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Specificity of training adaptation: time for a rethink?

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The key components of any training programme are the volume (how much), intensity (how hard) and frequency (how often) of exercise sessions. These ‘training impulses’ determine the magnitude of adaptive responses that either enhance (fitness) or decrease (fatigue) exercise capacity (Hawley, 2002). A long held view is that the training response/adaptation is directly related to the volume of exercise undertaken (Fitts et al. 1975). However, there is obviously a threshold volume/duration beyond which additional stimuli do not induce further increases in functional capacity. This ‘biological ceiling’ is important because it implies that the regulatory control mechanisms signalling adaptive responses are ultimately titrated by exercise duration (Booth & Watson, 1985). Competitive athletes are all too aware of this phenomenon: many elite performers walk a tightrope between chronic intensive training and inadequate recovery that can culminate in decrements in performance and the ‘overtraining syndrome.’ Biological scientists are also mindful that training volume and adaptation can be dissociated. Over 35 years ago Dudley et al. (1982) demonstrated that rats undertaking intense work bouts for shorter time induced similar increases in the maximal activities of several oxidative enzymes (i.e. cytochrome c) to those observed after more prolonged submaximal exercise training.

One of the key tenants of exercise physiology is the principle of training specificity, which holds that training responses/adaptations are tightly coupled to the mode, frequency and duration of exercise performed (Hawley, 2002). This means that the vast majority of training-induced adaptations occur only in those muscle fibres that have been recruited during the exercise regimen, with little or no adaptive changes occurring in untrained musculature. Furthermore, the principle of specificity predicts that the closer the training routine is to the requirements of the desired outcome (i.e. a specific exercise task or performance criteria), the better will be the outcome. In this issue of The Journal of Physiology, the results of study by Burgomaster et al. (2007) force us to rethink some of our long held beliefs regarding the concept of training specificity and response/adaptation, as well as providing a reminder that for certain individuals, very intense training can be a time-effective and potent stimulus for inducing many of the benefits normally associated with more prolonged, submaximal endurance-type workouts.

In their recent investigation Burgomaster et al. (2007) report that 6 weeks of low-volume, high-intensity sprint training induced similar changes in selected whole-body and skeletal muscle adaptations as traditional high-volume, low-intensity endurance workouts undertaken for the same intervention period. Specifically, they show that four to six 30 s sprints separated by 4–5 min of passive recovery undertaken 3 days per week results in comparable increases in markers of skeletal muscle carbohydrate metabolism (i.e. total protein content of pyruvate dehydrogenase), lipid oxidation (i.e. maximal activity of β-3-hydroxyacyl CoA dehydrogenase) and mitochondrial biogenesis (i.e. maximal activity of citrate synthase and total protein content of the peroxisome-proliferator-activated receptor-γ coactivator-1α) as when subjects undertook 40–60 min of continuous submaximal cycling a day for 5 days per week. These findings are particularly impressive given that weekly training volume was ~90% lower in the sprint-trained group (~225 versus 2250 kJ week−1) resulting in a total cumulative endurance time of ~1.5 versus 4.5 h per week. While the present study design did not incorporate a functional outcome measure of exercise capacity or performance, this same group (Gibala et al. 2006) using identical training protocols but a shorter intervention period (14 days), have previously reported no differences in the time to complete two discrete exercise performance tasks: one a short-term, high-intensity test lasting ~2 min and the other a longer trial of ~55–60 min duration. Taken collectively, the results from these studies are exciting, particularly as ‘lack of time’ is a common barrier to exercise participation and adherence regardless of sex, age or health status.

As with all studies, one should use caution when extrapolating the results beyond the specific conditions of the investigation. With regard to the time course of training-induced responses, it may be that high-intensity sprint training stimulates a more rapid up-regulation of selected physiological/metabolic markers than traditional low-intensity endurance training, but that over a longer period, the two training regimens elicit similar adaptations. Time course studies would resolve this question. Whether or not patients with risk factors for metabolic disease respond as positively to sprint training as young, healthy individuals also needs to be established. This is particularly relevant as continuous aerobic exercise has traditionally been recommended for fat loss because the proportion of lipid-based fuels oxidized during low-intensity exercise is greater than during high-intensity exercise. As obesity is strongly associated with a cluster of chronic metabolic disorders (Hawley, 2004), any reduction in lipid oxidation or total daily energy expenditure would not be a favourable outcome for these individuals. Notwithstanding these concerns, the novel findings of Burgomaster et al. (2007) provide a platform for exercise physiologists, exercise biochemists and molecular biologists to undertake a systematic and comprehensive evaluation of the specific adaptations induced by different training strategies in both healthy and diseased populations. As previously noted (Hawley, 2004) a determination of the underlying biological mechanisms that result from a wide variety of divergent exercise training protocols in association with appropriate functional outcome measures of exercise capacity is crucial in order to define the precise variations in physical activity that produce the most desired effects on targeted risk factors for disease and to aid in the development and subsequent implementation of such interventions.

References

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