

Hormonal and Metabolic Responses to High Intensity Interval Training

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In the past years there has been a lively discussion about High Intensity Training (HIT) and High Volume Training (HVT), their similarities and differences in adaptations. It was shown, that HIT is not only a useful tool for athletes, but also in terms of health prevention and rehabilitation [1,2]. Most studies focused on changes of endurance performance, muscle adaptations or health benefits, giving us a better idea of the molecular mechanisms of these adaptations [3]. Thereby, mainly two HIT protocols became common and were used in several published studies. First, a Wingate-based “all-out” protocol (also called Sprint Interval Training (SIT)) consisting of 4-6 × 30 sec intervals, mainly used by Gibala et al. [4], second, the 4x4 min protocol performed at an intensity of 90-95% of maximal heart rate, mainly used by Helgerud et al. [5].

The question is which role metabolic and hormonal changes play in this context. Studies using resistance exercise showed significant acute hormonal responses, which appear to be critical to tissue growth and remodeling (even more than chronic changes in resting hormonal concentrations) [6]. If we have a look at the power spectrum, some HIT protocols, especially the Wingate-based training (4 × 30 sec all-out), seem to be close to resistance training interventions. So we can speculate, that on the one hand, HIT induces relatively high mechanical stimuli, similarly known from strength training protocols; on the other hand previous studies showed that high metabolic stimuli (e.g. high lactate levels, large decreases in pH) are induced [7], which are known to be critical for “endurance adaptations” and for the acute hormonal responses as well. High intensity exercise studies showed that changes in the acid base status partly cause the cortisol response to Wingate-based training [7]. Two other studies also provided evidence for a physiological role of pH in the hGH responses to exercise [7,8]. Gordon et al. [8] demonstrated that with a 90 s maximal exercise bout, the post-exercise HGH responses were reduced by an orally induced alkalosis. Also Wahl et al. [7] demonstrated, that the consequence of an increase in circulating HGH levels is in part a decreased blood pH and that the HGH response is far higher compared to HVT. Similar results were shown for free testosterone [9]. A high intensity interval session caused greater increases in free testosterone than steady state endurance exercise. Also the response of other hormones and growth factors seems to be dependent on exercise intensity. Summing up the results of studies investigating Vascular Endothelial Growth Factor (VEGF), it seems that the VEGF response is dependent on the exercise intensity. High-volume low-intensity exercise causes no changes or a decrease in circulating VEGF levels [10-12]. On the other hand, higher intensities cause an increase in VEGF [12-14].

Besides the mechanical stimuli, metabolic perturbations seem to play a critical role in inducing acute hormonal responses and long term adaptations to HIT. Therefore, one aim could be to induce high metabolic stimuli and to prolong the time of exposure to metabolic perturbations during training. One way could be, to perform passive recovery rather than active recovery between HIT intervals. Normally, rest periods during interval training are carried out actively, performing low-intensity exercise to decrease lactate levels and to normalize pH faster compared to passive rest. But the faster elimination of lactate and the faster normalization of pH decreases the time of exposure to these potent metabolic stimuli, possibly leading to different stress/hormonal responses to the same training load. It can be speculated that also long-

term adaptations could vary depending on the arrangement of interval training and training intensity. This was shown by a recent study performing HIT over two weeks either with active or passive recovery. This study showed that passive recovery is superior to active recovery during a high intensity shock micro cycle in increasing performance [15].

We can speculate that the acute metabolic perturbations and hormonal increases after HIT might play a positive role in optimizing training adaptation and in eliciting health benefits [16]. Based on the data it appears that HIT (especially Wingate-based training) promotes anabolic processes. This does not necessarily mean muscle hypertrophy, but might mediate increased expression of aerobic enzymes or adaptations in other processes, such as erythropoietin or angiogenesis. These described hormonal reactions might contribute to the positive effects of HIT shown by previous studies using such exercise protocols. However, still open questions remain concerning the influences of different intensities, the duration of intervals and recovery and the work to rest ration on the acute hormonal response and especially on the long term adaptations.

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