7.1 Abstract

High-intensity interval training (HIT), involving short to long (~5-300 s) intensive work intervals interspersed by active or passive recovery periods, is frequently used in training programs of competitive athletes from various disciplines in order to improve (sport-specific) endurance performance. However, as a result of high metabolic and neuromuscular demands, HIT is also accompanied with acute feelings of fatigue. Ensuring that fatigue in HIT is adjusted appropriately is essential for both adaptations to training as well as competition performance. If the balance between appropriate training load and adequate recovery is disrupted, an abnormal training response may occur and a state of overtraining may develop. This applies especially for high-level athletes due to a process of intensifying training and competition loads in many disciplines in recent years. However, research on the acute responses and exercise-induced fatigue of different HIT-protocols that are commonly used in practice as well as on the diagnostic effectiveness of different markers for routine assessment of fatigue and recovery and on the effects of recovery interventions in connection with HIT is still lacking. Therefore, the purpose of the current doctoral thesis was to evaluate evidence-based guidelines for an appropriate managing of training load and recovery in HIT.

The methodology was based on three studies that were build on one another and were carried out in a chronological order. Each investigation was made up of an independent research approach, whereby the study design of part two and three were prepared each by using the findings of the previous sub-studies.

The aim of the first cross-sectional study was to evaluate the acute responses and exercise-induced fatigue of five different HIT-protocols adjusted by the maximum velocity obtained in the 30-15 Intermittent Fitness Test (VIFT). For this purpose, 16 well-trained intermittent sport players (mean ± SD; age, 24.6 ± 2.7 years; VO2max, 58.3 ± 5.9 ml·min·kg⁻¹) participated in five different running-based HIT-programs separated by six days in between (P240: 4 × 4 min at 80% VIFT; P120: 7 × 2 min at 85% VIFT; P30: 2 × 10 × 30 s at 90% VIFT; P15: 3 × 9 × 15 s at 95% VIFT; P5: 4 × 6 × 5 s sprints). Blood lactate (La) concentration, blood pH, serum concentration of creatinkinase (CK), heart rate (HR), session rating of perceived exertion (session-RPE), delayed onset muscle soreness (DOMS) and countermovement jump (CMJ) height were measured.

A significant main effect for protocol (p < 0.05) was found for the acute responses of HR, session-RPE and La concentration with values increasing in longer intervals from P15 to P120 and P240 while blood pH responded inversely. In contrast, P5 produced the highest La concentration and blood pH decreases. 24 h post exercise serum creatinkinase (CK), delayed onset muscle soreness (DOMS) and the decrease in countermovement jump (CMJ) height were significantly higher.
(p<0.05) after P_2 compared to all other protocols. Due to the highest work/rest ratio together with the high metabolic stress in P_240 and P_120, these protocols led presumably to the strongest depletion of skeletal muscle glycogen stores.

The second longitudinal study aimed to investigate the diagnostic effectiveness of different markers for routine assessment of fatigue and recovery in response to HIT. For this purpose, 22 well-trained male and female team sport athletes (mean ± SD; age, 23.0 ± 2.7 years; VO_2max, 57.6 ± 8.6 mL·min·kg^{-1}) participated in a six-day HIT-microcycle with a total of eleven running-based HIT-sessions that were adjusted by the V_IFT and designed to induce a temporary functional overload. Repeated sprint ability (RSA; criterion measure of fatigue and recovery), CMJ height, jump efficiency in a multiple rebound jump test (MRJ), 20-m sprint performance, muscle contractile properties, serum concentrations of CK, c-reactive protein (CRP) and urea as well as DOMS were measured pre and post the training program as well as after 72 h of recovery.

Following the microcycle significant changes (p<0.05) in RSA as well as in CMJ and MRJ performance could be observed, showing a decline (%Δ ± 90% confidence limits, ES = effect size; RSA: -3.8 ± 1.0, ES = -1.51; CMJ: 8.4 ± 2.9, ES = -1.35; MRJ: 17.4 ± 4.5, ES = -1.60) and a return to baseline level (RSA: 2.8 ± 2.6, ES = 0.53; CMJ: 4.1 ± 2.9, ES = 0.68; MRJ: 6.5 ± 4.5, ES = 0.63) after 72 h of recovery. Athletes also demonstrated significant changes (p<0.05) in muscle contractile properties, CK, and DOMS following the training program and after the recovery period. Further analysis revealed that the accuracy of markers for assessment of fatigue and recovery in comparison to RSA derived from a contingency table was insufficient. Multiple regression analysis also showed no correlations between changes in RSA and any of the markers.

The aim the third cross-over study was to examine the effect of a repeated us of active recovery (ACT) on HIT-induced markers of fatigue. For this purpose, eight elite mal junior tennis players (mean ± SD; age, 15.1 ± 1.4 years) with an international ranking between 59 and 907 (International Tennis Federation) participated in two four-day HIT-microcycles (each with a total of seven running-based HIT-sessions), which were interrupted by a four-month was-out period. After each training session, the players completed 15 min of either moderate jogging (ACT) or passive recovery (PAS). Running intensity both for HIT-sessions and for ACT was adjusted by the V_IFT. CMJ height, serum concentration of CK, DOMS, and perceived recovery and stress (Short Recovery and Stress Scale) were measured 24 h before and 24 h after each training program.

The HIT shock microcycle induced a large decrease in CMJ performance (ACT: -3.8 ± 2.9 cm, ES = -1.39, p < 0.05; PAS: -3.1 ± 2.4 cm, ES = -1.42, p < 0.05) and perceived recovery (ACT: ES = -1.79, p < 0.05; PAS: ES = -2.39, p < 0.05), as well as a moderate to large increase in CK levels (ACT: ES = 0.76, p > 0.05; PAS: ES = 0.81, p > 0.05), DOMS (ACT: ES = 2.02, p < 0.05; PAS:
ES = 2.17, \( p < 0.05 \), and perceived stress (ACT: ES = 1.98, \( p < 0.05 \); PAS: ES = 3.06, \( p < 0.05 \)), compared to the values before the intervention. However, no significant recovery intervention × time interactions or meaningful differences in changes were noted in any of the markers between ACT and PAS.

HIT protocols of different interval duration and intensity result in varying acute physiological and perceptual demands. Longer intervals lead to higher acute cardio-circulatory and metabolic responses, whereas sprint protocols induce the highest state of fatigue. Mean changes in measures of neuromuscular function (i.e., CMJ and MRJ performance as well as muscle contractile properties), CK and DOMS are related to HIT induced fatigue and subsequent recovery. However, low accuracy of a single or combined use of these markers requires the verification of their applicability on an individual basis. A repeated use of ACT during a HIT shock microcycle did not affect exercise-induced fatigue. Thus, athletes and their coaches are advised to focus on other recovery modalities to minimize the severity of fatigue after HIT. However, since ACT was not detrimental to the recovery process, individual preferences as well as experiences and beliefs concerning ACT may influence the choice of whether ACT is performed as a recovery method.