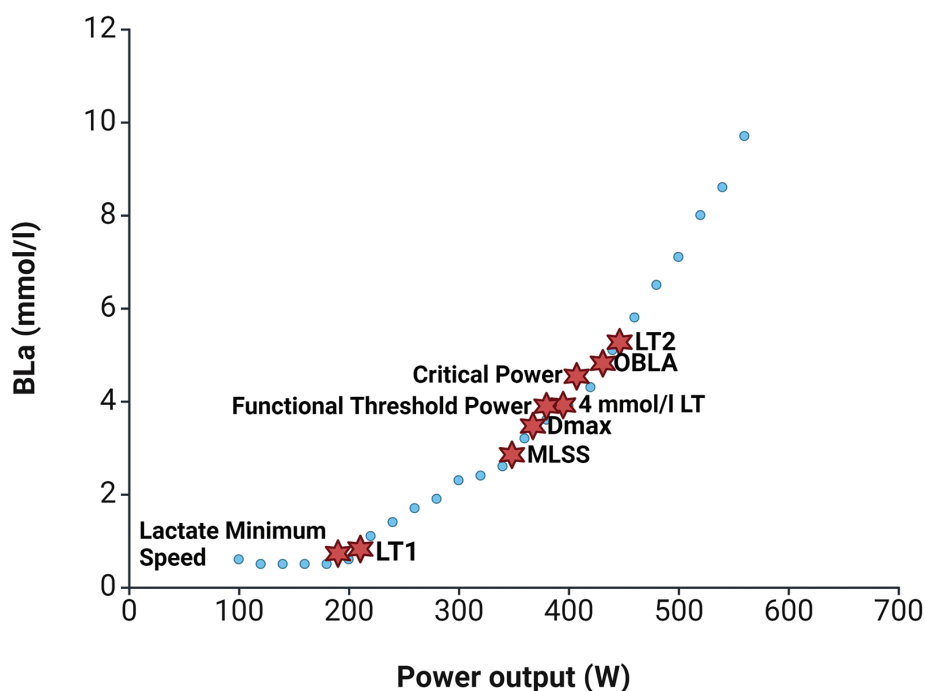
It is worth noting that for a long time it was considered a fact that lactate is also responsible for inducing the muscle acidosis seen in exercise, but that has also been challenged by Robergs et al.<sup>[20]</sup> His group, on the basis of earlier research, have argued that proton accumulation seen during exercise does not originate from lactate formation, but rather from the hydrolysis of ATP, which obligatory produces a free hydrogen proton.<sup>[21,22]</sup> Moreover, oxidative phosphorylation in mitochondria acts as a 'proton sink' which becomes less effective in extracting protons when vigorous exercise is performed above  $\dot{V}O_{2max}$ , and thus

still contributes to the acceleration of acidosis seen in exercise, irrespective of lactate.<sup>[20]</sup>

## The lactate threshold

As mentioned above, in the early 20th century, physiologists started using lactate as a measurement of intensity and plotting it against the work done during a graded exercise test, thus constructing the concept of the blood lactate curve. The first one to notice a specific point on that curve which signified an increase in the lactate concentration, owing to an increase in exercise intensity, was WH Owles<sup>[23]</sup>, which more or less is the first instance a 'lactate threshold' was acknowledged, although the term wasn't coined for another 35 years.<sup>[16]</sup> Scientists continued investigating the response of lactate concentration change in the blood to exercise, and the relation to intensity was further solidified by studies which showed the different lactate curves, matching graded intensity from light to severe.<sup>[24]</sup> Still, this remained a concept for the lab and it was not transferred to training until much later.

The first 'threshold' of any kind that was introduced was the anaerobic threshold (AT), coined by Wasserman and McIlroy<sup>[3]</sup> in 1964. They defined it as 'the metabolic point where bicarbonate concentration in the blood decreases and lactate concentration increases'. Although a valid assumption at the time, the researchers did not actually measure lactate levels in the blood during an exercise test – instead they measured the rate of gas exchange, the theoretical basis being that increased  $\dot{V}CO_2$  and respiratory exchange ratio (RER) above 1 signify the onset of anaerobic metabolism, which at the time was directly thought to be coupled with a blood lactate increase. The lactate threshold (LT) concept gained traction with the introduction of the enzymatic methods of measuring lactate from whole blood, which made it easier to assess lactate concentrations during laboratory exercise tests.<sup>[25]</sup> The invention of the test strip methods for measurement by Shimojo et al.<sup>[26,27]</sup> in the 1990s made it even more accessible and easy to implement and thus more popular between athletes. This accelerated research on the lactate threshold, using direct blood lactate measurements, but in time it produced a glaring problem – the lack of standardization and consensus on the terminology led to the inception of various threshold concepts, which aimed to describe the same physiological event but still differed enough so as not to be considered the same. As reviewed by Faude et al.<sup>[25]</sup>, up to 25 different LT concepts have been identified in the literature (**Fig. 2**). Generally, in the literature, there are agreed-upon and identified two main thresholds that signify two physiological events – the first LT, which marks the increase of blood lactate above resting levels, and the second LT, which marks the non-linear exponential increase in lactate concentration, which is thought of as the point where muscle metabolic acidosis accelerates. However, the debate is still revolving around the nature and exact meaning of the lactate thresholds more



**Figure 2.** Lactate threshold concepts. BLa: blood lactate; LT1, LT2: lactate threshold 1 and 2; MLSS: maximal lactate steady state; OBLA: onset of blood lactate accumulation. Created with Biorender.

so for the second LT than the first. Some concepts, however, stand out from the others as more popular or more accurate for determination of the second LT – the maximal lactate steady state (MLSS), onset of blood lactate accumulation (OBLA), the 4 mmol/l LT, the Dmax and modified Dmax methods.<sup>[28]</sup> MLSS is defined as the highest exercise intensity at which the arterial lactate concentration remains stable, albeit elevated from resting levels. The term was coined by Heck et al.<sup>[29]</sup> who developed a protocol for identifying this metric, which consists of several exercise bouts of ~30 minutes. Although accurate in measuring the aerobic-anaerobic transition around LT2, this method is not used often in everyday practice, as it is time and energy consuming. The OBLA is defined rather vaguely as the starting point of exponential increase in blood lactate concentration during graded exercise and it was again introduced to signify that metabolic shift.<sup>[30]</sup> Mader et al.<sup>[31]</sup> chose to show that shift with a specific value, hence why they developed the 4 mmol/l LT, which relies on the power at which 4 mmol/l of lactate is reached in blood, to determine the second LT, and that specifically had been an established long-standing belief among exercise scientists of where LT2 is. The Dmax and modified Dmax are mathematical methods for calculating the second LT by identifying the point on the third order polynomial lactate curve, which lies at the longest perpendicular distance from a line, drawn between the highest and lowest measured lactate values on that curve. The method was proposed by Cheng et al.<sup>[32]</sup> and modified by Bishop et al.<sup>[33]</sup> All those methods for determining the second threshold are valid, but the modified Dmax has been shown by Heuberger et al.<sup>[28]</sup> to

produce the least variance between successive calculations and also predict performance most accurately, when used to distribute intensity into zones, which are then used for exercise prescription.

## Application of theory into practice

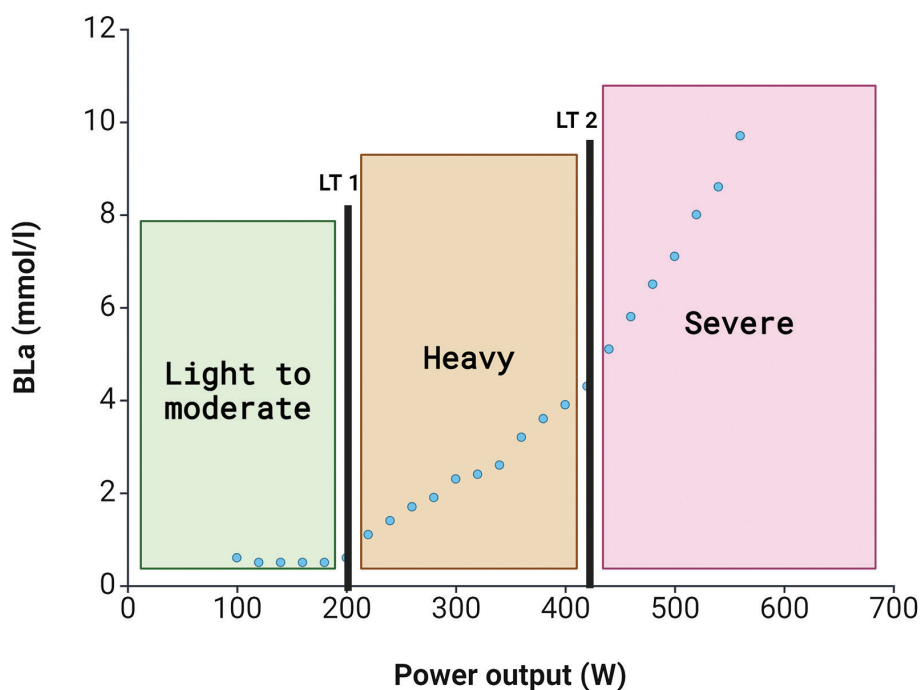
When the anaerobic threshold concept was introduced by Wasserman<sup>[3]</sup>, the main idea behind it was to quantify the physical capacity of cardiac failure patients. The first ones to propose the threshold concept for enhancing athlete performance were Kindermann et al.<sup>[5]</sup>, who appreciated that different intensity of training elicited different physiological responses and stimuli. They also acknowledged that the proposed ‘anaerobic’ threshold correlated with only slight increases in lactate up to 2 mmol/l and was actually a work rate that can be maintained for long periods of time, hence not anaerobic after all. This is why these authors proposed a new framework of two thresholds akin to the lactate thresholds discussed above – the aerobic and anaerobic threshold and also pioneered a line of thought of using that framework for prescribing exercise at different intensities, which could overall enhance performance. On that basis, other research in the field in the 1970s by Skinner and Mclellan<sup>[34]</sup> further developed this idea into a three-phase model of intensity distribution in graded exercise, describing in detail changes in metabolism, muscle, cardiovascular, and respiratory physiology. As discussed above, the nomenclature around the thresholds is quite unclear, owing to the fact that there is no standardization, but

the concept that Kindermann introduced and was further outlined and described by Skinner, has been tested in time and shown to be accurate enough for practical use and exercise prescription.<sup>[35]</sup>

An important advance in exercise science and, in general, in sport has been the introduction of training periodization at the start of the 20th century, which by the 1960s was well developed, especially in the USSR.<sup>[36]</sup> This training method tied well with the newly developed idea of intensity distribution in the 1980s and from this emerged the zonal models of intensity distribution. Although the concept was adopted earlier by some running coaches, it was not based on physiological metrics, but on pace, speed, or perceived exertion. The zonal distribution follows closely the model established by Skinner – three zones, where the transitions are marked by two thresholds, regardless of whether lactate or gas exchange is used to determine them (Fig. 3). Although models with up to seven zones exist, the one most firmly based on detectable physiological phenomena is the three-zone model; hence, the zones that are described later in this paper always refer to it. The first zone below the first threshold relates to light or moderate exercise intensity, the second zone between the two thresholds signifies a heavy intensity domain, and the third zone above the second threshold is termed as the severe intensity domain. The zonal intensity distribution is individual for every athlete and is obtained during a graded exercise test, where apart from lactate, other physiological metrics such as heart rate, ventilation rate,  $\text{VO}_2\text{max}$  and power output are also measured. These models of intensity distribution have grown to be very important in training of elite athletes, especially when talking about endurance sports. Virtually all profes-

sional athletes train according to some model and three stand out and are most commonly used: the pyramidal, polarized, and threshold.<sup>[37,38]</sup> The names of the models refer to the amount of training sessions performed at the three intensity levels. A pyramidal model includes a big percentage of training designated to zone 1, smaller to zone 2, and the least amount to zone 3. On the contrary, a polarized model prescribes training mostly between zone 1 and 3 in a 80:20 ratio and none in zone 2.<sup>[38]</sup> The use of these two models has been more popular since the 1990s and 2000s, but a newer version in the face of the threshold model has emerged in the last 15 years. It consists of a larger amount of training dedicated to zone 2 between the two lactate thresholds, with training sessions termed ‘threshold’, which are notorious with the need for rigorous on-field lactate testing.<sup>[39]</sup> This method was popularized mainly owing to the achievements of the Ingebrigtsen brothers<sup>[6]</sup> – European, World and Olympic champions in middle and long distance running; however, it is rarely adopted as a whole, even by them, since it brings a large training load and injury risk. Rather, some training elements and sessions are incorporated into the pyramidal model such as ‘double threshold’ sessions and active lactate testing during these specific sessions. These techniques have shown a profound effect on performance, although anecdotal, in other athletes such as the former Olympic champion in triathlon Kristian Blummenfelt.<sup>[7,40]</sup>

The scientific literature is equivocal on the most effective model when looking at performance results. There is conflicting evidence supporting the use of the polarized model<sup>[41]</sup>, but also such for the pyramidal model, while the threshold model, as mentioned, is rarely used, so direct



**Figure 3.** Domains of training intensity distribution. BLa: blood lactate; W: watts. Created with Biorender.

evidence is rather lacking.<sup>[38,40]</sup> Nonetheless, the above examples of athletes are in favor of using a key quality of the threshold approach – lactate-guided interval training, as an emerging novelty in endurance sports.<sup>[40]</sup>

Last but not least, another use of lactate measurement is as a component of estimating maximal anaerobic power, which is an important quality in sports where sustained sprinting is necessary. The specific metric in question is called maximal rate of lactate accumulation or  $VLa_{max}$  and it gives information on the ability of the muscles to use anaerobic glycolysis as a source of ATP resynthesis.<sup>[42,43]</sup> It is calculated in a sprinting test, where maximal exercise is performed for a short period of time, typically between 10 and 30 seconds.<sup>[44]</sup> Such a test theoretically stimulates maximally the ability of muscle fibers to use glycolysis. Lactate, as a main product of this process, is measured before and after the test and the resulting values are used to calculate the  $VLa_{max}$ .<sup>[42]</sup> It has been suggested that a high  $VLa_{max}$  value predicts better sprinting and short distance running performance.<sup>[43]</sup>

## Summary

The thought model on lactate's role in metabolism and exercise has shifted dramatically in recent years. This has opened the door for it to be considered as a valuable tool in the kit of exercise science professionals and athletes and it is now widely used for exercise testing, prescription and assessing performance capacity. It also has an emerging value as an on-field marker for quantifying intensity, which is the basis of lactate-guided training. It remains to be seen what the long-term performance effects of this type of training are, which is an intriguing topic for future research.

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## Competing interests

The author has declared that no competing interests exist.

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