

# The Paradox of Aerobic Fitness Prescription: A Facultative Anaerobe Sucks the Air Out of VO<sub>2</sub>max

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Have you ever thought about what it is exactly that drives improvement in aerobic work capacity? If you are like most people you really haven't felt compelled to ponder this. Even though I am trained pretty extensively in cardiovascular physiology and training theory I am an anaerobe and a musclehead. What makes muscle work, become stronger, bigger, or more powerful is my interest. That means that I hadn't, until recently, considered the question either. In fact, if I would have been asked that question by someone 2 years ago I probably would have pulled the answer out of some old aerobic dogma buried in my brain somewhere, obtained from reading texts and research journals or from sitting in a lecture hall somewhere. I accepted fairly unquestioningly, albeit with a few exceptions in programming issues, the conventional wisdom of aerobic training physiology. I was a happy camper. I didn't know I actually cared about a higher level of understanding pertaining to aerobic fitness.

When Mark Rippetoe and I decided to develop and publish a rational approach to strength training, it was in response to the vast amount of ill-conceived and poorly designed training models presented to the world as being authoritative. We both understood that many many people were lifting and programming incorrectly. We really didn't understand why what was obvious to a couple decent ex-competitors and pretty successful practitioners was not obvious to the rest of the weight training world. When we starting researching our books and digging into theory and authoritative documents it was extremely surprising to both of us to find a

tremendous lack of real and meaningful experimental data. It was virtually impossible to find well designed and well controlled experiments actually asking research questions, even simple ones, relevant to the practicing fitness professional or any trainee. It was also eye-opening to find so many people of all ilks defending the poorly founded conventional wisdom of "resistance training". Of course they didn't and don't know it is not a well founded doctrine.

Recently through Mark's professional practice, I have been fascinated by the CrossFit model of training. Seeing the improvements in the endurance aspect of fitness in the local CrossFitters has posed a new puzzle. Why did they get aerobically fit when they did not train in a manner that could be considered "aerobic"? Their amazing success didn't fit into the convenient box of aerobic training dogma. I asked other faculty with aerobic interests about what could be driving this fitness improvement and gained very little satisfaction. And as a professor who feels compelled to explain things to people, not being able to explain this phenomena really bugged me. This kind of stuff will wake a professor up at 3:00 am and compel them to search the National Library of Medicine on-line until dawn to find an answer to a piece of the puzzle that subconsciously emerged in sleep. So began a broader search for explanation, a search that demonstrated that the state of endurance training theory has uncanny parallels to the state of understanding in the strength arena. The answers to simple questions were hard to find and most of the literature didn't seem to stand up to scrutiny in respect to utility. It seems as though instead of asking what

drives adaptation in  $VO_2\text{max}$ , most researchers in exercise academic circles have been interested in what limits  $VO_2\text{max}$ . Understanding human limitations is a noble effort but fairly futile if you do not understand the process of inducing the physiological adaptations that move the body towards those limitations.

In 1936 Hans Selye proposed the General Adaptation Syndrome theory, an explanation of how the body responds to injurious and non-injurious stress. Selye proposed that the organism goes through a programmed series of physiologic responses and adaptations to ensure survival when the organism is exposed to the same or similar stress later in the life cycle. In the exercise and fitness sciences, this theory is well accepted but frequently misunderstood and misapplied. The crux of the correct application of Selye's theory is to understand that a disruption of homeostasis must occur in the physiological system of interest in order for adaptation and fitness improvement to occur in that same system.

One of the most apparent examples of the misuse, or more precisely, ignorance of the appropriate use of Selye's theory can be found on the holy ground of aerobic fitness. The fitness boon was born in the late 60's under the guidance of Jim Fixx and Kenneth Cooper. The idea was, and still is, simple. Run a lot and you will be fit and healthy. Over the decades, the mythology of running has firmly entrenched into conventional wisdom the idea that developing aerobic fitness (endurance) requires you to run – run long and run slow. The American College of Sports Medicine (ACSM) recommends 30-60 minutes of continuous low-to-moderate intensity aerobic activity in order to develop aerobic fitness. A problem immediately presents itself with this training concept. With low to moderate intensity running, the ultimate marker of aerobic fitness  $VO_2\text{max}$  – the

maximum amount of oxygen the body can consume at maximal effort – is not challenged. In the classical aerobic exercise prescription for improving aerobic fitness for the masses, the demand for oxygen at the working muscle is met by supply. The name itself says it all – aerobic – in the presence of oxygen. This means that by definition, this type of training cannot provide a disruption of oxygen homeostasis. With no homeostatic disruption there can be no adaptation and fitness gain. That is IF we believe Selye's theory. And of course we do. So it is obvious that the accepted prescription of long-slow-distance exercise for improving aerobic fitness lacks both substantial theoretical support and logic.

But tell any fitness trainer, exercise scientist, allied health professional, or physician that they are approaching the development of  $VO_2\text{max}$  incorrectly and they will claim heresy on your part and question your sanity, your IQ, and familial heritage. Just by writing these words for publication I am painting a target on my academic standing. It is an invitation to an open season on the aerobic heretic. But I will stick to my guns and here's why. Open discussion and objective examination of fact form the cornerstone of science and academia. It is my profession to pontificate. Even if I am wrongly assessing how the body responds to exercise every exercise professional, clinician, and scientist should be and is welcome to dissect and examine my thoughts and supporting data in an open forum. That's what academia is, exploration and explanation of the world around and within us – the search for truth. If we do not freely think and pose radically different ideas from convention when convention may be in err, then we are no longer academics, we are lemmings.

So I have posited here that everyone including the ACSM is approaching training for

improving  $\text{VO}_2\text{max}$  in a theoretically incorrect manner. That must mean that if I know that something is being done wrong, I must know how to do it right. Right? Of course I do, or at the least I have a very good explanation of what is actually being trained with conventional training methods.

We have considered in the previous discussion above the standard exercise prescription for the untrained and non-competitive subject. Let's turn our attention from the laboratory and clinic to the competitive field. Coaches do not use ACSM recommendations to improve  $\text{VO}_2\text{max}$  and performance in their athletes. They do not and would not have a trainee run at 70% of  $\text{VO}_2\text{max}$  for an hour in every training session. They know that performance is unaffected by this and what does not work in the field is abandoned in the field. Practical experience from more than a century past has demonstrated that this is an ineffective means of increasing  $\text{VO}_2\text{max}$  and performance. The only time you will see 70% runs being used is on a training day designated for recovery. A 70% workload cannot disrupt oxygen homeostasis. It is used for recovery training as it is easy enough on the body to allow for physiologic recovery from more rigorous training methods without losing neuromuscular condition.

To more fully examine the methods used in the field let's divide training for aerobic fitness into two basic types of training, long-slow-distance and interval training. There are many variations of both of these types but in large part the variants are fairly similar and can be described as follows (see table 1 for a more extended analysis). Long-slow-distance work is intended by convention to improve cardiovascular efficiency and  $\text{VO}_2\text{max}$  and interval training is intended to improve lactate tolerance/clearance and  $\text{VO}_2\text{max}$ . Both of these types of training have been

demonstrated to improve endurance performance and  $\text{VO}_2\text{max}$  and this is where it gets tricky. Two different training methods, two different sets of metabolic demands, and they both yield the same result. How can this be? Part of the answer can be found by considering the population on which the majority of research has been done, usually low to average fit individuals just starting a training program. In other words a beginner. A beginner is very far away from their genetic potential for performance and as such a very low level and non-specific stress can induce positive adaptations. We can basically have a beginner walk, jog, sprint, jump, twist, flex, wiggle, dance, swing, hang, roll, bounce, do virtually any activity, and improve endurance. This is so because any advancement of metabolic and oxygen demand beyond their sedentary lifestyle is a novel and disruptive stress and will induce an improvement in endurance and  $\text{VO}_2\text{max}$ . This concept of beginners responding to a non-specific stress is not unique to aerobic exercise. In the realm of strength development, you can have a beginner ride a bike and they will improve their squat performance. But put an intermediate, advanced, or elite trainee on a bike and they will not improve their squat. They need to do squats, they require specificity. Considering data from beginner populations to be relevant to trainees at every level of training advancement is a gross and progress defeating mistake.

If much of the data we have is flawed or uninformative, how are we supposed to know how to train people? We use our brains. Let's consider what specifically each of these two field methods of training do to the body. We've already established that long-slow-distance training cannot, by definition, disrupt oxygen delivery and utilization to a point of homeostatic disruption. But we also know that

endurance can be enhanced by this type of training. Why the incongruity? It's not really incongruent it's just confusing because of lax and complex terminology academics and clinicians have devised over the years. Endurance isn't just  $VO_2$ , there are more facets to it than that. But let's keep it simple here (**Keep It Simple Scientist**) and examine the two major facets of endurance, (1) energy and (2) oxygen.

Long-slow-distance training is energy substrate depleting in nature. It has been shown many times over that glycogen stores can be totally depleted with this type of training, and depletion of an energy substrate should be considered a fairly significant homeostatic disruption of metabolism. It would not be prudent to consider only complete depletion as a disruptive stress, partial depletions should be considered disruptive as well IF AND ONLY IF the depletion is larger than previously experienced by the trainee. This type of training can also exceed the body's ability to metabolize fat for energy. Driving a metabolic system beyond its normal range of operation or to failure is definitely a disruption of homeostasis. Combined, the stress of glycogen depletion below normally experienced levels while simultaneously exceeding fat metabolic capacity drives an improvement in storing and utilizing these two energetic substrates and results in being able to run longer – thus endurance has improved but  $VO_2$ max has not improved. This is a specific adaptation to a specific stress in a previously trained subject. This seems obvious, but most people fail to see this connection between aerobic exercise, metabolism, and performance and automatically, and incorrectly, attribute the improvement in endurance to an improvement in  $VO_2$ max.

The second common type of training done for aerobic fitness is interval training, shorter and more

intense segments of effort with little rest between repeats. It has been observed that lactic acid accumulates during this type of training and thus it is commonly accepted that intervals push the body to adapt to the presence of lactate by enabling it to tolerate higher concentrations. Alternatively, it is suggested that interval training may enable a quicker removal of lactate from the tissues and blood. This seems nice and logical, but it is off base. Although we are inundated with (mis)information that lactic acid is bad, lactic acid is an essential hydrogen ion acceptor in glycolytic metabolism. Sure the exercise conditions that are associated with its accumulation are a bit uncomfortable but correlation is not causation. So do we really care that lactate has accumulated? We really shouldn't, lactate isn't even part of aerobic metabolism and  $VO_2$ max, it is simply an inevitable consequence of the really important things happening here. With interval training, producing lactate is not important per se, exceeding oxygen consumption capacity is. Intervals are done in the glycolytic realm, whereas long-slow-distance is primarily oxidative. Running fast enough to require the primary metabolic systems for exercise support to be glycolytic, specifically anaerobic glycolysis, means that the working muscle cannot take up and use oxygen fast enough to meet exercise driven demand. If anything, significant lactic acid accumulation occurs coincident with disruption of oxygen homeostasis. The level of exertion where lots of lactate is produced is the level of exertion needed to drive improvements in  $VO_2$ max. It's the level of exertion where we have exceeded oxygen consumption capacity. The body will adapt to this stress by augmenting its ability to take up oxygen and to use it in the muscle - if this type of training is repeated chronically and progressively. It has been traditionally suggested that interval training should

account for about 5% of a runner's total mileage and this is a gross under-use of this training method. Lots of aerobic athletes use intervals. Many use them for the wrong reason. Regardless of the decision process used to justify their inclusion, they should likely do more, lots more. Most runners use interval intensities of between 85% to 105% of  $VO_2\max$  (usually calculated as a speed just slightly faster than race pace). Intervals need to be short and intense. Trained runners can run many miles at 85% of  $VO_2\max$ , so the low end of the common interval prescription is not useful. At the upper end, 105% is just barely enough intensity to drive any type of positive oxygen handling adaptation. Productive intervals will have intensities in the range of 150-250% of  $VO_2\max$ . To maximize gains trainees should run faster, a lot faster.

I've stated that it is the uptake and utilization of oxygen at the muscle that is the driving force of  $VO_2\max$  gain. And guess what? It really doesn't involve a great deal of cardiovascular adaptation. Rather, the adaptation must, by physiological necessity, be at and in the muscle. Changes in metabolic enzyme concentrations, membrane glucose transporters, myoglobin concentrations, and other phenomena localized to the working muscle enable more efficient extraction of oxygen from the blood and utilization in the cell. All these enable the muscle to consume more oxygen. Remember that the definition of  $VO_2\max$ , the absolute maker of aerobic fitness, has as a center piece of its definition "consumption of oxygen". It is not defined by the ability of the heart, lungs, and vasculature to deliver oxygen. Here lies the heresy. Consumption does not relate strongly to delivery. To state that to develop  $VO_2\max$  one does not need to significantly develop the heart and lungs through traditional aerobic training is not intuitive. So let's clarify with one important

piece of data to make sure this is correctly understood. At rest only a small amount of available oxygen in the blood is extracted for use at the cell. The remainder of hemoglobin bound oxygen stays associated with the red blood cells even after it has been exposed to the muscle at the capillary. Blood oxygen saturation is routinely 98% or better at rest. With long-slow-distance exercise, blood oxygen saturations are not significantly different from those at rest. It is rare to have a significant reduction in saturation with this type of training. Further, it has been proposed that the only way to induce a significant desaturation with long-slow-distance training is to do it at altitude (where there's less oxygen present to start with). Here's the rub, in a previously untrained individual, long-slow-distance training induces enough of an oxygen homeostatic disruption to drive improvement in  $VO_2\max$  for a short time. Statistically insignificant drops in blood oxygen saturation are an adequate adaptive stimulus in the beginner. But once the trainee has been consistently training for 3-9 months, long-slow-distance is no longer specific enough of a stress to drive oxygen handling adaptation. A beginner is adapted to no work, so any type of work above sedentary life will drive a spectrum of fitness related changes in structure and in function. Intermediate, advanced, and elite trainees cannot benefit similarly from such a non-specific training stress. In the intermediate trainee and beyond, it is the depression of oxygen saturation as a result of interval training that forces the muscle to adapt in its ability to extract and consume oxygen to power exercise. Oxygen saturation is a marker of the specific driving force of  $VO_2\max$  gain. If a beginner does long-slow-distance and blood oxygen saturations drop 1% or less to 97%, this is enough to drive adaptation. But intermediate, advanced, and elite trainees need more.

They need a drop in oxygen saturation to as low as 91%, maybe lower, for an elite athlete.

So here is where we stand today. The Swede's developed a system of intervals intended to improve fitness in the 1930's (the roots of Fartlek training). The godfather of Exercise Physiology Per Astrand investigated and proposed inclusion of intervals in aerobic fitness training as early as the 1960's. Continuing scientific data supports the concept of high intensity intervals driving  $VO_2$ max gain, especially in trained individuals. But the vast majority of the fitness industry and average exercisers continue to think that long-slow-distance exercise is the path to aerobic superiority. The chasm between science and practice is large here. The conventional wisdom is so entrenched into the public psyche that even the scientists who specialize in aerobic exercise and produce the data, blink the facts or fail to report them in deference to convention.

What is needed to rectify this problem is for the major professional organizations to discard convention that is not supported by fact, either experimental or experiential. What is needed is a large-scale experimental examination of physical fitness that asks appropriate questions about performance enhancement. We accept without question that being more fit makes us healthier and less likely to die. We will spend millions of research dollars trying to figure out the mechanism of that reduced mortality BUT we will not spend a penny on quality research on how to train to improve fitness and actually deliver that reduced mortality efficiently to the public. The failure of the government and granting agencies to fund performance research relegates this vital area of national health to small scale experiments that are limited in design quality and real-world utility. I propose the power of exercise

training to improve aerobic fitness and reduce mortality is likely found towards the anaerobic end of the metabolic spectrum. Experimentation is needed to prove this. More importantly, without a valid pool of evidence, practitioners must independently adopt a non-conventional point of view and training methodology to improve individual aerobic fitness and then national health.

<b>Training Method</b> (Common Name)	<b>Description</b>	<b>Accepted Intent or Disturbed Variable</b>	<b>Degree of VO<sub>2</sub> homeostatic disruption</b>	<b>Actual Disturbed Variable</b>	<b>End Result in Trained Individuals</b>
<b>Recovery</b>	20 to 60 minutes at approximately 70% of VO <sub>2</sub> max	Warm-up, Cool-down, recovery day <b>NOTE:</b> 20-60 minutes of aerobic activity is not a warm-up or a cool-down it is a workout (but not a very effective one)	None	None	Recovery of previous levels of performance, no improvement induced
<b>Long-Slow-Distance</b>	60 to 120 minutes at approximately 70% VO <sub>2</sub> max	Cardiovascular efficiency improvement	None	Oxidative Metabolism (Carbohydrate & Fat)	Improvement in stores of oxidative energy substrates and associated enzymes, can run longer but not faster
<b>Tempo (Interval type)</b>	20 minutes at approximately 85% VO <sub>2</sub> max	Improve lactate kinetics	None	Aerobic Glycolytic Metabolism (Carbohydrate)	Improvement in stores of aerobic glycolytic energy substrates and associated enzymes, delay of switch to anaerobic metabolism, can run a little longer a little faster
<b>Interval</b>	Up to 5 minutes at 95-100% VO <sub>2</sub> max	Improve VO <sub>2</sub> max, Improve lactate kinetics	Small	Primary - Aerobic glycolytic metabolic system, Secondary - Anaerobic Glycolytic Metabolism	Improvement in stores of aerobic glycolytic energy substrates and associated enzymes, delay of switch to anaerobic metabolism, improvement in anaerobic enzyme stores and function, can run a little longer a little faster (does not significantly improve VO <sub>2</sub> )
<b>Reps (Interval Type)</b>	30 to 90 seconds at slightly greater than VO <sub>2</sub> max	Improve speed and economy	Large	Anaerobic Glycolytic Metabolism and VO <sub>2</sub>	Improvement in anaerobic glycolytic storage and function, increased efficiency in O <sub>2</sub> consumption at the working muscle - Increased VO <sub>2</sub> max