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Mini-Review: Effect of exercise on cortisol synthesis, release, metabolism, and clearance

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Abstract

Cortisol is a glucocorticoid, synthesized in response to the action of adrenocorticotropic releasing hormone (ACTH) on adrenal glands (AG) and released from zona fasciculata. Increased cortisol is associated with psychological and physical stress. Several strategies have been used in the past to overcome stress in an athletic and healthy population. Among these strategies, exercise has been shown to beneficial for the management of stress. However, it is unknown if these benefits are due to the direct effect of exercise on cortisol levels. In this brief review, we have outlined the benefits of exercise and how exercise in previous literature has shown to affect cortisol levels in two ways. First, by directly activating the hypothalamus-pituitary axis (HPA) and second, by releasing ACTH which acts on AG and stimulates the release of cortisol. This mini-review will discuss the effects of type of exercise (aerobic and resistance) on cortisol synthesis and release, metabolism, and clearance.

Keywords: Cortisol, Exercise, Stress, Glucocorticoid, Hormone

1. Introduction

Cortisol is a glucocorticoid synthesized from cholesterol after a series of reactions in the adrenal glands (AG) (REF). Cortisol has been known to play an important role in stress, metabolism, and immune response (REF). In the presence of any form of stress, the hypothalamus releases a corticotrophin-releasing hormone (CRH) which acts on the pituitary gland and causes the release of an adrenocorticotropic releasing hormone (ACTH) from the anterior lobe (REF). Plasma ACTH acts on the adrenal cortex and causes the release of cortisol from the zona fasciculata region of the AG. Once inside the cell, cortisol binds to the glucocorticoid receptor (GR), attached to heat shock proteins (HSP), in the cytosol. The binding of cortisol to GR results in the dimerization of GR and separation of HSP. The Hormone-receptor complex (HRC) then translocates into the nucleus resulting in gene expression (Figure 1) and its other functions (REF). Cortisol is then metabolized into 5α-tetrahydrocortisol (5α-THF) and 5β-tetrahydrocortisol (5β-THF) in the liver and a proportion of cortisol is transported in the kidney where it is either excreted as urinary free cortisol (UFC) or is converted into cortisone (an inactive form of cortisol) in the presence of 11β-hydroxysteroid dehydrogenase 2 (11β-HSD2) enzyme (REF). Cortisone is then metabolized into 5β-tetrahydrocortisone (5β-THE) and excreted in urine or transported back to the liver where it is converted back into an active form, cortisol, in the presence of 11β-HSD1 (Figure 2) (REF). Maintaining normal cortisol levels are critical for normal metabolism, immunity, and stress maintenance. Chronic production of cortisol in excess levels (also reported in Cushing's syndrome patients) induces diseases like type II diabetes and obesity (1), and cardiovascular diseases (2,3). Conversely, low cortisol levels for a longer period of time (a condition called Addison's disease) also increases the risk of hypoglycemia (4), hypotension (5), and cardiovascular diseases (6).

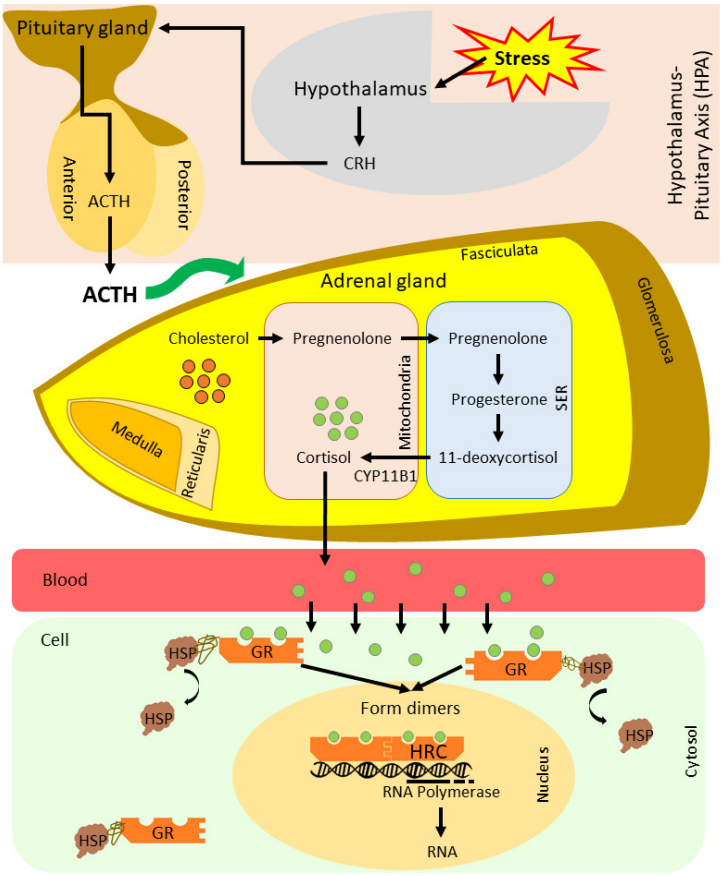

Figure 1: Synthesis, release, and gene expression of cortisol

Management through pharmacological intervention has shown to improve cortisol regulation in hypocortisolism (7) and hypercortisolism (8), however, these drugs primarily do not regulate cortisol action but rather treat comorbidities associated with hypo- and hypercortisolism. Exercise has been shown to affect cortisol levels by directly activating the hypothalamus-pituitary axis (HPA) and releasing ACTH which acts on AG and stimulates the release of cortisol (9). Exercises in different forms, types, and intensities have been shown to affect cortisol release differently (10). However, the effect of exercise at an individual step in the mechanism of cortisol synthesis, release, and clearance has not been discussed in the previous literature. The purpose of this paper is to

discuss the effect of different types/forms and intensities of exercises on different intermediate products in the mechanism of cortisol synthesis, release and clearance.

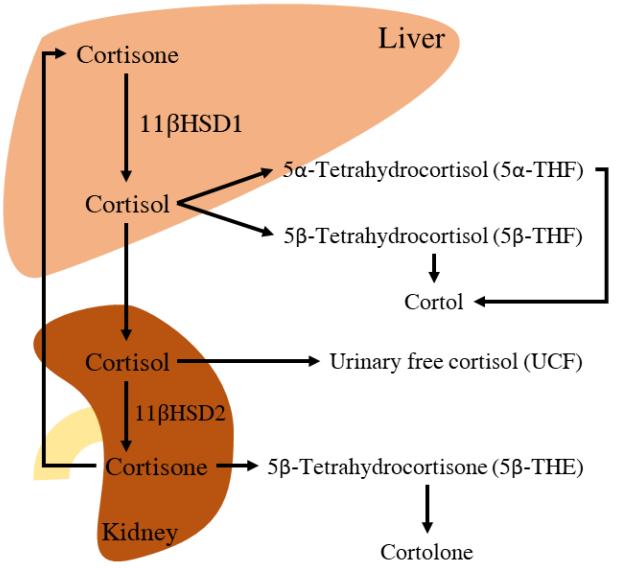

Figure 2. Cortisol metabolism and excretion

2. Effect of exercise on synthesis and release of cortisol

As summarized in figure 3, unlike other hormones, cortisol is not stored in the cells, rather its release is dependent on the rate of synthesis. Therefore, it is challenging to measure the synthesis and release of cortisol separately. However, as shown in figure 1, ß-hydroxylase (steroidogenic enzyme), encoded by CYP11B1 converts 11-deoxycortisol to cortisol and is a marker of cortisol synthesis.

Aerobic exercise:

Acute effects: When the effect of short-term aerobic exercises was observed on the release of cortisol, by measuring plasma cortisol levels, dose-dependent responses were observed (10). However, at least a certain threshold (e.g. greater than 60%) of exercises appeared to be needed to stimulate the release of cortisol (10,15,16) or longer duration exercises (more than 80 minutes or 120 minutes running) at low or moderate intensity exercise (55% VO_{2max}) were needed to increase the

release of cortisol (17). Moreover, cortisol release in response to threshold less than the aforementioned threshold was lower or disappeared with endurance training (15). This may be because, at lower threshold/intensities, clearance of cortisol (discussed in clearance session of this paper) may dominate synthesis and release (18). Once the threshold was at supramaximal levels (15,19) or above 65% VO_{2max} (20), cortisol release was greater in endurance-trained athletes and remained higher even during the recovery period (20).

Chronic effects: Limited studies reported the effects of exercise on mRNA expression of CYP11B1. Two weeks of voluntary running did not change CYP11B1 levels in male mice when measured at 07:30, 15:00, and 21:30 hours (11). However, male mice running voluntarily for 30 days on a wheel exhibited higher levels of CYP11B1 mRNA expression compared to sedentary mice (12). These effects were not consistent with female rats when exercised on a treadmill for 30 minutes/day for 5 days/week for 4 weeks i.e., CYP11B1 mRNA expression was not influenced by treadmill exercise in female rats (13) suggesting there may be a sex effect in cortisol synthesis in response to exercise. Interestingly, in humans, when genetic screening was performed in response to exercise training at 50% or 80% maximal oxygen consumption for blood pressure regulation, CYP11B1 mutation was linked in both white and black populations (14). Interestingly, with prolonged aerobic/endurance training, cortisol release was variable (21,22) with different forms of exercises, however, the majority of the studies reported a consistent increase in cortisol release in response to aerobic or power exercises (23) and submaximal and exhaustive exercises (22). However, an increase in cortisol release in response to exercise training appeared to be adaptable in trained individuals, i.e. long-term exercises or overtraining exhibited lower cortisol release during exercise (24). This is perhaps because individuals who are well trained does not activate the HPA axis, to an extent untrained individuals activate it, in repose to a bout of exercise (2-hour) and stress test, and therefore release cortisol in lower concentrations compared to untrained individuals (25).

Resistance training:

Acute effects: When the effect of resistance training was observed on cortisol release, inconsistent effects were observed with different exercise types/forms. Post-exercise increase in cortisol release was observed when a single bout (26,27) or 2 hours (28) resistance training was performed and remained elevated for 15 (29), 30 (27) and 60 minutes (28). However, cortisol release was affected when a bout of resistance training was performed by untrained individuals with no-rest or 30-seconds rest in between sets at 70-75% (30) or 80% 1RM until exhaust (31) suggesting cortisol release depends on exercise intensity and recovery period (32) both in men and women (33).

Chronic effects: With long-term exercise training, cortisol release varied with different protocols, 12 weeks of resistance training lowered cortisol release in overweight/obese individuals (34), and three weeks of training session caused an increase in cortisol release in strength athletes (35). However, the majority of other studies reported either no changes or decreased cortisol release following resistance training (33). This may be in part due to the duration of exercise training. The studies that observed no changes in the cortisol release were usually between 6 months to 2 years long of resistance training or follow-up (36-44) and studies that observed reduction in cortisol release were the studies that measured cortisol after 8-24 weeks of resistance training (38, 45-48). Suggesting cortisol release is increased in the early response to resistance training i.e. within the first three weeks, whereas between 8-24 weeks cortisol release appears to be falling which may be due to increased lean mass in response to resistance training (49). Once exercise training is performed for longer than 6 months, adaptation appears to occur (similar to aerobic exercise) and no changes are observed in cortisol release.

3. Effect of exercise on metabolism and clearance of cortisol

Metabolism and clearance of cortisol are vital components of cortisol regulation. Cortisol is metabolized into 5α-THF, 5β-THF, UCF, and THE in the liver and kidneys before it is excreted in the urine, sweat, and feces. Additionally, 11β-HSD2 deactivate cortisol to cortisone. Therefore, the measurement of the aforementioned metabolites can be

used to assess the clearance rate of cortisol. The majority of illness occurs not due to increase HPA induced cortisol synthesis and release but due to reduced metabolism by suppression of 11β-HSD2, preventing inactivation of cortisol to cortisone, 5α-THF, 5β-THF, and 5β-THE suggesting reduced metabolism and clearance of cortisol (50). Figure 3 summarizes these findings.

Aerobic exercise

Acute effect: Interestingly, when about (30 minutes) of aerobic exercise was performed by healthy male individuals at high intensity, until exhaustion, within 5 minutes of completion of the exercise, ACTH, cortisol, cortisone, corticosterone, and THF (combined 5α-THF and 5β-THF) were increased significantly (51) indicating, increased HPA cause increase in ACTH which acted on AG to induce cortisol production. However, cortisol was also metabolized instantaneously as evidenced by increased THF, suggesting one bout of exercise increased cortisol synthesis and metabolism. Additionally, 30 minutes after exercise, THF remained higher, and THE, metabolites of THF and THE (controls and cortolones) were also increased despite higher cortisol and cortisone levels, suggesting an increased rate of metabolism and clearance even after 30 minutes of exercise (51). No studies tested the chronic effects of aerobic exercise on cortisol metabolism and clearance.

Resistance training

Acute effects: In contrast, when cortisol and metabolites of cortisol were measured immediately after strength and endurance training, higher UCF and urinary 11β-HSD1 (responsible for conversion of cortisone to cortisol) levels were observed. Additionally, post-strength training, THE values were also increased after 48 hours post-exercise (52) suggesting increased cortisone metabolism. However, in untrained individuals only one study reported that one bout of eccentric/concentric exercise did not change the levels of cortisol metabolites, cortisone and their metabolites (54). These data suggest that the resistance trainng may effect cortisol metabolism at least after 48 hours.

Chronic effects: In untrained individuals, with 4 weeks of strength training (53) no changes were observed in the levels of cortisol metabolites, cortisone, or their metabolites in untrained males.

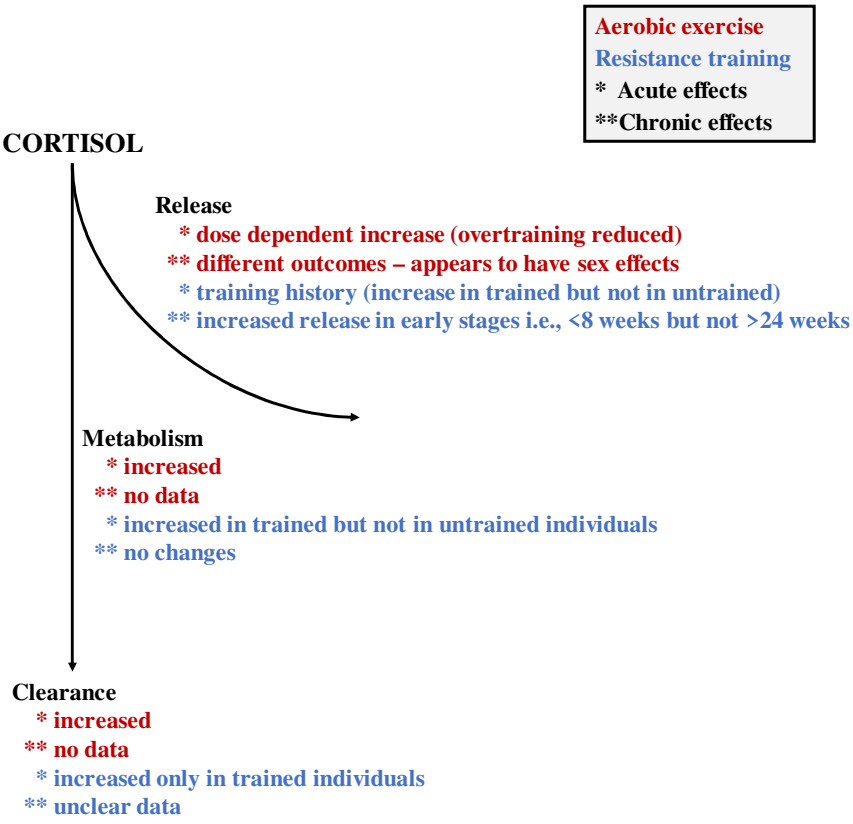

Figure 3: Summary of findings

In summary, as summarized in figure 3, cortisol synthesis increased with 30 days of voluntary running in male mice while in female rats, this effect was not observed. Mutation of the CYP11B1 enzyme was linked with individuals performing 50% or 80% $VO_{2max}$of exercise. Aerobic exercise exhibited a dose-response relationship with cortisol release but overtraining resulted in lower cortisol release. Resistance training exhibited mixed outcomes depending on duration and type of training. One bout of resistance exercise appeared to increase cortisol release, however, this effect was diminished when exercise was performed at

higher-intensities (70-80% 1 RM). While 8-24 weeks of resistance training reduced cortisol release, longer studies between 6 months to 2 years reported no changes. Lastly, the effect of exercise on metabolism and clearance is observed to be mixed. One bout of aerobic exercise exhibited increased metabolism and clearance rate of cortisol as evidenced by increased metabolites in plasma and urine and remained higher 30 minutes post-exercise. However, strength training and endurance training appear to have opposite effects.

Text in red denotes effects related to aerobic exercise. Text in blue denotes effects related to resistance training. One * denotes acute effects and two ** denotes chronic effects. Color of the * denotes type of exercise.

4. Conclusion

In conclusion, aerobic exercise appears to lower cortisol synthesis and release and increase its metabolism whereas resistance exercise appears to show mixed results. Even though several studies have been conducted on measuring the effects of exercise, from 30 minutes to 2 years, on cortisol release, limited studies are available to establish a relationship between the effect of exercise on metabolism and clearance of cortisol. Therefore, further studies are warranted in measuring the effects of exercise on the metabolites of cortisol.

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