

Christoph Zinner · Billy Sperlich *Editors*

Marathon Running: Physiology, Psychology, Nutrition and Training Aspects

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Chapter 1

Physiological Aspects of Marathon Running

Billy Sperlich

Abstract Marathon running has evolved as one of the world's popular running experiences. Independent of the runner's performance level the marathon event represent a major challenge to the runner's biology. Multiple integrated physiological processes operate to resist fatigue during marathon running. The physical preparation for a marathon involves a series of complex biological adaptations to counteract exercise induced fatigue. The following chapter aims at describing important physiological components that are proposed to constrain a champion's physiological capacity for ultimate endurance performance. Further, potential limiting factors of the lungs, cardio-vascular system, blood oxygen carrying capacity, muscle properties and metabolism are explained in order to understand the underlying mechanisms for developing specific training methods and to estimate the race pace during marathon running. Other important biological aspects involved in marathon running such as nutrition, thermoregulation, biomechanics will be discusses in detail in the following chapters.

Keywords Marathon • Oxygen uptake • Central limitations • Pulmonary diffusion • Blood oxygen carrying capacity • Muscle adaptation • Lactate threshold • Substrate regulation • Running • Running economy

1.1 Introduction

The question, “the two-hour marathon: who and when?” (Joyner et al. 2011) was mentioned in 2011, initiating a lively debate about who, how, and when an exciting barrier of human performance would be broken. The query instigated a search to identify an outstanding human with extraordinary physiological and mental stamina.

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During marathon running and prolonged endurance exercise, perceptions of mental and muscular fatigue eventually occur accompanied by a decline in muscular performance (Kay et al. 2001; Green 1997; Kay and Marino 2000; Wilmore and Costill 1999; Millet et al. 2000, 2002; Pinniger et al. 2000; Gibson et al. 2001). To date several cognitive, bio-chemical and mechanical models have been proposed to explain the process of fatigue (Noakes 2000, 2007; Hampson et al. 2001; Hunter et al. 2003). However, the fatigue-related process is a complex interplay of neuro-humoral, metabolic, cardio-vascular and cognitive factors. It has been suggested that the action of a central (brain-derived) neural regulator controls marathon performance in anticipation to avoid physical harm (Noakes 2007). This constellation of factors appears highly individual and remains under investigation (Bouchard et al. 1986).

The mental and physical fatigue occurring during marathon eventually defines the velocity achieved during marathon running. To date numerous physiological and mental components limiting the performance during marathon have been identified. So far, three important physiological components are proposed to constrain a champion's physiological capacity for ultimate endurance performance. These components include: the involvement of aerobic and anaerobic energy production—as reflected by the runner's peak oxygen uptake ($\dot{V}O_{2\text{peak}}$), velocity at lactate threshold (v_{LT}), and running economy (RE) (Joyner and Coyle 2008). More than 70 % of the inter-individual variance in long-distance running has been attributed to $\dot{V}O_{2\text{peak}}$, lactate threshold, and running economy (di Prampero et al. 1986) and together, can to a great extent, explain variation in marathon performance (Sjodin and Svedenhag 1985).

Of course, other factors such as climate, nutrition, mental stamina, and motivation also significantly affect marathon performance (Fig. 1.1 for more details). For this reason, we have dedicated separate chapters to each of the aforementioned aspects of marathon science.

It is well known that aerobic metabolism depends highly on oxygen availability of contracting muscle cells dictated by central (cardio-respiratory and hemodynamic) and peripheral (oxygen extraction) processes (Bassett and Howley 2000). A substantial body of evidence has shown the $\dot{V}O_{2\text{peak}}$ to be one of the most salient predictors of endurance performance since (i) $\dot{V}O_{2\text{peak}}$ determines the upper limit for endurance performance (i.e., a runner cannot sustain exercise above 100 % $\dot{V}O_{2\text{peak}}$ for prolonged bouts) (Bassett 2002) and (ii) there exists a strong correlation between $\dot{V}O_{2\text{peak}}$ and performance during a long distance run (Costill 1970).

However, significant variations in $\dot{V}O_{2\text{peak}}$ have been observed among runners with a similar level of performance, indicating that other components play an important role in inter-individual performance variability. From a physiological standpoint, the oxygen cost of running (expressed as oxygen uptake at submaximal running), and lactate threshold are additional factors effecting marathon performance. Basically, $\dot{V}O_{2\text{peak}}$ and lactate threshold define the duration that aerobic and anaerobic processes can be maintained, while the runner's economy regulates marathon velocity for a given amount of energy consumption.

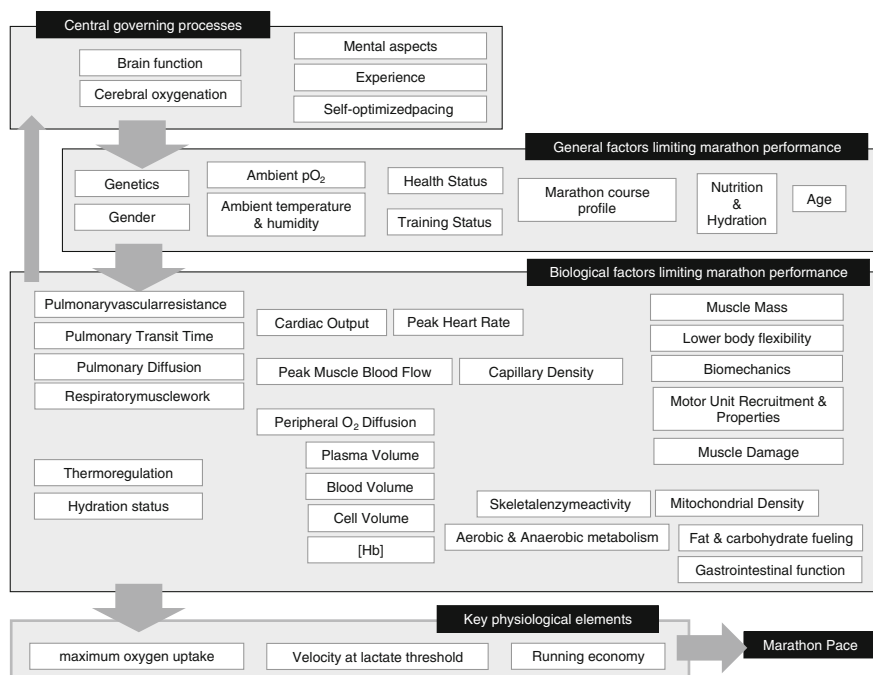


Fig. 1.1 Physiological and mental components limiting the performance during marathon (please note the list may not be exhaustive)

1.2 Oxygen Uptake During Marathon Running

A runner's $\dot{V}O_{2\text{peak}}$ is defined as the maximum amount of oxygen taken up and consumed by all tissue during exhaustive exercise (Bassett and Howley 2000). The $\dot{V}O_{2\text{peak}}$ consists primarily—although not exclusively—of the ability

- (i) to transport large amounts of blood (i.e. high cardiac output and total body hemoglobin),
- (ii) to distribute blood (i.e. by muscle blood flow) and
- (iii) to extract and utilize oxygen within the muscle cell.

(Bassett and Howley 2000; Joyner and Coyle 2008). The biological adaptation from long distance running allows an athlete to run a marathon at approximately 75–85 % of $\dot{V}O_{2\text{peak}}$ (Joyner and Coyle 2008; Bassett and Howley 2000). In contrast a 5 and 10-km run will be performed at 90–100 % $\dot{V}O_{2\text{peak}}$ explaining why marathon runners, when compared to middle-distance runners, possess a lower $\dot{V}O_{2\text{peak}}$. Male world-class marathon runners hold a $\dot{V}O_{2\text{peak}}$ of >80 ml/min/kg and female runners of >75 ml/min/kg.

1.3 Central Limitations to $\dot{V}O_{2\text{peak}}$

The primary limiting factors of $\dot{V}O_{2\text{peak}}$ are related to cardiac dimension, pulmonary diffusion and blood oxygen carrying capacity (Green et al. 1987, 1990).

1.3.1 Cardiac Dimensions

Endurance exercise provokes morphological, regulatory, and functional adaptation of the runner's heart (Pavlik et al. 2013). It is well known that endurance athletes' hearts adapt to training with a reduction in their rate at rest (resting bradycardia) resulting in heart rates <50 bpm (Underwood and Schwade 1977). Such a reduction is due to enhanced parasympathetic and reduced sympathetic activity. The reduction in resting heart rate following endurance training allows athletes' to perform at a lower heart rate with equal running velocity compared to pre-training.

Recreational endurance exercise produces a global cardiac enlargement, potentially increasing dimensions of all cardiac chambers but rarely produce left ventricular wall thickness greater than normal (Thompson 2007). However, in athletes performing extreme endurance disciplines such as ultra-runners, cardiac dimensions—especially chamber size and wall thickness (left ventricle internal dimensions, intraventricular septum thickness, posterior wall thickness)—may be significantly enlarged beyond normal (Nagashima et al. 2003; Thompson 2007).

The inter-individual difference in peak stroke volume (volume of blood expelled by the heart per beat) explains most of the variance in runners' $\dot{V}O_{2\text{peak}}$. It is noteworthy, that the peak values of heart rate are limited and decline with age, but stroke volume increases substantially especially with high-intensity exercise (Helgerud et al. 2007).

1.3.2 Pulmonary Diffusion

At sea level, pulmonary diffusion may limit performance, since in some cases, the ability of the pulmonary system to oxygenate blood may be insufficient (Dempsey et al. 1984; Powers et al. 1985). The runner's large cardiac output, that is, the volume of blood being pumped over time, permits the transportation of greater amounts of blood volume per heartbeat. The large blood transport however, may not allow the lungs to fully saturate hemoglobin with oxygen since the transit time of the blood within the lungs may be too short. Thus, well-trained athletes with large cardiac dimensions may show signs of "exercise-induced arterial hypoxemia" (Dempsey et al. 1984), especially when commencing heavy exercise.

1.3.3 Blood Oxygen Carrying Capacity

At the commencement of (endurance) exercise, the vascular system reallocates blood flow (within and between muscles) to metabolically active skeletal muscles, to ensure optimal O_2 -extraction. Increased blood allocation to the muscles is primarily achieved by elevated blood volume and red blood cells. Low-intensity endurance exercise has been shown to improve plasma volume (Green et al. 1987) and muscle blood flow (Coyle 1999) within a relatively short time period (after approximately 3 days of training (Green et al. 1987)), however a greater amount of exercise is necessary (approximately 3–5 weeks with 3–5 sessions/week) to further increase $\dot{V}O_{2peak}$ (Hickson et al. 1981) and capillary- and mitochondrial density (Hoppeler and Weibel 2000).

Reduced blood volume due to dehydration may compromise heat loss and improve thermal strain in marathon runners (Cheuvront and Haymes 2001). Remaining well hydrated during marathon (especially when performing in the heat) is (i) essential for optimal temperature regulation and (ii) upholds blood pressure and cardiac output for proper oxygen transport.

Altogether, the oxygen delivery to the exercising muscles may be limited by the aforementioned central factors however, several peripheral factors may also minimize endurance performance.

1.4 Peripheral Limitations to $\dot{V}O_{2peak}$

It is well documented that mitochondria enzyme activity as well as capillary density limit peripheral O_2 -utilization. Furthermore, the increase of enzymes as well capillaries improves peak oxygen uptake (Pringle et al. 2003; Kirkendall and Garrett 1998; Hoppeler and Weibel 2000; Tonkonogi et al. 2000; Bizeau et al. 1998) resulting in greater muscular oxygen extraction from the blood stream.

The trained, in contrast to untrained muscle, has three times greater capillary density, three to four times greater activity of aerobic enzymes, a larger content of slow twitch fibers (type I) (Henriksson 1992), and more efficient type I- (Linossier et al. 1993) and II-muscle fibers (Billat 2001). All aforementioned adaptations ultimately allow improved mitochondria ATP-synthesis leading to postponed onset of fatigue.

1.4.1 Muscle Adaptation and Lactate Threshold

The training goal of a marathon runner is to increase the velocity that can be maintained over 42.195 km by improving (i) the efficiency of bio-chemical process to transfer chemical into mechanical energy, (ii) biomechanics of locomotion, (iii) the ability to resist fatigue.

In endurance athletes, the velocity at the lactate threshold (v_{LT}) is closely linked to performance (Midgley et al. 2007; Bassett and Howley 2000). As a result, the

v_{LT} and fractional utilization of $\dot{V}O_{2peak}$ at v_{LT} ($\% \dot{V}O_{2peak}$) are regarded as good indicators of endurance performance between individuals of different ages, genders, and disciplines (Zinner et al. 2015). In untrained individuals, there is typically no increase in blood lactate $<60\%$ of peak oxygen uptake, although this value may vary individually (Scharhag-Rosenberger et al. 2010). In endurance-trained individuals, a rise in the concentration of blood lactate occurs at $75\text{--}90\%$ of peak oxygen uptake. It has long been suggested that the rise in blood lactate is the cause for muscle fatigue due to muscle hypoxia. However, as Cairns nicely summarized, lactate per se is not the reason for acidosis-induced muscle fatigue (Cairns 2006). As mentioned earlier, the relative contribution of anaerobic metabolism plays a minor role during marathon running; yet in 5 and 10 km events the contribution accounts for up to 20% of the total energy turnover (Joyner and Coyle 2008).

1.4.2 Substrate Regulation During Marathon Running

Depending on body size, gender, and age, the muscle mass of an endurance athlete totals approximately 15 kg (Hagerman 1984; Coyle et al. 1991). Examination of muscle biopsies have identified different muscle fiber types with different glycolytic and oxidative capacity (Saltin et al. 1977; Bassel-Duby and Olson 2006). Depending on the mode, frequency, intensity, duration of exercise, nutrition and environmental factors, the muscle has the potential to highly adapt to endurance training (Hawley et al. 1985; Zierath and Hawley 2004). Plasticity of adaptation however, seems (Holloszy 1967) genetically determined (Bouchard et al. 2011) and explains the large inter-individual variance in muscle distribution.

It is important to note, that for a marathon runner, fat oxidation during moderate or high intensity running is insufficient to satisfy the muscular ATP demands thereby favoring glycogen pathways. All marathon runners, independent of performance level, rely on carbohydrate fueling as evidenced by an average respiratory exchange ratio of >0.90 in the last half of the marathon (Spriet 2007).

With ongoing high-intensity running mitochondrial oxidation capacity is limited, consequently leading to the production of lactic acid (Holloszy et al. 1977; Robergs et al. 2004; Joyner and Coyle 2008). The oxidation system is highly adaptable to endurance training and explains why $\dot{V}O_{2peak}$ (Daussin et al. 2007, 2008; Gibala et al. 2006; Burgomaster et al. 2008; Akimoto et al. 2005; Kusuhabara et al. 2007; Serpiello et al. 2011; Laursen and Jenkins 2002) increases and well trained marathon runners may perform close to their $\dot{V}O_{2peak}$ without reaching V_{LT} (Holloszy et al. 1977).

The velocity during a marathon depends to a great extent on a high level of aerobic metabolism (Joyner and Coyle 2008). Depending on the analysis and modelling, aerobic metabolism will account for $>86\text{--}93\%$ of energy production in distance running (Duffield et al. 2005a, b). With increased running duration, the capacity of fueling contributes to fatigue since glycogen stores deplete with

prolonged exercise, thereby limiting oxidative ATP generation, and ultimately forcing the runner to reduce pace. In this case, special nutrition strategies before and during a marathon may counteract early depletion of glycogen stores by supplementing glycogen store and preventing hypoglycaemia (Coyle et al. 1983; Sherman and Costill 1984; Murray 1998).

Depending on the performance level, a marathon is usually run within 2–6 h. The main fuels for ATP production and muscle contraction derive from muscle and liver carbohydrates as well as muscle and adipose fat (van Loon et al. 2001; O'Brien et al. 1993). The depletion of muscle and liver glycogen inevitably lead to decreased performance (Green 1997; St Clair Gibson et al. 2001). For this reason, the intake of approximately 30–60 g CHO/h has been proposed during high-intensity exercise (Coyle and Montain 1992) to prevent muscle glycogen depletion (Stephens et al. 2001; Wojtaszewski and Richter 1998).

Prolonged muscle contraction, for example, during marathon running, requires sustained energy fueled by continuous generation of the universal energy compound ATP (adenosine triphosphate). ATP production during marathon running mainly relies on the breakdown of energy-rich molecules from glucose (also called as glycolysis) and mitochondrial respiration (ATP production by breakdown of carbohydrates and free fatty acids aerobic metabolism) (Romijn et al. 1993).

As a general rule, the use of carbohydrates for ATP production increases with intensity. Untrained runners generally use a higher relative amount of carbohydrates at a given intensity when compared to well-trained runners. With augmented fitness (i) the ability to take up free fatty acids from the plasma rises, (ii) the ability to store and oxidize intramuscular triacylglycerol increases and (iii) the maximal rates of carbohydrate and fat oxidation elevates (Hawley 2002). Collectively, such adaptations improve the reliance on fat oxidation at a given running velocity with a simultaneous sparing of carbohydrate fueling.

1.5 Running Economy

Recently, running economy has been deemed the “forgotten factor” in marathon running (Foster and Lucia 2007). Running economy [defined as the steady state $\dot{V}O_2$ expressed in ml/kg/min (Costill et al. 1973), or ml/min/m (di Prampero et al. 1986)] distinguishes between athletes of different performance levels (Conley and Krahenbuhl 1980). Runners with similar $\dot{V}O_{2peak}$ may perform differently during a marathon depending on their economy of running. Well trained runners display lower submaximal oxygen uptake at a given intensity when compared to less trained runners (Saunders et al. 2004; Barnes and Kilding 2015). Male world class runners have been shown to have a peak oxygen uptake of $\dot{V}O_{2max}$: 83 ml/min/kg need at 16 km/h ca. approximately 40 ml/min/km (Lucia et al. 2008), female runners with a $\dot{V}O_{2peak}$ of 75 ml/min/kg run 16 km/h with 44 ml/min/kg (Jones 2006). Recent reviews have nicely summarized which training strategies may be

most promising for increasing running economy in the range of 1–8 %, including high-intensity (uphill) running, different forms of strength training, and high altitude training (Barnes and Kilding 2015).

Ultimately however, the amount of accumulated years of running seems to have the greatest influence on improvements in running economy (Nelson and Gregor 1976; Svedenhag and Sjödin 1985; Jones 2006; Conley and et al. 1984). For example, a former female world class runner improved her economy of running by 14 % within 5 years (Jones 2006). As experimentally proven, the doubling of training volume does not lead to further gains in performance (Costill et al. 1988, 1991; Londeree 1997; Lake and Cavanagh 1996). It appears that once a certain level of adaptation is reached, further adaptations seem to be more dependent on high-intensity rather than on high-volume training (Londeree 1997).

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Chapter 2

Biomechanics of Marathon Running

Thomas Stöggl and Tobias Wunsch

Abstract This book section starts with an introduction towards the historical development of modern marathon running with respect to number of participants, gender and age distribution and the world record marathon times. This encompasses the development of the mean running velocities, details about the applied stride characteristics (e.g. stride rate, stride length), the applied strike patterns (e.g. forefoot strike vs. heel strike), the prevalence and causes of running related injuries. The second part gives an overview about general biomechanical principles which are relevant in running. In particular, specific definitions (phase definition of the running cycle, strike patterns), force components and the mathematical basics with respect to running energetics, joint loading, leg stiffness etc. are presented. The final part gives an overview about the state of the art knowledge about performance and injury related aspects with respect to marathon running from a biomechanical perspective. Special attention is paid to anthropometrics, running technique related aspects, fatigue effects and footwear.

2.1 Statistics About Marathon Running

2.1.1 *Historical Development of Marathon Running*

2.1.1.1 General Data About Marathon Running

Marathon running, covering the distance of 42.195 km is part of the Olympics since 1896. Since the New York City Marathon 1976, the sport has exploded on a global basis. Over the last 40 years, millions of runners participate in marathons in the world-metropolises like Tokio, Bosten, London, Berlin, Chicago and New York with more than 40,000–50,000 finishers, per each. These races, which belong to the

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