



Advanced Sports Medicine Concepts and Controversies

Lactate: Friend or Foe

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Abstract

Lactic acid has played an important role in the traditional theory of muscle fatigue and limitation of endurance exercise performance. It has been called a waste product of anaerobic metabolism and has been believed to be responsible for the uncomfortable “burn” of intense exercise and directly responsible for the metabolic acidosis of exercise, leading to decreased muscle contractility and ultimately cessation of exercise. Although this premise has been commonly taught, it is not supported by the scientific literature and has led to a great deal of confusion among the sports medicine and exercise science communities. This review will provide the sports medicine clinician with an understanding of contemporary lactate theories, including lactate’s role in energy production, its contributions to metabolic acidosis, and its function as an energy substrate for a variety of tissues. Lactate threshold concepts will also be discussed, including a practical approach to understanding prediction of performance and monitoring of training progress based on these parameters.

Introduction

Lactic acid has played an important role in the traditional theory of muscle fatigue and limitation of endurance exercise performance. It was thought that once exercise intensity exceeds the rate of maximal oxygen consumption (V_{O_2max}), then an “oxygen debt” occurs and metabolism switches from aerobic to anaerobic. This switch to anaerobic metabolism was thought to lead to an abrupt increase in blood lactate levels, resulting in metabolic acidosis. This lactic acidosis was believed to impair muscle contractility and, ultimately, to lead to fatigue, exhaustion, and cessation of exercise. The uncomfortable feelings within muscles working at these near-maximal efforts were believed to be directly associated with this lactic acidosis, as was the soreness that developed during subsequent days (now commonly referred to as delayed-onset muscle soreness). Thus lactic acid was believed to be little more than a metabolic waste product, the result of pushing our systems beyond our capacity to deliver an adequate oxygen supply to our working muscles. This line of thought led to the establishment of training programs that sought to increase maximal oxygen capacity through high-volume, lower intensity exercise and led many persons to be wary of exposing the

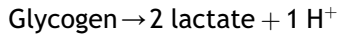
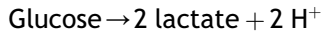
body too frequently to bouts of lactic acid–producing intensity.

Scientific thought has evolved during the past 30 years, and new understandings of the role of lactate in energy metabolism have altered these traditional teachings. Unfortunately, many misconceptions continue to permeate the sports medicine and exercise science communities. It is not uncommon to hear phrases such as “lactic acid burn” and “flushing out lactic acid” even among well-respected coaches in the endurance community. Although the exact mechanisms by which lactate metabolism affects endurance performances continue to be defined in the literature, several key concepts are important for all sports medicine clinicians to understand. It is also important to understand basic concepts of how lactate measurements are used in predicting performance and designing training programs and the inherent limitations of individual lactate measurement.

Energy Production and Lactate Kinetics***Lactic Acid Versus Lactate: An Important Differentiation***

Despite the ubiquitous use of the term “lactic acid” in both scientific and lay fitness and sports medicine

communities, the actual presence of meaningful quantities of lactic acid in the human body has been called into question. It is true that the glycolytic production of lactate is associated with hydrogen ion (H^+) production, as represented in the following summary equations [1]:



However, as detailed in the 2004 review of the biochemistry of exercise-induced metabolic acidosis by Robergs, Ghiasvand, and Parker, these summary equations do not imply that lactate is the source of H^+ , but rather that the proton release of glycolysis is likely associated with the non-mitochondrial hydrolysis of adenosine triphosphate (ATP) [1]. Although other explanations for H^+ formation have been proposed, most investigators now agree that lactic acid is not produced in muscle [2]. Although the construct of "lactic acidosis" appears intuitive and continues to be propagated in physiology texts and medical education, no convincing evidence exists in support of this theory. Regardless of whether this stance represents an "entrenched sloppy nomenclature" as suggested by Lindinger et al [2] or a true inherent misunderstanding of lactate's production, it undoubtedly leads to confusion among many sports medicine clinicians. For this reason, we will only use the term *lactate*.

Glycolysis, Metabolic Acidosis, and Lactate Production: What is the Connection?

Detailed reviews of glycolysis, metabolic acidosis, and lactate kinetics are beyond the scope of this review [1-4]. However, it is important to discuss the key concepts so that the role of lactate in energy production and exercise performance can be better understood.

The energy molecule ATP is required for muscle contraction. With increasing exercise duration, phosphocreatine stores decline and muscle glycogen, or circulating blood glucose, is shuttled through the glycolytic pathway, forming ATP and pyruvate [5]. Both glycolysis and glycogenolysis produce the same number of pyruvate, but glycolysis is associated with the net release of $2 H^+$, whereas glycogenolysis yields only $1 H^+$, but also an additional ATP [1]. The pyruvate is then shuttled into the mitochondria, where it undergoes oxidative phosphorylation, which produces an abundance of ATP to allow for ongoing muscle contraction (Figure 1). As exercise intensity increases, the mitochondria are unable to oxidize all the available pyruvate. The increasing concentrations of pyruvate then trigger the conversion of pyruvate to lactate via the enzyme lactate dehydrogenase [3]. It has been argued that the lactate dehydrogenase reaction not only supports ongoing glycolysis via maintenance of cytosolic

redox potential (oxidized nicotinamide adenine dinucleotide [NAD^+]/reduced NAD [$NADH$]) but that it also consumes a proton and effectively buffers against acidosis [1].

The origin of metabolic acidosis continues to be debated, but it seems clear that it is not directly related to lactic acid. Robergs et al [1] argue that non-mitochondrial ATP turnover is the source of H^+ , as previously described. Lindinger et al [2] have proposed that, based on physiochemical principles, the strong acid anions (namely, lactate⁻) that are produced with increasing glycolytic activity necessitate an increase in the net positive charge to maintain electroneutrality, and this positive charge is primarily provided by the dissociation of water.

More important than the exact mechanism of metabolic acidosis are the effects. Many of the misconceptions regarding lactate are directly related to the premise that acidosis is a primary cause of muscular fatigue and cessation of exercise. However, more recent studies have demonstrated limited effects on skeletal muscle contraction from induced acidosis, and in vitro studies have reported a protective effect of acidosis from hyperkalemic force depression in skeletal muscle [5]. Other beneficial effects of acidosis have been described, including greater release of oxygen from hemoglobin, ventilatory stimulation, enhanced muscular blood flow, and increased cardiovascular drive [5]. It is clear that the role of lactate in metabolic acidosis and fatigue must be reassessed.

Lactate Shuttles: What Happens to the Lactate Produced During Glycolysis?

Brooks [6] introduced the concept of cell to cell "lactate shuttles" more than 30 years ago. Ongoing research continues to expand and define the complex mechanisms at play both between cells and within cellular compartments. What has become clear is that lactate is not a waste product of anaerobic metabolism but rather an important fuel and potential signaling molecule that is continuously formed and utilized even under fully aerobic conditions [7].

The production of lactate, although likely oversimplified, was previously described. At this point, several pathways can be taken, all of which are facilitated by the monocarboxylate transport proteins (MCTs; Figure 2). Lactate can be transported into the mitochondria to be oxidized or transported into peroxisomes coupled with the reoxidation of NADH, which is required for function of β -oxidation [7].

Alternatively, lactate can be shuttled out of the cell via an MCT, possibly in conjunction with the extracellular transport of H^+ [1]. This blood lactate can then be taken up and used as fuel by adjacent skeletal muscle, as well as the heart, brain, liver, and kidneys [3,7]. During exercise, oxidation accounts for approximately

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