

The limits of human performance

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Abstract

Human performance, defined by mechanical resistance and distance per time, includes human, task and environmental factors, all interrelated. It requires metabolic energy provided by anaerobic and aerobic metabolic energy sources. These sources have specific limitations in the capacity and rate to provide re-phosphorylation energy, which determines individual ratios of aerobic and anaerobic metabolic power and their sustainability. In healthy athletes, limits to provide and utilize metabolic energy are multifactorial, carefully matched and include a safety margin imposed in order to protect the integrity of the human organism under maximal effort. Perception of afferent input associated with effort leads to conscious or unconscious decisions to modulate or terminate performance; however, the underlying mechanisms of cerebral control are not fully understood. The idea to move borders of performance with the help of biochemicals is two millennia old. Biochemical findings resulted in highly effective substances widely used to increase performance in daily life, during preparation for sport events and during competition, but many of them must be considered as doping and therefore illegal. Supplements and food have ergogenic potential; however, numerous concepts are controversially discussed with respect to legality and particularly evidence in terms of usefulness and risks. The effect of evidence-based nutritional strategies on adaptations in

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terms of gene and protein expression that occur in skeletal muscle during and after exercise training sessions is widely unknown. Biochemical research is essential for better understanding of the basic mechanisms causing fatigue and the regulation of the dynamic adaptation to physical and mental training.

Introduction

How fast, how high, how precise, what are the limits of human performance? This chapter identifies selected limiting factors and considers how biochemical approaches may help to improve performance.

Limiting factors of human performance

Limiting factors of human performance can be divided into three categories, the task, environmental and human factors, all interrelated. Therefore each performance is defined as a resistance requiring mechanical power (P_{mech}) carefully regulated over time. P_{mech} must be matched by metabolic power (P_{met}) which is determined by metabolic energy (E_{met}) available during the performance. P_{mech} , P_{met} , E_{met} and the interrelationship between all three factors may differ almost completely depending on task and environment. Environmental conditions such as temperature, wind, humidity and altitude may be supportive or detrimental. Only a fraction of E_{met} spent during a task can be transformed into the task-specific P_{mech} as defined by the task-specific biomechanical efficiency (η) (eqn 1).

$$P_{\text{mech}} = P_{\text{met}} \cdot \eta = E_{\text{met}} \cdot t^{-1} \cdot \eta$$

Force of a single maximal contraction of minimal duration is determined by the number of contractile filaments per cross-sectional area and their synchronized activity and therefore muscle mass. If numerous contractions are required, sustainability of the P_{met} becomes a performance-limiting factor enforcing a reduction in force and therefore in P_{mech} if performance time increases. The metabolic performance limit (P_{lim}) is the highest E_{met} that can be spent on a task (E_{lim}). Therefore P_{lim} can be described as a function of E_{lim} and time to exhaustion (t_{ex}) (eqn 2).

$$P_{\text{lim}} = E_{\text{lim}} \cdot t_{\text{ex}}^{-1}$$

As first described by Momod and Scherrer [1] P_{lim} and t_{ex} are inversely interrelated. A real-life example of this interrelationship is demonstrated by best performances of an international class athlete during a competitive season including winning a national championship and a world record (Figure 1).

Limits to provide metabolic energy

The universal metabolic source of metabolic energy is ATP generated via re-phosphorylation of ADP and AMP using different chemotroph reactions. In most cells, resting metabolic rate is fully aerobic. Any transitions from

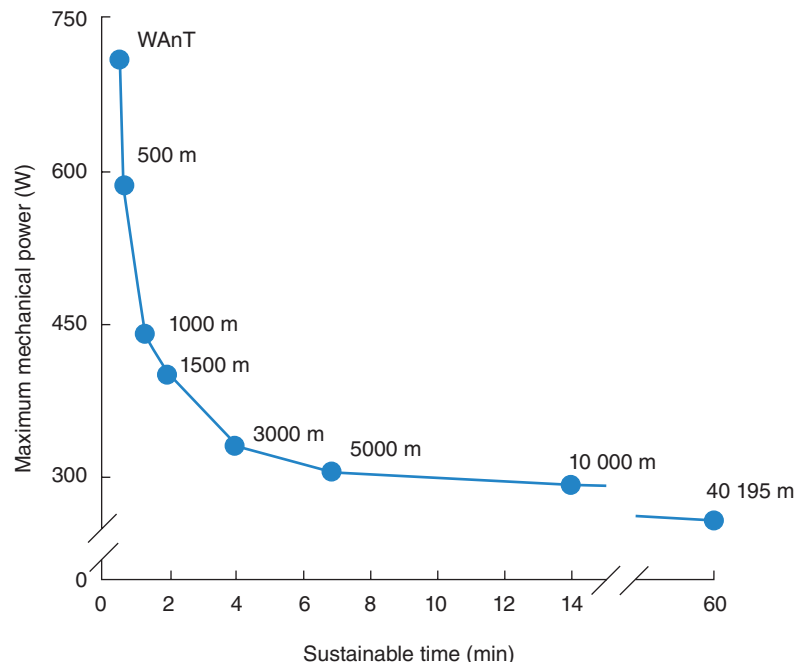


Figure 1. Maximal mechanical power as a function of sustainable performance time in selected performances of an international class athlete during a competitive season WAnT, 30 s Wingate Anaerobic Test; 500 m to 10 000 m, (National Champion) speed skating events; 40 195 m, 1 h unaccompanied speed skating (World Record).

rest to workloads above resting metabolic rate require anaerobic (E_{anaer}) and aerobic (E_{aer}) energy. The sum of E_{anaer} and E_{aer} reflects E_{lim} (eqn 3).

$$E_{\text{lim}} = E_{\text{anaer}} + E_{\text{aer}}$$

Sources of E_{anaer} are phosphocreatine and glycolysis, which can be utilized with immediate effect. However, the capacity of E_{anaer} is limited [2]. The corresponding E_{aer} is not fully instantaneously available. Due to the inertia of the aerobic system not only high-intensity short-duration events but also long-lasting endurance events have an anaerobic component. At high exercise intensities E_{aer} remains insufficient to meet P_{lim} . Within the limits of the dynamics of the oxygen uptake ($\dot{V}O_2$) maximum $\dot{V}O_2$ ($\dot{V}O_{2\text{max}}$) can be achieved and maintained for up to 7–9 min [3,4]. At events lasting longer than approx. 10 min aerobic metabolic rate may not achieve $\dot{V}O_{2\text{max}}$.

$\dot{V}O_2$ approaches the required metabolic rate exponentially with a time constant (τ) (Figure 2). Consequently, depending on E_{lim} and t_{ex} , the reliance on E_{anaer} and E_{aer} may be hugely different (Figure 3). A relatively simple model that defines P_{lim} combining eqn (2) and eqn (3) as a function of E_{anaer} and E_{aer} including the dynamic response of the $\dot{V}O_2$ is described by eqn (4):

$$P_{\text{lim}} = E_{\text{anaer}} \cdot t_{\text{ex}}^{-1} + E_{\text{aer}} \cdot t_{\text{ex}}^{-1} - [E_{\text{aer}} \cdot t_{\text{ex}}^{-1} \cdot \tau \cdot (1 - e^{-t_{\text{ex}}/\tau})] \cdot t_{\text{ex}}^{-1}$$

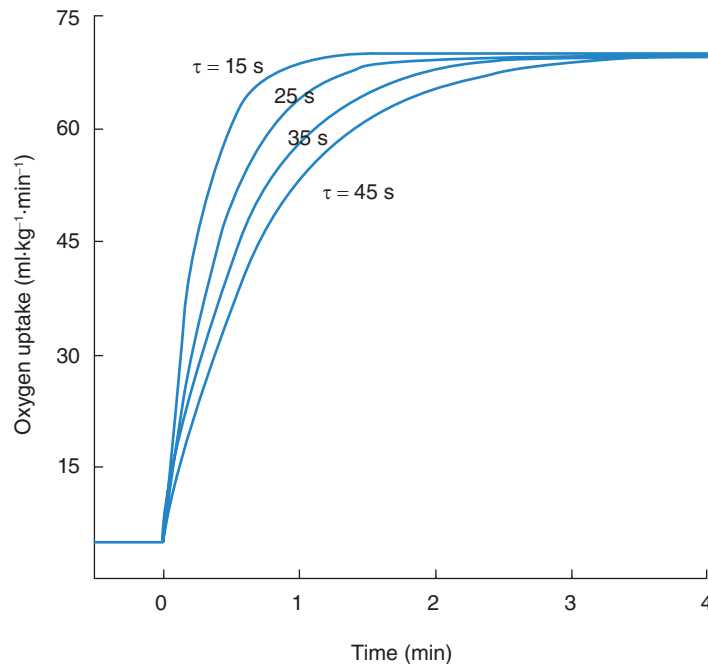


Figure 2. Oxygen uptake during 4 min of maximal performance as a function of time and time constants (τ) of oxygen onset kinetics

This model, which in principle is identical with the approach proposed by Wilkie [5], considers P_{lim} as a sustainable rate of metabolic demand. If P_{lim} equals P_{met} it defines the limit of P_{mech} (eqn 1). The dynamic response of E_{anaer} and E_{aer} is a function of maximum and sustainable anaerobic rate, achievable $\dot{V}O_2$ and τ and t_{ex} .

Eqn (4) is valid for contraction–relaxation cycles with cyclic loading and unloading of working limbs such as walking, running, cycling, speed skating (Figure 3) etc. These modes of exercise are well-defined in terms of the energy cost and efficiency and the corresponding E_{anaer} and E_{aer} are easily measurable [3,6–11]. The model does not consider any obstruction in muscular blood supply seen in events based on long-lasting static muscle contractions. Furthermore, the model does not consider any more complex kinetics of aerobic metabolism at sub-maximal exercise intensity, such as the slow component of $\dot{V}O_2$ [12]. The effect of such kinetics on the relative aerobic or anaerobic metabolic contributions at all-out performances is as yet unclear.

Maximal muscular performances with minimal aerobic energy provision lasting approx. 1 to maximally 10 s can reach refuelling rates of ATP of up to approx. 9 mmol per kg of dry muscle per s ($\text{mmol}\cdot\text{kg}^{-1}\cdot\text{s}^{-1}$) via phosphocreatine and of 2–9 $\text{mmol}\cdot\text{kg}^{-1}\cdot\text{s}^{-1}$ via glycolysis [2]. Muscular phosphocreatine content is in the magnitude of approx. 80 $\text{mmol}\cdot\text{kg}^{-1}$ of dry muscle⁻¹ and glycolytic capacity equivalent to an ATP provision of approx. 200 $\text{mmol}\cdot\text{kg}^{-1}$ of dry muscle⁻¹ [2]. Anaerobic capacity used may approach a maximum, may remain almost constant after 1–3 min and up to 7–15 min, or

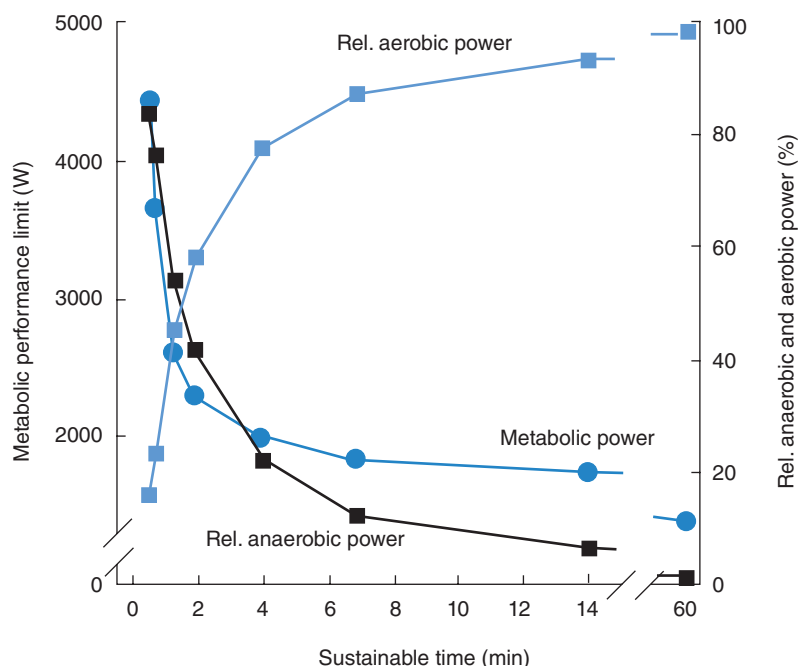


Figure 3. Estimated metabolic performance limit and relative aerobic and anaerobic energy contribution as a function of sustainable performance time

Data reflect a maximum anaerobic capacity of approx. $1.5 \text{ kJ}\cdot\text{kg}^{-1}$, which can be optimally utilized during 60–90 s performances, a $\dot{V}O_{2\text{max}}$ of approx. $70 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ($\tau=25 \text{ s}$). Metabolic performance limits match selected best performances of an international class athlete during a given season. WAnT, 30 s Wingate Anaerobic Test; 500 m to 10000 m, metric speed skating events; 40 195 m, 1 h unaccompanied speed skating (see Figure 1).

decrease asymptotically with performance time. The maximum duration of performances with more than 50% anaerobic metabolism appears to be 40–90 s [13]. The latter is modulated by maximal E_{anaer} and τ , which are usually between 1.2 and $1.8 \text{ kJ}\cdot\text{kg}^{-1}$ and 15 – 45 s . Effects of modulations in E_{anaer} are most prominent in sprint events (Figure 4). Changes in τ are most effective at events with a t_{ex} between 1 and 2 min (Figure 5) while changes in $\dot{V}O_{2\text{max}}$ or in the fraction of $\dot{V}O_{2\text{max}}$ that can be achieved without an increase in E_{anaer} after full adaptation of the $\dot{V}O_2$ to P_{met} modulates P_{lim} particularly in long-lasting endurance events.

The onset kinetics of aerobic metabolism or $\dot{V}O_2$ depends on aerobic fitness and training status [14]. However, there is an ongoing debate whether the velocity of $\dot{V}O_2$ -kinetics is limited at the pulmonary, cardiovascular or muscular level. Pharmacological minimization of convective and diffusive limits of oxygen delivery combined with elevation of the muscular blood flow adequate for a given exercise intensity before the start of exercise showed no effect on $\dot{V}O_2$ -kinetics and metabolism of an isolated muscle *in situ* during sub-maximal exercise intensity. However, at maximal exercise minimization of convective and diffusive limits of oxygen delivery and elevation of the muscular blood flow resulted in a small but significant and metabolically effective acceleration of muscular oxygen uptake kinetics [14].

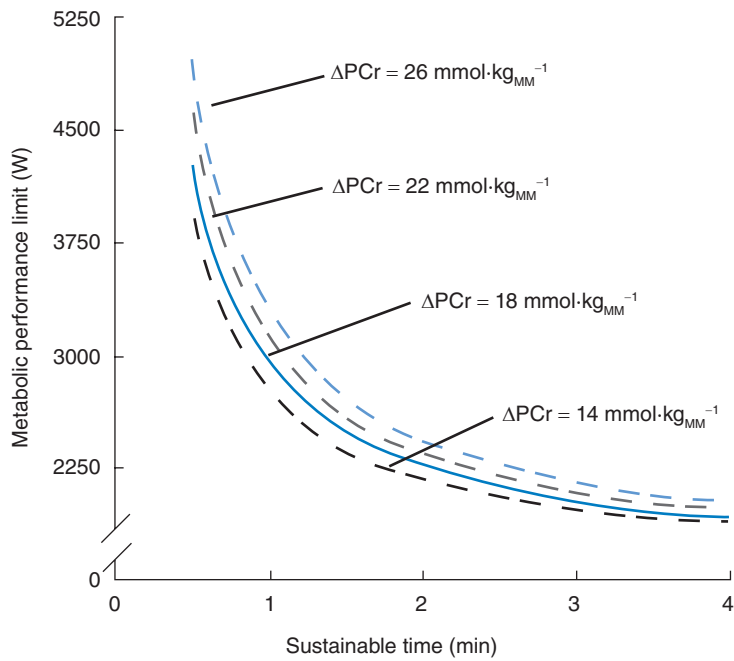


Figure 4. Maximum performance limit as a function of sustainable performance time and different phosphocreatine availability and a time constant of oxygen uptake of 25 s

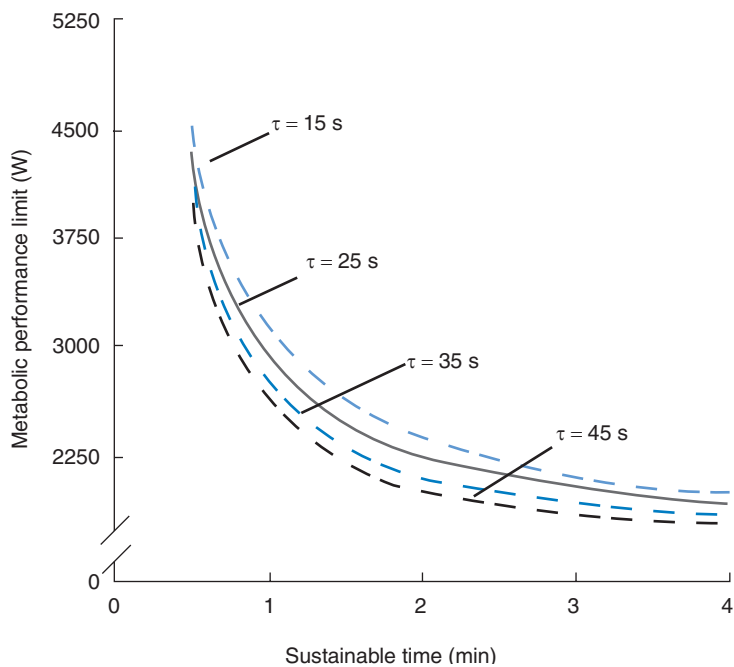


Figure 5. Maximum performance limit as a function of sustainable performance time and dynamics of oxygen uptake (Figure 2) and a given anaerobic capacity of approx. $1.5 \text{ kJ}\cdot\text{kg}^{-1}$

The latter maximal exercise experiment seems to support the concept that at maximal workload oxygen supply to the muscle cell is an independent performance-limiting factor, irrespective of whether the functional bottleneck is at pulmonary, cardiovascular, tissue or mitochondria level. Modelling the oxygen supply and utilization as a series of resistances in terms of decreases in oxygen partial pressure (pO_2) from ambient air to mitochondria level allows for a quantitative analysis of supply and utilization limits [15]. Working with large muscle groups under normoxic conditions, most healthy subjects will show an approx 50 mmHg decrease in pO_2 between ambient air and alveolar capillary, approx. 80 mmHg decrease between arterial and venous blood and approx. 20 mmHg decrease shared between muscle perfusion, diffusion and mitochondria capacity. In most athletes, the pO_2 decrease in the lungs can be functionally neglected because it is fully compensated for by the characteristics of the oxygen-haemoglobin binding curve. Consequently, during maximal exercise with large muscle groups for most athletes cardiac output appears to be the dominant limiting factor for aerobic metabolism. However, during exercise in environments with decreased ambient pO_2 , convective oxygen transport in the lungs may become a significant performance-limiting factor due to decreased alveolar diffusive transport. This may require even a decrease in cardiac output compared with normal ambient pO_2 conditions to increase erythrocyte transit times [16].

Selected healthy athletes demonstrate exercise-induced arterial hypoxaemia also at sea level and normal pO_2 [17]. Factors involved with exercise-induced arterial hypoxaemia are excessive alveolar to arterial pO_2 differences, inadequate compensatory ventilation, and acid- and temperature-induced shifts in O_2 dissociation at given pO_2 levels. Also expiratory flow limitations, ventilation-perfusion mismatch and diffusion limitations seem to be contributing factors; however, their physiological basis is not fully understood [18]. For these specific individuals the oxygen flow resistance of the lungs becomes functionally relevant and rate-limiting for aerobic metabolism.

At maximal work with small muscle groups the drop in pO_2 related to cardiovascular resistances decreases with any decrease in active muscle mass. The latter is combined with an increase in the fraction of pO_2 decrease on muscular level indicating the dominance of muscular factors as limits of maximal aerobic performance if the muscle mass stressed is sufficiently small. Although oxygen supply seems to reflect a limit of maximum aerobic metabolic rate, there is no fully convincing evidence for a lack of oxygen supply at exercise intensities below $\dot{V}O_{2max}$. The duration of sub-maximal performances sustainable for more than approx. 10 min is limited either by limitation of the adequate substrate for aerobic metabolism or is not fully understood.

Substrates for aerobic energy production are mainly carbohydrates or fat. In highly competitive athletes the percentage of body fat related to total body mass can vary by a factor of ten or even 15. Lowest body fat fractions, possibly borderline in terms of health, in the magnitude of approx. 2% have been seen in long-distance runners and in events regulated in weight categories like, for example, weightlifting, wrestling, judo and boxing, and highest values above

30% have been seen in selected shot putters [19]. Nevertheless, under the assumption of a total body mass of 60 kg, 1.8 kg of body fat and a typical energy cost of running of approx. $4 \text{ J}\cdot\text{kg}^{-1}\cdot\text{m}^{-1}$ [6], body fat could provide enough energy for a distance of almost 300 km. This distance is more than most world-class marathon runners cover during a weeks training. In a well-fed state corresponding glycogen stores of approx. 0.4 kg would last for just approx. 30 km, not sufficient to succeed a single marathon.

The interrelationship between glycolytic rate and aerobic pyruvate and fat combustion depends on the exercise intensity related to the maximal metabolic rate of an individual and thus P_{mech} , $\dot{V}\text{O}_2$ and aerobic metabolic rate increase almost linearly from rest to near maximum. Glycolytic rate adjusts non-linearly with a flat characteristic at low exercise intensities and steep increases at high workloads. This causes a deficit of pyruvate availability related to aerobic metabolic rate at low workloads. This lack of pyruvate is compensated for via the utilization of fatty acids [20]. As exercise increases, glycolytic rate increases more steeply and up-regulates pyruvate combustion ([21]; Figure 6).

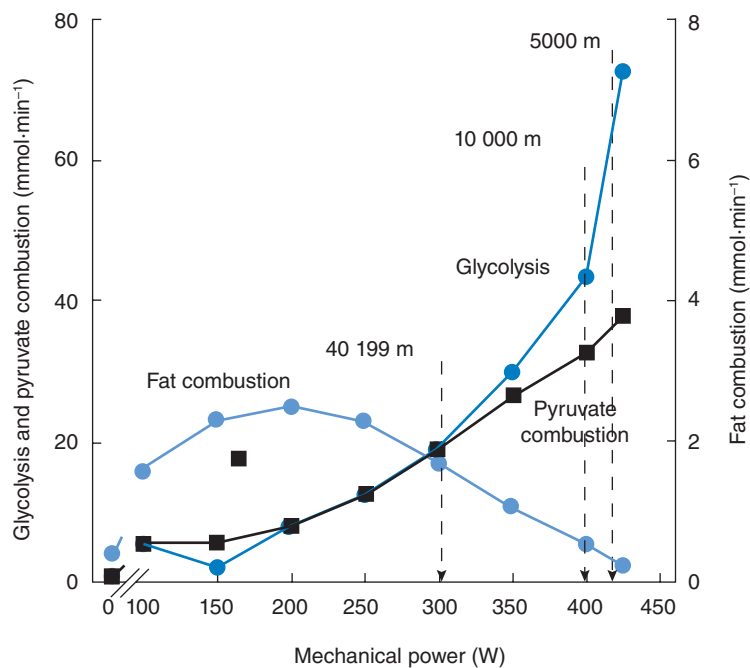


Figure 6. Estimated rates of glycolysis, and oxidative pyruvate and fat combustion as function of P_{mech} during incremental cycle ergometry related to speed skating performances of an international class athlete

Performances at 5000 m (approx. 7 min), 10000 m (approx. 14 min) and 1 h unaccompanied speed skating adjusted for difference in biomechanical efficiency between cycle ergometry and speed skating. The 5000 and 10000 m performances represent workloads where glycolytic rate clearly exceeds aerobic pyruvate combustion though $\dot{V}\text{O}_2$ is possibly below $\dot{V}\text{O}_{2\text{max}}$. The 1 h power output is near the threshold where glycolytic rate exceeds pyruvate combustion rate at a $\dot{V}\text{O}_2$ significantly below $\dot{V}\text{O}_{2\text{max}}$ (for details please see Beneke [25]).

There is evidence that pyruvate concentration serves as one activator of the pyruvate dehydrogenase irrespective of an increase or decrease in glycolytic rate [22–24]. Given limited glycogen stores and an almost unlimited availability of fat, the latter may help fine-tuning and optimize use of pyruvate as fuel for aerobic metabolism [25]. The steep increase of the glycolytic rate at higher exercise intensities exceeds the rate of aerobic pyruvate combustion at a metabolic rate that is significantly below $\dot{V}O_{2\max}$. There is a debate as to whether fat combustion reflects a relevant fraction of aerobic metabolism at and above the latter threshold. However, irrespective of a high between-subjects variability in exercise intensity, the duration of performances with glycolytic rates higher than pyruvate combustion are usually limited to significantly less than 1 h. During performances at this intensity domain, limits in the respiratory compensation of a corresponding metabolic acidosis seem to be relevant factors triggering symptoms of fatigue before glycogen depletion becomes a limiting factor. Induction of a respiratory alkalosis immediately pre-short-term maximal exercise can increase glycolytic energy supply by approx. 10% and performance by approx. 5% (R. Beneke, D. Böning and M. Hütler, unpublished work).

Limits to utilize metabolic capacity

As outlined above, the ability to provide metabolic energy in exercising muscle at the rate defined by P_{mech} and biomechanical efficiency seems to be highly predictive for performance limitations. However, with the exception of pathological conditions, maximal performance does not overstress biological systems to an extent that causes serious damage. Most situations that require a decrease in P_{met} or termination of performance do not show evidence of any clear biochemical limits. Consequently, the interrelationship between P_{mech} , P_{met} and performance time (Figures 1 and 3) does not necessarily fully stress the metabolic capacity of an athlete. It also includes a safety margin imposed in order to protect the integrity of the human organism.

The central nervous system clearly initializes any initial performance attempt. The brain may also have a central protective role as the perception of afferent input in terms of sensations and discomfort associated with heavy efforts leads to conscious or unconscious decisions to reduce P_{mech} or even terminate performance. The latter seems to be strongly supported by experiments with partial neuromuscular blockade [26]. However, the underlying mechanism of conscious or unconscious cerebral control and limitation of performance is not fully understood.

Cerebral limitation of performance may partly reflect restricted cerebral substrate availability and oxygen supply similar to that seen in peripheral muscles [26,27]. Brain hyperthermia, hypoglycaemia and decreased cerebral glucose uptake, changes in serotonergic activity, elevated ammonia levels, and alterations in regional dopaminergic activity may contribute to the impaired voluntary activation of the motor neurons after prolonged and strenuous exercise [28]. However, the fact that, even under conditions of maximal

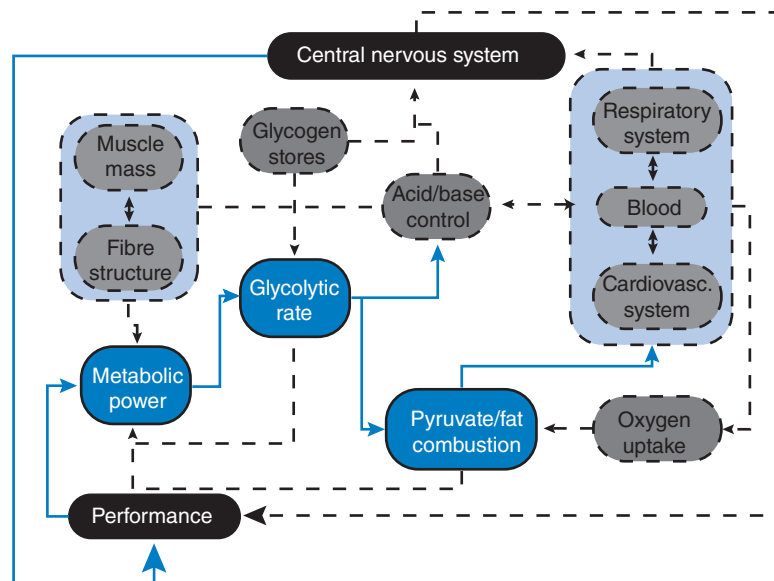


Figure 7. Performance as function of central nervous spatial and temporal recruitment of motor units, corresponding increases in metabolic power, anaerobic and aerobic metabolism (solid elements and lines); limitations and down-regulation of selected subsystems dependent on task and human factors (dotted elements and lines)

electrical stimulation of small muscles, muscular ATP cannot be decreased below approx. 60% of resting values clearly provides evidence that the theory of central control of performance limits can be heavily challenged [29]. The latter may support the idea that fine-tuning of performance, in terms of central nervous and peripheral management of stress and perception appears to be most probably a dynamic integration and multitasking of different peripheral and central mechanisms (Figure 7).

Moving the limits of performance

The idea to move borders of performance with the help of biochemicals is not new. Already two millennia ago, Plinius reported the performance-enhancing effect of shave-grass juice, and Philostratos and Galen mentioned positive effects of the ingestion of selected herbs, mushrooms and testicles [30]. During the past centuries, many biochemical findings resulted in highly effective pharmacological substances such as anabolic agents, anti-oestrogens, β -agonists, diuretics, stimulants, narcotics, glucocorticosteroids etc. Without any doubt, many of these pharmaceuticals have been widely used to increase performance in daily life, during preparation for sport events and during competition. However, many of such interventions must be considered as doping and therefore illegal [31]. Doping advice is no intention of this

chapter. Therefore the use of listed prohibited substances, blood doping including autologous, homologous or heterologous blood or red blood cells, haemoglobin products, the use of perfluorochemicals and any non-therapeutic use of cells, genes and genetic elements that are in conflict with anti-doping regulations will not be addressed (please see later chapters). A heavily marketed use of biochemicals in a broader sense is supplements and food with ergogenic potential. Many of these products and concepts are controversially discussed with respect to legality and particularly evidence in terms of usefulness and risks. Antioxidants, proteins, amino acids, vitamins and minerals are essential components of a balanced diet. However, additional oral supplementation does not necessarily increase endurance or strength.

Caffeine is ergogenic by sympathetic stimulation in certain aerobic activities. Creatine was found to be ergogenic in repetitive anaerobic sprints; however, it is unclear why mode of exercise seems to modulate its effectiveness [32]. Compared with non-vegetarians, supplementation of creatine may be more effective in vegetarians as their muscle creatine stores may be lower [33]. β -Hydroxy- β -methylbutyrate seems to have ergogenic potential in untrained individuals. However, to our knowledge, there are no studies on highly trained individuals. Attempts with pyruvate did not show any ergogenic effect. There is no evidence that androgenic precursors androstenedione and dehydroepiandrosterone increase any parameters of strength comparably with anabolic steroids. However, significant adverse effects similar to those seen after doping with anabolic steroids cannot be excluded [32]. Supplementation of (-)-Hydroxycitrate resulted in equivocal results on fat utilization during exercise [34]. More than two decades of research on carnitine supplementation failed to provide evidence for any performance-enhancing effect in healthy subjects [35]; however, there seems to be a convincing case that oral carnitine doses up to several grams are without toxic side effects [35]. *Eleutherococcus senticosus*, a species of small, woody shrub in the family Araliaceae native to Northeastern Asia, is lacking any evidence of the proposed improvement of fat metabolism and endurance [36].

There is a potential adverse effect of a vegetarian diet on iron status based on the low bioavailability of iron from plant foods. Given the vital role of iron for the red blood cell, iron supplementation may be necessary in vegetarians. Other nutrients that are candidates of concern particularly for vegetarians are zinc, vitamin B12, vitamin D and calcium. However, a vegetarian diet may increase antioxidant status in terms of vitamin C and E, and β -carotene [33].

Pre-exercise ingestion of bicarbonate may increase performance in events which require significant anaerobic energy [37]. Long- or medium-chain triacylglycerols have little or no effect on metabolism and do not alter subsequent exercise performance irrespective of time to exhaustion, medium-chain triacylglycerols may have a membrane-protective effect on red blood cells [38]. However, bicarbonate and triacylglycerols may cause gastrointestinal discomfort and therefore may induce a negative effect on performance.

Carbohydrate loading in the days before an endurance event lasting 90 min or more increases muscle glycogen and performance. Also 3–4 h before exercise, carbohydrate ingestion increases liver and muscle glycogen and enhances endurance performance. However, an increase in plasma insulin following carbohydrate ingestion especially in refined form in the hour before exercise inhibits lipolysis and liver glucose output, and can lead to transient hypoglycaemia during subsequent exercise in susceptible individuals [39]. If many purified carbohydrates like glucose or saccharose are consumed, there might be a relative lack of vitamin B1, which is important for decarboxylation of pyruvate.

The lack of evidence for successful use of the ergogenic potential of food and supplements reflects that the effect of nutritional strategies on molecular adaptations during and after exercise training sessions is widely unknown. Furthermore, the relationship between intensity, duration and mode of training on performance and underlying physiological and biochemical mechanisms is highly complex and not clear. Mathematical modelling has been suggested as a method of describing, analysing and predicting the effect of training and additional interventions on performance [40]. However, existing models lack specificity of input parameters and physiological and biochemical foundation [40]. An interesting modelling approach simulating gene and protein expression, protein synthesis and degradation in skeletal muscle as a function of muscular activity and inactivity requires sufficient evaluation and optimization [41].

Consequently, the key to improvement of performance is possibly not the development of new biochemicals and their application to athletes. Lack of a deeper understanding of the basic mechanisms related to acute response to exercise stress indicates one field where biochemistry may help more effectively. Tailored approaches specifically targeted on contractility, anaerobic and aerobic energy transfer, membrane transport and cellular integrity may become specific foci in the future. Particularly facilitated transmembrane transport may offer new avenues for maximal use of the multi-compartmentally organized system of stress response of the human organism. Hand in hand with transmembrane transport capacity goes a better understanding and possibly management of the inhomogeneity of physiochemical conditions in a specifically stressed tissue. There seems to be some potential in modulation and optimization of vasomotor control particularly in the lungs and in the brain.

Conclusion

Biochemical research seems to be one of the important keys for sustainable success in the movement of borders of performance. It is an essential tool for better understanding of the basic mechanisms causing fatigue. Furthermore, it appears to be specifically useful to clarify the process and regulation of the dynamic adaptation to physical and mental training.

Summary

- *Human performance depends on human, task and environmental factors, all interrelated.*
- *Performance defined by mechanical resistance and distance per time requires metabolic energy provided by anaerobic and aerobic metabolic energy sources.*
- *Capacity and rate limitations of different pathways to provide re-phosphorylation energy determine individual ratios of aerobic and anaerobic metabolic power and their sustainability.*
- *In healthy athletes, limits to provide and utilize metabolic energy are multifactorial and carefully matched.*
- *Limits of performance also include a safety margin, imposed in order to protect the integrity of the human organism under maximal effort.*
- *Perception of afferent input associated with effort leads to conscious or unconscious decisions to modulate or terminate performance; however, the underlying mechanisms of cerebral control are not fully understood.*
- *The idea to move borders of performance with the help of biochemicals is two millennia old.*
- *Biochemical findings resulted in highly effective substances widely used to increase performance in daily life, during preparation for sport events and during competition, but many must be considered as doping and therefore illegal.*
- *Supplements and food have ergogenic potential; however, numerous concepts are controversially discussed with respect to legality and particularly evidence in terms of usefulness and risks.*
- *The effect of evidence-based nutritional strategies on adaptations in terms of gene and protein expression that occur in skeletal muscle during and after exercise training sessions is widely unknown.*
- *The key to improvement of performance is possibly not the development of new biochemicals and their application to athletes.*
- *Biochemical research is essential for better understanding of the basic mechanisms causing fatigue and the regulation of the dynamic adaptation to physical and mental training.*

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