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The Paradox of the Aerobic Fitness Prescription

A Facultative Anaerobe Sucks the Air Out of VO_2max

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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 probably haven't really 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 had been asked that question two years ago, I probably would have pulled an 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.

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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 as authoritative. We both knew that many people were lifting and programming incorrectly. We really didn't understand why what was obvious to a couple decent ex-competitors and reasonably 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, we were both surprised to discover a tremendous lack of real and meaningful experimental data. It was virtually impossible to find well-designed and well-controlled experiments actually asking even simple research questions that are relevant to the practicing fitness professional or to 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 know that 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 endurance in the local

CrossFitters has posed a new puzzle. Why do they get aerobically fit when they do not train in a manner that would be considered "aerobic"? Their amazing success doesn't fit into the convenient box of aerobic training dogma (rhythmic and continuous exercise done for long durations at low to moderate intensity). I asked other exercise physiology faculty with aerobic interests about what could be driving this fitness improvement but gained very little satisfaction. And as a professor who feels compelled to explain things to people, not being able to explain this phenomenon really bugged me. This kind of stuff can wake a person up at 3:00 a.m. and compel him to search the National Library of Medicine online 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 with respect to utility. Instead of asking what drives adaptation in VO_2max, most researchers in exercise academic circles seem to have been interested in what limits VO_2max. 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 toward those limitations.

In 1936, Canadian endocrinologist 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 correctly applying Selye's theory is understanding that a disruption of homeostasis must occur in a physiological system 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 60s 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 20 to 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_2max—the maximum amount of oxygen the body can consume at maximal effort—is not challenged. In the conventional 20- to 60-minute prescription for improving aerobic fitness, the demand for oxygen at the working muscle is met by supply. The name itself says it all: aerobic means "in the presence of oxygen." This means that, by definition, this type of training does not—and cannot—provide a disruption of oxygen homeostasis. *With no homeostatic disruption, there can be no adaptation, and no fitness gain.*

But tell any fitness trainer, exercise scientist, allied health professional, or physician that they are approaching the development of VO_2max incorrectly and they will claim heresy on your part and question your sanity, your IQ, and your familial heritage. Just by writing these words for publication, I am painting a target on my academic standing. It is an invitation to 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 (though I don't think I am), every exercise professional, clinician, and scientist is welcome to dissect and examine my thoughts and supporting data in an open forum. That's what science 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 error, then we are merely lemmings.

So I posit here that everyone including the ACSM is approaching training for improving VO_2max in a theoretically incorrect manner. If I know that it is being done wrong, then, 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.

The discussion above considers 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 VO_2max and performance in their athletes. They do not and would not have a trainee run at 70% of VO_2max 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 VO_2max and performance. The only time 70% runs are prescribed 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: long-slow-distance and interval training. There are many variations of both of these types, but in large part the variants are fairly similar (see table 1 for a more extended comparison). Long-slow-distance work is intended by convention to improve cardiovascular efficiency and VO_2max, and interval training is intended to improve lactate tolerance/clearance and VO_2max. Both have been demonstrated to improve endurance performance and to improve VO_2max, 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 individuals of low to average fitness just starting a training program. In other words, beginners. Beginners are far from their genetic potential for performance and therefore a very low-level and non-specific stress can induce positive adaptations. We can have beginners walk, jog, sprint, jump, twist, flex, wiggle, dance, swing, hang, roll, bounce, or do virtually any activity, and their endurance will improve. 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 VO_2max. 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 beginners ride bikes and their squats will improve (but not so for intermediate, advanced, or elite trainees, who require specificity to make further improvements). Considering data from beginner populations to be relevant to trainees at every level of training advancement is a gross and progress-retarding mistake.

If much of the data we have is flawed or uninformative, how are we supposed to know how to train people? Well, let's consider what specifically each of these two methods of training—long slow distance and intervals—do to the body. We've already established that long-slow-distance training cannot, by definition, stress oxygen delivery and utilization systems to the 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 max; there are more facets to it than that. But let's keep it simple here and examine the two major facets of endurance: energy and 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 disruption of metabolic homeostasis. It would not be prudent to consider only complete depletion as a disruptive stress; partial depletions should be considered disruptive as well, but if and only if the depletion is greater than that previously experienced by the trainee. Long-slow-distance 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 depleting glycogen stores and simultaneously exceeding fat metabolic capacity drives an improvement in storing and utilizing these two energetic substrates and results in improved endurance. So, endurance has improved, but VO_2max has not. 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 instead automatically, and incorrectly, attribute the improvement in endurance to an improvement in VO_2max.

The second common type of training done for aerobic fitness is interval training, shorter and more intense segments of effort with short rest periods between repeats. It has been observed that lactic acid

Training method *(Common name)*	**Description**	**Intended Result or disturbed variable**	**Degree of VO_2**	**Actual disturbed variable**	**End results in trained individuals**
Recovery	20 to 60 minutes at approximately 70% of VO_2 max	Warm-up, cool-down, recovery day *Note:* 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
Long Slow Distance	60 to 120 minutes at approximately 70% of VO_2 max	Improve cardiovascular efficiency	None	Oxidative metabolism (carbohydrate and fat)	Improvement in stores of oxidative energy substrates and associated enzymes; athlete can run longer but not faster
Tempo (interval type)	20 minutes at approximately 85% VO_2 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; athlete can run a little longer a little faster
Interval	Up to 5 minutes at 95-100% of VO_2 max	Improve VO_2 max; improve lactate kinetics	Small	Primary: aerobic glycolytic metabolism 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; athlete can run a little longer a little faster (but does not significantly improve VO_2)
Reps (interval type)	30 to 90 seconds at slightly greater than VO_2 max	Improve speed and economy	Large	Anaerobic glycolytic metabolism and VO_2	Improvement in anaerobic glycolytic storage and function; increased efficiency in O_2 consumption at the working muscle (i.e., increased VO max)

Table 1 Conventional types of training for aerobic fitness

accumulates during this type of training and thus it is commonly posited 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 the (mis)information that lactic acid is bad, actually it 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, since lactate isn't even part of aerobic metabolism and VO_2max but is simply an inevitable consequence of the really important things happening here. With interval training, producing lactate is not the important effect; exceeding oxygen consumption capacity is. Intervals are done in the realm of glycolytic metabolism, whereas long-slow-distance is primarily oxidative. Running fast enough to require the body to use primarily glycogen to fuel the activity (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 that produces lots of lactate is the level of exertion needed to drive improvements in VO_2max. It's the level of exertion where the athlete exceeds oxygen consumption capacity. The body adapts to this stress by augmenting its ability to take up oxygen and to use it in the muscle. At least, it does 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; this is a gross underuse of this training method. Lots of aerobic athletes use intervals. Many use them for the wrong reason and/or at the wrong intensities. Regardless of their reasons for including interval training, most athletes should likely do more, lots more. Most runners who do them use interval intensities of between 85% and 105% of VO_2max (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_2max, 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% to 250% of VO_2max. 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_2max 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 VO_2max, the absolute marker of aerobic fitness, has as the centerpiece of its definition "ability to consume oxygen." It is not defined by the ability of the heart, lungs, and vasculature to deliver oxygen.

Here lies my heresy. Consumption does not relate strongly to delivery. To state that to develop VO_2max 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. When the body is 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 though. In a previously untrained individual, long-slow-distance training induces enough of an oxygen homeostatic disruption to drive improvement in VO_2max 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 training consistently for 3 to 9 months, long-slow-distance is no longer sufficiently specific 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 to improve its ability to extract and consume oxygen to power exercise. Oxygen saturation is a marker of the specific driving force of VO_2max gain. If a beginner does long-slow-distance work 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 even lower for an elite athlete.

So here is where we stand today. In the in the 1930s the Swedes developed a system of intervals intended to improve fitness (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 1960s. Continuing scientific data supports the concept of high-intensity intervals driving VO2max 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 in the public psyche that even the scientists who specialize in aerobic exercise and produce the data tend to blink the facts or fail to report them in deference to convention.

What is needed to rectify this problem, and to make exercise physiology relevant, 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 that the power of exercise training to improve aerobic fitness and reduce mortality is likely found toward the anaerobic end of the metabolic spectrum. Experimentation and clinical data are needed to prove this. More importantly, without a valid pool of evidence to drive changes in the conventional wisdom, practitioners must independently adopt a non-conventional point of view and training methodology to improve individual aerobic fitness and then national health.

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