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RESEARCH ARTICLE

ACTH AND CORTISOL RESPONSE TO SUB MAXIMAL AEROBIC EXERCISE IN NON-SMOKER AND SMOKERS

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ABSTRACT

Aims and Objectives: This study was performed to investigate the acute effect of the submaximal aerobic exercise upon adrenocorticotropic hormone and cortisol levels in chronic smokers and non-smoker.

Materials and Methods: The study was carried out upon 8 regular (15> cigarettes/day) smoker untrained male along five years and 9 never smoker untrained male. Subjects performed an endurance exercise that continues 40 minutes at 70% maximal heart rate. There were 15cc venous blood samples extracted from the forearm pre-exercise (PRE), post-exercise (POST), post-exercise 2 hours (2h), post-exercise 24 hours (24h) to measure of ACTH and cortisol levels.

Results: It is determined that in POST ACTH levels of smokers and non-smokers were shown a significant increase according to PRE levels ($p<0,05$). 2h cortisol levels of smokers and non-smokers were significantly lower than PRE levels ($p<0,05$). In comparison between-groups, all measurements (PRE, POST, 2h and 24h) indicated that differences in ACTH and cortisol levels of non-smokers were not significant ($p>0.05$).

Conclusion: The study revealed the fact that, in terms of non-smoker and smoker groups cortisol and ACTH showed similar tendency in time analysis. Smoking habit did not make a difference on the hypothalamic-pituitaryadrenal axis during exercise. The results demonstrate that it may be helpful to investigate this relationship with different types of exercises in different intensities in more controlled subsequent studies.

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INTRODUCTION

Endocrine system helps the major regulator, the nervous system, to maintain homeostasis that is more difficult in rigorous exercise. It consistently monitors the internal environment of the body and response quickly to keep homeostasis constant and inhibits dramatic disruptions. As the body transitions from resting to an active state with the increase in metabolism, fine-tuning physiological responses is the responsibility of the endocrine system which affects the body slowly and longer-lasting (Kenney *et al.*, 2015). In response to any external stimulus such as intense physical activity, smoking, climatic environment change, inflammation; these are perceived as a threat to homeostasis (stress), activation of the autonomic nervous system occurs and plasma cortisol levels increase as a result of activation of the hypothalamo pituitary–adrenal (HPA) axis (Warren and Constantini, 2000) Adrenal cortisol hormones are release due to pituitary hormonal stimulation by adrenocorticotropic hormone (ACTH) which is under influence of hypothalamic level of corticotrophin-releasing hormone (CRH). Cortisol acts at both pituitary and hypothalamic level via negative feedback to inhibit ACTH and CRH (Kraemer and Rogol, 2005). Exercise, whether it is an acute episode of strenuous exercise or chronic endurance training is also known to activate the HPA axis as well as smoking (Kraemer and Rogol, 2005). Tobacco smoking and nicotine (nicotine has endocrine-like effects on hypothalamus, postsynaptic cholinergic sites, presynaptic action on monoaminergic neurons) have pronounced effects on endocrine function (Pickworth and Fant, 1998). It activates hypothalamic-pituitaryadrenal axis (HPA), stimulates ACTH secretion and enhances ACTH effect on adrenal cortex which results in stimulation of cortisol production (Steptoe and Ussher, 2005).

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Table 1. The physical properties of smoker and non-smoker groups

	Smoker Mean ± SD	Non-smoker Mean ± SD
Age (years)	28,44 ± 3,94	29,62 ± 3,46
Body Height (cm)	175,45 ± 4,70	173,87 ± 3,96
Body Weight (kg)	73,11 ± 6,95	71,12 ± 5,61
BMI (kg/m^2)	23,17 ± 1,96	22,72 ± 2,72

*Significant difference between groups (p<0.05).

Table 2. Within-group and between-group comparisons based on alterations of cortisol and ACTH in time

	n	PRE	POST	2h	24h	time*group F	p
CORTISOL							
N-S	8	157,55 ± 29,85^a	162,89 ±47,31^a	122,38 ± 27,30^b	146,65 ± 28,82^a	0,642	0,602
Smokers	8	161,54 ± 27,06^a	149,14 ± 18,09^a	117,50 ± 18,92^b	163,20 ± 33,88^a		
ACTH							
N-S	8	23,21 ± 6,96^b	45,50 ±20,68^a	19,22 ± 8,59^b	26,04 ± 5,09^b	0,602	0,626
Smokers	8	22,26 ± 5,46^b	33,30 ± 14,83^a	21,40 ± 6,99^b	21,40 ± 6,99^b		

Within group comparison in the same row a>b N-S: non-smokers.

Among other things, this activation is associated with the lower sensitivity of nicotine receptors in central nervous system. Prolonged discontinuation of smoking leads to changes in HPA activity and a steep decrease in serum cortisol (Frederick *et al.*, 1998; Kirschbaum *et al.,* 1992) but in the short-term, smoking cessation does not correct the diminished adrenocortical responses to stress caused by chronic smoking (Berlin, 2009). Although a simple problem about the chronic effects of smoking addiction on basal cortisol levels has not been satisfactorily answered because of conflicting evidences, a study with 167 adult participants revealed that smokers (average of 12 cigarettes per day) have approximately 40% and 35% higher cortisol levels than non-smokers in work and weekend days (Steptoe and Ussher, 2005). Smoking habit, on the other hand, can affect negatively the exercise performance with different ways. Acute or chronic abstinence of smoking have important improvements on athletic performance. For example, oxygen cost of breathing decrease between %13-79 and hearth rate lower 5-7% with smoking abstinence for habitual smokers after one day (Rode and Shephard, 1971) and air resistance increases three-fold after 15 puffs (Nadel and Comroe, 1961). During graded exercise testing on treadmill seven days smoking abstinence produce significant reduction in heart rate and increase performance by the time to exhaustion (Hashizume *et al.,* 2000). In the long run, chronic cigarette smokers tend to have lower fitness levels and sedentary lifestyles than counterparts (Sidney *et al.,* 1993). Females who exercise and quit smoking made fitness improvements greater than trained but did not quit smoking counterparts (Albrecht *et al.*, 1998). Smoking enhances dependence on carbohydrate substrate for energy supply in exercise for some reason and decreases lung capacity (McArdle and Katch, 2009). All these findings reveal that while one day of abstinence decrease the cost of breathing and enhance performance (McArdle and Katch, 2009), it is well documented that there are several important negative adaptations of long-term tobacco smoking including lipid profiles, immune function, central adiposity, bone mineral density and reproductive function (Steptoe and Ussher, 2005). Because of these findings of previous studies that it is wondered whether functional handicaps arising from long-term tobacco addiction will be reflected in the level of cortisol during aerobic exercise. In this regard, it was hypothesized that the excessive physiological stress elements such as elevated hearth rate, respiration rate, air resistance, oxygen cost etc. which occurs with chronic smoking increase the cortisol level when it combined with aerobic exercise more than non-smokers.

MATERIAL AND METHODS

Eight regular smokers (28,44 ± 3,94 age, 175,45 ± 4,70 cm, 71,12 ± 5,61 kg) for five years (≥15 cigarettes per day) and nine non-smokers (29,62 ± 3,46 age, 173,87 ± 3,96 cm, 73,11 ± 6,95 kg) participated in this study. The subjects with any symptoms (hypertension, thyroid, diabetes, cardiac etc.) and those involved any training program was not included in study. The participants were given detailed information about the objectives of the study in accordance to the Helsinki Medical Declaration and they gave their full content. This study was carried out according to the approval of Selçuk University Faculty of Sports Science Non Enterprising Ethical Committee. Subjects performed an endurance exercise on treadmill (Dunlop EL900) that continues 40 minutes in 70% maximal heart rate – (approximately%55-63 MaxVO_2) (15). To verify and record the duration and intensity of exercise, participants wore a heart rate monitor (Polar RS400, Polar, Kempele, Finland). There were 15 cc venous blood samples extracted from the forearm pre-exercise (PRE), post-exercise (POST), post-exercise 2 hours (2h), post-exercise 24 hours (24h) to measure of ACTH and cortisol levels. The blood samples were first centrifuged at a rate of 5000 revolution/minute and the upper phases were transferred to eppendorf tubes and kept at -80 ^{0}C until the use. Serum cortisol and ACTH levels of all subjects were analyzed PRE-POST. ACTH and cortisol concentrations were studied with the immunochemiluminescence method using the DiaSorin Liaison autoanalyzer kit. Distributions of the variables according to the groups were analyzed and the normality of the distribution and the homogeneity of the variances were determined with the Mauchly' Sphericity Test and Levene test. Within-group and smoking-effect analysis were carried out with the repeated measures ANOVA, level of significance was accepted as 0,05.

RESULTS

In POST ACTH levels of smokers and non-smokers were shown a significant increase according to PRE levels (p<0,05). 2h cortisol levels of smokers and non-smokers were significantly lower than PRE levels (p<0,05). In comparison

between-groups, all measurements (PRE, POST, 2h and 24h) indicated that differences in ACTH and cortisol levels of non-smokers were not significant (p>0.05) (Table 2 – Figure 1).

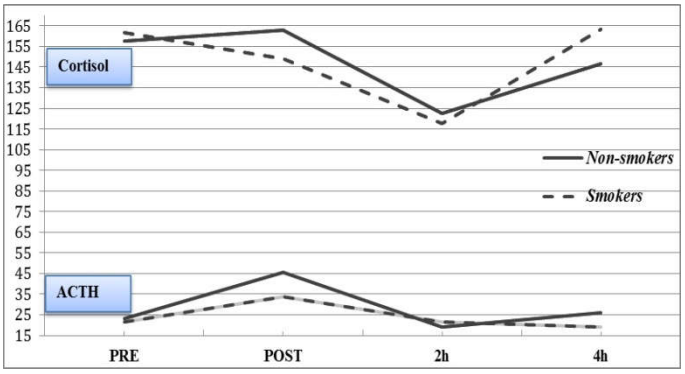

Figure 1. Alterations of cortisol and ACTH in times

Conclusion

ACTH increased immediately after exercise and in the following two measurements returned to the baseline for both groups. The increase in cortisol after exercise was seen in non-smokers and it was not observed in smokers but this difference was not reflected in statistical analysis. After 2h exercise, cortisol decreased for both groups and after 24h, it returned to baseline. Although there was no difference between smokers and non-smokers in terms of time*group analysis, the difference immediately after exercise measurements appears to be the highest among four between group comparisons for each times. This may suggest that exercise may cause some different responses in ACTH and cortisol between smoker and non-smokers, depending on exercise intensity and load. In this study, it was aimed to implement the duration and intensity of exercise in a borderline intensity and load based on the findings of previous studies. In this respect, while it was aimed to maintain the exercise for 40 minutes close to the anaerobic threshold pace. But findings in previous studies about the intensity of exercise required to stimulate the HPA axis reveal different results. Both acute high intensity exercise (Witten *et al.,* 1991) and prolonged submaximal exercise (Inder *et al.,* 1998) are associated with increases in plasma ACTH and cortisol levels. High-intensity exercise favors arginine vasopressin release and that prolonged duration favors CRH release, which is the mechanism of ACTH release occurs in exercise (Inder *et al.*, 1998). In Inders' study exercise intensity was the same (70% VO2max) but the duration was 20 min longer than present study. In another study with a mean HR of 153 HR and 60% VO2max, ACTH significantly increased from 12,3 to 20,1 (Hill *et al.,* 2008). This may explain why the ACTH increased but that did not reflect cortisol levels immediately after exercise.

Besides ACTH triggered increase in cortisol cannot be expected until after a certain time-lag due to the on of the adrenocortical enzymes (Schwarz and Kindermann, 1990). With the incremental graded exercise (step test), although the exercise continued 10 min more after anaerobic threshold (occurred at 200-250 watt) cortisol levels did not change immediately after exercise compared to basal levels. The considerable change occurred after 10 min measurements. In another study, similar to this, ACTH increased with the end of mean 21 minute exercise (Schwarz and Kindermann, 1990). This increment defined by mostly anaerobic components of physical exercise which formed when the anaerobic threshold had been exceeded or at the point of an over proportionate increase in lactate concentration. Some studies where participants have exercised below anaerobic threshold does not result in activation of the HPA (Kindermann *et al.*, 1982; Schwarz and Kindermann, 1990). In prolonged exercise, the activation of HPA is mostly dependent on carbohydrate deficit (Utter *et al.*, 1999), before exercise energy sources and diet (Langfort *et al.*, 1996), maintenance of glucose homeostasis (Kraemer and Rogol, 2005) and lactic acid response. 3h cycling at 50% VO2max elicited an increase in ACTH and cortisol in the latter part with negative correlation of blood glucose (Tabata *et al.*, 1990). In the present study, the duration of the exercise was 40 minutes or until exhaustion with the intensity of 70% HRmax which was hard to improve a disruption on glucose homeostasis in normal nutritional condition. The intensity was almost equal to 65-70% of VO2max for untrained subjects (Pollock *et al.,* 1998). This intensity may be sufficient to activate HPA axis as seen in previous studies (Inder *et al.,* 1998; Luger *et al.*, 1987). Some studies have revealed that the intensity below 60% (Davies and Few, 1973; Hill *et al.*, 2008) or 70% of VO2max does not induce familiar rise over baseline (Inder *et al.*, 1998; Jacks *et al.,* 2002; Luger *et al.,* 1987). These findings explain the rise of ACTH at the end of running in the present study. The decline of cortisol after exercise was revealed at the end of submaximal (Scerbo *et al.,* 2016) and maximal intensity efforts (Few, 1974) as it is seen 2h after measuring in present study. In the other hand, normalization of plasma levels after endurance exercise may take up to 18-24h (Lutoslawska *et al.*, 1991)which explains the return to baseline for both hormones after 24h.

Research involving the effects of cigarette smoking on exercise has primarily focused on the compounds nicotine and carbon monoxide. It has been demonstrated that each of these compounds has independent effects on the body that can influence the responses of the body to exercise (Hoyt, 2013). Cigarette smoking is known to activate the sympathoadrenal system, increasing heart rate, blood pressure, and circulating levels of catecholamines and corticosteroids. Scerbo et al. (2010), reported that the normal cortisol decrease during abstinence from cigarettes was attenuated by a 15-min bout of vigorous intensity running, for up to 30 min post-exercise, and Ho (2009) found that plasma adrenocorticotropic hormone (ACTH), serum cortisol, heart rate, and systolic blood pressure were all elevated post-resistance exercise when compared to both a passive condition during abstinence and in an ad libitum smoking condition. There is no experiment investigating the cortisol, ACTH alterations and HPA axis activation associated with physical activity/exercise in smokers and non-smokers. In this study pre-exercise and 24h post-exercise cortisol amounts seem to have a higher in smokers than the counterparts. But this difference is not statistically significant for any between-group comparison, for any hormones and any times. Furthermore, time*group comparison analysis in Table 2 reveal that there is no difference in cortisol and ACTH alterations in time in terms of smokers and non-smokers. While it was hypothesized before the experiment that the response to exercise-induced stress was mediated by smoking-induced stress would stimulate higher and earlier HPA response in smokers; all measurements in our study show the indifference between smokers and non-smokers during exercise and basal conditions. This unexpected result has emerged in previous evidences which has shown that cortisol

and ACTH levels for habitual smokers is contradictive so far (Steptoe and Ussher, 2005). Some of them showed that cortisol output was elevated in smokers (Baron *et al.*, 1995; Field *et al.,* 1994)and no difference in others (Gossain *et al.*, 1986) as it is in this study. As we know, ACTH and cortisol profile is depending on lots of factors such as day-time, nutrition, gender, age, training status, smoking, metabolic rate etc. In this respect, this uncontrolled nature of the endocrinal experiments may explain the complicated and different results. As a result, the hypothesis that cortisol and ACTH response in smokers are more prominent during exercise was rejected. It may be helpful to investigate this relationship with different types of exercises in different intensities in more controlled subsequent studies.

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