ORIGINAL ARTICLE

Acute metabolic, hormonal and psychological responses to cycling with superimposed electromyostimulation

Patrick Wahl · Markus Hein · Silvia Achtzehn · Wilhelm Bloch · Joachim Mester

Abstract

Purpose The purpose of the present study was to evaluate the effects of superimposed electromyostimulation (E) during cycling on the acute hormonal and metabolic response, as E might be a useful tool to intensify endurance training without performing high external workloads.

Methods Thirteen subjects participated in three experimental trials each lasting 60 min in a randomized order. (1) Cycling (C), (2) cycling with superimposed E ($C + E$) and (3) E. Human growth hormone (hGH), testosterone and cortisol were determined before (pre) and 0′, 30′, 60′, 240′ and 24 h after each intervention. Metabolic stimuli and perturbations were characterized by lactate and blood gas analysis (pH, base excess, bicarbonate, partial pressure of oxygen, partial pressure of carbon dioxide). Furthermore, changes of the person's perceived physical state were determined.

Results $C + E$ caused the highest increases in cortisol and hGH, followed by C and E. Testosterone levels showed no significant differences between $C + E$ and C. Metabolic stress was highest during $C + E$, followed by C and E. $C + E$ was also the most demanding intervention from an athlete's point of view.

Conclusion As cortisol and hGH are known to react in an intensity dependent manner, the present study showed that superimposed E is a useful method to intensify endurance training, even when performing low to moderate external workloads. Even at lower exercise intensities, additional E may allow one to induce a high (local) stimulus. It can be speculated, that these acute hormonal increases and metabolic perturbations, might play a positive role in optimizing long-term training adaptations, similar to those of intense training protocols.

Keywords Cortisol · HGH · Testosterone · Endurance training · Perceived physical state

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Abbreviations

AMP	Adenosine monophosphate
ANOVA	Analysis of variance
ATP	Adenosine triphosphate
BE	Base excess
C	Cycling
$C + E$	Cycling with superimposed electromyostimulation
E	Electromyostimulation
Hb	Haemoglobin
HCO_3^-	Bicarbonate
Hct	Hematocrit
hGH	Human growth hormone
HIT	High-intensity training
PCO_2	Partial pressure of carbon dioxide
PEPS	Person's perceived physical state
pO_2	Partial pressure of oxygen
PPO	Peak power output

PV   Plasma volume
RPE   Rating of perceived exertion
T/C ratio Testosterone/cortisol ratio
VC   Voluntary contraction
VO_2max Maximal oxygen uptake

Introduction

The intensification of endurance training by short intense intervals [high-intensity training (HIT)] has been shown to be a promising and time-saving strategy to improve endurance performance in various groups of subjects (Wahl et al. 2010a, b). It has been suggested, that the major stimulus of HIT is metabolic stress in terms of an altered energy status in muscle associated with reductions in adenosine triphosphate concentrations and relatively large concomitant rise in adenosine monophosphate, high lactate levels and large decreases in pH (Laursen 2010). Furthermore, it was shown that HIT induces high acute hormonal responses, which are known to be critical for adaptational processes (Wahl et al. 2013).

Electromyostimulation (*E*) is an alternative training method mainly performed in strength training and is used for its intensification. Only a few studies used isolated *E* in matters of improvements of endurance performance (Kim et al. 1995). Paillard (2008) described that isolated *E* is highly demanding on muscle metabolism and can enhance energy consumption and carbohydrate oxidation more than voluntary contraction (VC) can. According to the preferential recruitment of larger fibers, *E* strongly activates anaerobic glycolysis for energy production with lactate formation and acidifies more cytoplasm than VC leading to early fatigue (Bergstrom and Hultman 1988; Hamada et al. 2004; Spriet et al. 1987). In previous studies, we were also able to show greater metabolic changes [lactate, respiratory exchange ratio, base excess (BE), bicarbonate (HCO_3^-), partial pressure of carbon dioxide (PCO_2)] during cycling with superimposed *E* compared with normal cycling (Wahl et al. 2012). Due to this increased metabolic stress, the additional application of *E* during endurance exercise might lead to greater adaptations and greater improvements of performance when compared with traditional endurance training. Furthermore, acute hormonal responses appear to be critical for adaptations (Goto et al. 2005; Kraemer and Ratamess 2005). Training-induced increases of the anabolic hormones testosterone and human growth hormone (hGH) might be beneficial for endurance performance, as they stimulate erythropoiesis and enhance lactate transport from the muscle cell (Enoki et al. 2006; Shahidi 2001). However, up to now no study looked for hormonal changes after superimposed *E* during endurance training. Therefore the aim of the present study was to compare the hormonal responses (cortisol, testosterone, hGH), metabolic disturbances (lactate, blood gas) and the athletes' perceived physical state to cycling (*C*), to cycling with superimposed *E* (*C + E*) and to *E* (*E*).

Materials and methods

Subjects

Thirteen healthy, nonsmoking sport students (mean ± SD, age 24.8 ± 3.7 years, weight 79.4 ± 7.5 kg, height 186.3 ± 4.1 cm, relative VO_2max 51.6 ± 5.4 ml min^{-1} kg^{-1}) volunteered and gave written informed consent to participate in this study, which was approved by the University's ethics review board.

Exercise study protocol

Before participation, subjects performed a step test to determine VO_2max (Zan 600, Zan Messgeräte, Oberthulba, Germany) and peak power output (PPO) in order to determine the proper intensities for each subject for the main experiments. The step test consisted of cycling at a cadence ≥80 rpm with an initial workload of 100 W for 5 min and incremental 40-W increases every 5 min until volitional exhaustion was reached.

Subjects participated in three experimental trials each lasting 60 min and each separated by 1 week in a randomized order. (1) Cycling (*C*), (2) cycling with superimposed *E* (*C + E*) and (3) *E*. *C* and *C + E* were carried out on a bicycle ergometer (Schoberer Rad Meßtechnik SRM GmbH, Jülich, Germany) both times adjusted to 70 % PPO and a cadence between 80 and 85 rpm. *E* was carried out in a sitting position on a chair. For the two interventions including *E* (circular-) electrodes were placed all around the thigh (44 × 4 cm) and the calf (27 × 4 cm) stimulating the major muscles of the thigh and the calf. Two electrodes (13 × 10 cm) were placed at the buttocks. A bipolar impulse with an impulse width of 400 ms was continuously applied with 60 Hz. The settings of the E-device (miha bodytec, Emersacker, Germany) were kept identical, including the intensity, for *C + E* and *E*. The intensity was set individually for each subject at the maximum tolerated intensity according to each athlete's discomfort threshold and at an intensity at which proper pedaling was still possible. Electrical stimulation was continuous and independent from cycling. Before each experimental trial, subjects warmed up for 5 min at an intensity of 50 % PPO on a cycle ergometer.

Before each experiment, subjects had a standardized resting-phase of 1 h in which 500 ml of water was ingested in order to assure a well-hydrated status and to provide a good

conductance of the skin where the electrodes are placed. During each session, environmental conditions (temperature and humidity) were kept constant and all three tests were carried out at the same time of day in order to prevent diurnal variations in performance and the hormonal status.

The food intake before the tests was standardized to the extent that subjects recorded their food intake on the day before the first test, and then were advised to reproduce their diet before each test day. In addition carbohydrate-rich foods were recommended to the subjects. A last snack was allowed 2 h before the test. The subjects were not allowed to perform strenuous exercise 24 h before testing. 30 min after each of the three tests, subjects received 500 ml of a low fat chocolate milk and additional energy-bars. Food intake was adjusted so that energy intake matched the calculated energy expenditure of each trial. After the ingestion, subjects were only allowed to drink water until the 240′ blood sample was withdrawn.

Measurements

Venous blood samples were collected for the determination of cortisol, hGH, and testosterone. One venous blood sample was taken before exercise (pre), and five post-exercise samples were taken at 0 min (0′), 30 min (30′), 60 min (60′), 240 min (240′) and 24 h (24 h) after cessation of each of the three tests. 8.5 ml of blood was collected by the Vacutainer blood withdrawal system (Becton–Dickinson). After storage at 7 °C for ~30 min for deactivation of coagulation factors, the blood samples were centrifuged for 10 min at $1.861g$ and 4 °C (Rotixa 50, Hettich Zentrifugen, Mühlheim, Germany). The serum was stored at −80 °C till analysis. Serum levels of cortisol (ng ml^{-1}), hGH (mlU ml^{-1}) and total testosterone (ng ml^{-1}) were determined by using human ELISA kits (Cortisol ELISA EIA-1887; hGH ELISA EIA-3552; Testosterone ELISA EIA-1559; DRG Instruments GmbH Germany). All samples for each parameter were analyzed in duplicate, and the mean was used for statistical analysis. All results of the growth factor analysis were adjusted for changes in plasma volume (PV): PV changes in percentage of pre values = [(Hb$_{pre}$/Hb$_{post}$) × (100 − Hct$_{post}$)/[(100 − Hct$_{pre}$) − 1]] × 100.

During each intervention capillary samples from the earlobe were collected for blood gas (pH, BE, HCO$_3^-$, pO$_2$, PCO$_2$) (AVL Omni 6; Roche Diagnostics GmbH, Mannheim, Germany) and lactate analysis (EBIOplus; EKF Diagnostic Sales, Magdeburg, Germany) under resting condition (pre), and every 15 min during exercise (15′, 30′, 60′). Rating of perceived exertion (RPE) was asked every 15 min during exercise.

Before (pre), directly after (0′), 240 min after (240′) and 24 h (24 h) after exercise, a scale was used to assess

a person's perceived physical state (PEPS) (Kleinert 2006). The subjects were asked to judge spontaneously to what extent 20 given adjectives coincide with their current physical feeling. Each of the four dimensions of the PEPS includes five adjectives: perceived physical energy (e.g., flabby, washed out), perceived physical fitness (e.g., well trained, strong), perceived physical flexibility (e.g., flexible, elastic), and perceived physical health (e.g., sick, injured). Subscale values of the PEPS were computed by a mean function over all items of the belonging subscale.

Statistics

Statistical analyses of the data were performed by using a statistics software package (Statistica for Windows, 7.0, Statsoft, Tulsa, OK, USA). Descriptive statistics of the data are presented as mean ± SD. To assess the effect of the three different interventions on circulating cortisol, testosterone, and hGH, a 2-factor [intervention (C, $C + E$, E); time (pre, 0′, 30′, 60′, 240′, 24 h)] repeated-measures analysis of variance (ANOVA) with Fisher post hoc test was used. For each growth factor we reported the p value corresponding to the main intervention effect, time effect and intervention × time. Statistical differences were considered to be significant for $p < 0.05$. Furthermore, the effect size "partial η^2" was calculated according to the main effect over time for each intervention and parameter. An effect size of 0.1 represents a small effect; 0.25, a medium effect; and 0.4, a large effect.

Results

Lactate and blood gas analysis

Lactate (Fig. 1a) showed significantly higher, BE (Fig. 1c), HCO$_3^-$ (Fig. 1d), and pCO$_2$ (Fig. 1f) significantly lower values during $C + E$ compared to C. HR (Fig. 1g) showed no significant differences between C and $C + E$, but both interventions showed significantly higher levels compared to E. During E, lactate levels were significantly lower, BE, HCO$_3^-$ and pCO$_2$ significantly higher compared to C and $C + E$ respectively. pO$_2$ (Fig. 1e) and pH (Fig. 1b) showed no significant differences between the three interventions.

Hormonal analysis

hGH

Over all ANOVA showed a significant time effect, intervention effect and interaction effect (intervention × time) (all $p < 0.001$). Post-hoc analysis revealed that $C + E$ and
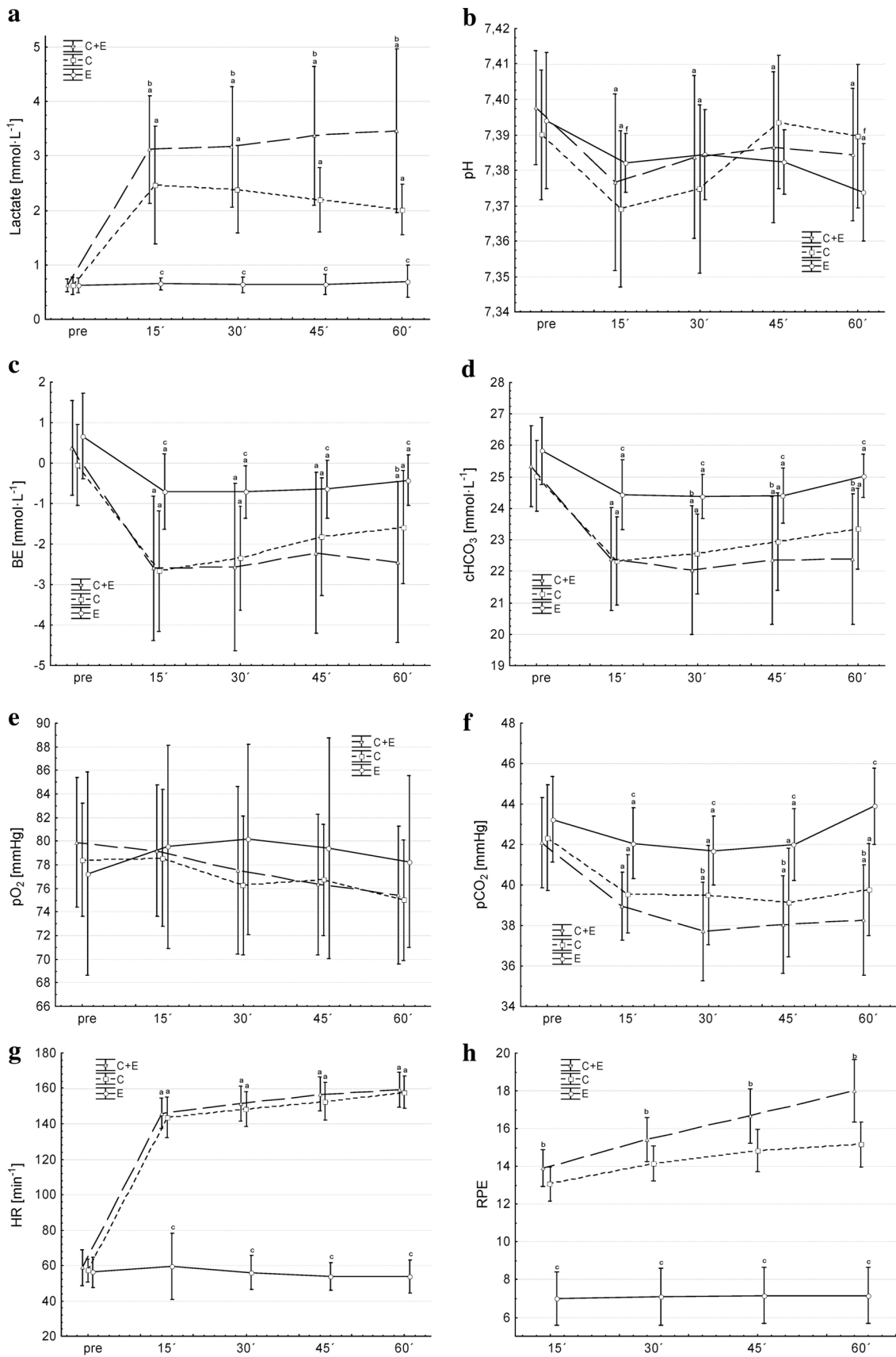
◀**Fig. 1** Changes in lactate (**a**), pH (**b**), BE (**c**), cHCO$_3^-$ (**d**), pO$_2$ (**e**), pCO$_2$ (**f**), HR (**g**), RPE (**h**) before (pre) and during (15′, 30′, 45′, 60′) $C + E$ (*triangles, broken line*); C (*squares, dotted line*) and E (*circles, solid line*). *a* Significant different to 'pre' of the same condition, *b* significant difference between C and $C + E$ at respective time points, *c* significant difference between E and $C + E/C$ at respective time points, *f* significant difference between C and E at respective time points. Values are presented as mean ± SD

C significantly increased cortisol levels 0′ and 30′ after exercise compared to pre, whereas E induced no significant changes. 0′ after exercise $C + E$ showed significantly higher values compare to C. hGH levels were significantly lower 0′ and 30′ after E compared to $C + E$ and C (Fig. 2a). C and $C + E$ showed large effects, E small effects on hGH levels over time, when considering the variations over the six measurements (partial η^2: $C = 0.50$, $C + E = 0.86$, $E = 0.14$).
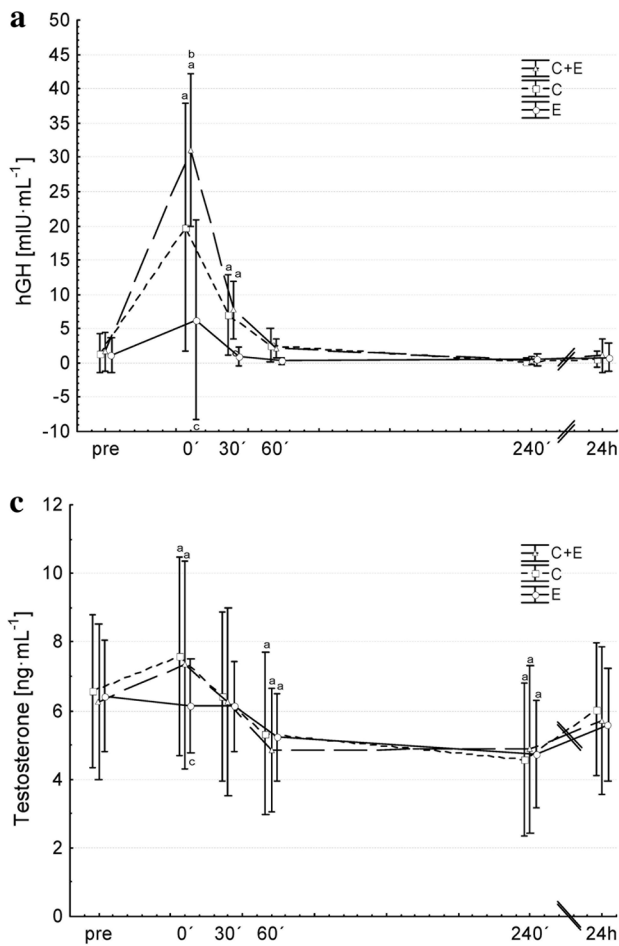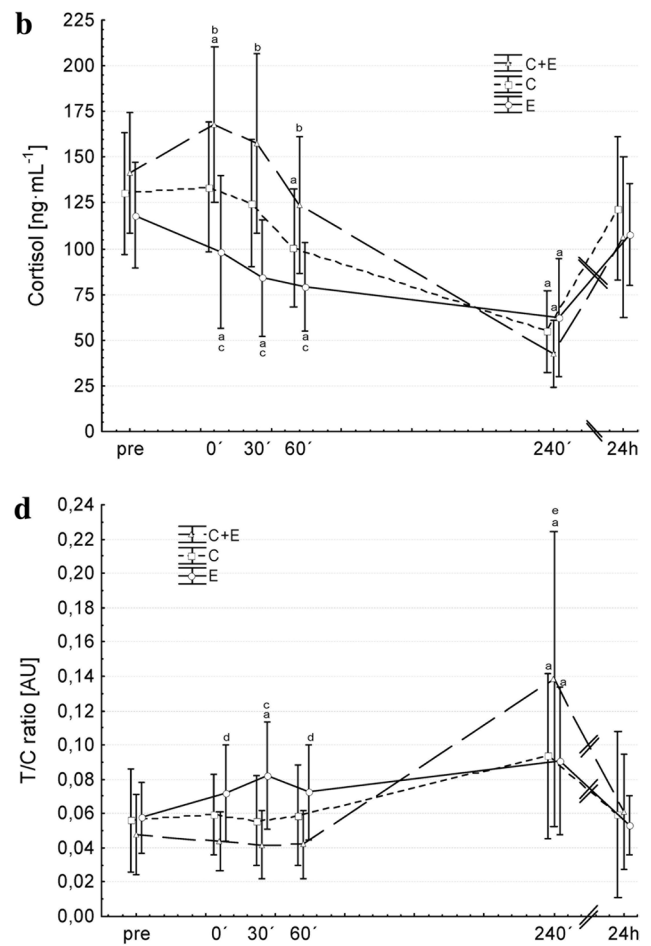
Cortisol

Over all ANOVA showed a significant time effect, intervention effect and interaction effect (intervention × time) (all $p < 0.001$). Post-hoc analysis revealed that $C + E$ significantly increased cortisol levels 0′ after exercise compared to pre, whereas C and E had no or decreasing effects. All interventions differed significantly 0′, 30′ and 60′ between each other (Fig. 2b). All three interventions showed large effects on cortisol levels over time, when considering the variations over the six measurements (partial η^2: $C = 0.66$, $C + E = 0.73$, $E = 0.49$).

Testosterone

Over all ANOVA showed a significant time effect ($p < 0.001$), no intervention effect ($p = 0.58$) and a

Fig. 2 Changes in circulating hGH (**a**), cortisol (**b**), and testosterone (**c**) levels and the *T/C* ratio (**d**) before (pre) and after (0′, 30′, 60′, 240′, 24 h) each intervention: $C + E$ (*triangles, broken line*); C (*squares, dotted line*) and E (*circles, solid line*). *a* Significant different to 'pre' of the same condition, *b* significant difference between C and $C + E$ at respective time points, *c* significant difference between E and $C + E/C$ at respective time points, *d* significant difference between $C + E$ and E at respective time points, *e* significant difference between $C + E$ and C/E at respective time points, *f* significant difference between C and E at respective time points. Values are presented as mean ± SD
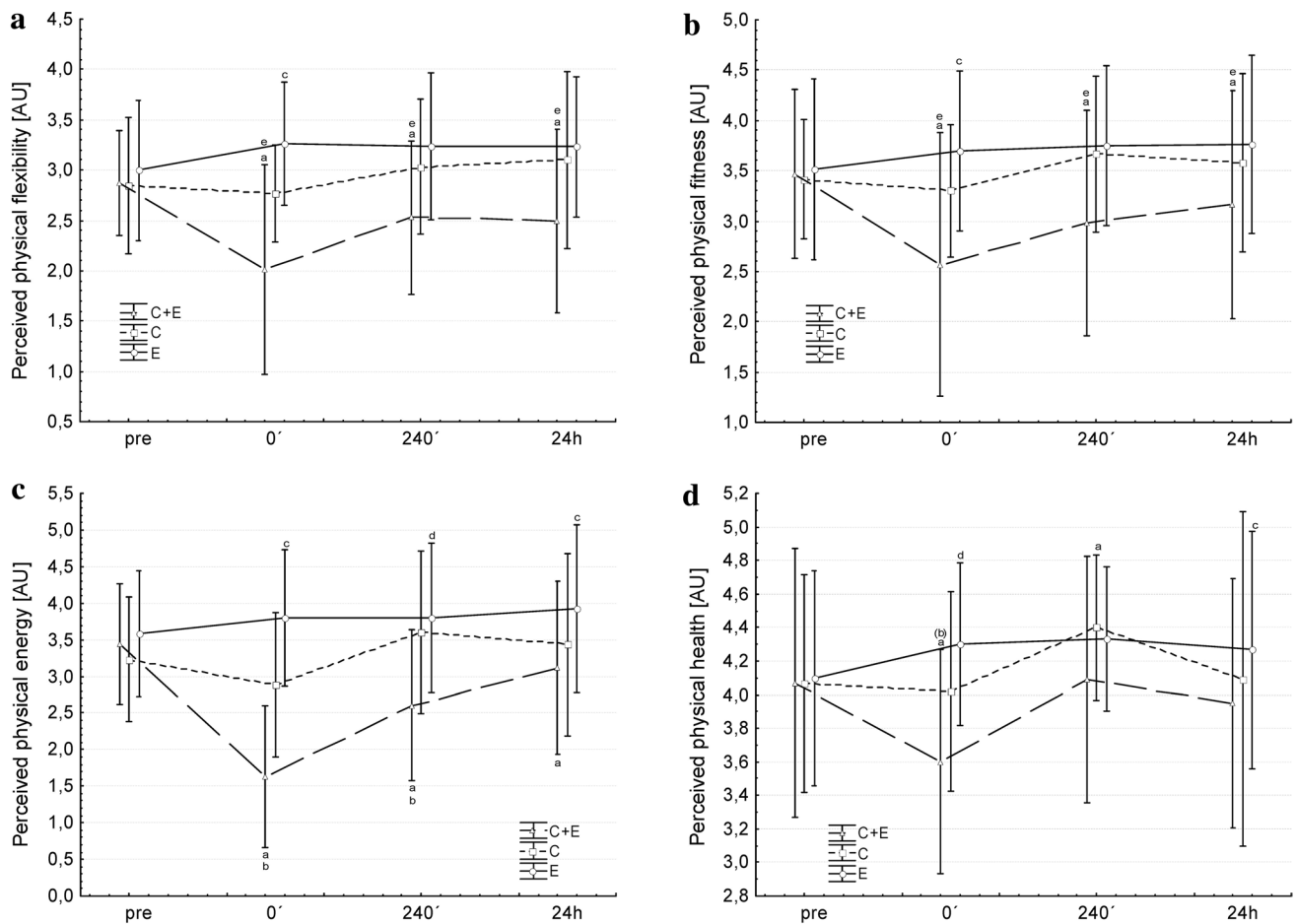
Fig. 3 Changes in perceived physical flexibility (**a**), perceived physical fitness (**b**), perceived physical energy (**c**) and perceived physical health (**d**) before (pre) and after (0′, 240′, 24 h) each intervention: $C + E$ (*triangles, broken line*), C (*squares, dotted line*) and E (*circles, solid line*). *a* Significant different to pre of the same condition, *b* significant difference between C and $C + E$ at respective time points, *c* significant difference between E and $C + E/C$ at respective time points, *d* significant difference between $C + E$ and E at respective time points, *e* significant difference between $C + E$ and C/E at respective time points, *f* significant difference between C and E at respective time points. Values are presented as mean $\pm$ SD

significant interaction effect (intervention × time) ($p < 0.05$). Post-hoc analysis revealed that $C + E$ and C significantly increased testosterone levels 0′ after exercise compared to pre, whereas E induced no significant changes. $C + E$ and C showed significantly higher values 0′ compared to E (Fig. 2c). All three interventions showed large effects on testosterone levels over time, when considering the variations over the six measurements (partial η^2: $C = 0.54$, $C + E = 0.51$, $E = 0.43$).

Testosterone/cortisol ratio (T/C ratio)

Over all ANOVA showed a significant time effect ($p < 0.001$), no intervention effect ($p = 0.13$) and a significant interaction effect (intervention × time) ($p < 0.001$). Post-hoc analysis revealed that all interventions significantly increased the *T/C* ratio 240′ after exercise. However,

$C + E$ showed significantly higher values 240′ post compared to C and E. 0′, 30′ and 60′ the *T/C* ratio was significantly higher after E compared to $C + E$ and 30′ also to C (Fig. 2d). $C + E$ showed large effects, C and E medium effects on the *T/C* ratio over time, when considering the variations over the six measurements (partial η^2: $C = 0.28$, $C + E = 0.55$; $E = 0.37$).

PEPS and RPE

RPE showed significant differences between all interventions and was highest during $C + E$ (Fig. 1h). In order to characterize the three interventions from the athlete's point of view, changes of the PEPS are shown in Fig. 3. In general, the $C + E$ intervention was the most demanding intervention for the athletes with significant changes over time and with significant differences compared to C

and *E* mainly over all time points. *C* or *E* had no significant effects over time (Fig. 3).

Discussion

The present study investigated the effects of *C*, *E* and *C* + *E* on the concentration of 2 anabolic hormones (hGH and testosterone) and one catabolic hormone (cortisol) in order to quantify the intensification of endurance training with *E*. The major findings of the present study are that *C* + *E* caused the highest increases in cortisol and hGH followed by *C* and *E*. Serum testosterone levels were significantly increased by *C* and *C* + *E*, whereas *E* had no effects. Metabolic stress, characterized by values of lactate and blood gas measurements (pH, BE, HCO_3^-, PO_2, PCO_2) was highest during *C* + *E* followed by *C* and *E*. RPE was highest during *C* + *E*. The PEPS was most affected by *C* + *E* even up to 24 h post exercise, affecting nearly all dimensions of the scale, whereas *C* and *E* had no significant effects.

In previous studies, we were able to show that cortisol and hGH clearly react in an intensity dependent manner (Wahl et al. 2013). Therefore, the present results of the hormonal response underline the results of the higher metabolic disturbances and show that it is possible to induce an intensification of endurance training with superimposed *E* during cycling. The exact mechanisms and factors that affect the exercise-induced release of hGH are not known yet, even though several possibilities, such as differences in neural stimuli, accumulation of catecholamines, ammonia and lactate, acid–base balance and nitric oxide are discussed (Godfrey et al. 2003; Stokes 2003; Wahl et al. 2010c). Although the mechanism by which hGH secretion is stimulated by acid–base changes and lactate is yet to be fully understood, it was shown that the activation of the hypothalamus-pituitary axis by afferent signals from muscle metaboreceptors plays an essential role in the control of hormone responses during exercise (Goto et al. 2005; Takarada et al. 2000). Hence, an exercise regimen with greater metabolic stress, like the one with superimposed *E* of the present study, should cause greater hGH responses to exercise. Another factor which could explain the higher hGH levels after *C* + *E* compared with *C* is a direct neural input to the anterior pituitary. Gosselink et al. (1998) suggested that afferents originating in fast-twitch motor units increase hGH release. As *E* recruits additional type 2 fibers and motor units, this stimulation might as well explain the higher hGH levels and the increased metabolic changes during and after *C* + *E*. However, this mechanism might only play a minor role, as *E* alone did only induce a slight but not significant increase.

Exercise stress evaluated by cortisol levels was highest after *C* + *E*, whereas *C* and *E* had no or even decreasing effects on cortisol levels directly after exercise, which corresponds with the athletes RPE and perceived physical state. As previous studies showed that exercise intensity positively deflects cortisol concentrations (Wahl et al. 2013), the present data indicate that *E* clearly induced an intensification. A slightly higher energy turnover, due to the enhanced recruitment of type 2 fibers, and the greater disturbances in the acid–base balance (Wahl et al. 2010c) might explain the higher cortisol levels after *C* + *E* compared to *C* and *E*. The exercise-induced increase in the blood cortisol concentration seems to be essential for the metabolic response to exercise and in the process of adaptation (Viru and Viru 2004), as cortisol is involved in the maintenance of blood glucose levels by acting upon skeletal muscle and adipose tissue (Galbo 2001; Wolfe 2001). Furthermore, post-exercise increases in cortisol are essential because of their catabolic action on the proteins that were damaged during exercise, thus leaving a "pool" to the synthesis of new amino acids (Viru and Viru 2004). In previous studies, we were able to show the higher skeletal muscle damage after cycling with superimposed *E* compared to normal cycling (Wahl et al. 2012), may be leading to an increased protein turnover, explaining furthermore the higher cortisol levels after *C* + *E* compared to *C* and *E* of the present study.

As previously explained, exercise with enhanced metabolite accumulation enhances concomitant hGH secretion (Goto et al. 2005; Takarada et al. 2000); however, this was not shown for testosterone (Viru et al. 1998). Viru et al. suggested that the control of the concentrations of hGH/cortisol seems to differ from the control of testosterone. This might explain the present findings of similar testosterone increases after *C* + *E* and C. However, the *T/C* ratio showed the highest increases after *C* + *E*. The *T/C* ratio has been suggested to be an indicator of the anabolic/catabolic state (Viru and Viru 2004). However, the significance of this ratio has been questioned and the interpretation of a general anabolic or catabolic status seems to be an oversimplification. Anyway, in the present study, the increase in the *T/C* ratio may indicate an anabolic state. The time course between *C* + *E* and *C/E* was different, showing later and higher anabolic reactions after *C* + *E*. This might be due to the more demanding character of *C* + *E* and therefore the need of a longer time of recovery, which is in line with previous studies showing an intensity dependent reaction of the *T/C* ratio (Wahl et al. 2013). Furthermore, the higher muscle damage that we showed in a previous study to occur during *C* + *E* (Wahl et al. 2012), may require a longer anabolic response to recover the damage.

As metabolic changes and acute hormonal responses are important for the induction of cellular signaling cascades and adaptations (Coffey and Hawley 2007), the present results indicate that adding *E* to cycling may be an

enhancing stimulus for expanding aerobic capacity in athletes, and for patients who cannot perform high workloads. As the physiological effects of hGH, testosterone, and cortisol (growth, turnover of muscle, bone and collagen, regulation of metabolic function) are well known (Godfrey et al. 2003; Viru and Viru 2004), we can speculate that an acute increase as shown in the present study, might play a positive role in optimizing endurance training adaptations and in eliciting health benefits. Even at lower exercise intensities, additional *E* may allow one to induce a high local stimulus on skeletal muscle. The nonselective recruitment may provide (clinical) advantages in that all fibers, regardless of type, have the potential to be activated at relatively low exercise intensities, which might lead to greater skeletal muscle adaptations and improvements of endurance performance, especially in fast-twitch fibers (Gregory and Bickel 2005). However, depending on the stimulation intensity, *E* might only stimulate superficial and not deeper fibers (Adams et al. 1993).

Nevertheless, one has to consider the negative impact of superimposed electrical stimulation on perceived fitness, flexibility, energy and health, which might decrease the compliance, especially of less trained people to this kind of training and which might lead to worsened effectiveness. However, this seems to be more a general problem of intense training protocols, as a significantly inferior perceived physical state was also shown for HIT in comparison to high-volume low-intensity training sessions (Wahl et al. 2013). Furthermore, we are aware that a more refined approach would be to trigger stimulation to coincide with muscle EMG activity during the exercise, so that *E* contributes to the cycling (to volitional contractions), and does not interfere with normal contraction/relaxation cycles.

Conclusion

In conclusion, the results of the present investigation showed that superimposed *E* is a useful method to intensify endurance training, even when performing low to moderate external workloads. It suggests that $C + E$ promotes anabolic processes more that *C* or *E*, due to higher increases of hGH and the *T*/*C* ratio. As these hormones are involved in inducing endurance adaptations (erythropoiesis, expression of aerobic enzymes), the described hormonal reactions may contribute to positive effects, similar to those of intense training protocols (HIT). Moreover, the present results provide further evidence of the association between the metabolic stress and the hormonal responses. It can be speculated, that these acute hormonal increases and metabolic perturbations, might play a positive role in optimizing long-term training adaptations.

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