

---

# Chasing Pheidippides

---

## The Science of Endurance

BY JASON R. KARP, PH.D.

---

**F**rom the time the ancient Greek runner Pheidippides ran from Marathon to Athens to announce the Greeks' victory over Persia in the Battle of Marathon, humans have had a compelling interest with endurance. Indeed, humans have repeatedly tried to push the limits of endurance. From 50 marathons in 50 days and the 300 miles of nonstop running by Dean Karnazes, to the average speed of 26 miles per hour by Lance Armstrong during the three-week Tour de France, to the average marathon pace of 5 minutes and 10 seconds per mile by England's Paula Radcliffe and 4 minutes and 44 seconds per mile by Ethiopia's Haile Gebrselassie, to the 1,544 miles covered by Norwegian Borge Ousland in crossing Antarctica in 64 days, to the ascent of Mount Everest without supplemental oxygen, humans' feats of endurance have been nothing short of remarkable. As a coach, you're in the business of improving endurance. Let's take a look at what your athletes' endurance is composed of.

### CARDIOVASCULAR FACTORS

The main cardiovascular factors that influence endurance are cardiac output and blood flow to the muscles. Cardiac output is the volume of blood pumped by the heart's left ventricle per minute, and is the product of stroke volume and heart rate. Stroke volume is the amount of blood the heart pumps with each contraction of its left ventricle, and is determined by the return of blood back to the heart through the venous circulation (venous return), the heart's ability to contract quickly and forcefully (con-

tractility), the amount of pressure in the left ventricle (preload) and in the aorta (afterload), and the size of left ventricle. The larger the left ventricle, the more blood it can hold; the more blood it can hold, the more blood it can pump. One of the hallmark adaptations to cardiovascular endurance training is an increase in the size of the left ventricle. So characteristic is a large heart of genetically gifted and highly trained endurance athletes that it is considered a physiological condition by the scientific and medical communities called Athlete's Heart (Naylor et al. 2008).

Once the blood leaves the heart, its flow to the muscles depends on a number of factors, including the redistribution of blood away from other, less important tissues to the active muscles; the resistance of blood flow through the blood vessels; adequate dilation of blood vessels, which depends on the interplay between the sympathetic and parasympathetic nervous systems and their associated hormones; oxygen transport capacity of the blood, which is determined by red blood cell volume and the amount of hemoglobin; the amount of myoglobin, which transports oxygen in the muscles; and the density and volume of capillaries that perfuse the muscle fibers, which determine the time available for diffusion into the muscle mitochondria as blood transits the capillary network.

Men have a greater stroke volume and cardiac output to send more blood and oxygen to the muscles and more hemoglobin in their blood to transport oxygen, which gives them greater cardiovascular endurance than women.



**MUSCULAR FACTORS**

Once oxygen is delivered to the muscles, the muscles have to use the oxygen to regenerate energy (ATP) for muscle contraction. The amount of oxygen extracted and used by the muscles is largely dependent on the muscles' mitochondrial and capillary volumes. The more capillaries that perfuse the muscle fibers, the shorter the diffusion distance for oxygen from the capillaries to the mitochondria, which contain the enzymes involved in aerobic metabolism. The number of mitochondrial enzymes is also an important determinant of endurance, since enzymes, through their catalyzing effect on chemical reactions, control the rate at which ATP is produced.

Together, the cardiac output and the amount of oxygen extracted and used by the muscles determine aerobic power ( $VO_2\text{max}$ ), the maximum volume of oxygen that your athletes' muscles can consume per minute.  $VO_2\text{max}$  is considered the best single indicator of a person's aerobic fitness. Since it was first measured in humans in the 1920s, it has become one of the most often measured physiological variables in exercise physiology. In 1930, David Dill and his colleagues were among the first physiologists to suggest that there are marked differences in the amount of oxygen different people use when running at the same speeds, and that these differences in "economy" of oxygen use could be a major factor explaining differences in endurance performance (Dill, 1930). Running economy is the volume of oxygen ( $VO_2$ ) your athletes use to run at a given speed, and is influenced by their biomechanics, proportion of slow-twitch muscle fibers, mitochondrial density, and body weight. It is an important indicator of endurance. For example, if two of your athletes have the same  $VO_2\text{max}$ , but Runner A uses 70 percent and Runner B uses 80 percent of that  $VO_2\text{max}$  while running at 7:00 mile pace, the pace feels easier for Runner A because Runner A is more economical. Therefore, Runner A can run at a faster speed before feeling the same amount of fatigue as Runner B.

**METABOLIC FACTORS**

Endurance is influenced by a number of metabolic factors, including the removal of lactate and the buffering of metabolic acidosis. At slow running speeds, lactate is removed from the muscles as quickly as it is produced. At faster speeds, there is a greater reliance on anaerobic glycolysis for the production of ATP, and aerobic metabolism (Krebs cycle and electron transport chain) can't keep up with the production of pyruvate from glycolysis. So pyruvate is converted into lactate and lactate removal starts lagging behind lactate production, causing lactate to accumulate. Concomitant with lactate accumulation is the accumulation of hydrogen ions in muscles and blood, causing metabolic acidosis and the development of fatigue. The lactate threshold (LT) is the fastest running speed above which lactate production begins to exceed its removal, with blood lactate concentration beginning to increase exponentially.

The LT demarcates the transition between running that is almost purely aerobic and running that includes significant oxygen-independent (anaerobic) metabolism. (All running speeds have an anaerobic contribution, although when running slower than LT pace, that contribution is negligible.) Thus, the LT is an important determinant of endurance performance since it represents the fastest speed your athletes can sustain aerobically.

The ability to perform prolonged endurance exercise is strongly influenced by the amount of carbohydrate (glycogen) stored in skeletal muscles (Ahlborg et al., 1967), with fatigue coinciding with glycogen depletion (Sahlin et al. 1998). Thus, the ability to metabolize fat also influences endurance since the muscles' preferred fuel – carbohydrate – is limiting, providing enough energy for only about 100

minutes of marathon running (Newsholme 1981). By contrast, humans' store of fat is virtually unlimited, with enough to fuel about five days of marathon running (Newsholme 1981) or about 1,000 miles of walking for a 145-pound person with 18 percent body fat (Coyle 2000). At slow running speeds, some of carbohydrate's metabolic responsibility for ATP regeneration is relieved by fat, in the form of free fatty acids in the blood and intramuscular triglyceride. Even with the contribution of fat oxidation helping to delay the depletion of glycogen, moderate-intensity running (70-75 percent  $VO_2\text{max}$ ) can only be sustained for two to three hours (Coyle et al. 1986).

While women are at a definite cardiovascular disadvantage to men, they seem to have a greater capacity to metabolize fat and conserve glycogen (Tarnopolsky 1998), which may give them an advantage for very long endurance activities. Indeed, in 2002 and 2003, Pam Reed beat all the men at the 135-mile Badwater Ultramarathon.

**NEUROMUSCULAR FACTORS**

There are a number of steps that occur for muscles to contract and produce force. First, the central nervous system sends a signal to a motor neuron, which integrates with a number of muscle fibers, creating a motor unit. When this signal reaches the end of the axon of the motor neuron, the neurotransmitter acetylcholine is released at the neuromuscular junction. This causes a change in polarity of the muscle membrane (called depolarization), as sodium ions rush in and potassium ions rush out. The signal, now called an action potential, propagates deep into the muscle to the sarcoplasmic reticulum, which stores calcium ions. The calcium diffuses from the sarcoplasmic reticulum into the area of the contractile proteins—actin and myosin – and binds to a protein called troponin that integrates with actin. Upon calcium binding to troponin, another protein called tropomyosin is removed from the active binding sites on actin, exposing those sites to myosin. Myosin then binds to actin, forming a cross-bridge. Finally, an ATP molecule that is contained inside the myosin is broken down into its constituents, releasing the energy contained within that molecule, allowing the muscle to contract. For force production to continue, and for your athletes to maintain their pace, the central nervous system has to increase the number of motor units recruited and increase the frequency of stimulation of the motor units.

**TRAINING ENDURANCE**

Endurance training stimulates many physiological, biochemical, and molecular adaptations, including a greater storage of fuel (glycogen) in the muscles; an increase in intramuscular fat use; an increase in the number of red blood cells and hemoglobin, which improves blood vessels' oxygen-carrying capability; a greater capillary network for a more rapid diffusion of oxygen into the muscles and, through the complex activation of gene expression, an increase in mitochondrial density and the number of aerobic enzymes, which increases aerobic metabolic capacity.

**CARDIOVASCULAR FACTORS**

Long intervals (3 to 5 minutes) run at the velocity at which  $VO_2\text{max}$  occurs ( $vVO_2\text{max}$ ) provides the greatest cardiovascular load because your athletes repeatedly reach and sustain their maximum stroke volume, cardiac output, and  $VO_2\text{max}$  during the work periods. Long intervals are the most potent stimulus for improving  $VO_2\text{max}$  (Billat 2001; Midgley et al. 2007). However, short intervals (2 minutes) can also improve  $VO_2\text{max}$ , as long as the intervals are performed at a high intensity and with short, active recovery periods to keep  $VO_2$  elevated throughout the workout (see Methods for Improving

Endurance). The higher your athletes'  $\text{VO}_2\text{max}$ , the higher their aerobic ceiling. The more trained your athletes, the more important the intensity of training becomes to improve  $\text{VO}_2\text{max}$  because the more cardiac-limited  $\text{VO}_2\text{max}$  becomes.

For highly trained runners,  $\text{vVO}_2\text{max}$  is about 3,000-meter race pace. If using heart rate as a guide, your athletes should come close to reaching their maximum heart rates by the end of each work period.

#### MUSCULAR FACTORS

A large volume of endurance training may be the simplest way to increase the muscular factors associated with endurance (mitochondrial and capillary density and enzyme activity). Interval training has also been shown to increase aerobic enzyme activity (Talanian et al. 2007).

#### METABOLIC FACTORS

Running at the LT increases it to a faster speed and higher percentage of  $\text{VO}_2\text{max}$ , making what was an anaerobic intensity before now high aerobic. LT training can be done as a continuous workout or as intervals performed at LT intensity with short rest periods. LT pace is about 10 to 15 seconds per mile slower than 5K race pace (or about 10K race pace) for slower runners (slower than about 40 minutes for 10K). If using heart rate (HR), the pace is about 75 to 80 percent max HR. For highly trained and elite runners, LT pace is about 25 to 30 seconds per mile slower than 5K race pace (or about 15 to 20 seconds per mile slower than 10K race pace) and corresponds to about 85 to 90 percent max HR. The pace should feel "comfortably hard."

Long runs present a threat to the muscles' survival by depleting their store of glycogen. Depleting muscle glycogen forces muscles to rely on fat as fuel. The human body responds rather elegantly to situations that threaten or deplete its supply of fuel, synthesizing and storing more than what was previously present, thus increasing endurance for future efforts. Empty a full glass, and you get a refilled larger glass in its place. The more glycogen your athletes have packed into their muscles, the greater their ability to hold a hard pace.

#### NEUROMUSCULAR FACTORS

In addition to increasing mitochondrial and capillary density, a large volume of endurance training may have a neuromuscular benefit. It is possible that, just as repetition of the walking movement decreases the jerkiness of a toddler's walk to the point that it becomes smooth, repetition of a specific movement has an under-recognized neural component. With countless repetitions, motor unit recruitment patterns, all of the steps involved in muscle contraction, and possibly even the relationship between breathing and stride rate are optimized to minimize the oxygen cost and improve economy.

Neuromuscular factors and aerobic economy can also be targeted by power training. Studies have shown that explosive strength training with heavy weights and plyometric training improve economy in endurance athletes (Hoff et al. 2002; Jung, 2003; Paavolainen et al. 1999; Spurrs et al. 2003; Turner et al. 2003). When strength training, make sure your athletes use a very high intensity and very few reps to focus on neural adaptation rather than muscle hypertrophy (which would decrease running economy by adding muscle mass).

Understanding the science of endurance will help you



KIRBY LEE PHOTOGRAPH



train your athletes. And if they train long enough, they'll undoubtedly have the greatest endurance among all their competitors, good enough perhaps to even chase Pheidippides.

Dr. Jason R. Karp is owner of RunCoachJason.com, a coaching, personal training, and fitness consulting company, providing science-based coaching to runners of all levels, fitness training to the public, and consulting to coaches and fitness professionals. He has a Ph.D. in exercise physiology, and is director and coach of REVO2LT Running Team.

**METHODS FOR IMPROVING ENDURANCE**

$vVO_2\max$  = velocity at  $VO_2\max$ ; LT = lactate threshold.

**CARDIOVASCULAR FACTORS**

- 5 x 1,000 meters at  $vVO_2\max$  (95-100% max heart rate) with  $1\leq 1$  work-to-rest ratio
- 4 x 1,200 meters at  $vVO_2\max$  (95-100% max heart rate) with  $1\leq 1$  work-to-rest ratio
- 16 x 400 meters at  $vVO_2\max$  with  $1\leq 1$  work-to-rest ratio

**MUSCULAR FACTORS**

High mileage, with progressive increases in volume (days per week and duration) over time

**METABOLIC FACTORS**

- 3-6 miles at LT pace
- 5-7 x 1 mile at LT pace with 1 minute rest
- Long runs of 10 to 15 miles

**NEUROMUSCULAR FACTORS**

- Strength training: 3-4 sets of 3-5 reps at  $>85\%$  1-rep max with 3 minutes rest
- Plyometrics (box jumps, squat jumps, leg bounds, bleacher hops, etc.)

**REFERENCES**

Ahlborg, B., et al. (1967). Muscle glycogen and muscle electrolytes during prolonged physical exercise. *Acta Physiologica Scandinavica*. 70:129-142.

Billat, V. (2001). Interval training for performance: A scientific and empirical practice. Special recommendations for middle- and long-distance running. Part I: Aerobic interval training. *Sports Medicine*. 31(1):13-31.

Coyle, E.F. (2000). Physical activity as a metabolic stressor. *American Journal of Clinical Nutrition*. 72(Suppl):512S-520S.

Coyle, E.F., et al. (1986). Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. *Journal of Applied Physiology*. 61(1):165-172.

Dill, D.B., Talbot, J.H., and Edwards, H.T. (1930). Studies in muscular activity. VI: Response of several individuals to a fixed task. *Journal of Physiology*. 69:267-305.

Hoff, J., Helgerud, J., and Wisløff, U. (2002). Endurance training into the next millennium: muscular strength training on aerobic endurance performance. *American Journal of Medicine in Sports*. 4:58-67.

Jung, A.P. (2003). The impact of resistance training on distance running performance. *Sports Medicine*. 33(7):539-552.

Midgley, A.W., McNaughton, L.R., and Jones, A.M. (2007). Training to enhance the physiological determinants of long-distance running performance. *Sports Medicine*. 37(10):857-880.

Naylor, L.H., et al. (2008). The Athlete's Heart: A contemporary appraisal of the 'Morganroth Hypothesis.' *Sports Medicine*. 38(1):69-90.

Newsholme, E.A. (1981). The glucose/fatty acid cycle and physical exhaustion. *Ciba Foundation Symposium*. 82:89-101.

Paavolainen, L., et al. (1999). Explosive-strength training improves 5-km running time by improving running economy and muscle power. *Journal of Applied Physiology*. 86(5):1527-1533.

Sahlin, K., Tonkonogi, M., and Söderlund, K. (1998). Energy supply and muscle fatigue in humans. *Acta Physiologica Scandinavica*. 162:261-266.

Spurrs, R.W., Murphy, A.J., and Watsford, M.L. (2003). The effect of plyometric training on distance running performance. *European Journal of Applied Physiology*. 89(1):1-7.

Talanian, J.L., et al. (2007). Two weeks of high-intensity aerobic interval training increases the capacity for fat oxidation during exercise in women. *Journal of Applied Physiology*. 102:1439-1447.

Tarnopolsky, M.A. (1998). Gender differences in lipid metabolism during exercise and at rest. In M.A. Tarnopolsky (Ed.), *Gender Differences in Metabolism: Practical and Nutritional Implications*. Boca Raton, FL: CRC Press, pp. 179-199.

Turner, A.M., Owings, M., and Schwane, J.A. (2003). Improvement in running economy after 6 weeks of plyometric training. *Journal of Strength and Conditioning Research*. 17(1):60-67.

