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CURRENT OPINION

Do Olympic Athletes Train as in the Paleolithic Era?

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Abstract Every 4 years, approximately 10,000 athletes participate in the Olympic Games. These athletes have dedicated several years of physical training to achieve the best possible performance on a given day. Their preparation has been supported by expert coaches and an army of sport scientists, whose overall responsibility is to ensure that the athletes are in peak condition for their event. Although every athlete prepares specifically for the unique physiological challenges of their event, all athletes have one common characteristic: they are *Homo sapiens*. They share a unique genome, which is the result of evolutionary forces beyond their individual control. Although studies on the influence of different genetic polymorphisms on selected athletic events have been proven to be of limited utility, a body of evidence—from molecular biology to whole-body measures—suggests that training adaptations are enhanced when the stimulus closely resembles the activity pattern of human ancestors. Because genetic evolutionary changes occur slowly in *Homo sapiens*, and the traditional physical activity and dietary patterns of *Homo sapiens* have undergone rapid and dramatic changes in previous centuries, we propose that modern humans are physiologically better adapted to training modes and nutritional strategies similar to the ones that their hominid ancestors evolved on, rather than those supported by modern societies. Such an ancestral pattern was mainly characterized by the prevalence of daily bouts of prolonged, low-intensity, aerobic-based activities interspersed with periodic, short-duration, high-intensity bursts of activity. On some occasions, such activity patterns were undertaken with low carbohydrate availability. Specific activities that enhanced strength and power were typically performed after aerobic activities. We present scientific evidence to support the appropriateness of this model, and we propose that future studies should address this hypothesis in a multitude of different sporting activities, by assessing the genetic responses to and performance-based outcomes of different training stimuli. Such information would provide data on which sport scientists and coaches could better prepare athletes and manage their training process.

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1 Introduction

The evolution of biological complexity beyond single-celled organisms was linked temporally with the development of an oxygen-rich atmosphere, combined with the capacity of selectable replicating organisms to transfer free energy, which is obligatory for transformation [1]. Subsequently, the impact of natural selection modelled the role of oxidative metabolism for survival in our *Homo*

predecessors, who perpetuated their genes under selective environmental pressure [2]. Furthermore, it has been suggested that the hunter–gatherer lifestyle adopted by our human ancestors required a large increase in aerobic activity, which could also have influenced human neurobiology [3]. Thus, the hereditary characteristics of our species are theoretically those that fit better with environmental demands for survival, as they are the product of millions of years of gene–environment interaction.

A robust body of evidence suggests that modern cardiovascular and metabolic diseases are linked to the change in human lifestyle that occurred in recent centuries, as a consequence of the Industrial Revolution [4–8]. This in turn promoted a sedentary lifestyle, which is opposite to the physically active way of life of our ancestors. There is some consensus about the greater impact of physical inactivity and its role in the prevalence of modern pandemics and life expectancy [9]. The new lifestyle could have disrupted the genetic pool and environmental requirements for survival, as our genetic endowment has not been significantly altered since the Paleolithic Era, when *Homo sapiens* were still hunter–gatherers [10]. In fact, our ancestors lived as hunter–gatherers for approximately 84,000 generations [4, 5]. The adaptations selected for survival during that wide timeframe may have become maladaptations under the current environmental change of physical inactivity [11]. This dissonance between Stone Age conditions and modern environments is the basis of the so-called *mismatch hypothesis* [8, 12, 13], which has been used by evolutionary medicine to explain most current diseases. The mismatch hypothesis has also been utilized in discussion of the controversy surrounding barefoot running [14].

While the relationship between genetic endowment, lifestyle and health seems clear, researchers have paid less attention to the possible influence of our genetic pool on the physiological adaptations to athletic training and subsequent sport performance. Sport genetics is a growing field, which has provided some gene candidates for greater responsiveness to various training modalities [15, 16]. However, the link between those genes, the training process and, more importantly, sport performance could be limited [17]. Alternatively, it may be interesting to analyze the evidence linking training stimuli and physiological responses with the pattern of physical activity that modelled the human genome via selection pressure. In other words, an appealing hypothesis could be that activities that favoured survival before and during the Paleolithic Era may evoke greater physiological adaptations and subsequent performance than other training stimuli [18]. If this assumption is correct, a more robust genetic response to training modalities more similar to the Paleolithic pattern of physical activity could be expected. Thus, the training load and the specific physiological responses to such training stimuli should be defined. This approach would contribute to more precise characterization of this activity pattern and the subsequent limits of human training adaptation and performance.

The aim of this review was to analyse the available evidence for the hypothesis that humans respond better to training stimuli that are similar to the exercise patterns of our ancestors than they respond to other training interventions.

2 Exercising in the Paleolithic?

2.1 Energy Intake

The most important characteristic of human physical activity is its intimate link to the energy requirements for survival [12]. That is, caloric expenditure is intended to guarantee caloric availability [19]. However, the niche of early hominins—and thus the nutritional composition of the early human diet—are still heavily debated. Integration of data from various disciplines suggests that for a long time period in evolution, hominins derived large amounts of energy from (terrestrial and aquatic) animal fat and protein [20]. The majority of carbohydrates were obtained from fresh fruits and vegetables, together with roots and tubers, and very little intake came from cereal grains or refined carbohydrates [21]. This was reversed by the onset of the Neolithic agricultural revolution—a remarkable economic transformation that could have been preceded by expanding diet breadth in the later Paleolithic, according to the 'Broad Spectrum Revolution' hypothesis [22]. Little is known about what our Paleolithic ancestors ate each day or in each season in any specific habitat, but it is suggested that anatomically modern *Homo sapiens* relied on a variety of food sources in varying environments. This nutritional flexibility may have been central to human evolution across seasonal variations, during climatic fluctuations and through famine times. Hominins moved to eat, whereas modern athletes eat to move.

Technological advances favoured a great increase in hard food—including proteins, which have been linked to the important increase in brain mass of our species. More importantly, Cordain et al. [23] reported that the diets of studied hunter–gatherer populations were higher in protein (19–35 %), lower in carbohydrates (22–40 %) and equivalent or even higher in dietary fat (28–58 %) than current diets. The lesser presence of carbohydrates in the prehistoric diet than in the Western diet could be one of its most relevant characteristics. From an exercise perspective, maintenance of prolonged, intense exercise (i.e. above the lactate threshold) could not be expected in those ancient

times, as glycogen availability would have been limited, resulting in greater reliance on fat metabolism [19]. Thus, lipolytic and phosphagen metabolic pathways would have been less limited by food availability than glycolysis [24].

2.2 Physical Activity for Survival

The main activities for survival could have involved low-intensity tasks performed on a regular basis. These daily activities could have included social interactions; maintenance of shelter and clothing; and gathering of wild plants, grains, and fruits, among other vegetables, for eating or for making tools [5, 11, 19]. As these raw materials were more abundant in the forest, our ancestors probably walked medium-to-long distances to look for them, as well as for hunting. This issue is important, since the prevalence of open environments in the proximity of hominin fossil sites has been documented recently, suggesting that woods covered less than ~40 % of hominin habitats [25]. There is anecdotal evidence of daily game pursuits of 10–15 km, with estimated and measured energy expenditures of ~3,000–5,000 kcal/day in modern hunter–gatherers [5, 10, 19, 26].

It appears that multiple hominoid lineages evolved in African highlands at altitudes of 1,000–2,000 m. Thus, evolution of human locomotor physiology may have occurred under conditions of mild hypobaric hypoxia [27], with hominid locomotion probably involving intermittent activities [28]. The greater reliance on aerobic pathways and the coupling efficiency between energy production and energy demand observed in high-altitude natives and endurance-trained athletes could partly account for the functional advantages of the so-called *lactate paradox* phenomenon (i.e. the attenuation of lactate accumulation despite maintained hypoxia) [29]. This controversial aspect of the metabolism of lactate could be interpreted as the result of directional selection for the early emerging *hypoxia tolerance/endurance performance phenotype* in human phylogeny [30].

An interesting hypothesis is that endurance running is a particular adaptive characteristic of the *Homo* genus [31]. The evidence supporting this unique adaptation may include various anatomical and functional characteristics [32–34], suggesting that the *Homo* genus is both an excellent endurance runner and a bad sprinter when compared with other species. Some authors have suggested that running could be important for scavenging and/or predator pursuit [31, 35]. Moreover, unlike other species, humans exhibit a constant energy cost of locomotion at different velocities [36], which allows good locomotor economy, independent of the frequent changes in velocity that are necessary for the aforementioned activity pattern. A key component is the probable link between human locomotion and access to a protein-rich diet, which could allow the important brain growth of our species. In this respect, endurance running could have increased the chance to encounter new environments, thus favouring the need for superior cognitive abilities for adaptation [36]. This assertion supports the role of physical activity as a mediator of neuronal plastic adaptations that could have had an important evolutionary role [3], with cognitive and motor skills considered as complementary within a general concept of 'activity' [37].

As hunting was the best source of energy intake and nutrients of quality, most of the habitual activities of our ancestors were probably related to hunting. More specifically, hunting could be broken down into various activities such as searching for and pursuing animals, throwing, sprinting, and carrying the game after taking the prey [5, 19]. Overall, such a pattern could be interpreted in terms of a polarized intensity distribution, with the predominance of prolonged low-intensity activities interspersed with some energy bursts of explosiveness in a predictable sequence in most cases (see Fig. 1). This polarized profile of physical activity could also be mediated by the aforementioned metabolic limitations associated with food availability in those ancient times.

It should be pointed out that the physiological differences between men and women could be related to differences in daily physical activity, with women being excluded from hunting large game animals [5, 38]. It is important to note that the division of labour has been linked to the origin of humankind [5, 8, 38, 39], and this salient characteristic of the human condition may have involved specific environment–gene interactions resulting in different age- and sex-dependent adaptations during millions of years of evolution. However, further comparative evidence supporting this hypothesis is needed.

3 'Paleolithic Training' for Athletic Success

The physiological demands of any sport are not necessarily similar to the physical activity demands of the prehistoric niche. More importantly, the *principle of allocation* predicts that excellence in one task can be attained only at the expense of average performance in all other tasks, which has been previously confirmed by Van Damme et al. [40] in an analysis of the records of top-level decathletes. Our main hypothesis suggests that the physical demands of our ancestors modelled our genome and therefore our capacity to respond better to training stimuli, independently of the physiological demands of competition. This means that although our ancestors' activities would be more similar to the current training activities of endurance athletes, other competitive athletes (e.g. team sport players, sprinters) would also benefit from this phylogenetic profile

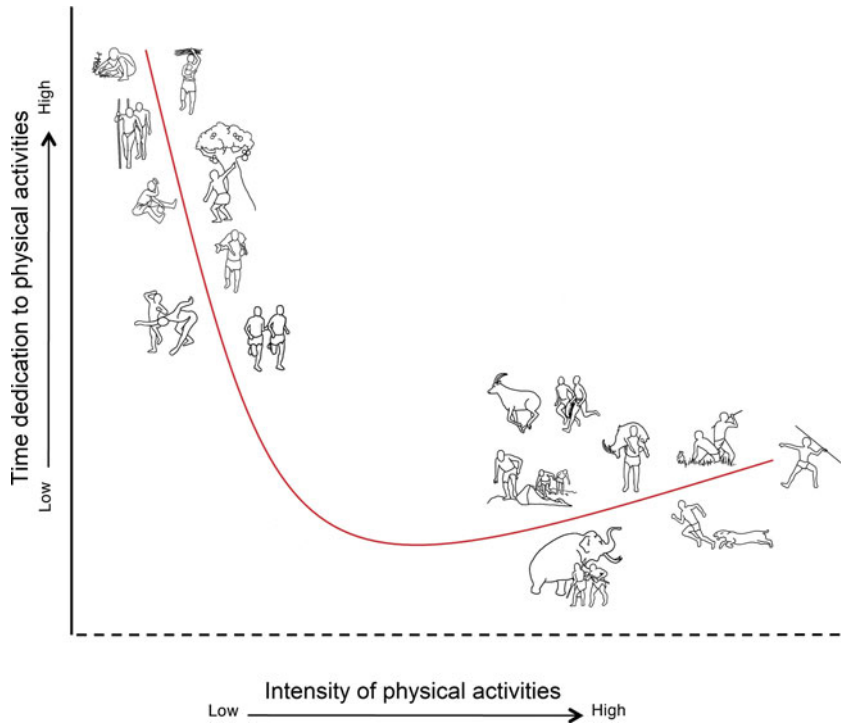

Fig. 1 Hypothetical distribution of *Homo sapiens*' physical activities during the Paleolithic Era

considering the specific demands of their competitive events in accordance with the *principle of allocation*. For instance, it is well known that various physiological adaptations related to higher aerobic capacity would also enhance repeated sprint ability [41].

The concept of *dose response* establishes the necessity to elaborate on what factors determine the better training load for a better performance. In this respect, Kiely [42] has recently pointed out the absence of strong evidence supporting the validity of widely used *periodization models* to plan and organize athletic training. We suggest that training programmes should take into consideration our ancestors' activity pattern, in which they probably self-regulated their daily physical activity, depending on their caloric requirements [19]. It may be expected that our predecessors naturally decided to rest or perform light activities after hard days to be better prepared for the next hard day(s) [5, 19]. This approach is in agreement with recent studies that have described a better training outcome in subjects who regulated their training load, depending on the state of their autonomic nervous system [43, 44]. Furthermore, previous exercise training studies have reported that low-stress participants experienced a significantly greater increase in performance [45, 46]. Therefore, preservation of homeostasis in the face of different sources of stress plays a pivotal role in chronic training adaptations. This could also explain why *periodization models* often fail to be effective, as they are not adapted to individual responses to training, which mainly depend on homeostatic control for subsequent adaptations.

Elite athletes may represent an artificial selection, with endurance athletes having a more adapted genotype for survival that is suitable for health and longevity [47–49]. However, the hypothesis of a lower risk of disease has not been confirmed [50]. On the other hand, O'Keefe et al. [5] have pointed out that the current training loads undertaken by athletes are far beyond those required by our ancestors for survival. While there is no doubt that greater cardio-respiratory fitness is related to better health and maybe longevity [51], it appears there is an optimum level of physical activity that might not be very different from that performed by our ancestors. In contrast, modern elite runners train for more than ~20 km a day, with daily energy expenditures of ~6,000–8,000 kcal. Furthermore, the limits of human endurance are far beyond these exercise levels, with extreme caloric expenditures estimated at ~1 million kcal over a 159-day Antarctic expedition [52]. Therefore, the physical activity levels of hunter–gatherers were presumably far below those currently performed in elite sport. However, this is not necessarily a contradiction, as Olympic athletes could be considered very specialized '*Homos*', who waste minimal resources in other stressful activities different from that of their training and competition.

The training volumes performed by elite endurance athletes could well be related to the intensity distribution of their training loads. Observational studies on various endurance sports [53–57] have systematically reported that *polarized intensity* is the most frequent training intensity distribution and the optimal way to attain sporting

excellence in world-class elite athletes, as well as performance improvements in well-trained athletes [58, 59]. This model implies that ~80 % of the training sessions are dedicated to exercise below the lactate threshold, with the remaining ~20 % targeting high-intensity training (HIT). In this sense, various studies [56, 60] with endurance runners have demonstrated that the volume of training performed below or very close to the first ventilatory threshold is directly related to sport performance. This is apparently a paradoxical finding, given that the vast majority of competitive endurance events are performed at intensities between the lactate threshold and maximal oxygen uptake (VO_{2max}). However, these observations fit perfectly well with our hypothesis, with an intensity distribution similar to that undertaken by our *Homo* ancestors (see Fig. 1). Interestingly, endurance athletes frequently avoid training too often at the lactate threshold, with HIT often performed at maximal [61], and supramaximal intensities [62]. Within this picture, we speculate that the intensities that correspond with the severe domain of exercise may be poorly tolerated by athletes in comparison with other training intensities. This could be a consequence of the induced *homeostatic crisis* at these exercise intensity levels, as inferred from previously reported metabolic, autonomic, neuromuscular and psychological disturbances [63–66]. This outcome would be interpreted in terms of a genetic limitation for these metabolic demands [18], which agrees with the previously suggested lesser importance of the glycolytic pathway for survival of our ancestors [19, 24]. In this respect, the trainability of lactate dehydrogenase activity is lower than that of citrate synthase activity [67].

The impact of daily low-intensity activities has also been confirmed by Hautala et al. [68], who observed that the amount of daily light physical activity correlated with gains in VO_{2max} in a group of physically active men who performed a specific endurance training programme. This outcome is in agreement with a previous study by Ross and McGuire [69], who found a significant relationship between incidental physical activity and cardiorespiratory fitness. It is interesting to note that a polarized intensity pattern of physical activity fits perfectly well with observational data on the spontaneous physical activity of children [70, 71] and also team sport match activities [72, 73], with low-intensity activities being predominant but frequently interspersed with brief bursts of explosive actions. Collectively, these observations reinforce the necessity to take into account incidental physical activity, programmed exercise and competitive efforts when comparing the outcomes of different training regimes in athletes.

From a molecular point of view, the greater effectiveness of both light and very intense exercise on aerobic phenotypic adaptations [61] could be linked to activation of intracellular signalling cascades, which are well-characterized upstream modulators of peroxisome proliferator-activated receptor-γ coactivator-1α (PGC-1α) expression in skeletal muscle. In this respect, HIT can induce skeletal muscle metabolic and functional performance adaptations comparable to traditional low-intensity training [74, 75], confirming the key role of PGC-1α in aerobic phenotypic adaptations after both low- and high-intensity training stimuli [61].

3.1 Concurrent Activity Patterns

The ancestral physical activity pattern included a mixture of different activities, which is in contrast to the high specialization level required for elite sport. This issue has been pointed out by Cordain and Friel [19] who suggested that our ancestors were the historical equivalent of cross-training athletes. In this respect, we have suggested [18] a possible explanation, based on an interference phenomenon during concurrent strength and endurance training [76]. This model suggests that there is an adaptive conflict during concurrent training performed at intensities in the severe and heavy domains for aerobic exercise, and heavy submaximal loads for resistance exercise, thereby exhibiting a greater level of interference and a weaker dose–response relationship. Conversely, aerobic exercise below the lactate threshold performed concurrently with strength training at maximum intensities would exhibit a lower level of interference and thus a stronger dose–response relationship. Additionally, this interference phenomenon could be associated with training modes that produce low levels of blood lactate as a consequence of lesser activation of the glycolytic pathway.

Endurance athletes improve their performance significantly when they perform high-intensity strength training based on heavy resistance and plyometric exercises, or a combination of both. Such improvements have also been observed in spite of an important reduction (~30 %) of the sport-specific training volume [77, 78]. It seems that during concurrent training, the intra-session sequence of aerobic exercise + resistance exercise is better for aerobic performance than resistance exercise + aerobic exercise [79]. The same could be concluded when comparing both between-session training sequences, as running performance was better maintained following an endurance–strength sequence training day [80]. It is interesting to note that high-intensity strength training favours greater muscle power mainly via neural adaptations with no or very little hypertrophic response. From an evolutionary point of view, this could be a key phenotypic adaptation, as greater skeletal muscle mass is more energy demanding and therefore less suited for survival. Moreover, an improvement in power performance immediately after different

endurance exercises has been described [81, 82], maybe suggesting that acute muscular potentiation after endurance running may be also an adaptive characteristic.

These observations are reinforced by recent studies on molecular responses to exercise. For instance, the complex protein mammalian target of rapamycin (mTOR) is thought to be another key factor that integrates signals of the energetic status of the cell and environmental stimuli to control cell growth. Although it has been proposed that activation of PGC-1α and mTOR signalling pathways is responsible for the specific adaptive responses that have been shown after aerobic exercise and resistance exercise, various recent studies have demonstrated that there is no clear distinction in exercise-specific signalling pathways [83–85]. Furthermore, Lundberg et al. [86] have recently reported that the skeletal muscle anabolic environment was reinforced under concurrent training conditions, as aerobic exercise 6 hours prior to resistance exercise did not impair signalling of mTOR-related proteins. Moreover, and in contrast with the traditional notion, PGC-1α expression was also increased 3 hours after resistance exercise. These results suggest that the widespread idea that aerobic exercise and resistance exercise responses could be incompatible from a molecular point of view may be too simplistic and could be better understood by considering the order in which they are performed.

3.2 Train Low, Compete High

Chakravarthy and Booth [87] have suggested that the oscillations of muscle glycogen and triglyceride levels with physical activity–rest cycles during feast–famine cycles throughout evolution selected some genes for oscillating enzymatic regulation of fuel storage and efficiency during fuel usage. In this sense, various studies have shown that low levels of pre-exercise glycogen [88], as well as training twice every second day, may be more efficient in enhancing muscle glycogen stores and enzymatic activity and in improving exercise performance than training daily [89, 90]. Moreover, the current evidence suggests that there is enhancement of intracellular signalling pathways (e.g. 5′ adenosine monophosphate-activated protein kinase) and reduced reliance on carbohydrate utilization when exercising with low glycogen stores [91]. However this nutritional strategy has not been effective in improving performance and could compromise the health status of athletes and their training and competitive performances in high-intensity sports [92, 93]. Therefore, while the impact of these adaptations on performance enhancement is yet to be determined—especially when competing under normal or supercompensated glycogen levels—it seems that low pre-exercise glycogen levels favour better training adaptations.

4 Conclusion and Future Perspectives

From the scientific evidence presented here, we suggest that the genetic heritage of our species could strongly influence the capacity of Olympic athletes to adapt to diverse training. This could explain the difficulties in achieving a consensus on the role of different polymorphisms in athletes' performance. The existence of an 'ideal' physical activity pattern inherited from our ancestors does not exclude the need for training individualization, with consideration of both athletes' characteristics and the specific demands of individual athletic events. However it appears that the more similar a training regime is to our ancestors' activity profile, the greater the adaptations and subsequent performance are. Further studies should address this hypothesis, paying special attention to the link between molecular responses and performance outcomes that take place when long-lasting training regimes respect this phylogenetic template. Athletes' dietary intake should be prescribed to support the energy needs and adaptation to training programmes that often mimic the activity pattern of our ancestors. Additionally, studies on epigenetics [94] could also help us to determine the extent to which the mismatch between the early developmental environment and that experienced during growth and mature life accounts for interindividual variability in training-induced adaptations.

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References

1. Koch LG, Britton SL. Aerobic metabolism underlies complexity and capacity. J Physiol. 2008;586(1):83–95.
2. Wood B, Harrison T. The evolutionary context of the first hominins. Nature. 2011;470(7334):347–52.
3. Raichlen DA, Polk JD. Linking brains and brawn: exercise and the evolution of human neurobiology. Proc Biol Sci. 2013;280(1750):20122250.
4. O'Keefe JH, Vogel R, Lavie CJ, et al. Achieving hunter–gatherer fitness in the 21st century: back to the future. Am J Med. 2010;123(12):1082–6.
5. O'Keefe JH, Vogel R, Lavie CJ, et al. Exercise like a hunter–gatherer: a prescription for organic physical fitness. Prog Cardiovasc Dis. 2011;53(6):471–9.
6. Booth FW, Chakravarthy MV, Spangenburg EE. Exercise and gene expression: physiological regulation of the human genome through physical activity. J Physiol. 2002;543(Pt 2):399–411.

7. Booth FW, Chakravarthy MV, Gordon SE, et al. Waging war on physical inactivity: using modern molecular ammunition against an ancient enemy. J Appl Physiol. 2002;93(1):3–30.
8. Eaton SB, Konner M, Shostak M. Stone agers in the fast lane: chronic degenerative diseases in evolutionary perspective. Am J Med. 1988;84(4):739–49.
9. Booth FW, Gordon SE, Carlson CJ, et al. Waging war on modern chronic diseases: primary prevention through exercise biology. J Appl Physiol. 2000;88(2):774–87.
10. Cordain L, Gotshall RW, Eaton SB, et al. Physical activity, energy expenditure and fitness: an evolutionary perspective. Int J Sports Med. 1998;19(5):328–35.
11. Booth FW, Lees SJ. Fundamental questions about genes, inactivity, and chronic diseases. Physiol Genomics. 2007;28(2): 146–57.
12. Eaton SB, Strassman BI, Nesse RM, et al. Evolutionary health promotion. Prev Med. 2002;34(2):109–18.
13. Williams GC, Nesse RM. The dawn of Darwinian medicine. Q Rev Biol. 1991;66(1):1–22.
14. Lieberman DE. What we can learn about running from barefoot running: an evolutionary medical perspective. Exerc Sport Sci Rev. 2012;40(2):63–72.
15. Puthucheary Z, Skipworth JR, Rawal J, et al. The ACE gene and human performance: 12 years on. Sports Med. 2011;41(6): 433–48.
16. Puthucheary Z, Skipworth JR, Rawal J, et al. Genetic influences in sport and physical performance. Sports Med. 2011;41(10): 845–59.
17. Timmons JA, Knudsen S, Rankinen T, et al. Using molecular classification to predict gains in maximal aerobic capacity following endurance exercise training in humans. J Appl Physiol. 2010;108(6):1487–96.
18. Boullosa DA, Nakamura FY, Ruiz JR. Effectiveness of polarized training for rowing performance (letter). Int J Sports Physiol Perform. 2010;5(4):431–2.
19. Cordain L, Friel J. The Paleolithic athlete: the original cross trainer. In: Sands RR, Sands LR, editors. The anthropology of sport and human movement: a biocultural perspective. Lanham: Lexington Books; 2010. p. 267–76.
20. Kuipers RS, Joordens JC, Muskiet FA. A multidisciplinary reconstruction of Palaeolithic nutrition that holds promise for the prevention and treatment of diseases of civilisation. Nutr Res Rev. 2012;25(1):96–129.
21. Konner M, Eaton SB. Paleolithic nutrition: twenty-five years later. Nutr Clin Pract. 2010;25(6):594–602.
22. Stiner MC. Thirty years on the "Broad Spectrum Revolution" and paleolithic demography. Proc Natl Acad Sci USA. 2001; 98(13):6993–6.
23. Cordain L, Miller JB, Eaton SB, et al. Plant-animal subsistence ratios and macronutrient energy estimations in worldwide hunter–gatherer diets. Am J Clin Nutr. 2000;71(3):682–92.
24. Hochachka PW. Fuels and pathways as designed systems for support of muscle work. J Exp Biol. 1985;115:149–64.
25. Cerling TE, Wynn JG, Andanje SA, et al. Woody cover and hominin environments in the past 6 million years. Nature. 2011;476(7358):51–6.
26. Pontzer H, Raichlen DA, Wood BM, et al. Hunter–gatherer energetics and human obesity. PLos One. 2012;7(7):e40503.
27. Hochachka PW, Gunga HC, Kirsch K. Our ancestral physiological phenotype: an adaptation for hypoxia tolerance and for endurance performance? Proc Natl Acad Sci USA. 1998;95(4): 1915–20.
28. Dudley R. Limits to human locomotor performance: phylogenetic origins and comparative perspectives. J Exp Biol. 2001;204(Pt 18):3235–40.
29. Hochachka PW, Beatty CL, Burelle Y, et al. The lactate paradox in human high-altitude physiological performance. News Physiol Sci. 2002;17:122–6.
30. Hochachka PW, Rupert JL, Monge C. Adaptation and conservation of physiological systems in the evolution of human hypoxia tolerance. Comp Biochem Physiol A Mol Integr Physiol. 1999;124(1):1–17.
31. Bramble DM, Lieberman DE. Endurance running and the evolution of Homo. Nature. 2004;432(7015):345–52.
32. Lieberman DE, Raichlen DA, Pontzer H, et al. The human gluteus maximus and its role in running. J Exp Biol. 2006;209(Pt 11):2143–55.
33. Pontzer H, Rolian C, Rightmire GP, et al. Locomotor anatomy and biomechanics of the Dmanisi hominins. J Hum Evol. 2010; 58(6):492–504.
34. Rolian C, Lieberman DE, Hallgrímsson B. The coevolution of human hands and feet. Evolution. 2010;64(6):1558–68.
35. Liebenberg L. Persistence hunting by modern hunter–gatherers. Curr Anthropol. 2006;47(6):1017–25.
36. Lieberman DE, Bramble DM, Raichlen DA, et al. Brains, brawn, and the evolution of human endurance running capabilities. In: Grine FE, Fleagle JG, Leakey RE, editors. The first humans: origin and early evolution of the genus Homo. New York: Springer; 2009. p. 77–98.
37. Kempermann G, Fabel K, Ehninger D, et al. Why and how physical activity promotes experience-induced brain plasticity. Front Neurosci. 2010;4(189):1–9.
38. Hurtado A, Hawkes K, Hill K, et al. Female subsistence strategies among Ache hunter–gatherers of eastern Paraguay. Hum Ecol. 1985;13(1):1–28.
39. Panter-Brick C. Sexual division of labor: energetic and evolutionary scenarios. Am J Hum Biol. 2002;14(5):627–40.
40. Van Damme R, Wilson RS, Vanhooydonck B, et al. Performance constraints in decathletes. Nature. 2002;415(6873):755–6.
41. Bishop D, Girard O, Mendez-Villanueva A. Repeated-sprint ability—part II: recommendations for training. Sports Med. 2011;41(9):741–56.
42. Kiely J. Periodization paradigms in the 21st century: evidence-led or tradition-driven? Int J Sports Physiol Perform. 2012;7(3): 242–50.
43. Kiviniemi AM, Hautala AJ, Kinnunen H, et al. Endurance training guided individually by daily heart rate variability measurements. Eur J Appl Physiol. 2007;101(6):743–51.
44. Kiviniemi AM, Hautala AJ, Kinnunen H, et al. Daily exercise prescription on the basis of HR variability among men and women. Med Sci Sports Exerc. 2010;42(7):1355–63.
45. Ruuska PS, Hautala AJ, Kiviniemi AM, et al. Self-rated mental stress and exercise training response in healthy subjects. Front Physiol. 2012;3:51.
46. Bartholomew JB, Stults-Kolehmainen MA, Elrod CC, et al. Strength gains after resistance training: the effect of stressful, negative life events. J Strength Cond Res. 2008;22(4): 1215–21.
47. Ruiz JR, Morán M, Arenas J, et al. Strenuous endurance exercise improves life expectancy: it's in our genes. Br J Sports Med. 2011;45(3):159–61.
48. Sanchis-Gomar F, Olaso-Gonzalez G, Corella D, et al. Increased average longevity among the "Tour de France" cyclists. Int J Sports Med. 2011;32(8):644–7.
49. Fiuza-Luces C, Ruiz JR, Rodríguez-Romo G, et al. Are 'endurance' alleles 'survival' alleles? Insights from the ACTN3 R577X polymorphism. PLoS One. 2011;6(3):e17558.
50. Gómez-Gallego F, Ruiz JR, Buxens A, et al. Are elite endurance athletes genetically predisposed to lower disease risk? Physiol Genomics. 2010;41(1):82–90.

51. Kokkinos P, Myers J, Kokkinos JP, et al. Exercise capacity and mortality in Black and White men. Circulation. 2008;117(5): 614–22.
52. Noakes TD. The limits of human endurance: what is the greatest endurance performance of all time? Which factors regulate performance at extreme altitude? Adv Exp Med Biol. 2007;618: 255–76.
53. Mujika I, Chatard JC, Busso T, et al. Effects of training on performance in competitive swimming. Can J Appl Physiol. 1995;20(4):395–406.
54. Steinacker JM, Lormes W, Lehmann M, et al. Training of rowers before world championships. Med Sci Sports Exerc. 1998;30(7): 1158–63.
55. Fiskerstrand A, Seiler KS. Training and performance characteristics among Norwegian international rowers 1970–2001. Scand J Med Sci Sports. 2004;14(5):303–10.
56. Esteve-Lanao J, San Juan AF, Earnest CP, et al. How do endurance runners actually train? Relationship with competition performance. Med Sci Sports Exerc. 2005;37(3):496–504.
57. Zapico AG, Calderón FJ, Benito PJ, et al. Evolution of physiological and haematological parameters with training load in elite male road cyclists: a longitudinal study. J Sports Med Phys Fitness. 2007;47(2):191–6.
58. Ingham SA, Fudge BW, Pringle JS. Training distribution, physiological profile, and performance for a male international 1500-m runner. Int J Sports Physiol Perform. 2012;7(2):193–5.
59. Neal CM, Hunter AM, Brennan L, et al. Six weeks of a polarized training-intensity distribution leads to greater physiological and performance adaptations than a threshold model in trained cyclists. J Appl Physiol. 2013;114(4):461–71.
60. Esteve-Lanao J, Foster C, Seiler S, et al. Impact of training intensity distribution on performance in endurance athletes. J Strength Cond Res. 2007;21(3):943–9.
61. Laursen PB. Training for intense exercise performance: high-intensity or high-volume training? Scand J Med Sci Sports. 2010;20(Suppl. 2):1–10.
62. Iaia FM, Bangsbo J. Speed endurance training is a powerful stimulus for physiological adaptations and performance improvements of athletes. Scand J Med Sci Sports. 2010; 20(Suppl. 2):11–23.
63. Škof B, Strojnik V. Neuromuscular fatigue and recovery dynamics following prolonged continuous run at anaerobic threshold. Br J Sports Med. 2006;40(3):219–22.
64. Seiler S, Haugen O, Kuffel E. Autonomic recovery after exercise in trained athletes: intensity and duration effects. Med Sci Sports Exerc. 2007;39(8):1366–73.
65. Faude O, Kindermann W, Meyer T. Lactate threshold concepts: how valid are they? Sports Med. 2009;39(6):469–90.
66. Ekkekakis P, Parfitt G, Petruzzello SJ. The pleasure and displeasure people feel when they exercise at different intensities: decennial update and progress towards a tripartite rationale for exercise intensity prescription. Sports Med. 2011;41(8): 641–71.
67. Issurin VB. Generalized training effects induced by athletic preparation: a review. J Sports Med Phys Fitness. 2009;49(4): 333–45.
68. Hautala A, Martinmaki K, Kiviniemi A, et al. Effects of habitual physical activity on response to endurance training. J Sports Sci. 2012;30(6):563–9.
69. Ross R, McGuire KA. Incidental physical activity is positively associated with cardiorespiratory fitness. Med Sci Sports Exerc. 2011;43(11):2189–94.
70. Bailey RC, Olson J, Pepper SL, et al. The level and tempo of children's physical activities: an observational study. Med Sci Sports Exerc. 1995;27(7):1033–4.
71. Duncan JS, Badland HM, Schofield G. Combining GPS with heart rate monitoring to measure physical activity in children: a feasibility study. J Sci Med Sport. 2009;12(5):583–5.
72. Rampinini E, Coutts AJ, Castagna C, et al. Variation in top level soccer match performance. Int J Sports Med. 2007;28(12): 1018–24.
73. Dogramaci SN, Watsford ML, Murphy AJ. Time-motion analysis of international and national level futsal. J Strength Cond Res. 2011;25(3):646–51.
74. Burgomaster KA, Howarth KR, Phillips SM, et al. Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. J Physiol. 2008;586(1):151–60.
75. Gibala MJ, Little JP, van Essen M, et al. Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. J Physiol. 2006;575(Pt 3):901–11.
76. Docherty D, Sporer B. A proposed model for examining the interference phenomenon between concurrent aerobic and strength training. Sports Med. 2000;30(6):385–94.
77. Paavolainen L, Häkkinen K, Rusko H. Effects of explosive type strength training on physical performance characteristics in cross-country skiers. Eur J Appl Physiol Occup Physiol. 1991;62(4): 251–5.
78. Paavolainen L, Häkkinen K, Hämäläinen I, et al. Explosive-strength training improves 5-km running time by improving running economy and muscle power. J Appl Physiol. 1999;86(5): 1527–33.
79. Chtara M, Chamari K, Chaouachi M, et al. Effects of intra-session concurrent endurance and strength training sequence on aerobic performance and capacity. Br J Sports Med. 2005;39(8): 555–60.
80. Doma K, Deakin GB. The effects of strength training and endurance training order on running-economy and -performance. Appl Physiol Nutr Metab. 2013;38(6):651–6.
81. Vuorimaa T, Virlander R, Kurkilahti P, et al. Acute changes in muscle activation and leg extension performance after different running exercises in elite long distance runners. Eur J Appl Physiol. 2006;96(3):282–91.
82. Boullosa DA, Tuimil JL, Alegre LM, et al. Concurrent fatigue and potentiation in endurance athletes. Int J Sports Physiol Perform. 2011;6(1):82–93.
83. Coffey VG, Zhong Z, Shield A, et al. Early signaling responses to divergent exercise stimuli in skeletal muscle from well-trained humans. FASEB J. 2006;20(1):190–2.
84. Coffey VG, Pilegaard H, Garnham AP, et al. Consecutive bouts of diverse contractile activity alter acute responses in human skeletal muscle. J Appl Physiol. 2009;106(4):1187–97.
85. Wilkinson SB, Phillips SM, Atherton PJ, et al. Differential effects of resistance and endurance exercise in the fed state on signalling molecule phosphorylation and protein synthesis in human muscle. J Physiol. 2008;586(Pt 15):3701–17.
86. Lundberg TR, Fernandez-Gonzalo R, Gustafsson T, et al. Aerobic exercise alters skeletal muscle molecular responses to resistance exercise. Med Sci Sports Exerc. 2012;44(9):1680–8.
87. Chakravarthy MV, Booth FW. Eating, exercise, and "thrifty" genotypes: connecting the dots toward an evolutionary understanding of modern chronic diseases. J Appl Physiol. 2004; 96(1):3–10.
88. Pilegaard H, Saltin B, Neufer PD. Effect of short-term fasting and refeeding on transcriptional regulation of metabolic genes in human skeletal muscle. Diabetes. 2003;52(3):657–62.
89. Hansen AK, Fischer CP, Plomgaard P, et al. Skeletal muscle adaptation: training twice every second day vs. training once daily. J Appl Physiol. 2005;98(1):93–9.

90. Yeo WK, Paton CD, Garnham AP, et al. Skeletal muscle adaptation and performance responses to once a day versus twice every second day endurance training regimens. J Appl Physiol. 2008;105(5):1462–70.
91. Baar K, McGee S. Optimizing training adaptations by manipulating glycogen. Eur J Sport Sci. 2008;8(2):97–106.
92. Hawley JA, Tipton KD, Millard-Stafford ML. Promoting training adaptations through nutritional interventions. J Sports Sci. 2006;24(7):709–21.
93. Burke LM. Fueling strategies to optimize performance: training high or training low? Scand J Med Sci Sports. 2010;20(Suppl. 2):48–58.
94. Ehlert T, Simon P, Moser DA. Epigenetics in sports. Sports Med. 2013;43(2):93–110.