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VIEWPOINT

Commentaries on Viewpoint: Resistance training and exercise tolerance during high-intensity exercise: moving beyond just running economy and muscle strength

RESISTANCE TRAINING AND EXERCISE TOLERANCE DURING HIGH-INTENSITY EXERCISE: CAN WE MOVE FROM THE LABORATORY TO THE TRACK?

TO THE EDITOR: A consistent increase in endurance performance has often been observed after resistance training (RT) (4). Based on the critical power (CP) concept, Denadai and Greco (1) recently proposed an interesting model to explain this RT-induced improvement in endurance performance. According to these authors, the gains (35–60%) in the curvature constant of the power-duration hyperbola (W′) could explain the performance improvements during constant-workload exercises performed above the CP after a RT program. However, it is important to highlight that during most athletic events, the intensity of the exercise is not previously fixed, but self-selected by the athletes. The intensity distribution during middle- and long-distance running races has often been characterized by a U-shaped pacing profile, with start and finish intensities being higher than in the middle part of the race (5). This U-shaped pacing makes the W′ use more complex, because athletes might switch from one exercise intensity domain to another throughout the race (3). This could indicate that the increase in W′ with RT might be more relevant for some specific parts of the race, in which athletes perform at intensities above the CP, such as during the fast start and the final sprint. This suggestion is in agreement with previous findings showing that RT can counteract fatigue during the last part of a running race (2). Therefore, further research in this exciting area is necessary to elucidate the influence of RT on W′ and its possible relationship with changes in specific parts of self-paced, real races.

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COMMENTARY ON VIEWPOINT: HYPOXIA COULD FURTHER ENHANCE PERIPHERAL MUSCLE ADAPTATIONS TO RESISTANCE TRAINING AND BOOST W′

TO THE EDITOR: We appreciate the Viewpoint of Denadai and Greco (1) questioning the physiological mechanisms by which resistance training may increase the amount of work performed in the severe-intensity domain (W′) and, thereby, exercise tolerance. This enhancement of exercise capacity may be related to improvement in buffer capacity and/or reduction in group III/IV afferent sensitivity subsequent to resistance training. In our opinion, the use of hypoxia as an ergogenic tool can further boost these peripheral adaptations to promote greater or faster improvement in W′. We have demonstrated that resistance training in hypoxia (RTH) leads to greater gains in muscle hypertrophy (4) and maximal strength (3, 4) compared with similar training in normoxia, contributing to enhanced exercise tolerance at a given intensity. Evidences show that the low tissue O_2 partial pressure accelerates metabolite accumulation (e.g., blood lactate, growth hormone) and gene transcription (e.g., hypoxia-inducible factors, mammalian target of rapamycin), leading to earlier and greater recruitment of higher-threshold motor units (4). Furthermore, the hypoxia-induced vasodilation and subsequent microvascular O_2 delivery may cause type II muscle fibers to behave more like their oxydatively efficient type I counterparts (2). This would potentially attenuate peripheral locomotor muscle fatigue and augment the sensory tolerance limit. Despite that W′ was recently shown to remain unchanged up to a simulated altitude of ~4,000 m (5), one may speculate that RTH would affect it positively. Future research could elucidate the "optimal" characteristics for successful implementation in athletic and patient populations to further enhance exercise capacity and quality of life.

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TIME TO MOVE BEYOND THE CRITICAL POWER PARADIGM

TO THE EDITOR: We commend Denadai and Greco (3) for proposing the need to investigate alternative mechanisms for the enhancement of endurance performance following a period of resistance training. Denadai and Greco (3) suggest the physiological adaptations to resistance training could increase W′ and thus lead to increased exercise tolerance in the severe-intensity domain. However, this discussion seems premature when the basic tenets of the critical power model and the underpinning mechanisms related to critical power and W′ remain to be firmly established. Moreover, it is important to recognize that the critical power concept is based on the mathematical relationship between power and time, and so it is problematic to associate it with specific physiological parameters. Indeed, Denadai and Greco's own research challenges the basis of W′ as a fixed amount of work that can be performed at intensities above critical power by suggesting that it increases depending on the nature of its expenditure (2). Accordingly, despite the 3-min all-out test having a sound theoretical background (1), it overestimates critical power and performance in competitive cyclists (4), suggesting performance is a complex phenomenon that cannot be entirely predicted by a two-variable model. Furthermore, Salam and coworkers (5) recently showed mental fatigue reduced time to exhaustion proportionally across different exercise durations, indicating a decrease in W′ by purely psychological factors (5). In light of the recent findings (5), and some inconsistencies already recognized (2, 4), we wonder whether there is enough evidence to move beyond the critical power paradigm to articulate new hypotheses.

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COMMENTARY ON VIEWPOINT

TO THE EDITOR: The field of sports science is relatively new and so many points are yet to be elucidated and resolved. The proposal to present my views on the work in question (3) seemed tempting. Every vision and integrative analysis seems to me to be extremely valid in the area of sport, precisely because performance is a function of several components (2). I thought of pursuing an approach more closely linked to pure physics, biomechanics, or biochemistry. However, what struck me most was that a paper used by the authors (1) presents some flaws in the interpretation of the data. Table 2, of the article (1), for example, analyzing effect size, pre and post intervention, results in a very different vision from the authors. Hence my question: to what extent are many of our discussions anchored in misinterpretations? Is it not time to take a step back and do a major review of the main works to verify how much we can rely on the conclusions obtained by that analysis of the data?

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COMMENTARY ON VIEWPOINT

TO THE EDITOR: The work by Denadai and Greco (2) proposes several possible mechanisms for resistance training to improve W′ and exercise tolerance, but several questions remain unanswered. For example, the type of resistance training utilized may determine the magnitude of alterations in W′, as discrepant results exist on the influence of heavy vs. explosive resistance exercise (1, 4). Furthermore, the long-term impact of concurrent training (i.e., strength + aerobic) on W′ is unknown. Although a 2008 review (5) on concurrent training supports the authors' conclusions, the longest intervention included in the review lasted 14 wk. The influence of resistance training on W′ beyond 14 wk and throughout a training and competitive sports season remains unclear. We wonder how a long-term dose of resistance training affects W′ and performance gains, especially in comparison to a long-term dose of traditional endurance training. Additionally, as Berryman et al. (1) used subjects with no history of strength training, we wonder if improvements in W′ occur for individuals who already utilize resistance training or if there are diminishing

returns such that increasing the dose results in lesser performance gains. Finally, the appropriate endurance athlete population must be considered. While the authors correctly include 800–3,000 m athletes in their discussion, we caution the reader not to extend the application to those running longer than ~30 min, as this results in an intensity lower than critical speed and thus theoretically no utilization of D′ (3).

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FAST-TWITCH SPECIFIC MUSCLE FIBER ADAPTATIONS WITH RESISTANCE TRAINING MAY IMPROVE HIGH-INTENSITY AEROBIC EXERCISE TOLERANCE

TO THE EDITOR: A call for consideration of physiological mechanisms through which resistance training improves tolerance to high-intensity exercise was recently put forth (1). We speculate that the addition of resistance training to an endurance training regimen may condition fast-twitch muscle fibers to better tolerate high-intensity aerobic exercise, thereby improving performance. Superior endurance performance is not typically considered dependent on fast-twitch fibers, but intensified endurance training in competitive athletes results in fast-twitch specific morphological and functional deficits (2). Conversely, reduced training volume (taper) after heavy training appears to mediate performance enhancement by what is seemingly a compensatory rebound in fast-twitch size and power (5). Targeted improvements in fast-twitch muscle fiber size (and likely power) with resistance training (see supplementary table in Ref. 3) could help ameliorate fast-twitch specific decrements that can occur with heavy endurance training, thereby improving high-intensity exercise tolerance. Importantly, reducing aerobic training volume to account for supplementary resistance exercise-training might further optimize high-intensity work capacity (4). Enhanced high-intensity exercise tolerance in this scenario could admittedly be attributed to the avoidance of overtraining and general fatigue that can occur with high aerobic training volumes, but fast-twitch specific adaptations from resistance training should not be overlooked. Collectively, our hypothesis that resistance training supports improvements in high-intensity work capacity through fast-twitch specific muscle fiber adaptations is plausible and deserving of more detailed exploration.

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COMMENTARY ON VIEWPOINT

TO THE EDITOR: In their Viewpoint, Denadai and Greco (3) challenge the idea that resistance training improves middle-distance endurance exercise performance consecutive to alterations in running economy. Rather, they suggest that this improvement might be related to an increase in W′, a component of the power-time relationship (3). As a possible mechanism, they propose that a desensitization of group III/IV muscle afferents might occur following resistance training, leading to greater W′ while central fatigue would be reduced, resulting in exercise performance improvement (3). On the one hand, previous observation of a greater peripheral fatigue tolerance while central fatigue was unchanged following endurance training indirectly supports the idea that a desensitization of group III/IV muscle afferents might occur (5). On the other hand, studies using pharmacological attenuation of group III/IV muscle afferent feedback showed not only greater spinal motoneuronal output during cycling exercise (less central fatigue) but also reduced cardiovascular and respiratory response to exercise (less muscle O_2 delivery leading to increased rate of peripheral fatigue), and failed, in fine, to show any improvement in exercise performance (1, 2). Accordingly, a desensitization of group III/IV muscle afferents is likely not the main mechanism responsible for the improved W′ during endurance exercise following resistance training. Alternatively, altered central processing of group III/IV muscle afferents (4) and/or increased muscle buffer capacity (3) are other potential mechanisms to explain an increase in W′. These mechanistic propositions are difficult to explore in humans but definitely require further investigations.

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COMMENTARY ON VIEWPOINT

TO THE EDITOR: The viewpoint by Denadai and Greco (1) raised pertinent questions regarding critical power (CP) and W′ in several populations. A primary characteristic of aging and "at-risk" populations is exercise intolerance. Therefore, important mechanistic information related to CP/W′ within these populations is warranted. Running/walking "economy" may not account for all the improvements in W′ due to resistance training (1); however, other mechanisms may be involved.

One hypothesis is that resistance training, which primarily targets glycolytic type II muscle fibers, stimulates nitric oxide (NO) bioavailability via improvements in nitrite or neuronal nitric oxide synthase (nNOS) flux (2). Indeed, nNOS appears specific to type II fibers (2) and exercise derived NO liberated from nNOS attenuates alpha-adrenergic vasoconstriction (5), thus optimizing perfusion. Furthermore, plasma nitrite is reduced in aged/diseased populations, which may be related to exercise intolerance. Additionally, the appearance of the VO_2 slow component of oxygen uptake kinetics during heavy-to-severe intensity exercise has been attributed to extra-recruitment of type II fibers, corresponding to CP/W′ (4). Therefore, increasing NO bioavailability via resistance training may optimize the oxygen cost of contractions (2), consequently improving exercise tolerance/W′.

Mechanistically, NO is also involved in attenuating the exaggerated exercise pressor response (EPR) in "at-risk" populations, as reductions in NO within the nucleus tractus solitarii of the brain contribute to the exaggerated EPR (3). As Denadai and Greco (1) hypothesize, resistance training-induced improvements of EPR could change the "sensory tolerance limit." This may be attributed to improvements in NO bioavailability within the brain, enhancing exercise tolerance and W′.

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THE ROLE OF CENTRAL COMMAND IN RESISTANCE-TRAINING-INDUCED IMPROVEMENT IN EXERCISE TOLERANCE DURING HIGH-INTENSITY EXERCISE

TO THE EDITOR: Given that critical power (CP) and the total work performed above CP (W′) are derived from performance data, any change in performance determines a change in CP, W′, or both. While Denadai and Greco (1) report data suggesting that resistance training increases W′ but not CP, there are also apparently paradoxical data showing no change in W′ after high-intensity interval training that can be explained by methodological rather than physiological reasons (5). This warrants caution when interpreting the mechanisms underlying performance adaptations by means of the CP model.

Leaving aside the methodological issues associated with the W′ concept, we wish to raise attention to an overlooked mechanism underlying the improvement in exercise tolerance after resistance training. The increase in muscle strength and the decrease in exercise-induced muscle fatigue that occur with this training intervention point to a reduction in the central neural drive needed to sustain a given constant workload (2). Consequently, a resistance-training-induced decrease in the magnitude of central command (the activity of motor and premotor areas of the brain relating to voluntary muscle contraction) probably contributes to enhancing endurance performance. While directly measuring central command during endurance exercise is impractical, measuring respiratory frequency and perceived exertion can help shed some light on the role of central command in exercise tolerance, because both variables are regulated by central command during high-intensity exercise (2–4). From this perspective, the findings reported by Denadai and Greco (1) can be interpreted in the light of the psychobiological model of endurance performance (2).

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COMMENTARY ON VIEWPOINT: RESISTANCE TRAINING AND EXERCISE TOLERANCE DURING HIGH-INTENSITY EXERCISE: MOVING BEYOND JUST RUNNING AND MUSCLE STRENGTH

TO THE EDITOR: Denadai and Greco (1) provide an interesting Viewpoint about the effects of resistance training on the curvature constant of the critical power (W′) and physiological mechanisms underlying exercise tolerance. From a practical standpoint, we agree that the implementation of specific resistance training programs reduce the energy demands of running economy. However, from a more theoretical perspective, the study of running economy should include comprehensive structural and biomechanical (joint kinetics, muscle actions, vertical oscillation, change in velocity, etc.) analyses (2) to discern potential metabolic differences observed among different subject groups exposed to resistance training. An important consideration for the discussion is related to the variability of the prescribed resistance exercises. What are the intensity, duration, and frequency of training that improve W′ in untrained, trained, and clinical populations? What is the role of peripheral factors in limiting exercise tolerance? It is important to consider the behavior of the power-duration relationship in hypoxemic patients with different degrees of impairment and the effective strategies on specific determinant of the endurance capacity. These factors may be variable in patients with different disease entities or severity and ultimately depend on the degree of intrinsic peripheral muscle dysfunction and weakness (3), imbalance between different physiological systems (4), and imbalance between energy demands and supplies to working skeletal and respiratory muscles (5).

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COMMENTARY ON VIEWPOINT

TO THE EDITOR: The Viewpoint by Denadai and Greco (3) provided an insightful explanation of how resistance training may improve high-intensity aerobic exercise tolerance, proposing an attenuated H^+ accumulation-lowered firing rate of group III/IV afferents after a resistance training period. In this regard, a blood flow restriction (BFR) study observed an exacerbated metabolic stress as indicated by a decreased pH level when individuals performed low-intensity resistance exercises associated with BFR (5), therefore suggesting that chronic resistance exercise + BRF training may lead to an attenuated H^+ accumulation. However, it is possible that low-intensity aerobic training associated with BFR may also constitute as an alternative to enhance the high-intensity aerobic exercise tolerance, possibly through similar mechanisms to those hypothesized by Denadai and Greco (3) (i.e., metabolic changes in muscle milieu-improved "sensory tolerance limit"). For example, improvements in muscle strength, $\dot{V}O_{2max}$ and time to exhaustion were observed after eight weeks of a low-intensity aerobic (40% $\dot{V}O_{2max}$) + BFR training (1). Furthermore, 2 weeks of intermittent walk + BFR training led to greater $\dot{V}O_{2max}$ and exercise tolerance (4). Together, the adaptations observed in these studies (1, 4) may suggest that increased high-intensity aerobic exercise tolerance is also attainable in low-intensity aerobic exercise + BFR training, perhaps due to a BFR training-attenuated H^+ accumulation (2). The possibility of improving high-intensity exercise tolerance through a low-intensity aerobic exercise training + BFR is of interest for clinical population with restriction to resistance training as well as for endurance athletes wanting to maximize training adaptations. Future studies are required to confirm this hypothesis.

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MIDDLE DISTANCE RUNNING EFFICIENCY—QUANTIFYING INTERVENTIONS AT WORKLOADS ABOVE CRITICAL VELOCITY—MORE THAN JUST A PHYSIOLOGICAL PURSUIT?

TO THE EDITOR: The demands of world class 800 m running competition require exceptional tolerance of high-intensity running speeds far beyond critical velocity (CV) (11.0 s/100 m sectors; 5). In agreement with field observations, modifications in running economy measured during submaximal exercise may not translate to improved running performance over middle-distance race speeds (2). Therefore, quantification of running efficiency at speeds above CV may allow better assessment of adaptive responses to resistance training interventions.

The impact that a resistance training intervention imparts on 800 m "race pace" running may be difficult to define and quantify. Underlying improvements in high-intensity (>CV) running efficiency following a resistance training intervention may stem from biomechanical and anthropometric adaptations that include inter and intramuscular coordination and postural control as well as changes in passive stiffness of relevant tissue (and, as a result, leg spring stiffness) that ultimately reduce energy leakage during running (1, 4) to optimize the utilization of the available D′. In addition, resistance training can enhance ground reaction force application in both vertical and horizontal planes, both of which provide cocoordinative challenges that limit the ability to perform faster running speeds and may extend an athletes tolerance to high-intensity work. A multidisciplinary perspective that incorporates the sciences of biomechanics and motor control, alongside physiology, may allow us to view middle-distance running more as a skill, whereby an applied resistance training intervention can foster the development of the underlying components of importance for efficient high-intensity running and D′ of middle-distance runners (3).

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CARDIOVASCULAR CHANGES RELATED TO RESISTANCE TRAINING ARE GREATER THAN TYPES III/IV AFFERENTS FOR EXERCISE TOLERANCE AND W′

TO THE EDITOR: The recent Viewpoint (1) postulates that resistance training can enhance exercise tolerance and W′ by reducing firing frequency of types III/IV afferents. However, both skeletal muscle vascularization and blood flow capacity can also play crucial roles in oxygen transport to muscle fibers, maximal oxidative metabolic rate, and clearance of fatigue-related metabolites, which have been shown critical to W′ (2, 3). Importantly, hemodynamic changes such as increase in blood flow, total capillary/arteriolar proliferation, and cross sectional area can occur in both trained and untrained skeletal muscle (2–4) and are greater in shorter concentric/eccentric (e.g., workout training) compared with longer (e.g., isometric/muscle stretching) muscle contractions (3–5). Overall, these components have been shown to exert more influence over critical power than firing frequency of types III/IV afferents or other components of fatigue, even in COPD and CHF patients (3, 4). Therefore, resistance training might be capable to improve muscle functions more likely by a broad spectrum of cardiovascular adaptations [e.g., neuronal nitric oxide synthase (nNOS), arteriogenesis, angiogenesis, microvascular O_2 transport and muscle blood flow] than the regulation over types III/IV afferents activity (2–4). Accordingly, cardiovascular adaptations need to be taken into account beyond firing frequency muscle types III/IV afferents on improvements of the exercise tolerance and W′, even in clinical populations.

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COMMENTARY ON VIEWPOINT

TO THE EDITOR: Although guidelines recommend exercise training as an adjunctive therapy in patients with heart failure (HF). The recently published Viewpoint (1) elegantly pointed out underrated mechanisms involved in the resistance training effects in the improvement of aerobic capacity and on exercise tolerance at high intensities in these patients. Indeed, HF patients display decreased muscular strength, which may explain the reduced aerobic power. Therefore, it has been suggested that physical disabilities and HF-associated comorbidities might be reduced by neuromuscular electrical stimulation, as well mentioned in the Viewpoint (1). Thus, whether neuromuscular electrical stimulation promotes enhanced exercise tolerance, probably occurs due to neural adaptations (2), thereby further supporting the benefits of resistance training on muscle strength and improvement in the W'. However, great attention should be paid to the prescription of high-intensity resistance training for this population, since the prediction of the maximal exercise intensity might be deeply influenced by the HF-associated comorbidities (3). Another contributing factor to the exercise intolerance in HF patients is the severe endothelial dysfunction. Recently, our group demonstrated a high positive correlation ($r = 0.91$) between resistance exercise intensity and endothelial-dependent vasodilation (4), which also could help in increase of exercise tolerance in these patients. Along with the underlying effects of resistance training on the removal of metabolites and muscle buffer capacity, it is important to highlight that cardiac adaptation occur after resistance training, with improvement of cardiac output and in health-related quality of life in these patients (5).

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FACTORS AFFECTING THE CURVATURE CONSTANT OF POWER-TIME HYPERBOLA

TO THE EDITOR: Critical power (CP) and the curvature constant (W′) characterize the power-duration hyperbola well known in exercise physiology. The invariance of these two parameters for a given class of subjects has been in general acceptance, but recent reports (1, 2) show basis for its possible revision. First of all, the power hyperbola, which can be linearized in the work-time plane or in the log-power log-time domain arises from having to yield to a dynamic equilibrium prevailing in the physiological system. The authors also believe that it has deeper connections with the excitability of muscle/nerve tissues on account of its resemblance with strength-duration relationship wherein the parameters of chronaxie and rheobase serve well for comparison.

There is paucity of literature revealing what factors influence W′. Attempts were made to manipulate W′ and CP prompted by the belief that concurrent training and other variations in exercises would spur the anaerobic and aerobic mechanisms and siphon additional energy reserves (3). Jones et al. (4) established that phosphorylcreatine and pH balance was intact below CP but was offset above it during high intensity exercises. If CP is thus unaltered, the only factor that possibly enhances W′, has to do with time. Recently (1) reported marked enhancement in W′ following induced alkalosis among subjects under both hypoxic and normoxic conditions. They also found a fall in CP in participants administered with simulated hypoxia. So pretreatment with phosphocreatine or some alkaline drink (5) is supposed to extend the time margin before reaching the task failure, thus increasing W′.

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