The cortisol awakening response reflects fatigue caused by high-intensity resistance exercise

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Research Article

Keywords: exercise stress, endocrine, physical adaptation-maladaptation, biomarker

Posted Date: April 11th, 2022

DOI: https://doi.org/10.21203/rs.3.rs-1520784/v1

Abstract

Background

The cortisol awakening response (CAR) reflects the physical adaptation to exercise and training loads and has been reported the response especially by high-intensity exercise. However, few studies have examined the effects of resistance exercise. The purpose of this study was to investigate the effect of high-intensity resistance exercise on the CAR.

Methods

Thirteen healthy male university students performed 1 repetition maximum (1 RM) tests and an experimental session for 4 consecutive days: sedentary control sessions (Day 1 and 2), high-intensity resistance exercise (bench press) session at 75% of 1 RM (HIRE; Day 3), and recovery session (Day 4). Saliva samples for Day 1–3 were collected: 1) before and 2) immediately after the session, 3) 10 min, 4) 20 min, and 5) 30 min into the recovery period from each session, 6) 9 pm, and 7) 11 pm at night after the session, and 8) immediately after awakening and 9) 15 min and 10) 30 min post-awakening the day after the session (Sampling on Day 4 only upon awakening).

Results

The results showed that the HIRE session did not change the acute cortisol levels; however, the CAR showed significantly different responses in those who increased ($n = 7$; Responders) and those who did not increase ($n = 6$; non-Responders). In addition, a significant negative correlation was found between the rate of increase in area under the curve for the whole of CAR (AUC_g) and the amount of change of physical fatigue from control days (Day 1 and 2) to Day 3 and 4 ($r = -.478$, $p = .014$).

Conclusions

These results suggest that both increased and no increased CAR may result from physical adaptation and maladaptation to the HIRE session. Therefore, CAR has the potential to assess the physical adaptation to the loads of local resistance exercise as well, which may help to plan future training loads and recovery.

Background

Exercise training has positive effects that led to improved physical functions such as enhanced energy metabolism and circulation (adaptation), but also negative effects such as causing overtraining (maladaptation) (1). The duality of exercise effects (adaptation-maladaptation) can be explained by the concept of allostasis – the ability to maintain stability through change (2). When there is a proper

exercise and training load and/or recovery period from the load, it functions in allostasis and provides the positive effect of improving physical functions. If these are not effectively performed, the allostatic load, limiting factor in allostasis, may increase, which results in the negative effect of disruption of physical functions.

Cortisol, which sensitively reflects the activity of the hypothalamic-pituitary-adrenal (HPA) axis, has been studied extensively as one of the evaluation indicators of the allostatic process (3, 4). It has a diurnal rhythm that was high in the morning and low at night (5) and occurs in acute increases in concentration as a response to stressful stimuli such as high-intensity exercise and greater threat psychological stress (6, 7, 8). As such, contextual and environmental events can influence cortisol levels during the daytime.

In addition to these responses, an increase in cortisol levels was observed in the 30–45 minutes after awakening, independent of the circadian rhythm of cortisol (9). This phenomenon is known as the cortisol awakening response (CAR) and has been investigated in association with a variety of psychological and physical conditions as an indicator reflecting chronic stress (10). CAR is generally governed by hypothalamic and pituitary hormone regulation and is thought to be regulated by recognizing daily demands, and can be critical in giving the necessary energy while switching from a resting to an active state (9, 11). Recently, CAR has also been studied in the context of exercise and training, suggesting that exercise and training load can influence its responses. After a seven-day hard training phase, CAR noticeably increased, and the higher the CAR, the lower the rate of decline in performance among elite soccer players (12). The training load from the previous day had an association with CAR, but it had no discernible relationship with psychological indicators (13). In addition, the study, which compared CAR in athletes diagnosed with overtraining syndrome, healthy athletes, and sedentary controls, reported blunted CAR in athletes diagnosed with overtraining syndrome (14). These results may reflect the progressive process of adrenal disturbance-adaptation-maladaptation, consistent with the “prolonged response” model of allostatic loads since over-or under-activity of the HPA axis can occur depending on the degree of physical condition (15). Therefore, there are suggestions that increased and blunted CAR may indicate physical adaptation and maladaptation to exercise and training and may be an early predictor of overtraining.

The main findings of CAR reactions were not experimentally investigated because most earlier studies on CAR were field investigations. In this context, Ogasawara et al. (16) experimentally examined the effects of three different intensities of exercise (40%, 60%, and 80% of VO_2 max) with a cycle ergometer on CAR the following day in healthy men. The results showed that high-intensity exercise resulted in higher CAR on the following day, suggesting that CAR may be influenced by the threshold of exercise intensity. However, exercise and training situations may incorporate resistance exercise to improve muscle hypertrophy and maximal muscle strength into their training program, in addition to aerobic exercise to improve cardiorespiratory endurance. Considering that CAR is used as an indicator to assess physical adaptation-maladaptation to exercise and training loads, it should also examine the effects of resistance exercise.

With these considerations in mind, the purpose of this study was to experimentally investigate the effect of resistance exercise on CAR in healthy male university students. CAR was positively correlated with the training load of the previous day (13), and higher values were observed the day after high-intensity exercise with a cycle ergometer (16). On the other hand, the acute effect of resistance exercise on the HPA axis is less pronounced in the resistance exercise paradigm compared to endurance exercise because the metabolic stimulus for cortisol secretion is less prominent (17). We hypothesized that 1) CAR would increase with high-intensity resistance exercise as well as previous study using a cycle ergometer, and 2) CAR would not change through resistance training even at a high intensity level due to the lack of stimulation of the HPA axis.

Methods

Study Design and Setting

To investigate the effects of high-intensity resistance exercise on CAR, this study consisted of strength testing and four consecutive days of experimental session (Figure 1). The first visit (strength testing) was used for screening and measuring their one-repetition maximum (1RM). The next four days were conducted experimental session that continued as follows: two days of sedentary control sessions (Day 1 and Day 2), a high-intensity resistance exercise session until exhaustion (HIRE; Day 3), and a recovery session considering the effect of delayed onset muscle soreness which occurs 8 to 72 hours after exercise (Recovery; Day 4) (18). Strength testing and experimental sessions were separated by at least 72 hours. Since bench press exercises have been commonly used among various trained individuals and the load can be easily controlled to ensure safety (19), a bench press exercise was selected as a high-intensity resistance exercise session in this study. In addition, we first examined the effect of intensity aimed at muscle hypertrophy (75% of 1 RM), which was observed to increase greater acute cortisol levels in previous studies (20, 21). The CAR was measured during the day after each session, and each response was compared. Additionally, as CAR has high intra-individual stability, whereas high inter-individual variability (12), we also analyzed focusing on the inter-individual variability.

[Insert Figure 1]

Participants

Before the experiment, all participants were provided with an oral and written informed consent. Fourteen healthy male university students with at least 1 year of resistance exercise experience participated in this study (mean ± SD; 21.9 ± 0.9 years, 171.2 ± 5.8 cm, 68.3 ± 6.0 kg, 23.3 ± 1.4 BMI, 88.08 ± 17.8 1 kg bench press 1RM). Exclusion criteria included a history of hormonal disorders, mental illness, smoking, a diet chronically low in carbohydrates, use of anabolic steroids, or chronic nonsteroidal anti-inflammatory drug use. One participant was excluded since he was affected by other exercises during the experimental period. Therefore, 13 participants' data were used for the further analyses. This research was approved by the Ethics Committee at Osaka University of Health and Sport Sciences (approval number: 20-19).

Procedures

Strength Testing

At least 72 hours prior to the experimental session, each participant had their 1 RM determined on the bench press exercise. The 1 RM tests were conducted using the National Strength and Conditioning Association protocol for 1 RM testing (22). The protocol required the participants to progressively increase the resistance across several trials until the 1 RM was achieved. During the trials, the participants were asked to raise to maximum speed for all the trials. The movement velocity (MV) was measured on each repetition to determine the 1 RM based on the load-velocity relationship. The MV showed on the last successful repetition was defined as the movement velocity threshold (MVT) (23), which was used to evaluate the training load during the HIRE session.

Experimental Session

On Day 1 and Day 2, a sedentary control session (20 minutes) was conducted each day to measure their baseline values. On Day 3 (HIRE session), the participants performed bench press exercises with (a) 20 kg for 10 repetitions and (b) 40% for 10 repetitions as a warm-up exercise. After the warm-up, they performed 10 repetitions of bench press exercises at 75%. Following a 90-second rest, they performed 5-8 sets again until exhaustion. On Day 4, a Recovery session without any physical activity for a day was conducted for an additional day after the HIRE session.

As shown in Figure 1, measurements for the control and the HIRE sessions were taken based on previous research (16); before (Pre) and immediately after (Post-0) the experimental session, during the recovery period of the session (every 10 min for 30 min; Post-10, Post-20, and Post-30), the night after returning home from the session (9 pm, 11 pm; Recovery 1 and Recovery 2), and upon waking up the day after the session. The Recovery session of Day 4 was conducted only to measure upon waking up the next day. All participants completed the sessions at the same time of the day (6 pm ± 30 min) to control for biological variation. They were asked to eat similar meals during the experiment and to refrain from eating at least 4 hours before the experiment and no later than 8 pm after the experiment. In addition, the participants were required to refrain from exercise, caffeine, and alcohol during the experiment.

Instruments

Evaluation of bench press exercise

To evaluate whether the HIRE session was performed to exhaustion, MV and the rating of perceived exertion (RPE) of the upper limb were measured. MV was measured during each repetition of the HIRE session using accelerometers (PUSH Band 2.0: PUSH Inc., Canada). The RPE of the upper limb was determined using Borg's 6-20 points rating of perceived exertion scale, a validated Japanese questionnaire (24). The HIRE session was considered valid and reliable if the participants met three of the following criteria: 1) MV at the last repetition showed equal to or lower than MVT (23), 2) the RPE of the

upper limb rating was of 19 or greater, and 3) participants were unable to lift the bench press by themselves.

Cortisol measurements

Saliva samples were collected (Figure 1) by the passive drool techniques. Prior to the experiment, the participants were informed of the guidelines for saliva collection. Participants collected saliva in their mouths for 2 minutes before pouring it into a tube using a straw. To avoid saliva dilution, no mouthwash was utilized. Participants were asked not to drink, wash their teeth, or bathe for 1 hour before saliva collection for Recovery 1 and 2. During the wakeup phase, they were also not allowed to eat, drink, brush their teeth, engage in vigorous physical activity, or return to sleep, but they were allowed to engage in other normal morning activities. On a Google form, the participants were asked to report their sleeping time, waking time, saliva collection time, and sleeping status. Participants were asked to preserve saliva samples in a commercial freezer at minus −4°C for 48 hours before submitting them to the laboratory. To precipitate particulate particles, the saliva samples were centrifuged at room temperature for 5 minutes at 3000g and kept at minus −80°C until analysis. Following that, cortisol concentrations were determined using an enzyme-linked immunosorbent test (Cortisol ELISA Kit (RE52611), IBL, Japan). The magnitude of change (CAR_c), the relative change (CAR%), and the area under the curve were determined in two ways from the samples collected upon awakening: relative to the ground (compared to a "zero" concentration; AUC_g) and relative to the increase (related to the first concentration; AUC_i) (13, 9).

$$CAR_c = \text{Maximum value of } [C_{15}] \text{ or } [C_{30}] - [C_0]$$

$$CAR\% = \frac{CAR_i}{[C_0]} \times 100$$

$$AUC_g = \frac{([C_0]+[C_{15}]) \times 15}{2} + \frac{([C_{15}]+[C_{30}]) \times 15}{2}$$

$$AUC_i = AUC_g - [C_0] \times 30$$

Factors influencing the CAR

To explore the factors that changed the CAR resulting from the HIRE session, physical conditions (perceived physical fatigue and muscle soreness of the upper limb) were assessed using a visual analogue scale (VAS). In addition to excluding confounding factors, factors that could influence CAR were measured upon awakening. The degree of stress and sleep quality were evaluated by VAS. The psychological condition was assessed with a total mood disturbance (TMD) score calculated from the short form of the Profile of Moods States Second Edition (POMS2-S), which has been translated into Japanese (25). Participant graded a set of 35 items related to the mood on a Likert scale from 0 (not at all) to 4 (extremely) to such question "How do you feel at this moment?" to assess 7 dimensions: Anger-Hostility (AH), Confusion-Bewilderment (CB), Depression-Dejection (DD), Fatigue-Inertia (FI), Tension-Anxiety (TA), Vigor-Activity (VA), and Friendliness (F).

Data Analysis

Data were expressed as mean ± standard error. A paired t-test was used to compare the MVT and MV at the last repetition of the HIRE session. Two-way repeated-measures ANOVA tests were conducted to analyze the changes in cortisol concentrations on the day of the experimental session (session [4] × sampling point [7]) and CAR on the day after the session (session [4] × sampling point [3] and session [3] × sampling point [3]). One-way repeated measure ANOVAs were used to analyze the difference between experimental sessions in CAR data (CAR_{σ}, CAR%, AUC_{g}, and AUC_{i}), VAS (physical fatigue, muscle soreness, stress, and sleep quality), the TMD score during awakening, sleeping hours, and awakening time. Mauchly's test was used to assess the sphericity assumption. If the assumption was violated, Greenhouse-Geisser epsilon values were used to adjust the degrees of freedom. Bonferroni corrections were used for post-hoc tests. Estimate of effect size was quantified partial eta squared (η_p^2), where η_p^2 = 0.01, 0.06, and 0.14 were estimated for a small, moderate, and large effect, respectively (26). We also conducted correlation analyses (Pearson correlation) to assess the relationship between the rate of change in CAR data (CAR_{σ}, CAR%, AUC_{g}, and AUC_{i}) and the amount of change in perceived physical condition data (physical fatigue and muscle soreness).

The level of significance was set to $\alpha \leq .05$. In addition, a more liberal α value of $\leq .10$ was used to determine marginal significance to avoid Type error (27). All statistical analyses were performed using SPSS version 27.0 (IBM, Japan).

Results

There was no significant difference in MV at the last repetition compared to MVT ($t(12) = 1.648$, $p = 0.125$, $d = 0.355$). In addition, the RPE value of the upper limb at the last repetition was 19.8 ± 0.1 (Table 1).

Table 1
Exercise data of each participant

Case no.	RPE of the upper limb								MVT (m/s)	MV at final rep (m/s)
	1st set	2nd set	3rd set	4th set	5th set	6th set	7th set	8th set		
1	15	17	18	19	19	19	20	-	0.18	0.17
2	13	15	18	19	19	20	-	-	0.21	0.21
3	13	16	17	19	19	19	20	20	0.20	0.16
4	13	18	19	19	20	20	-	-	0.18	0.20
5	13	15	18	19	19	20	-	-	0.21	0.21
6	13	14	17	20	20	-	-	-	0.20	0.19
7	5	18	19	20	20	-	-	-	0.16	0.16
8	17	19	20	20	20	-	-	-	0.19	0.15
9	14	16	17	17	19	-	-	-	0.17	0.20
10	13	13	15	16	17	19	-	-	0.24	0.22
11	12	12	16	19	20	20	-	-	0.22	0.20
12	13	15	16	17	18	20	-	-	0.15	0.14
13	13	16	18	19	20	-	-	-	0.22	0.20
mean	12.8	15.7	17.5	18.7	19.2	19.6	20.0	20.0	0.19	0.19
SE	0.7	0.5	0.4	0.3	0.2	0.1	0.0	0.0	0.007	0.007
MV: Mean concentric velocity is defined as the average velocity taken from all the velocities recorded during the entire concentric portion of an exercise (23).										
MVT: The mean concentric velocity produced on the last successful repetition of a set to failure performed with maximal lifting effort (23).										

[Insert Table 1]

Cortisol Data Throughout The Experimental Session

The changes in salivary cortisol concentration on the day of the experimental session and CAR on the day after the experimental session are shown in Fig. 2.

Salivary cortisol concentration on the day of the experimental session showed that the main effect was significant at sampling point (F (6, 72) = 10.704, p = .002, ε = .242, η_p^2 = .471). The main effect of session (F (2, 24) = 0.554, p = .582, η_p^2 = .044) and the interaction between session and sampling point (F (12, 144) = 0.765, p = .497, ε = .201, η_p^2 = .060) were not significant. The results of multiple comparisons at the sampling points were significantly lower for Recovery 2 compared to Pre-test (p = .031).

CAR on the day after the experimental session showed that the main effect was significant at sampling point (F (2, 24) = 5.567, p = .025, ε = .642, η_p^2 = .317). The main effect of session (F (3, 36) = 0.518, p = .582, η_p^2 = .041) and the interaction between session and sampling point (F (6, 72) = 1.800, p = .168, ε = .471, η_p^2 = .130) were not significant. The results of multiple comparisons at the sampling points showed higher values at C_{30} compared to C_0 and C_{15} (p = .079, p = ,023, respectively).

[Insert Fig. 2]

Factors That Influenced The Car

Factors that influenced the CAR measured upon awakening are shown in Table 2 and Table 3. The main effect was significant in muscle soreness (F (3, 36) = 18.109, p = .000, ε = .621, η_p^2 = .601) at session. Post-hoc tests of muscle soreness confirmed that it was significantly higher on Day 3 than both Day 1 and 2 (p = .002) and Day 4 than Day 1 (p = .001) and Day 2 (p = .000). There were no other significant main effects.

Table 2
Factors influencing CAR as measured at awakening ($M \pm SE$)

Measure	Day 1 (Control)	Day 2 (Control)	Day 3 (HIRE)	Day 4 (Recovery)
Physical fatigue	12.0 ± 3.0	10.8 ± 2.7	21.7 ± 5.2	24.4 ± 5.4
Muscle soreness	5.5 ± 3.0	2.1 ± 1.1	36.5 ± 6.8*	35.5 ± 5.0*
Stress	13.9 ± 3.3	14.6 ± 3.3	12.5 ± 2.8	18.2 ± 4.9
Sleep quality	77.3 ± 5.6	58.3 ± 6.5	68.3 ± 6.1	57.3 ± 7.4
TMD score	98.6 ± 3.3	98.2 ± 2.9	97.0 ± 3.1	97.7 ± 3.2
Muscle soreness was significantly higher on Day 3 and Day 4 than on Day 1 and Day 2, respectively (*p < .05). Values are mean ± SE.				

Table 3
Sleeping hours, awakening time, and first sampling point during the experimental session ($M \pm SE$)

Measure	Day 1 (Control)	Day 2 (Control)	Day 3 (HIRE)	Day 4 (Recovery)
Sleeping Hours (h)	7.0 ± 0.4	6.9 ± 0.3	7.0 ± 0.3	7.0 ± 0.4
Awakening Time	7:40 ± 0:21	7:53 ± 0:26	8:28 ± 0:22	8:02 ± 0:27
First Sampling Time	7:42 ± 0:21	7:56 ± 0:27	8:29 ± 0:22	8:03 ± 0:27

[Insert Table 2]

[Insert Table 3]

The Result Of Considering Individual Adaptation

The CAR data (CAR_c, CAR%, AUC_i, and AUC_g) of each participant can be seen in Fig. 3. There were no significant main effects for any of the CAR data, but the individual data resulted in differences in inter-individual variability from the training session: several subjects increased CAR data, whereas other subjects showed no increases (or even decreases) in the same variables.

[Insert Fig. 3]

To distinguish between different individual adaptations to the training sessions, the subjects were separated into two groups based on arbitrary criteria used in previous study (12): Responders ($n = 7$) and non-Responders ($n = 6$). For this purpose, the mean of AUC_g of control days (Day 1 and 2; Control) and the change of percentage in AUC_g from Control (the mean of Day 1 and 2) to Day 4 were calculated. Then, in the case of subjects with variable control values (Day 1 and 2), as shown in Fig. 4, the data of the factors influencing CAR between two days were compared, and one day with a stronger influence was excluded. Finally, the median change of percentage in AUC_g (24.7%) was used to separate the two groups.

[Insert Fig. 4]

The Responders group showed the significant main effect of session ($F(2, 12) = 11.655$, $p = .002$, $\eta_p^2 = .660$) (Fig. 5). The interaction of session and sampling point was also significant ($F(4, 24) = 3.880$, $p = .014$, $\eta_p^2 = .393$). No significant main effect was found for sampling points ($F(2, 12) = 1.275$, $p = .315$, $\eta_p^2 = .393$). Post-hoc tests of session confirmed that the HIRE and the Recovery sessions were significantly higher than Control at C_{15} ($p = .045$, $p = .013$, respectively). In addition, the Recovery session was significantly higher than Control at C_{30} ($p = .022$). Post-hoc tests of sampling points showed no significant differences in any session. The non-Responders group, however, showed the significant main

effect of sampling points (F (2, 10) = 12.317, p = .002, η_p^2 = .711). The main effect of session (F (2, 10) = 1.626, p = .245, η_p^2 = .245) and the interaction between session and sampling point (F (4, 20) = 0.244, p = .910, η_p^2 = .047) were not significant. The results of multiple comparisons at the sampling points showed higher values at C_{15} and C_{30} compared to C_0 (p = .022, p = ,039, respectively).

[Insert Fig. 5]

The correlation analysis showed a significant negative relationship between the change of percentage in AUC_g and the amount of change in physical fatigue from Control to Day 3 and 4 (r = − .478, p = .014), whereas no association was found with the amount of change in muscle soreness (r = − .120, p = .560) (Fig. 6). There was no significant relationship between other CAR data (the change of percentage in AUC_i, CAR_o, and CAR%) and physical condition data (the amount of change in physical fatigue and muscle soreness).

[Insert Fig. 6]

Discussion

This study aimed to investigate the effect of high-intensity resistance exercise on CAR in healthy participants. Our results showed that the HIRE session did not change the cortisol concentration on the day of the experiment; however, the CAR showed different responses in those who increased and did not increase. In addition, a significant negative correlation was found between the rate of change in the area under the curve for the whole of CAR (AUC_g) and the amount of change of physical fatigue from control days (the mean of Day 1 and 2) to Day 3 and 4. These results suggest that an increase and no increase of CAR may respectively result from physical adaptation and maladaptation to the HIRE sessions. This was the first study to demonstrate that CAR reflected physical adaptation to a single experimental resistance exercise.

All participants performed the HIRE sessions to exhaustion. This was clear from the results of the MV and the local RPE. Nevertheless, salivary cortisol levels did not change in the HIRE session. This result was different from our previous study, showing that CAR was higher the day after high intensity (80% VO_2max) exercise with a cycle ergometer (16). The metabolic stimulation of cortisol secretion was found less prominent in the resistance exercise paradigm than in endurance exercise (17). Therefore, the acute effects of resistance exercise on the HPA axis are not typically as pronounced. In fact, training intensities aimed at improving muscle strength/power (88% of 1 RM) did not produce acute changes in cortisol levels (20, 21).

On the other hand, metabolic demanding higher total work protocols (i. e., high volume, moderate to high intensity with short rest periods) elicited a greater acute cortisol response as part of a larger remodeling process in muscle tissue, even in the context of resistance exercise (28). Training intensities aimed at improving muscle hypertrophy (75% of 1RM) and muscular endurance (60% of 1RM) resulted in acute

increases in cortisol concentrations (20, 21). The HIRE sessions in this study were performed at the intensity of 75% of 1RM aiming at muscular hypertrophy, which did not increase the cortisol levels. In the above previous study, the participants performed exercises that mobilized larger amounts of muscle mass (squat lifts, full-body exercises combining multiple disciplines) compared to the bench press exercises performed in this study. Therefore, the bench press exercise, which mobilizes a small amount of muscle, might have been a factor that did not cause an acute cortisol response. The decreased level of cortisol from pre to Recovery 2 was perhaps not because of resistance exercise but rather due to the diurnal variation of cortisol, which was high in the morning and low at night (5).

The results of CAR on the day after the experimental session showed no change for overall participants; however, there were two types of participants at the individual level: the ones whose CAR increased and the other whose CAR did not increase. Interestingly, such changes were observed in the CAR while the HIRE session did not produce any acute physiological response. Considering the relationship with other data (especially physical condition), it seems that the results supported the hypothesis that the HIRE session would increase CAR, but not the hypothesis that it would not change CAR. It is generally considered that resting cortisol concentrations reflect the adaptation to chronic training stress (28), but chronic resistance exercise did not produce consistent cortisol secretion. There have been reports of unchanged (29, 30), decreased (31, 32), and increased (33) resting cortisol concentrations during normal strength and power training in men and women during short-term overreaching, respectively. In the context of the CAR study, there were also participants whose CAR increased and did not increase (or even decreases) during the 7-day strength training period (12). In the study, the rate of decrease in performance was positively correlated with the respective CAR data, suggesting that increased CAR may be the result of training load-induced hyper-responsiveness of the HPA axis. In contrast, unchanged and decreased CAR might reflect dysregulated adaptation to training load due to a subclinical form of hypocortisolism. In this study, the HIRE session caused delayed onset muscle soreness, and a negative correlation was found between the rate of change in AUC_g and the amount of change in physical fatigue. The lack of correlation between the rate of change in AUC_g and the amount of change in muscle soreness probably reflected the results of previous studies (34, 35), showing that there were individual differences in perceived muscle soreness (subjective evaluation) and poor correlation with objective indicators. Single overload resistance exercise caused acute exhaustion and a relative decline in performance (36). Considering the association with physical fatigue, the increase and unchanged/decrease in CAR observed in this study may indicate hyper-responsiveness and dysregulation of the HPA axis, shown in previous studies (12). However, no previous study has examined the effect of a single resistance exercise session on CAR, and the current study did not examine the response of CAR to training load based on their performance. Thus, the physical adaptation or maladaptation to the training load could not be completely concluded from the response of CAR in this study.

The limitations of the present study should be noted. First, the experimental protocol that was conducted on consecutive days made it difficult to measure the control value of CAR. The experiment was conducted on consecutive days with two control sessions within individuals to examine the effect of the

HIRE sessions on CAR. During these two control sessions, several participants were not stable in CAR. It has been argued that two to six days are needed to obtain reliable trait measures since the CAR of a single day is greatly determined by situational factors and biased data from a single day (37). Some studies also measured control values according to identical regimens on two consecutive days or on separate days (38, 12). To determine reliable control values, it may be necessary to increase the number of control sessions or to measure control values on other days when the same regimen can be maintained. Second, the results were based on a single resistance exercise and did not consider their performance. The increased and unchanged CAR, which were observed in this study are similar to a previous work that they might be interpreted regarding the “prolonged response” model of allostatic loads proposed by McEwen (2). However, making such a conclusion from only a single resistance exercise and subjective assessment of physical condition would be difficult. Considering that CAR is an indicator reflecting chronic stress, the effects of prolonged exercise and training should also be examined. Finally, it would be worthwhile to examine the effects of resistance exercises that mobilized other, larger amounts of muscle mass or at different intensities based on the total work of the training.

Conclusions

The results of this study demonstrated that resistance exercises may influence the response of CAR. Although an issue of obtaining reliable control values of CAR when the experiment is conducted on consecutive days currently remains, CAR has the potential to assess physical adaptation to the local training loads (i.e., bench press exercise) if accurate control values can be obtained. This suggests that CAR may respond very sensitively to various exercises and training loads. In sum, CAR could be widely used as an indicator for monitoring the physical condition in a variety of training situations, which may help individuals involved in training plan future training loads and recovery.

Abbreviations

ANOVA
Analysis of variance
AUC_g
The area under the curve relative to the ground
AUC_i
The area under the curve relative to the increase
CAR
Cortisol awakening response
CAR_c
The magnitude of change in CAR
CAR%
The relative change of CAR
HIRE

High-intensity resistance exercise
HPA
Hypothalamic-pituitary-adrenal
MV
Movement velocity
MVT
Movement velocity threshold
1 RM
One-repetition maximum
POMS2-S
Profile of Moods States Second Edition
RPE
rating of perceived exertion
TMD
Total mood disturbance
VAS
Visual analogue scale
VO_2 max
Maximal oxygen consumption

Declarations

Ethics approval and consent to participate

This research was conducted according to the declaration of Helsinki and was approved by the Ethics Committee at Osaka University of Health and Sport Sciences (approval number: 20-19). Oral and written informed consent was obtained from all participants.

Consent for publication

Not applicable.

Availability of data and materials

The data that support the findings of this study are available from the corresponding author on reasonable request.

Competing interests

The authors, Yui Ogasawara, Takuma Yoneyama, Takeaki Ikuji, Hironobu Tsuchiya, and Takayuki Sugo, declare that they have no competing interests.

Funding

This work was supported by JSPS KAKENHI Grant Number 19K11511 (T. Sugo).

Authors' contributions

All authors contributed to the study conception and design. Data collection and analysis were performed by YO and TY. The first draft of the manuscript was written by YO and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Acknowledgements

Not applicable.

Authors' information

Not applicable.

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Figures

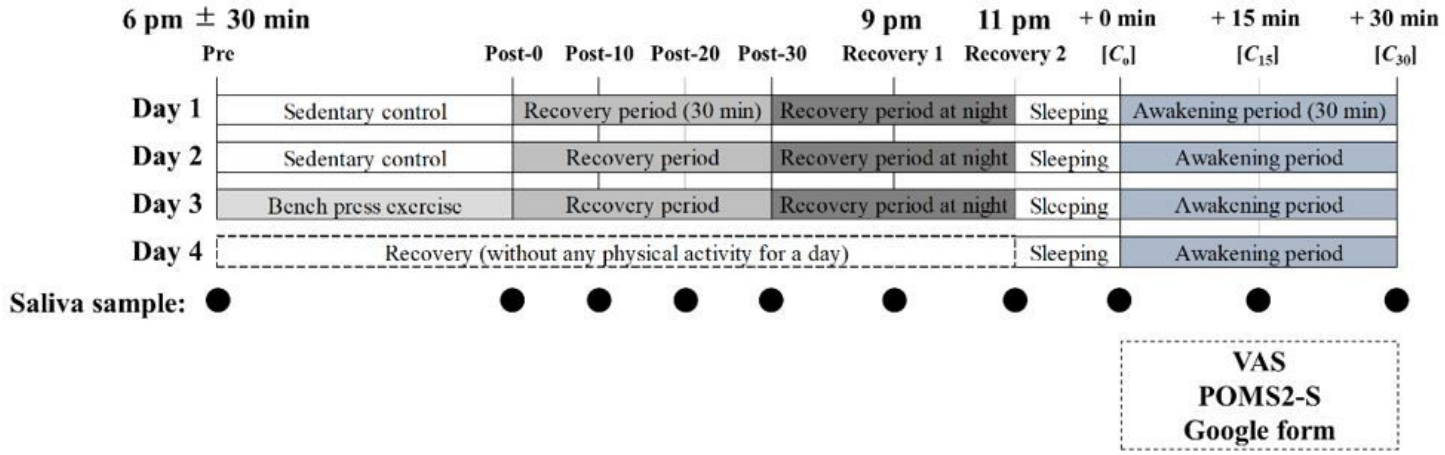

Figure 1

Study protocol

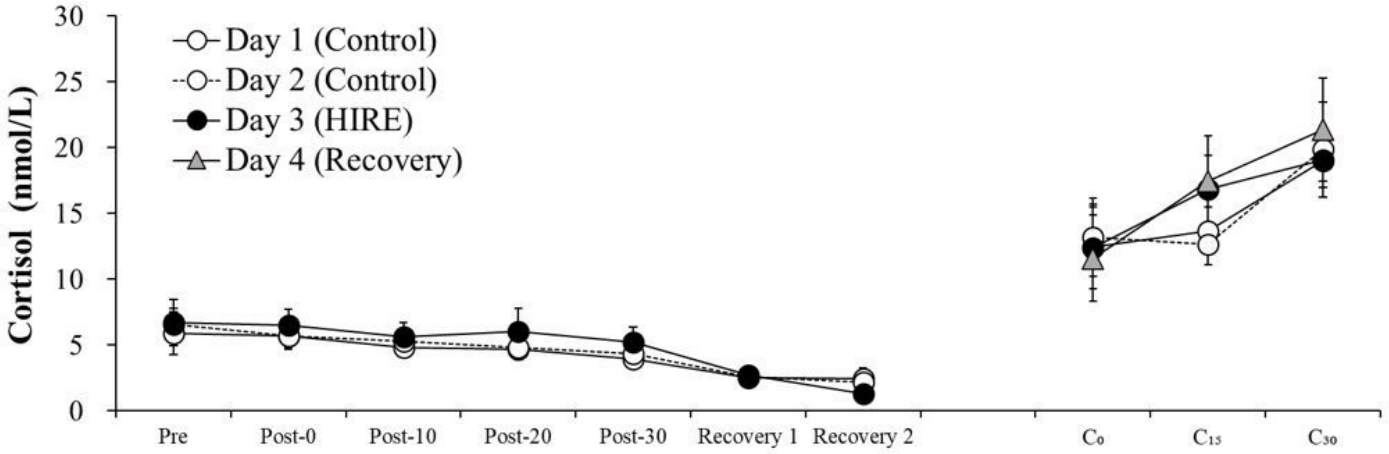

Figure 2

Cortisol data throughout the experimental session (*M ± SE*)

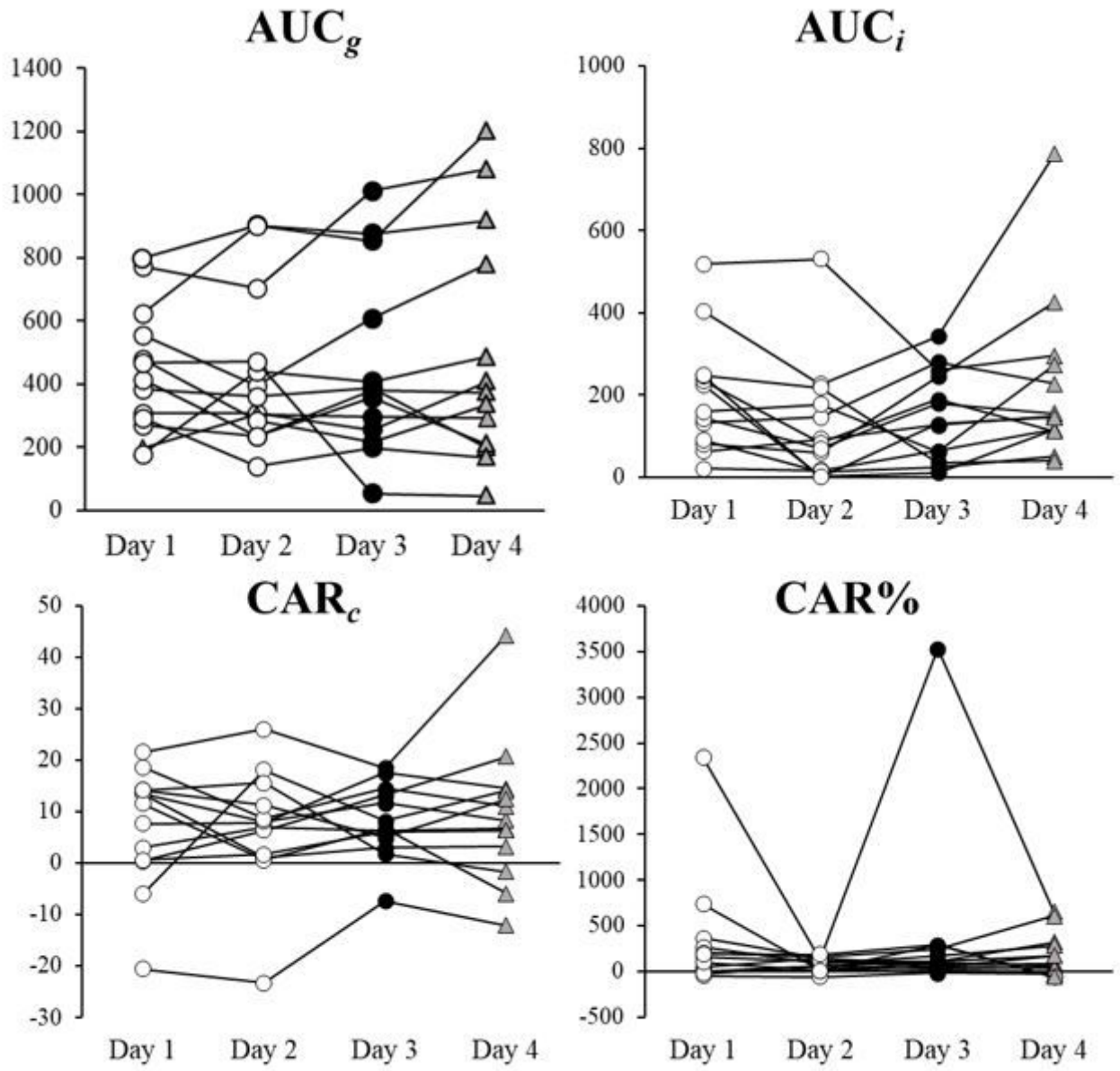

Figure 3

Individual CAR data (the area under the curve, and magnitude and relaive change of CAR)

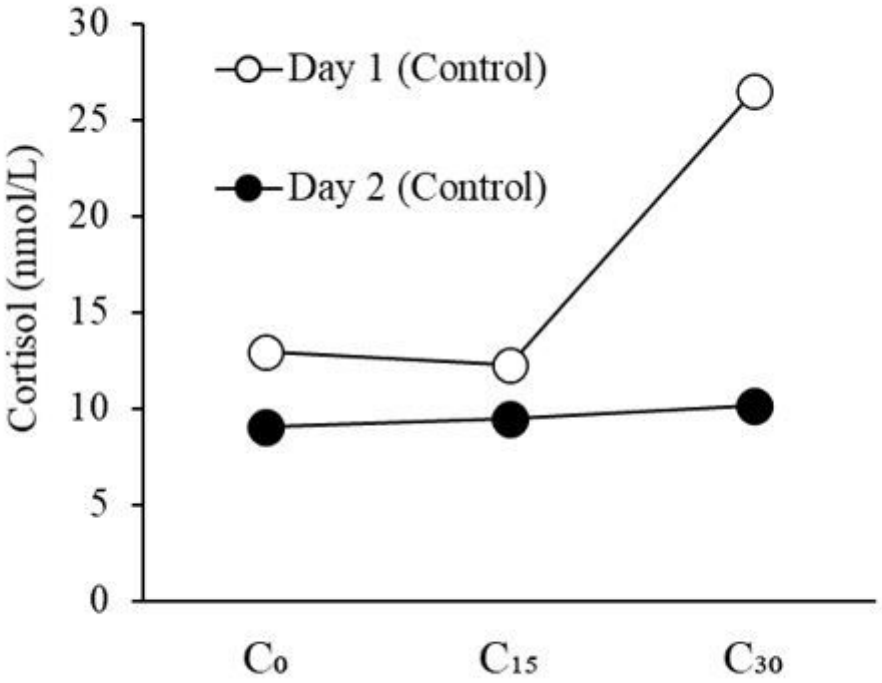

Figure 4

Screening of control values.

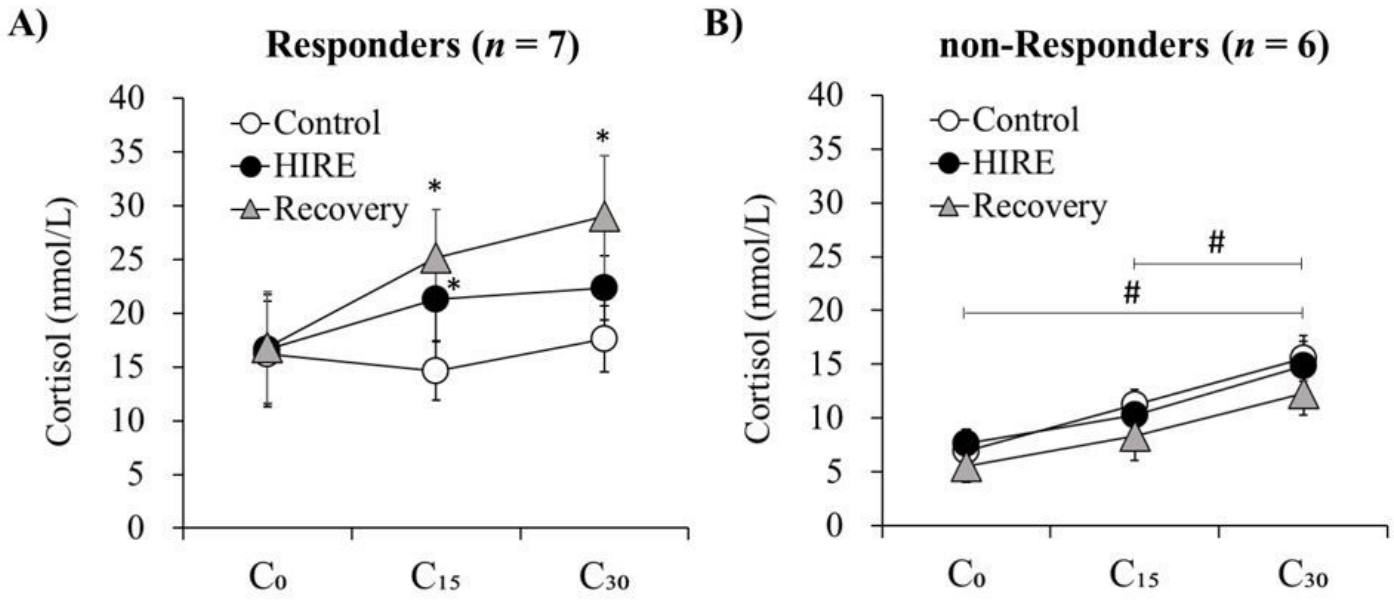

Figure 5

Mean (± *SE*) levels of CAR in A) Responders and B) non-Responders.Control data represent mean values of Day 1 and Day 2 after screening of control value.

* Significant session effect ($p < .05$, vs. Control)

Significant sampling point effect ($p < .05$)

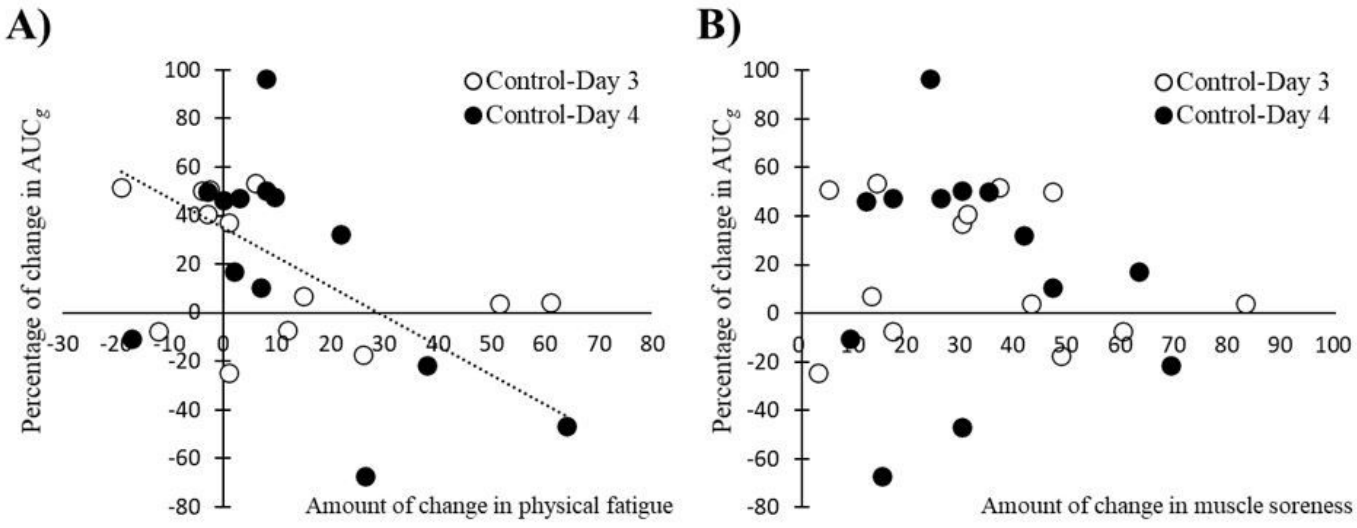

Figure 6

Relationship between percentage of change in AUC_g and the amount of change in A) physical fatigue and B) muscle soreness from Control to Day 3 and Day 4. The horizontal axis shows the amount of change in physical fatigue and muscle soreness, respectively, and the vertical axis shows the percentage of change in AUC_g.