

Physiological Determinants of Endurance Exercise Performance

Edward F Coyle
Human Performance Laboratory,
Department of Kinesiology and Health Education
The University of Texas, Austin, USA

Coyle, E.F. (1999). Physiological determinants of endurance exercise performance. *Journal of Science and Medicine in Sport* 2 (3): 181-189.

Performance in endurance events is typically evaluated by the power or velocity that can be maintained for durations of 30 min. to four hours. The two main by-products of intense and prolonged oxidative metabolism that can limit performance are the accumulation of hydrogen ion (i.e. lactic acidosis) and heat (i.e. hyperthermia). A model for endurance performance is presented that revolves around identification of the lactate threshold velocity which is presented as a function of numerous morphological components as well as gross mechanical efficiency. When cycling at 80 RPM, gross mechanical efficiency is positively related to Type I muscle fiber composition, which has great potential to improve endurance performance. Endurance performance can also be influenced by altering the availability of oxygen and blood glucose during exercise. The latter need forms the basis for ingesting carbohydrate at 30-60 grams per hour during exercise. In laboratory simulations of performance, athletes fatigue due to hyperthermia when esophageal is approximately 40°C, in association with near maximal heart rate and perceived exertion. It is likely that the central nervous system is involved in the aetiology of fatigue from hyperthermia. Dehydration during exercise promotes hyperthermia by reducing skin blood flow, sweating rate and thus heat dissipation. The combination of dehydration and hyperthermia during exercise causes large reductions in cardiac output and blood flow to the exercising musculature, and thus has a large potential to impair endurance performance. Endurance performance is optimized when training is aimed specifically at developing individual components of the model presented and nutritional supplementation prevents hypoglycemia and attenuates dehydration and hyperthermia. Indeed, the challenge at the transition to a new millennium is to synergistically integrate these physiological factors in training and competition.

Introduction

Even before Pheidippides is alleged to have run from the plains of Marathon to Athens to proclaim the Greek victory over the Persians, men and women have awed at their ability to exercise for long durations. They have also become baffled by their physiological limitations. The remarkable scientific advances of the past century have identified a plethora of important individual physiological factors that relate to athletic performance. The goal of the present review, written at the transition to a new millennium, is to discuss our current understanding of how numerous physiological factors and systems interact in establishing the limits of human endurance performance ability.

Determinants of Endurance Performance

Overview and Definitions

This review will focus on durations of exercise that can be endured for 30 min. to four hours. Performance can be evaluated by the amount of time required to complete a given amount of work (i.e. power) or by the length of time that a given power output can be maintained (i.e. capacity). Figure 1 presents a simple but useful scheme of the energetics of exercise under these conditions and the major sites of fatigue (Coyle, 1995). Almost all of the energy (i.e. ATP) utilized under aerobic conditions will be resynthesized in the mitochondria via oxidative metabolism of carbohydrate and fat. Therefore, performance can be directly influenced by altering availability of oxygen, carbohydrate and fat. Furthermore changes in muscle mitochondrial density, as occurs with endurance-training, serve to regulate oxidative metabolism (Holloszy, 1967; Holloszy & Coyle, 1984).

The two main by-products of intense and prolonged oxidative metabolism that can limit performance are the accumulation of hydrogen ion (i.e. acidosis) and heat (i.e. hyperthermia). Hydrogen ion accumulation in muscle, and thus acidosis, is typically reflected by muscle and blood lactic acid accumulation. This can be a sensitive measure of the degree to which the exercising musculature is stressed regarding its ability to maintain prolonged aerobic ATP production. In practical terms, identification of the exercise intensity eliciting the 'blood lactate threshold' is very predictive of endurance performance ability and it will be the central focus of the physiological model developed below (Coyle, 1995).

Heat is produced both in the chemical reactions needed to resynthesize ATP as well as by the process of ATP hydrolysis (Kushmerick, 1983). In fact, more than 75% of the energy liberated in the oxidation of carbohydrate and fat during exercise is converted to heat, with less than 25% being converted to actual power and movement (Coyle et al., 1992). As a result, the contracting muscles impart a huge heat load to the body. If this heat is not transferred from the body to the environment, largely from blood flowing to the skin that is cooled by evaporating sweat, a person can increase their core temperature to lethal levels with just a few minutes of intense exercise. Needless to say, hyperthermia has great potential to limit endurance performance and impair health especially when environmental conditions are hot and/or humid (Costill, 1972).

High sweating rates (e.g.: 1-2 litres per hour) during prolonged exercise cause dehydration if fluid replacement is not adequate. It appears that the major mechanisms by which dehydration impairs endurance performance are by causing hyperthermia and impairing cardiovascular function, which seem to

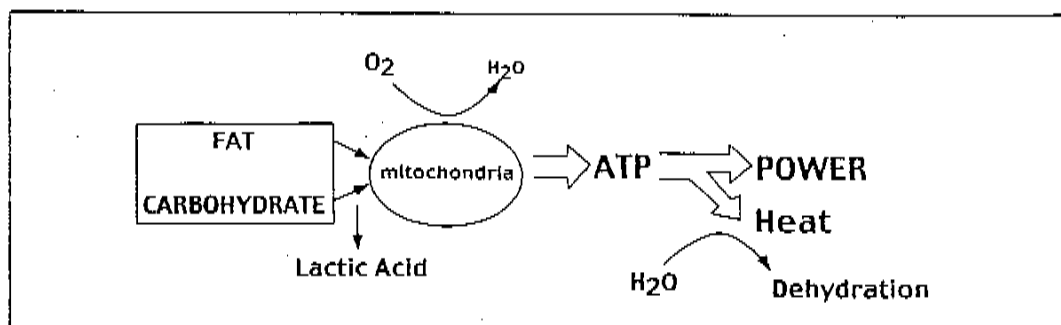


Figure 1: Scheme of aerobic energy production and by-product accumulation.

Determinants of Endurance Performance

cause added hormonal and metabolic stress (Febbraio et al., 1994; González-Alonso et al., 1995; González-Alonso et al., 1997; González-Alonso et al., 1998; González-Alonso et al., 1999). In addition to drinking water during prolonged exercise, endurance athletes benefit by ingesting carbohydrate in order to maintain blood glucose oxidation in an attempt to preserve the substrate supply (i.e. carbohydrate) needed to exercise intensely (Coggan & Coyle, 1991). Endurance performance is maximized when schedules for fluid and carbohydrate supplementation are developed and practised.

Model for Endurance Power and Velocity

Figure 2 presents a model of the numerous physiological factors that integrate to determine the Performance-Velocity that can be maintained in a motivated athlete who is not dehydrated, hyperthermic or carbohydrate depleted (Coyle, 1995). This model revolves around the concept that the blood "lactate threshold (LT)" provides information which is most indicative of the stress experienced by the exercising person as it relates to Performance-Velocity. Since the early study of Margaria, Edwards and Dill (1933), it has been recognized that the production of lactic acid by muscle is indicative of muscle metabolic stress. It appears that marathon runners and other endurance athletes who compete for durations of approximately 2-3 hours, set a pace that is slightly faster (e.g. 75% VO_2max) than the intensity of the lactate threshold (e.g. 70% VO_2max ; Costill & Fox, 1972; Farrell et al., 1979). The pace appears to be selected according to the sensation of stress in the exercising musculature that is proportional to muscle and blood lactate concentration and indicative of the rate of muscle glycogenolysis. It should

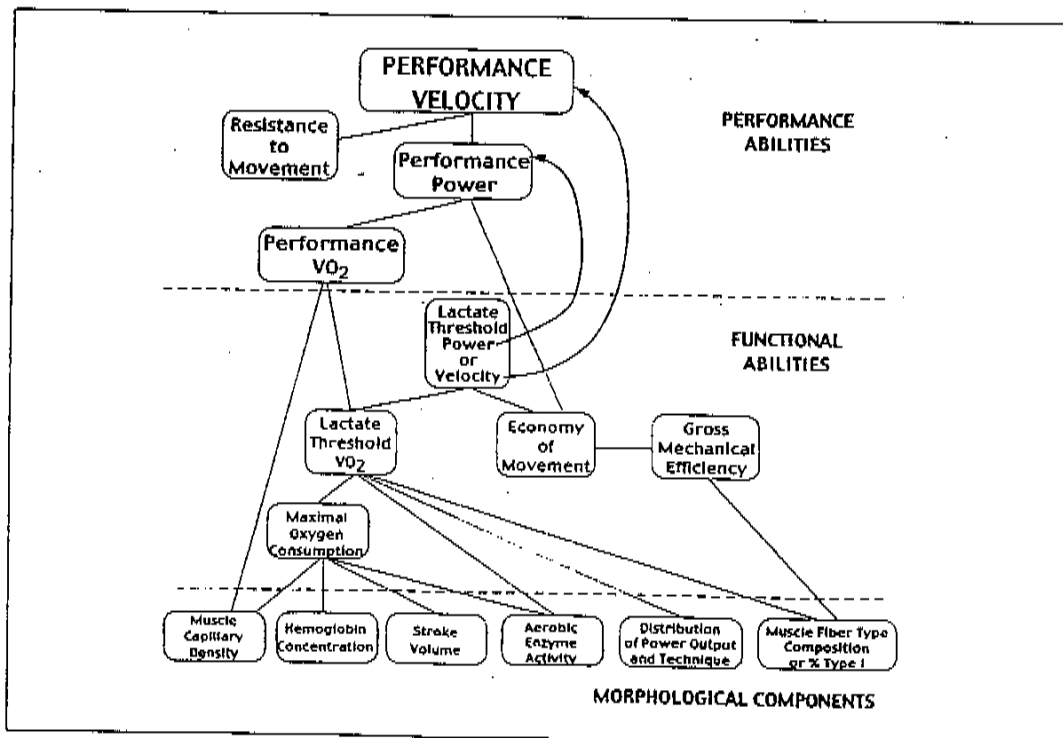


Figure 2: Model of the interrelationships of the physiological factors determining endurance performance ability (modified from Coyle, 1995).

Determinants of Endurance Performance

be noted that in events lasting approximately one hour, trained cyclists maintain an intensity of 85-95% VO_2max , which is well in excess of their blood lactate threshold (i.e. 73-84% VO_2max) and produces a constant blood lactate concentration of 7 mM on average (Coyle et al., 1991).

The model further indicates that Lactate Threshold Power is a function of the Lactate Threshold VO_2 and Gross Mechanical Efficiency (or Economy of Movement). Lactate Threshold VO_2 is a physiological measure in that it is the rate of oxygen consumption that stresses the exercising musculature to accelerate glycogenolysis. The numerous morphological components and systems that establish Lactate Threshold VO_2 are shown in Figure 2 and are integrated in the discussion below. However, it is useful to first illustrate how Lactate Threshold VO_2 and Gross Mechanical Efficiency combine to establish Lactate Threshold Power and ultimately Performance Power.

Cycling Performance and Mechanical Efficiency are Related to the Percentage of Type I

Muscle Fibres

Gross Mechanical Efficiency is calculated simply as the ratio of worked accomplished per min. relative to energy expenditure per min. As shown in Figure 3, Gross Mechanical Efficiency when cycling at 80 RPM ranged from 18.3 to 22.6% in a population of endurance trained cyclists and it was significantly correlated with % Type I muscle fibres ($r = 0.75$; Coyle et al., 1992). This finding should be interpreted in light of the velocities of contraction employed. Peak muscular efficiency generally occurs at a velocity of approximately one-third of the maximal shortening velocity in both type I and type II fibres (Kushmerick, 1983). It is likely that type I fibres were found to be more efficient when cycling at 80 RPM because this velocity is closer to their velocity of peak efficiency compared with type II fibres.

The influence on Performance Power of Gross Efficiency when cycling is illustrated by comparison of subjects grouped according to their % Type I muscle fibres (Horowitz, 1993). The normal % type I group was indeed similar to the

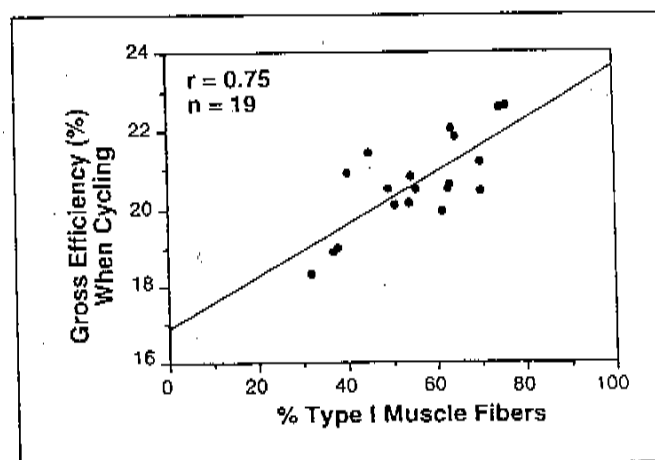


Figure 3: The relationship between % type I muscle fibres and gross mechanical efficiency when cycling at 80 RPM (modified from Coyle et al., 1992).

Determinants of Endurance Performance

average composition of the general population with a mean of 48% type I with a distribution of 38-54%. The mean value of 73% in the high type I group also represents values reported in some exceptional endurance athletes (Horowitz et al., 1993). Despite having almost identical Lactate Threshold VO_2 as well as Performance- VO_2 (i.e. 4.5 l/min) subjects in the high % type I group generated 9% more Performance-Power compared with the low % type I group (342 vs 315 watts; Horowitz et al., 1993). A relatively high % of Type I fibres and superior Gross Mechanical Efficiency can allow cyclists with a below average Lactate Threshold VO_2 and Performance- VO_2 , to perform as well as athletes with much higher Lactate Threshold VO_2 and Performance- VO_2 , yet only normal % Type I fibres. We have seen that the coefficient of variation (SD/mean) for Efficiency and Gross Mechanical Efficiency is approximately 4-6% in competitive cyclists. However, the coefficient of variation in other functional abilities (i.e. Lactate Threshold VO_2 and Performance- VO_2) is relatively greater averaging 8-10%. This greater variability in Lactate Threshold VO_2 and Performance- VO_2 is probably due to the fact that it is composed of more morphological components compared with Economy (Figure 2).

Morphological Components of Lactate Threshold VO_2

Figure 2 indicates that several morphological components integrate into Lactate Threshold VO_2 through their influence on Maximal Oxygen Consumption (i.e. $\text{VO}_{2\text{max}}$). Stroke volume and hemoglobin concentration influence oxygen delivery to muscle and have great potential to improve endurance performance ability. This is illustrated by the findings that elevation of hemoglobin concentration from 15.7 to 16.7 g/100 ml, via red blood cell infusion, increased $\text{VO}_{2\text{max}}$ and greatly increased running time to exhaustion at high intensities (i.e. 7.2 min vs. 9.65 min; Buick et al., 1980).

There is much evidence supporting the idea introduced by Holloszy et al. (1967) that mitochondrial density or Aerobic Enzyme Activity is a major determinant of the degree of muscle stress during exercise (Fink et al., 1977, Holloszy & Coyle, 1984, Ivy et al., 1980). Ivy et al. (1980) found that muscle respiratory capacity, which reflects mitochondrial enzyme activity, is highly related to the Lactate Threshold VO_2 when examining a heterogeneous group of people. In addition, the time course with which Aerobic Enzyme Activity (i.e. citrate synthase) declines over the course of 84 days of detraining is closely paralleled by reductions in Lactate Threshold VO_2 (Coyle et al., 1985). Therefore, there is strong theoretical and direct support for the idea that mitochondrial Aerobic Enzyme Activity is a major determinant of Lactate Threshold VO_2 . The concept is that as energy expenditure is increased, the VO_2 eliciting the lactate threshold represents the rate of energy expenditure at which muscle cell homeostasis is disturbed sufficiently to more markedly stimulate glycogenolysis and lactate production. The more mitochondria sharing in the power output, the higher the absolute VO_2 needed to elicit lactate threshold. As discussed below, methods for increasing the number of mitochondria sharing in the oxidative power output include not only increasing the number of mitochondria in a given muscle mass, but also increasing the amount of muscle mass sharing in the power output (e.g. Distribution of Power Output).

The amount of muscle mass active when cycling can be estimated as the quotient of the total mmoles of muscle glycogen oxidized by the whole body and the mmoles of glycogen used per kg of muscle, obtained from muscle biopsies (i.e.

Determinants of Endurance Performance

vastus lateralis), during 30 min of cycling at 79% VO_2max (Coyle et al., 1988; Coyle, 1995). We have estimated that a group of subjects with a high Lactate Threshold VO_2 are able to distribute the power output of cycling over an approximately 22% greater amount of muscle mass (Coyle et al., 1988; Coyle, 1995). We think it likely that the high lactate threshold group has developed the ability to better utilize their hip extensors muscles for powering the cycling down-stroke, possibly as a result of their greater number of years spent training for cycling (Coyle et al., 1988; Coyle, 1995). It seems most likely to us that greater muscle fibre recruitment would result in higher peak muscle force. Our pedal torque data support this interpretation in that elite cyclists with a high Lactate Threshold VO_2 maintained an 11.2% higher power output during the 1h performance (i.e. Performance Power) test by generating more torque during the cycling down-stroke (Coyle et al., 1988; Coyle, 1995). This was accomplished by generating 22% more peak torque compared with other competitive cyclists, with peak torque occurring slightly before the cycle crank was horizontal in both groups (i.e. 86-90 degrees). The greater peak torque generated by elite cyclists is derived by producing 24% higher vertical force directed "down-ward" and 14% higher horizontal force directed "forward". Simply put, they pushed down harder. Elite cyclists display this pattern even when cycling at a given absolute power output (Coyle et al., 1988; Coyle, 1995).

Exercise time to fatigue is also related to Muscle Capillary Density (i.e. capillaries per mm^2), which interacts with Lactate Threshold VO_2 . Together these two factors accounted for more than 92% of the variance in time to fatigue when cycling at intensities well above lactate threshold (Coyle et al., 1988). A high Muscle Capillary Density has been postulated to increase muscle perfusion by reducing diffusing distances and thus aid in removing lactate from muscle. We interpret this relationship to indicate that time to fatigue during exercise, at an intensity above the lactate threshold, is related to both lactate production (as reflected by Lactate Threshold VO_2 or % VO_2max at LT) and removal from muscle (as related to Muscle Capillary Density).

The model presented predicts endurance performance in highly motivated athletes who are not carbohydrate depleted, hypoglycemic, hyperthermic or dehydrated. The remaining portion of this review will discuss how these factors impair performance. It should be noted that discussion of the psychological factors determining endurance performance is beyond the scope of this review. It is assumed that highly motivated athletes can exercise to the point where physiological factors reliably predict performance. This is not to say, however, that psychological factors can't extend physiological performance even in highly motivated athletes.

Carbohydrate Feeding During Exercise

People fatigue after one to three hours of continuous exercise at moderate intensities (i.e. 60-80% VO_2max) in a cool environment due largely to depletion of carbohydrate as a substrate (Coggan & Coyle, 1991). Carbohydrate feedings (i.e. glucose, maltodextrins or sucrose) during exercise will delay fatigue by 30-60 minutes (Coggan & Coyle, 1991). However, this improvement in performance is not due to a sparing of muscle glycogen use during cycling, but instead it appears that the exercising muscles rely mostly upon blood glucose for energy late in exercise (Coyle et al., 1986; Coggan & Coyle, 1991). Substrate oxidation changes during prolonged exercise at 65-75% VO_2max in endurance trained subjects when

Determinants of Endurance Performance

fasted overnight. Approximately 50% of the energy for exercise at 70% VO_2max is derived from fat, with equal contributions from plasma FFA and intramuscular triglycerides during the early period. There is a small increase in plasma FFA contribution over time. The remaining 50% of the energy is derived from carbohydrate. During the early portions of exercise, the majority of carbohydrate energy is from muscle glycogen. As exercise progresses, muscle glycogen is reduced and contributes less to the carbohydrate requirements of exercise and there is increased reliance upon blood glucose. Therefore carbohydrate should be ingested to supply the blood with exogenous glucose at approximately 1 g/min, late in exercise. Studies which have observed carbohydrate ingestion throughout exercise to improve performance have fed subjects at a rate of 30-60 g/h, beginning early in exercise (Coggan & Coyle, 1991).

The shortest duration of moderate to high intensity endurance exercise that benefits from carbohydrate feeding is not entirely clear. Blood glucose concentration declines during the second hour of exercise and therefore it is generally believed to be beneficial for durations lasting longer than two hours. However, carbohydrate feedings also benefit intense exercise (i.e. 80-90% VO_2max) lasting one hour in duration (Below et al., 1995).

Hyperthermia

Heat production during intense exercise can elevate core and muscle temperature rapidly and it seems that hyperthermia is an independent cause of fatigue. It has long been recognized that no single factor poses a greater threat to an endurance athlete's health and performance than does hyperthermia (Costill, 1972). Athletes in laboratory studies seem to cease exercise at relatively homogenous esophageal temperatures and muscle temperatures (i.e. 40-41°C; González-Alonso et al., 1999). These points were recently emphasized in a study that exercised subjects in an environment that caused hyperthermia by limiting heat dissipation and thus prevented subjects from exercising the long durations typical in cooler

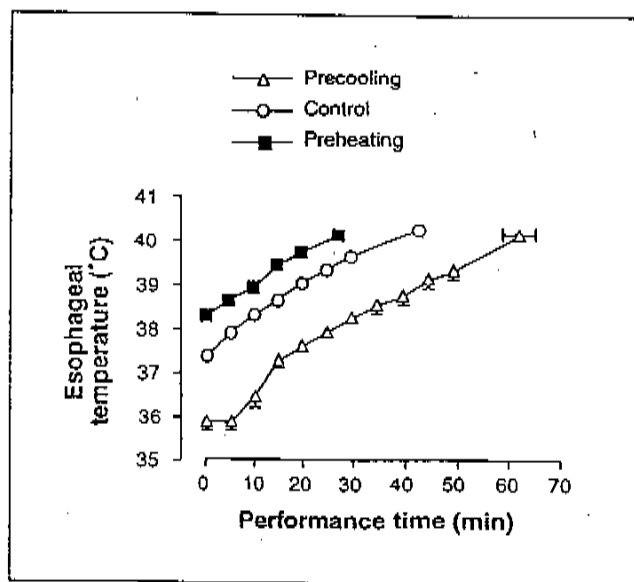


Figure 4: Performance time during exercise begun with differing esophageal temperatures (modified from González-Alonso et al., 1999).

Determinants of Endurance Performance

environments (e.g. >2 hours). Body core temperature was lowered or elevated before exercise (González-Alonso et al., 1999). As shown in Figure 4, exercise time to exhaustion was inversely related to the initial body temperature (i.e. 63, 46, and 28 min). Furthermore, increases in heart rate and reductions in stroke volume paralleled the rise in core temperature (González-Alonso et al., 1999).

There are several mechanisms by which hyperthermia might cause fatigue. It could affect areas of the central nervous system such as the hypothalamus, the motor cortex or muscle efferents that cause perceived exertion to increase and muscle fibre recruitment to become increasingly difficult (Bruck & Olschewski, 1987; Fuller et al., 1998).

Dehydration

Sweating rate during intense exercise is typically 1-2 litres per hour and thus athletes can become dehydrated if fluid replacement is not sufficient (González-Alonso et al., 1995; Noakes, 1993). It rarely is because a sufficient voluntarily drinking rate is usually only about one-half the sweating rate (Noakes, 1993). The major detrimental effects of dehydration are hyperthermia and impaired cardiovascular function. Dehydration causes very large reductions in stroke volume that can not be sufficiently compensated by increasing heart rate and as a result cardiac output declines during exercise at a constant power output and oxygen consumption (Sawka et al. 1979; González-Alonso et al., 1995; Montain & Coyle, 1992). Furthermore, blood flow to the exercising muscles declines (González-Alonso et al., 1998), thus stressing metabolism by reducing the delivery of oxygen and substrates. Impaired cardiovascular function from dehydration and hyperthermia, possibly sensed as a reduction in arterial blood pressure (González-Alonso et al., 1995), triggers a large catecholamine response to exercise which appears to elicit added metabolic stress (Febbraio et al., 1988). Needless to say, dehydration and the concomitant hyperthermia have great potential to impair endurance performance and endanger health.

References

- Below, P.R., Mora-Rodríguez, R., González-Alonso, J., & Coyle, E.F. (1995) Fluid and carbohydrate ingestion independently improve performance during 1 h of intense exercise. *Medicine and Science in Sports and Exercise* 27: 200-210.
- Buick, F.J., Gledhill, N., Froese, A.B., Spriet, L., & Meyers, E.C. (1980) Effect of induced erythrocythemia on aerobic work capacity. *Journal of Applied Physiology* 48: 636-642.
- Coggan, A.R., & Coyle, E.F. (1991) Carbohydrate ingestion during prolonged exercise: effects on metabolism and performance. *Exercise and Sports Science Reviews* 19: 1-40.
- Costill, D.L., & Fox, E.L. (1969) Energetics of marathon running. *Medicine and Science in Sports* 1: 81-86.
- Costill, D.L. (1972) Physiology of marathon running. *JAMA* 221: 1024-1029.
- Costill, D.L., Thomason, H. & Roberts, E. (1973) Fractional utilization of the aerobic capacity during distance running. *Medicine and Science in Sports* 5: 248-252.
- Coyle, E.F., Martin, W.H., Bloomfield, S.A., Lowry, O.H., & Holloszy, J.O. (1985) Effects of detraining on responses to submaximal exercise. *Journal of Applied Physiology* 59: 853-859.
- Coyle, E.F., Coggan, A.R., Hemmert, M.K., & Ivy, J.L. (1986) Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. *Journal of Applied Physiology* 61: 165-172.
- Coyle, E.F., Coggan, A.R., Hopper, M.K., & Walters, T.J. (1988) Determinants of endurance in well trained cyclists. *Journal of Applied Physiology* 64: 2622-2630.
- Coyle, E.F., Feltner, M.E., Kautz, S.A., Hamilton, M.T., Montain, S.J., Baylor, A.M., Abraham, L.D., & Petrek, G.W. (1991) Physiological and biomechanical factors associated with elite endurance cycling performance. *Medicine and Science in Sports and Exercise* 23: 93-107.
- Coyle, E.F., Sidossis, L.S., Horowitz, J.F., & Beltz, J.D. (1992) Cycling efficiency is related to the

Determinants of Endurance Performance

- percentage of Type I muscle fibres. *Medicine and Science in Sports and Exercise* **24**: 782-788.
- Coyle, E.F. (1998) Integration of the physiological factors determining endurance performance ability. *Exercise and Sports Science Reviews* **23**: 25-63.
- Febbraio, M.A., Lambert, D.L., Starkie, R.L., Proietto, J., & Hargreaves, M. (1988) Effect of epinephrine on muscle glycogenolysis during exercise in trained men. *Journal of Applied Physiology* **64**: 465-470.
- Febbraio, M.A., Snow, R.J., Stathis, C.G., Hargreaves, M., & Carey, M.F. (1994) Effect of heat stress on muscle energy metabolism during exercise. *Journal of Applied Physiology* **77**: 2827-2831.
- Farrell, P.A., Wilmore, J.H., Coyle, E.F., Billing, J.E., & Costill, D.L. (1979) Plasma lactate accumulation and distance running performance. *Medicine and Science in Sports and Exercise* **11**: 338-344.
- Fink, W.J., Costill, D.L., & Pollock, M.J. (1977) Submaximal and maximal working capacity of elite distance runners. Part II. Muscle fiber composition and enzyme activity. *Annals of the New York Academy of Sciences* **301**: 323-327.
- Fuller, A., Carter, R.N., & Mitchell, D. (1998) Brain and abdominal temperatures at fatigue in rats exercising in the heat. *Journal of Applied Physiology* **84**: 887-883.
- González-Alonso, J., Mora-Rodríguez, R., Below, P.R., & Coyle, E.F. (1995) Dehydration reduces cardiac output and increases systemic and cutaneous vascular resistance during exercise. *Journal of Applied Physiology* **79**: 1487-1496.
- González-Alonso, J., Mora-Rodríguez, R., Below, P.R., & Coyle, E.F. (1997) Dehydration markedly impairs cardiovascular function in hyperthermic endurance athletes during exercise. *Journal of Applied Physiology* **82**: 1229-1236.
- González-Alonso, J., Calbet, J.A.L., & Nielsen, B. (1998) Muscle blood flow is reduced with dehydration during prolonged exercise in humans. *Journal of Physiology* **513**: 895-905.
- González-Alonso, J., Teller, C., Andersen, S.L., Jensen, F.B., Hyldig, T., & Nielsen, B. (1999) Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *Journal of Applied Physiology*, **86**: 1032-1039.
- Holloszy, J.O. (1967) Biochemical adaptations in muscle: effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle. *Journal of Biological Chemistry* **242**: 2278-2282.
- Holloszy, J.O., & Coyle, E.F. (1984) Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology* **56**: 831-838.
- Horowitz, J.F., Sidossis, L.S., 199 & Coyle, E.F. (1993) High efficiency of Type I muscle fibres improves performance. *International Journal of Sports Medicine* **15**: 152-157.
- Ivy, J.L., Withers, R.T., Van Handel, P.J., Elger, D.H., & Costill, D.L. (1980) Muscle respiratory capacity and fiber type as determinants of the lactate threshold. *Journal of Applied Physiology* **48**: 523-527.
- Kushmerick, M.J. (1983) Energetics of muscle contraction. In: L.E. Peachey, Adrian, R.H., Geiger, S.R. (Eds.) *Handbook of Physiology, Section 10: Skeletal Muscle*. Bethesda, MD: American Physiological Society, pp. 189-236.
- Margaria, R., Edwards, H.T., & Dill, D.B. (1933) The possible mechanisms of contracting and paying the oxygen debt and role of lactic acid in muscle contraction. *American Journal of Physiology* **106**: 689-715.
- Montain, S.J., & Coyle, E.F. (1992) The influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *Journal of Applied Physiology* **73**: 1340-1350.
- Noakes, T.D. (1993) Fluid replacement during exercise. *Exercise and Sports Science Reviews* **21**: 297-330.
- Romijn J.A., Coyle, E.F., Sidossis, L., Gastaldelli, A., Horowitz, J.F., Endert, E., & Wolfe, R.R. (1993) Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity. *American Journal of Physiology* **265**: E380-E391.
- Sawka, M.N., Knowlton, R.G., & Critz, J.B. (1979) Thermal and circulatory responses to repeated bouts of prolonged running. *Medicine and Science in Sports* **11**: 177-180.