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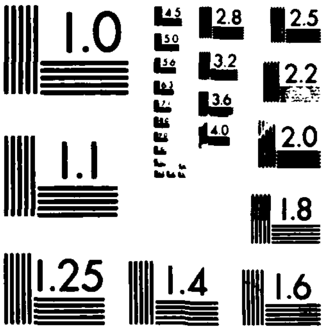

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ENDOCRINOLOGICAL RESPONSES
TO EXERCISE IN STRESSFUL ENVIRONMENTS

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INTRODUCTION

The metabolic, thermoregulatory, and fluid-regulatory adjustments which occur during exercise, even under relatively moderate environmental conditions, may be concomitant with endocrine and neuroendocrine responses involving the hypothalamus, pituitary, adrenal, thyroid, sex glands, and pancreas. Reviews of studies investigating these relationships have been published previously ~~(22,66,87,153)~~; the imposition of an environmental stress in the form of heat, cold, or high terrestrial altitude in many cases exacerbates the intensity of these endocrinological responses in man and higher animals. The responsivity and lability of these hormonal adjustments, the availability and accessibility of the biological medium in man (plasma, serum, urine), and the recent development of specific quantitative techniques for micro-assay (high-pressure liquid chromatography, radioimmunoassay) have combined to produce numerous reports on the human endocrine/neuroendocrine response to exercise during heat, cold, or hypoxic stress.

Environmental Stress

As early as 1968 Collins and Weiner (22) reviewed the effects of heat exposure on endocrinological responses, and concluded that thyroid hormones, corticosteroids, mineralocorticoids, and antidiuretic hormone levels were all affected in man by sedentary exposure to heat stress. Collins (20) later reviewed data indicating that acute exposure to cold stress in man resulted in elevated norepinephrine excretion. Budd and Warhaft (13) reported that when test subjects were challenged by a cold stress test following nearly 6 months residence in Antarctica, the prior cold exposure resulted in elevated urinary 17-hydroxycorticosteroids and 17-ketosteroids, but urinary norepinephrine and epinephrine increments were similar to those recorded in midsummer in Australia. Eastman et al. (36) reported increments in circulating levels of triiodothyronine (T_3) and thyroxin (T_4) upon exposure of 4 lightly clad test subjects to an ambient temperature of 6°C for 4 days.

Analogously, acute exposure to high altitude hypobaric hypoxia has been often reported to stimulate the activity of the sympathoadrenocortical axis. Mackinnon et al. (99) reported that within 24 hours of exposure to an altitude of 4300 m, urinary 17-hydroxycorticosteroids were significantly elevated with an apparent return toward baseline levels by 5 days of exposure. Similar results were reported by Moncloa et al. (109) who also observed a transient (4-7 d) elevation in urinary 17-hydroxycorticosteroids upon translocation of sea level natives to an altitude of 4300 m. During successive sojourns to altitude (3800 m) Timiras et al. (146) had earlier noted not only an attenuating trend of 17-hydroxycorticosteroids after several days at altitude, but also a reduced magnitude of response during consecutive altitude sojourns. Later, Becker and co-workers (5,6,30) described an elevated epinephrine and hydroxymethoxymandelic acid excretion upon exposure to a simulated altitude of 4000 m; these authors also discussed other factors which might affect endocrine/neuroendocrine responses to environmental stressors: physical activity, confinement, discomfort, affect state. We later reported (48) that elevated levels of plasma cortisol and urinary 17-hydroxycorticosteroids prior to simulated high altitude exposure can significantly modulate the response elicited by the environmental stressor. In an attempt to explain the electrolyte and water fluxes which occur upon acute exposure to hypobaric hypoxia, Hogan et al. (73) reported decreased plasma renin activity and urinary aldosterone excretion after exposure of test subjects to 3660 m simulated altitude. Apparently, early studies which examined the effects of high altitude on human endocrinological responses concentrated on the sympathicoadrenal axis.

EXERCISE AND TRAINING

As early as 1952 von Euler and Hellner (152) reported that not only is physical exercise accompanied by an increase in the urinary excretion of epinephrine and norepinephrine, but also that the excretion rate may be correlated with the work intensity. Gray and Beetham (65) demonstrated that plasma norepinephrine levels were markedly elevated immediately after acute exhaustive work and returned to normal levels within 15-30 min of exercise completion while Peronnet et al. (116) reported that both epinephrine and norepinephrine increased significantly after several intensities of exercise. Further von Euler (151) attributed the increased catecholamine secretion during exercise to the need for fuel mobilization as well as the homeostatic maintenance of blood pressure by vasoconstriction in non-active areas to compensate for the hyperemia and vasodilation in actively contracting muscle groups.

The issue of hormonal responses to exercise prior and subsequent to a training program has been addressed in experimental subjects ranging from rats to humans. Although most of the relevant studies did not include the imposition of an environmental paradigm, a brief summation of important observations may be appropriate for this review. Tharp and Buuck (143) used *in vitro* techniques to demonstrate that the adrenal glands from rats which had been trained for 8 weeks responded to ACTH stimulation with less corticosterone production than glands removed from non-trained control animals. Winder et al. (161) and Ostman and Sjostrand (114) demonstrated also in rats that, following training, stress-induced or exercise-induced increments in urinary norepinephrine as well as plasma norepinephrine and glucagon were attenuated in the trained groups. In humans various investigators have reported that exercise-induced increments in norepinephrine (69), glucagon and catecholamines (162), and plasma renin activity (62) were moderated during exercise subsequent to physical training. Our own work (52,53) has indicated that heat acclimation can modulate the responses of stress hormones and fluid regulatory hormones to a heat stress test.

Karagiorgios et al. (84) observed that both continuous and intermittent exercise elicited significant elevations in plasma growth hormone levels in moderately fit volunteers. Johnson et al. (82) investigated the effects of training on hormonal responses, and reported that in racing cyclists elevations of growth hormone and catecholamines were attenuated while insulin, frequently reported to decrease during muscular activity (19,79,89), fell less during exercise in this group in comparison with a less fit control group. Concurrent with decrements in plasma insulin levels during exercise are apparent elevations in circulating levels of glucagon (9,40). Analogously, Sutton (138) reported that unfit subjects were characterized by a greater elevation of growth hormone and cortisol than fit subjects when all subjects exercised at a fixed rate. Cronan and Howley (29) were unable to demonstrate any effects of training on norepinephrine and epinephrine excretion, but Howley (77) later reported a clear correlation between norepinephrine excretion and work load with less consistent responses for epinephrine. Daniels and Chosy (32) had earlier imposed a moderate altitude stress (2200 m) on fit athletes during a training program and observed no changes in epinephrine excretion and a moderate increase in urinary norepinephrine by 2 days of altitude exposure which persisted through 3 weeks at altitude. Later, White et al. (156) reported that the physiological strain of increasingly intense exercise, as manifested in serum corticosteroid levels, can be attenuated by increased physical fitness.

In female test subjects Boyden et al. (11) reported that increasing the weekly training distance from 21.7 km to 48.3 km effected a decrease in circulating triiodothyronine (T_3), reverse T_3, and a greater thyroid stimulating hormone response to thyroid releasing hormone treatment. However, all of these apparent alterations were reversed when weekly training distances increased from 48 km to 80 km (10). Irvine (80) reported that physical training significantly increased the turnover of circulating thyroxin (T_4), and Balsam and Leppo (4) noted differential effects of training on the turnover of T_4 and T_3. As early as 1954 Lashof et al. (95) had reported no effects of moderate or intense exercise on the concentration or clearance of T_4. Later DeNayer et al. (34) demonstrated a slight, but significant, decrement in free T_4 30 min after strenuous exercise while Terjung and Tipton (142) concluded that moderate (61% $\dot{V}O_2$ max) exercise resulted in an increasing trend of free T_4 which failed to achieve statistical significance. Thus, it appears likely that the intensity of either the exercise or the training regimen may affect the thyroidal response to exercise under normal environmental conditions.

The effects of exercise, fitness, and training regimens on the response of circulating testicular hormones have also been evaluated. For example, in highly trained swimmers and rowers Sutton et al. (139) reported increments in serum androgens (predominantly testosterone) during intense exercise. While Galbo et al. (58) observed slightly increased levels of testosterone with increased $\dot{V}O_2$, they demonstrated much larger increments 40 min after running at 75% $\dot{V}O_2$ max which, interestingly, declined after 60 and 80 min of exercise consonant with the decrements observed by Dessypris et al. (35) subsequent to a marathon run. Wilkerson et al. (157) exercised men for 20 min at 5 intensities up to 90% $\dot{V}O_2$ max; they reported that apparent increments in plasma testosterone concentrations were negated when decreases in plasma volume were considered. The latter results may be compatible with those of Galbo et al. (58) which indicated the requirement for more prolonged exercise to elicit a true testosterone effect, but Wilkerson et al. (157) concluded that the effects of exercise duration and intensity, as well as physical training, are less than clear.

ALTITUDE AND EXERCISE

The environmental and endocrinological literature contains a plethora of reports describing the sympathicoadrenal response to real or simulated high altitude exposure (67,74,94,98,99,108,109,146). As early as the 1940's several groups of investigators (91,145) reported the effects of adrenocortical extracts on the ability of small animals to withstand extreme hypoxia. Roosevelt et al. (125) updated these earlier studies and reported that sham-adenalectomized and sham-hypophysectomized rats survived extreme hypoxia significantly longer than adenalectomized and hypophysectomized animals. Further, they reported (125) that cortisol administration to adrenalectomized animals attenuated the increases in circulating lactate levels induced by hypoxia. The latter observation could have practical application to exercise endurance during altitude exposure, but subsequent studies have apparently not been performed.

INSERT FIGURE 1

Moreover, the application of exercise or work stress supplemental to exposure to altitude or simulated hypobaric hypoxia has been reported to induce a multiplicity of hormonal responses, many of which are associated with the stimulated activity of the hypothalamic-pituitary-adrenal axis. However, Moncloa et al. (108) examined adrenal function during exercise after acute exposure to high altitude, and reported a significant decrement in plasma cortisol levels after exercise. They attributed this observation to an increased hepatic blood flow and clearance of cortisol during high altitude exercise. In their report on the ascent of Mt. Paril, Guilland et al. (67) also reported a significantly increased clearance and catabolism of cortisol with exercise at high altitude. Alternatively, Humpeler et al. (78) reported progressively increasing levels of cortisol upon 10 days exposure to 2850 m when test subjects walked an average of 12.7 $km.day^{-1}$; however, it should be noted that blood was taken for these measurements before the initiation of exercise. In a more recent study Maresh et al. (103) examined the adrenal responses of low altitude natives (373 m or less) and moderate altitude natives (1800-2200 m) to maximal bicycle ergometry at a simulated altitude of 4270 m. These investigators reported (103) that serum cortisol levels were increased following exercise in both groups of test subjects; however, in keeping with the traditional difficulty of comparing results among experiments, it must be noted that the exercise regimen imposed in the latter experiments was a maximal test of very short duration.

Maresh et al. (103) also reported that exercise at either low, moderate, or high altitude induced significant elevations of plasma aldosterone levels, but levels both pre- and post-exercise were moderated during high altitude exposure. Earlier, Maher et al. (102) had reported that increasing intensity of exercise at 4300 m was accompanied by an increase in circulating levels of aldosterone. Further, they observed that aldosterone levels were reduced by acute (14 hours) exposure to high altitude, and that with increasing sojourn (11 days) at high altitude, levels recovered toward those recorded at sea level. At a lower altitude (2040 m) Humpeler et al. (78) did not observe this acute decrement in aldosterone levels. Milledge et al. (107) studied subjects who exercised (hill walking) for 6-7 hours each day at 3100 m, and reported that aldosterone levels were significantly elevated immediately after exercise, but this increment was attenuated over the 5 days of altitude exposure. Maher et al. (102) acknowledged the apparent dichotomy of decreased aldosterone levels at a time of apparent contracture of plasma volume upon acute exposure to high altitude; they attributed this response to a possible peripheral venoconstriction, thus decreased vascular capacity, which could be sensed as an increase in relative blood volume. Hogan et al. (73) had previously speculated that decreased aldosterone secretion at moderate altitudes is a direct result of diminished plasma renin activity.

INSERT FIGURE 2

Generally, investigators have examined the renin-angiotensin-aldosterone systems simultaneously, and have observed close correlative responses between angiotensin I levels and aldosterone (122). Angiotensin I levels are ordinarily estimated from plasma renin activity following appropriate incubation periods during which angiotensin converting enzyme is inhibited. Thus, Milledge et al. (107) reported increments in plasma renin activity following exercise at altitude; like aldosterone responses, these increments moderated with increasing time of altitude exposure. The data of Maher et al. (102) were essentially identical to those which they noted for aldosterone: plasma renin activity was reduced by acute altitude exposure and increased during exercise with chronicity of exposure. Alternatively, Humpeler et al. (78) reported that plasma renin activity was unaffected by exposure to moderate altitude although the effects of exercise could not be directly assessed because blood was drawn after an overnight fast and 2 hours of supine bed rest. Thus, while aldosterone and plasma renin activity levels may be reduced or not greatly affected by altitude exposure, they are both increased by exercise under either sea level or altitude conditions; further, the chronicity of the altitude exposure and the intensity of the exercise may both affect these responses.

In his report on the scientific and medical aspects of the Australian Andean expedition, Sutton (136) described the effects of exercise and altitude acclimatization on circulating levels of insulin, glucagon, cortisol, thyroxin, and growth hormone. Of these variables the "most interesting finding" was the response of plasma growth hormone levels to exercise at sea level, during acute exposure to simulated altitude of 4550 m, and following 3 months acclimatization in the Andes (4540 m). Significant elevations in plasma growth hormone levels during submaximal exercise were observed during the trial at simulated altitude, but no such increments occurred at sea level or after altitude acclimatization. Later, Gutton and Garmendia (140) reported that maximal physical exercise was necessary to induce a growth hormone response in sea level dwellers who had been acclimatized to 4500 m for 3 months. These investigators also observed an increased baseline level of growth hormone in high altitude natives, a finding which was later confirmed by Raynaud et al. (121). The latter workers compared the effects of exercise at sea level, after a 5 day sojourn at 2850 m, and again at sea level but while test subjects breathed a gas mixture with O_2 equivalent to 2850 m. These test subjects (121) were lowland natives and exercised on a bicycle ergometer for 1 hour in each of the three conditions. Again, under all 3 conditions plasma growth hormone levels increased significantly during exercise, most rapidly and to the greatest degree under high altitude conditions. During 1 hour of recovery under each environmental condition growth hormone levels returned to normalcy. While several studies have indicated an association between the growth hormone response to exercise and high altitude exposure (136,137), elevations in growth hormone levels may be more closely correlated with the intensity and duration of exercise (141).

A limited number of studies have addressed the thyroidal response to exercise at altitude. The most detailed of these is probably the report of Stock et al. (132) who examined the effects of moderate exercise (20 min) prior to, during, and after a 3 week sojourn at high altitude (3650 m). Generally, the results indicated that the combination of altitude exposure and exercise were stimulatory to thyroidal activity. Other observations were a slightly increased thyroidal response when subjects were fasting and a noteable decreasing trend of responsivity after 3 weeks exposure to high altitude in comparison with 1 week residence. The next year Wright (164) also reported an increased thyroidal activity as manifested in plasma T_4 and reverse T_3 levels during a high altitude trek for 13 days between 1000 and 5000 m. Altitude exposure in the absence of exercise has been reported (120) to elicit increases in plasma levels of T_3 and T_4. The combination of exercise/high altitude exposure apparently effects increased metabolic demands concomitant with elevated thyroidal activity.

Increased sympathicoadrenal activity pursuant to exercise under high altitude conditions has been reported in animals as small as rats (104) and as large as steers (8). In humans Guilland et al. (67) have reported that urinary levels of epinephrine, norepinephrine, metanephrine, and vanillylmandelic acid were increased during exercise at high altitude; the increments were maximal above 6000 m, and persisted during the descent to sea level. Even earlier, Becker and Kreuzer (5,6) observed that when high altitude exposure was combined with a physical work regimen, then increased norepinephrine excretion occurred in the absence of significant effects on epinephrine excretion. The same investigators (5,6) noted that sedentary exposure to simulated high altitude (3000-4000 m) for 90 minutes resulted in no effects on either norepinephrine or epinephrine secretion. Thus, the stimulation of the sympathicoadrenal axis by altitude exposure and exercise may be affected by the elevation, the level of physical activity, and also the degree of acclimation to the selected altitude.

The effects of exercise and altitude exposure on circulating levels of several additional hormones have been described; several discrepancies persist. For example, Humpeler et al. (78) described a signficant elevation in testosterone levels 2 days after arrival at 2040 m and in combination with daily walking over 12.7 km to 2850 m. Interestingly, luteinizing hormone and follicle stimulating hormone fell under the same conditions. While Vander et al. (149) observed similar results at 4300 m, Guilland et al. (67) described "hypoandrogenicity" during work at high altitude as manifested in urinary testosterone and metabolites of testosterone. The latter workers (67) state that the "duration, altitude, and the level of physical exertion" are all important variables probably affecting results. In their report Sutton and Garmendia (140) observed that high altitude natives manifested a higher basal concentration of glucagon, and elevations in glucagon were correlated with length of time of altitude exposure. Interestingly, both glucagon (14) and glucocorticoids (125) have been reported to have salutary effects in protecting in vitro heart preparations from severe hypoxia.

Thus, it can be concluded that the combination of hypoxia and exercise generally stimulates the secretory activity of the hypothalamic-hypophysial sympathicoadrenal axis, manifested in elevations in circulating levels of respective hormones and neuroendocrines. Further, the increased metabolic demands, not only of exercise, but also of altitude exposure (63,85), are compatible with the increments reported in hormones which are associated with elevated metabolic rate. There are several additional points, however, which are worth emphasizing. Increased circulating levels of any hormone may indeed be the direct result of heightened secretory activity; but, it should be remembered that such could also result from a decreased uptake by target organs, a decreased catabolism, decreased excretion, or increased release rate in the absence of de novo synthesis. All of these variables may be affected by type and intensity of exercise, fitness, duration and level of altitude exposure, origin and acclimatization of test subjects, and real or simulated high altitude exposure. Further complicating an already overwhelming battery of potential variations are time of day of sampling, time of sampling with respect to exercise duration or completion, sample handling and storage, particularly in field experiments, and assay technique and variability.

HEAT AND EXERCISE

Exercise in a hot environment can be expected to induce endocrinological alterations designed to reduce urinary fluid loss, increase peripheral vasodilation, promote heat dissipation, maintain blood flow to the exercising muscles, and simultaneously preserve plasma volume and cardiac stability. One of the many physiological responses among humans and higher animals to achieve these sometimes disparate goals is a stimulated secretion of vasopressin (VP) or antidiuretic hormone which reduces urinary water loss and conserves body fluids and electrolytes (49). Moreover, in the absence of adequate fluid replenishment, exercise in the heat may lead to progressive dehydration with significant effects on plasma volume and plasma osmolality (28,128). This increased osmolality may be sensed by hypothalamic osmoreceptors and result in stimulated secretion of pituitary VP (see 22) with striking results. For example, Strydom et al. (133) reported that during an 18 mile road march in the heat mean urine volume was reduced to only 134 ml while sweat losses were greater them 4 liters. When Shvartz et al. (130) administered orthostatic tilt tests to subjects prior and subsequent to exercise in the heat for 8 days (heat acclimation), they observed a 50-fold elevation of VP during the tilt test before acclimation and a 75% decrement in this response following acclimation. They attributed this decrement to the increased plasma volume ordinarily elicited by heat acclimation.

Convertino et al. (24) attempted to separate the exercise and thermal factors effecting the increased plasma volume, and concluded that increments in VP levels with exercise contributed significantly to the elevated plasma volume. However, with severe heat stress (70-75°C) sedentary exposure has also been reported to induce significant increments in VP (124), while acclimation, in the absence of further exercise or heat stress, had no effects on plasma VP levels (126). Studying hidromeiosis (sweat suppression) during prolonged and repeated heat exposures, Candas et al. (15) concluded that changes in sweat rates were not associated with alterations in plasma levels of VP. The results generally indicate that acute exposure to intense heat or exercise in the heat stimulates the synthesis and release of VP to reduce urinary fluid loss, that hyperhydration may reduce the intensity of this response, and that this response may be related to plasma osmolality (61,105).

Widespread reports of elevations in plasma aldosterone levels during single or consecutive exercise trials in a hot environment prompted Braun et al. (12) to examine the effects of exogenous aldosterone administration on the acquisition of heat acclimation. Despite the fact that these investigators found no shortening in the time required for acclimation, they did report some beneficial effects on heart rates and rectal temperatures. Even in the absence of exercise, heat stress alone has been frequently reported to induce significant increments in circulating aldosterone levels (46,90). When heat exposure was combined with consumption of a low sodium diet, then increments in circulating aldosterone levels were exacerbated (3,46). While exercise in the heat is ordinarily accompanied by rapid increments in plasma aldosterone levels (27), this response is apparently attenuated following acclimation of the test subjects (25,42,52). Further, whereas plasma volume expansion (51), glucose-electrolyte replacement solutions (55), or saline ingestion (33) also moderated the effects of exercise in the heat on plasma aldosterone levels, we have demonstrated that hypohydration (52) and increasing intensity of hypohydration (54) may exacerbate these responses. Thus, the stimulated secretion of aldosterone during a heat exposure/exercise contingency is clearly adaptive to water and sodium conservation.

Insert Figure 3

Similarly, exercise in the heat is accompanied by a reduction in renal blood flow, some degree of hypohydration, and, if prolonged, decrements in total body sodium content, all of which are conducive to elevated plasma renin activity (PRA). As with aldosterone, sedentary exposure to high ambient temperature has been reported (3,90) to elicit significant elevations in PRA. Introduction of an acute exercise regimen to the heat stress generally results in further elevation in PRA (7,43,54), and these increments may be affected by state of heat acclimation (42,43,52), hydration status (52,54), sodium balance (3,43,55), physical conditioning (61), and even age (115). Since plasma levels of angiotensin II may be partially responsible for the control of plasma aldosterone concentrations, it is not unanticipated that alterations in plasma renin activity may be mirrored in plasma aldosterone fluxes during exercise in the heat; however, environmental and exercise conditions as well as the physiological status of the test volunteers may preclude such correlations (41).

Collins and Weiner (22) reviewed data which indicated that the adrenocorticotrophic response to exercise/heat exposure may be affected by the intensity of the stress, its duration, and the physiological strain induced by this combination. Just a year later Collins et al. (21) provided evidence that the stimulation of glucocorticoid secretion in man may be closely related to achieving a "critical" rectal temperature during the experimental protocol. Follenius et al. (45) hypothesized that increasing plasma cortisol levels may indeed be a useful metric of heat intolerance. Generally, our own results have indicated that mild exercise in a hot environment can be tolerated with minor effects on glucocorticoid hormones when the men are reasonably fit and well hydrated. For example, we have observed (50) that exercise (5.6 $km.h^{-1}$) in the heat (49°C db) prevented the normally occurring circadian reductions in plasma cortisol (93), but did not elicit increments in these levels. We later reported that during exercise in the heat plasma cortisol levels may be increased by hypohydration (53), but if test subjects were well heat-acclimated, then exercise/heat stress may again only prevent the aforementioned circadian reductions (54).

These effects of heat acclimation are consistent with the report of Davies et al.(33) who also observed attenuated effects on plasma cortisol levels during exercise in the heat following heat acclimation. In a recent review Viru (150) argued that exercise in the heat may be accompanied by a decrement in adrenocortical activity in humans, but one of the references cited (70) attributed the observed reduction to differences in pre-heat exposure adrenocortical activity. Earlier, Sulman et al. (134) attributed catecholamine deficiency in patients during prolonged and recurrent exposure to heat stress to an adrenal exhaustion syndrome, but it is unlikely that young healthy test subjects exposed to acute exercise/heat regimens in a natural or chamber environment would manifest such symptoms.

Using a comprehensive study design, Sedgwick et al. (127) compared the effects of smoking, psychological stressors, heat, exercise, and fat ingestion on the neuroendocrine response profiles of 12 healthy test subjects. They reported that while norepinephrine excretion rate was increased by exercise (bicycle ergometer, 3-18 min periods, HR 130-140 b.min $^{-1}$), heat exposure (1h, 42.5°C db, 28.8°C wb) had no effects on either epinephrine or norepinephrine excretion. Using consecutive daily heat exposures in combination with light exercise, Polozhentsev et al. (117) reported that norepinephrine excretion was increased sharply on the first experimental day in the exercising group, but this increment disappeared in the ensuing experimental days. Also noteable in this experiment was an apparent anticipatory response both upon initiation and completion of the 12-day scenario. Powers et al. (118) attempted to separate the thermal and exercise effects on catecholamine levels, and reported that exercise combined with heat elicited an increase in plasma norepinephrine greater than the sum of the increments induced by exercise or passive heating alone with smaller effects on epinephrine responses. Maher et al. (101) reported that the acquisition of heat acclimation reduced the urinary norepinephrine levels noted following exercise in the heat.

As noted earlier (134), there have appeared reports of adrenal exhaustion syndrome in individuals exposed to extreme heat for prolonged periods; the authors reported that treatment of such patients with monoamine oxidase inhibitors was effective in reducing the extensive symptomatology of the disorder. Since the manifestations of exercise in the heat (dehydration, increased perceived exertion and core temperature, hypoglycemia) may all affect catecholamine secretion and are generally attenuated by heat acclimation, it is reasonable that the magnitude of the catecholamine response may be affected by the intensity and duration of the exercise, the ambient temperature, the fitness levels, and the degree of acclimation of the test subjects.

Considering the role of thyroid hormones in stimulating oxidative metabolism and heat generation, it is probably not surprising that in their early review of endocrinological responses to heat exposure Collins and Weiner (22) first discussed the rather extensive literature on depressed thyroid activity during heat exposure. More recently, Epstein et al. (38,39) have reported that light work in the heat is accompanied by significant decrements in T_3 levels while reverse T_3, the noncalorigenic metabolite, actually increased in serum. Gertner et al. (60) examined thyroid gland activity in winter and summer in Israeli laborers and reported lower circulating levels of T_3 during the summer. Since T_4 levels were unaffected, they concluded that extra-thyroidal conversion of T_4 to T_3 may be integral to the regulation of energy metabolism. Earlier, Yoshimura et al. (165) had speculated that the seasonal variation of basal metabolic rate in Japanese may be related to reduced thyroid activity during the summer season, and Sulman et al. (135) attributed the symptomatology of heat stress syndrome to hyperthyreosis. The results generally indicate that T_3 levels may be affected before T_4 and that work under more chronic conditions induced the more physiologically significant changes in human thyroid activity.

Winter (163) used growth hormone deficient adolescents to demonstrate that during prolonged exercise growth hormone (GH) release is necessary for maintenance of free fatty acids as a fuel source. In the absence of a work paradigm, Leppaluoto et al. (97) reported an increase in circulating growth hormone levels shortly after exposure to severe heat. When we combined exercise with a hot-dry or hot-wet environment, we observed inconsistent responses of growth hormone which were affected by hydration (53) and, to a much lesser degree, by acclimation (50). Frewin et al. (56) reported a marked difference in plasma growth hormone responses to exercise when the exercise was carried out at $40^{o}C$ vs. $10^{o}C$. At the hot temperature there occurred a significant increase in GH while at $10^{o}C$ no such increment occurred. However, it should be noted that GH responses of humans to exercise/environmental stress are markedly variable and, in the report of Frewin et al. (56), levels of GH ranged from $3ng.ml^{-1}$ to $70ng.ml^{-1}$ during exercise in the heat. Weeke and Gunderson (154) also observed increments in GH induced by heating while cool immersion repressed plasma GH levels. In summary, it should be pointed out that in humans growth hormone secretion is episodic in nature (155), and interindividual variability in the sporadic pattern and timing of growth hormone secretion may contribute to the divergence of results reported as well as the inability to achieve consistent responses during exercise in cool environments.

Thus, there is abundant evidence to demonstrate the myriad of hormonal adaptations which occur in humans during exercise in the heat. The most consistent responses are designed to conserve body fluids and electrolytes, and, quite logically, these alterations are exaggerated by hypohydration and electrolyte deprivation. It should be duly noted that in most of the studies cited, test subjects were young, healthy, and moderately fit, all of which could affect test results. For example, in our own studies mild/moderate exercise in a hot environment usually does not elicit a significant glucocorticoid response when the subjects are well hydrated. It is conceivable, however, that in a group of less fit, older, or heavier subjects, stress perception and reality could be far greater, and adrenocorticotrophic responses could likewise be more prominent. An imminent study has been designed to assess the effects of age on some endocrinological responses to exercise in the heat.

COLD AND EXERCISE

In a recent volume in this series Horvath (75) reviewed the physiological responses of humans to exercise in a cold environment and specifically noted the paucity of information on the endocrinological effects induced by this regimen. Research reports over the last six years indicate that the apparent imbalance between investigations concerned with hormonal responses of humans to exercise in hot vs. cold environments has persisted. Manuscripts on the endocrinological responses to work in a cold environment remain limited; infact, in his review Horvath (75) reported on the endocrine responses to cold water immersion only. The limited interest in this area of investigation may be surprising considering the enormous metabolic demands elicited by work in a cold environment which are met by hormonal adaptations stimulating oxidative processes (129).

For example, when Timmons et al. (147) examined fat metabolism in humans during exercise at -10° and +22°C, they reported that oxygen consumption, energy utilization, and fat expenditure were all significantly higher in the cold environment. Analogously, Jacobs et al. (81) compared glycogen depletion in men exercising (50-65 watts) on a bicycle ergometer at 21°C and 9°C. While they reported no change in glycogen content at 21°C, the same exercise at 9°C elicited a 23% decrement in glycogen. Hormonal profiles were not reported in these studies.

Fisher (44) observed that even neonatal or premature infants can respond to cold stress with elevations in thyroid stimulating hormone and T_3, deiodination of T_4 to T_3, and elevations in basal metabolic rate. Considering their thermogenic role (31), it is probably not surprising that thyroid hormones in subjects ranging from rats (96) to humans (63,113) are elevated pursuant to cold exposure although the literature is not wholly consistent on this point (154). In relevant human investigations Nagata et al. (110) observed that work in a cold (4-6°C) environment for 3 hours had no effects on T_4 or T_3, but individuals who worked in the cold more chronically (i.e. the winter season) did manifest elevated T_3 levels. Alternatively, Wilson (159) had earlier reported that minor changes in protein-bound iodine and cellular/plasma ratios of T_3 during increased physical activity in a cool environment were attributable to decreases in plasma volume. Premachandra et al. (119) investigated the effects of distance swimming and cycle ergometry on thyroid hormones and reported relatively minor effects on T_3 during swimming; Terjung and Tipton (142) had previously reported minor effects of cycle ergometry (30 minutes) on thyroid hormones including thyroid stimulating hormone. During 3.5 hours of bicycle ergometry O'Connell et al. (111) observed increased concentrations of reverse T_3 which was moderated by dextrose infusion. Thompson et al.(144) administered exogenous thyroid hormones to cold-exposed test subjects, and demonstrated that heat production increased in 3 of 4 subjects receiving T_3 and one hypothyroid subject administered T_4. In humans brief (0.5-3 hours) cold exposure without physical activity had either no (71,159,160) or minor (148) effects on thyroid hormone levels. Clearly, the thyroid responses to exercise and cold exposure may be affected by the duration and intensity of the cold stress as well as the exercise, the absorptive and glycemic status of the subject, and the type of physical exercise.

INSERT FIGURE 4

Studying both hyper- and hypothyroid subjects, Copinschi et al. (26) reported a remarkably close association between plasma levels of thyroid hormones and cortisol although no reports have addressed the correlative responses of thyroid and glucocorticoid hormones to exercise in a cold environment. In their comprehensive paper Galbo et al. (57) investigated the effects of water temperature and prolonged swimming in humans on circulating hormones, and reported that cortisol levels fell 15 min. after a 1 hour swim in 21°C water while increments occurred after swimming at 27 and 33°C. We (47) and others (64) had previously reported that cold stress can disrupt the consistent daily periodic oscillations in circulating cortisol levels, but no physiologically significant changes were demonstrated. Hartley et al. (69) used heavy (98% $\dot{V}O_2$ max) bicycle exercise to elicit a significant elevation in circulating cortisol while both mild (42%) and moderate (75%) work failed to elicit any effects on this variable; physical conditioning also affects this response (156). Moreover, cold exposure in the absence of a work regimen did elicit significantly elevated cortisol levels (158, 160). Surprisingly enough, no studies were identified which compared the glucocorticoid reponses in humans during increasing intensity or chronicity of exercise under cold ambient conditions.

Mager and Robinson (100) exposed men to 4°C intermittently for 5 weeks and observed significantly increased excretory rates of norepinephrine initially which returned to control levels after 7 days; alternatively, circulating epinephrine was unaffected during the entire cold exposure. Of course, catecholamine responses to the cold pressor test have been monitored frequently, and generally significant increments in both circulating norepinephrine and epinephrine have been reported (2,123). While a considerable number of reports have documented the responsivity of circulating norepinephrine and epinephrine to acute exercise stress (65,69,72), whole body exercise in combination with cold stress has not been extensively investigated for these variables in humans. In rats Chin et al. (17) and Harri et al. (68) have demonstrated that exercise training can attenuate the increments in plasma and urinary norepinephrine and epinephrine pursuant to exposure of the animals to cold stress. Exogenous administration of norepinephrine to Korean diving women in winter elicited a statistically significant, but physiologically minimal, increase in oxygen consumption of a magnitude insufficient to conclude that nonshivering thermogenesis had developed in these test subjects (83). Galbo et al. (57) reported that during the first 30 min. of swimming there occurred significant elevations in circulating norepinephrine and epinephrine which appeared to reach steady-state levels between 30-60 min.; it is also relevant to note that the greatest absolute increments in these variables occurred during swimming in the coldest water (21°C vs 27°C or 33°C). A similar study at various air temperatures has not been executed.

Christensen et al. (18) observed that when men exercised sufficiently to increase rectal temperature by 1°C, small but significant elevations of growth hormone were noted; however, when the same exercise intensity and duration was duplicated in a cold room with no increase in Tre, then growth hormone release was repressed. Similarly, Galbo et al.(57) reported unchanged levels of growth hormone when their subjects swam in 21°C water while swimming at either 27°C or 33°C induced elevations in this variable. Generally, the same results have been reported during passive exposure to cold temperatures (64) although Okada et al. (112) did observe significant increments in plasma growth hormone in both male and female test subjects upon rewarming following no changes during 1-2 hours of cold stress.

The repressive effects of the combination of cold exposure and exercise on growth hormone levels may be related to the concurrent insulin/glucose response to exercise in the cold. For example, both cold exposure and exercise have been extensively reported to depress circulating insulin levels in a variety of experimental species to maintain circulating glucose levels (37,72,88,131). Hypoglycemia is ordinarily stimulatory to growth hormone secretion. Thus, if normo- or hyperglycemia is maintained during exercise in the cold, then an important stimulus for growth hormone release may be neutralized.

In 1981 Horvath (75) wrote: "It is hoped that investigations into the area of physiological adjustments to cold will receive the same degree of attention as has been given to hot environment studies." Over the past six years it appears that little progress has been made in addressing this apparent imbalance. Thorough literature searches have revealed a dearth of studies investigating the effects in humans of exercise on hormonal responses in cold air environments. The hormonal adaptations necessary to support the increased metabolic demands of muscular work and heat production during exercise in the cold are clearly identified as an area of environmental physiology requiring further research effort.

ENDOGENOUS OPIOIDS, EXERCISE, AND ENVIRONMENTAL STRESS

The burgeoning research field of endogenous opioid polypeptides and their relationship to stress physiology is relatively new, but certainly deserves note in this review. According to Akil et al. (1), there are three opioid peptide families - the B-endorphins, enkephalins, and alpha-endorphins, all of which may be found in appreciable concentrations in the "stress" glands frequently mentioned in this review - the hypothalamus, the pituitary, and the adrenal. These endogenous opioids are hypothesized to act as pain moderators, and may be intimately involved in the development of stress- induced analgesia.

Colt et al. (23) measured B-endorphin immunoreactivity in 35 runners after mild and moderate runs and reported significant increments in plasma extracts which were probably related to the intensity of the run. At approximately the same time Gambert et al. (59) reported similar results in both men and women exercising at fairly heavy intensities for short durations; they suggested that increments in B-endorphin immunoreactivity may be associated with the so-called "runner's high."

The potential of these endogenous opioids to act as modulators of pain or the affect state has, quite naturally, been adequate stimulus for a flurry of research activity to quantitate and characterize these responses during various exercise intensities (92), in various levels of physical training (16), in women (76), and during altered physiological states (106). It should be noted that, to date, very few studies have addressed the response of these peptides to exercise during any type of environmentally stressful condition. However, Kelso et al. (86) did examine the effects of cycling (50% $\dot{V}O_2$ max) at 24^oC and 35^oC on plasma B-endorphin/B-lipotropin levels, and observed slightly increased concentrations at the warmer environmental condition. To the best of my knowledge no reports have appeared examining the combination of exercise and altitude or cold stress on these responses, but this area of investigation is certainly identified as fertile for additional studies. Responses to such a combination of stressors might be extremely helpful in identifying the role of these compounds in physical training and in mood modulation.

CONCLUSION

Clearly, the physiological or fitness status of an individual may play a pivotal role in the initiation, duration and intensity of a hormonal response to exercise in a stressful environment. Given the wide variability in the fitness and acclimation levels of a population of potential test subjects, interindividual response differences in such variables as circulating hormones are inevitable. Thus, when evaluating the endocrinological responses to exercise in a stressful environment, it must be noted that the perception and the reality of stress will vary among subjects according to their level of training, hydrational status, acclimation level, nutritional condition, and, importantly, the novelty of the situation. Equally or more divergent is the number of experimental conditions which can be applied to the exercise/environmental scenario. While it is likely and desirable that future research reports maintain this diversity of independent variables, investigators should provide maximal physical and physiolgical information relative to test subjects and logistical information relative to test scenarios. Such information will be invaluable to the interpretation of results and useful in explaining inconsistencies in results.

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Figure Legend

Fig. 1. Effects of exercise at sea level and a simulated altitude of 4550 m on circulating levels of cortisol, growth hormone, and insulin. This figure reproduced from: J.R. Sutton, J. Appl. Physiol. 42, 587, 1977. (Reprinted with permission of the author and the American Physiological Society.)

Fig. 2. Effects of exercise (hill-walking) and altitude (3100m) on plasma levels of aldosterone and renin activity. Blood samples at altitude were taken immediately upon completion of the exercise. This figure reproduced from: J.S. Milledge et al., J. Appl. Physiol. 55, 413, 1983. (Reprinted with the permission of the author and the American Physiological Society.

Fig. 3. Effects of hypohydration (5%) and acclimation on responses of circulating cortisol, plasma renin activity, and aldosterone during exercise in a hot-dry environment. Top figure taken from: R. Francesconi et al., Aviat. Space Environ. Med. 55, 365, 1984; middle and bottom figures taken from: R. Francesconi et al., J. Appl. Physiol. 55, 1970, 1983. (Reprinted with permission of the author, the Aerospace Medical Association, and the American Physiological Society.)

Fig. 4. Effects of exercise (breast stroke swimming) in three water temperatures (21, 27, 33°C) on serum or plasma hormone levels. Reproduced from: H. Galbo, Acta. Physiol. Scand. 105, 326, 1979.

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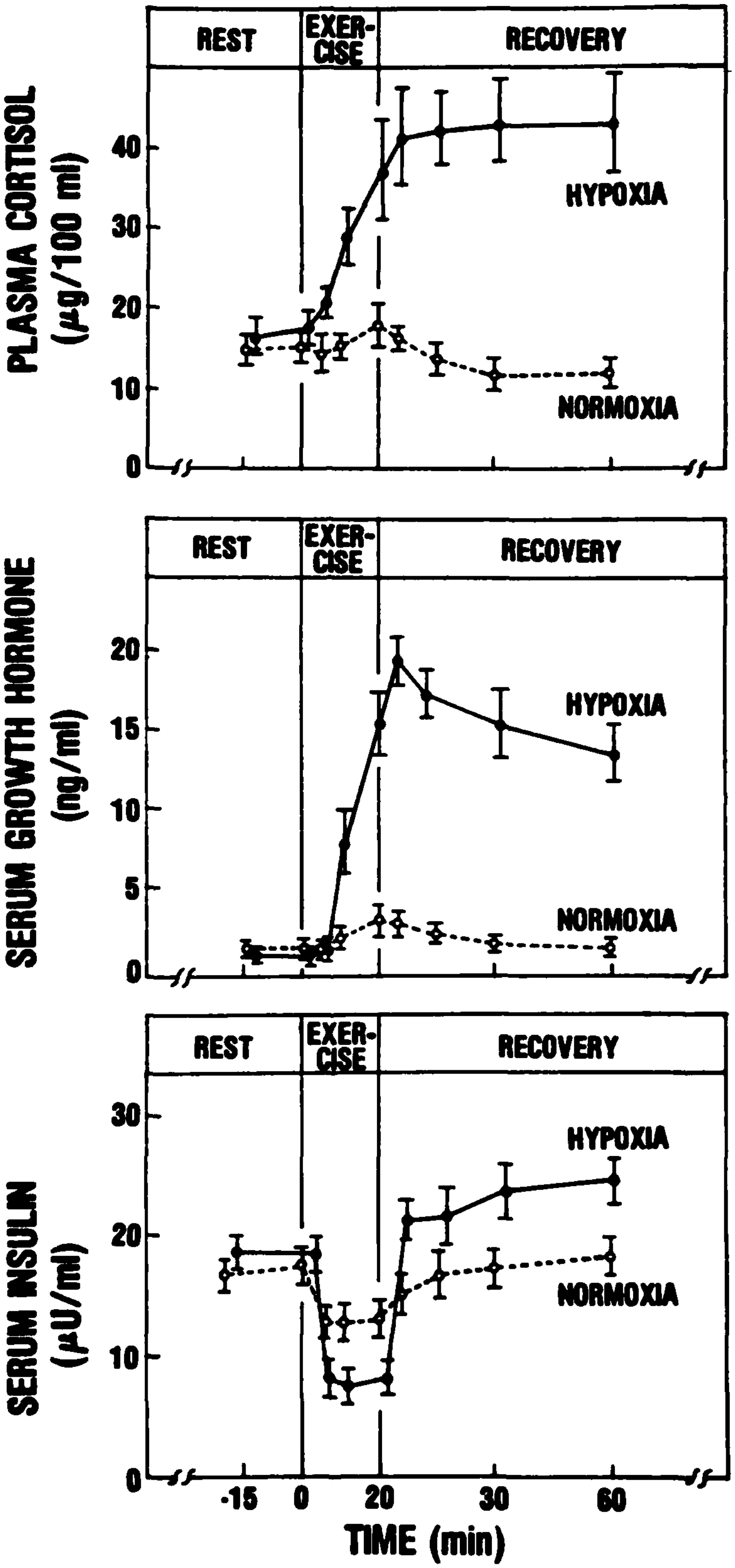
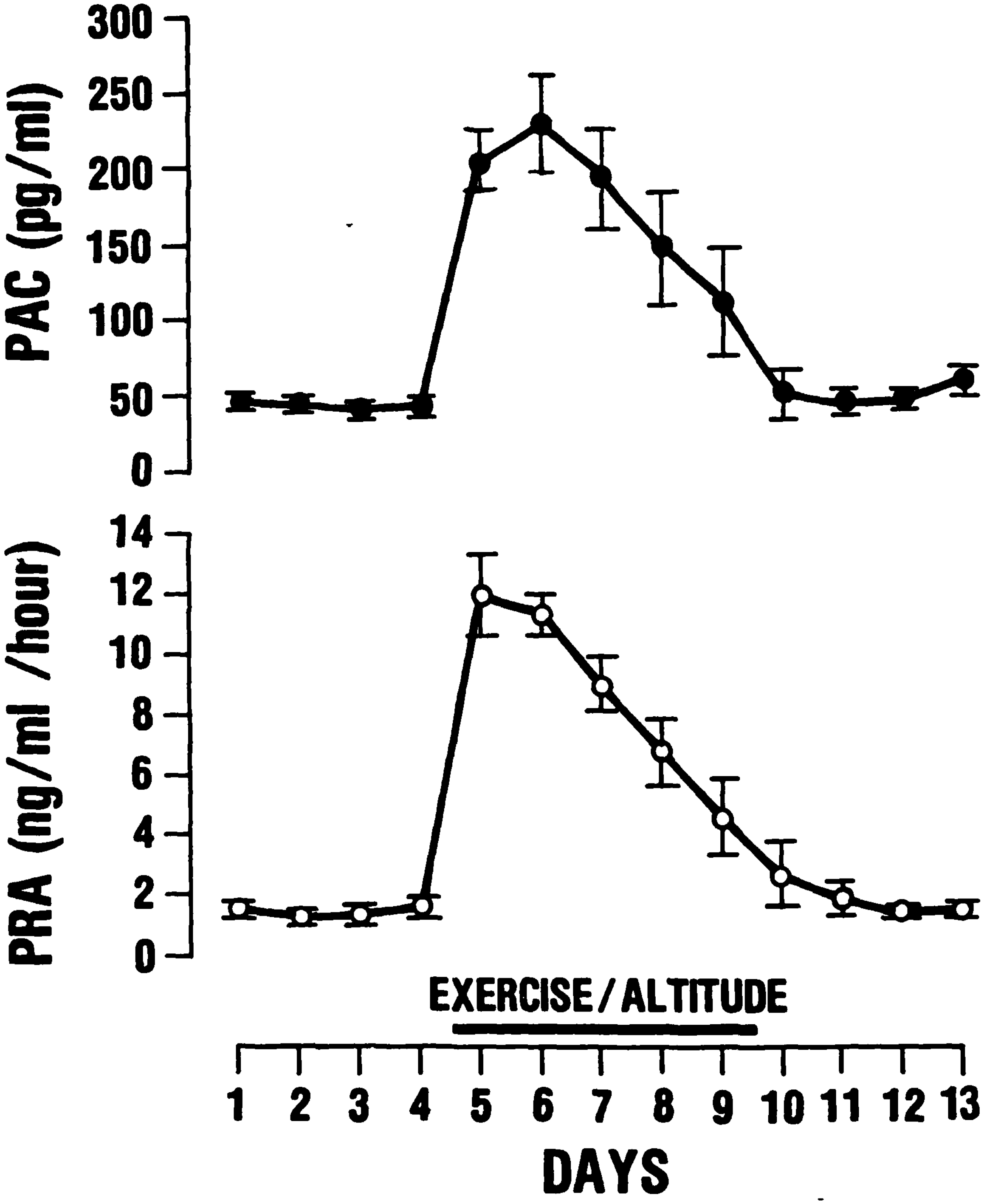


Francesconi Fig 1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100
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PRA (ng/ml /hour)
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12
10
8
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EXERCISE / ALTITUDE
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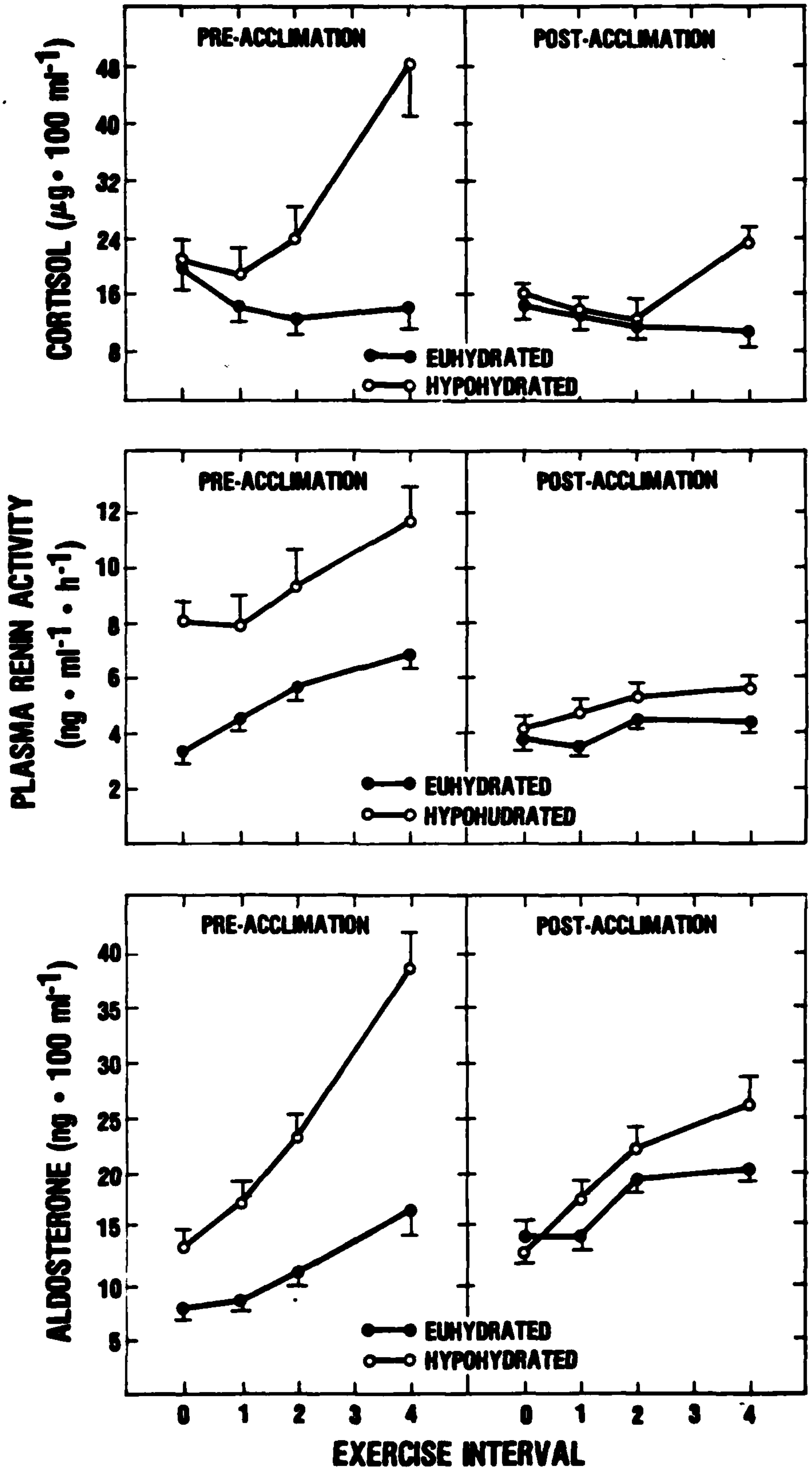
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POST-ACCLIMATION
CORTISOL (μg • 100 ml^{-1})
48
40
32
24
16
8
● EUHYDRATED
○ HYPOHYDRATED
PRE-ACCLIMATION
POST-ACCLIMATION
PLASMA RENIN ACTIVITY (ng • ml^{-1} • h^{-1})
12
10
8
6
4
2
● EUHYDRATED
○ HYPOHUDRATED
PRE-ACCLIMATION
POST-ACCLIMATION
ALDOSTERONE (ng • 100 ml^{-1})
40
35
30
25
20
15
10
5
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EXERCISE INTERVAL

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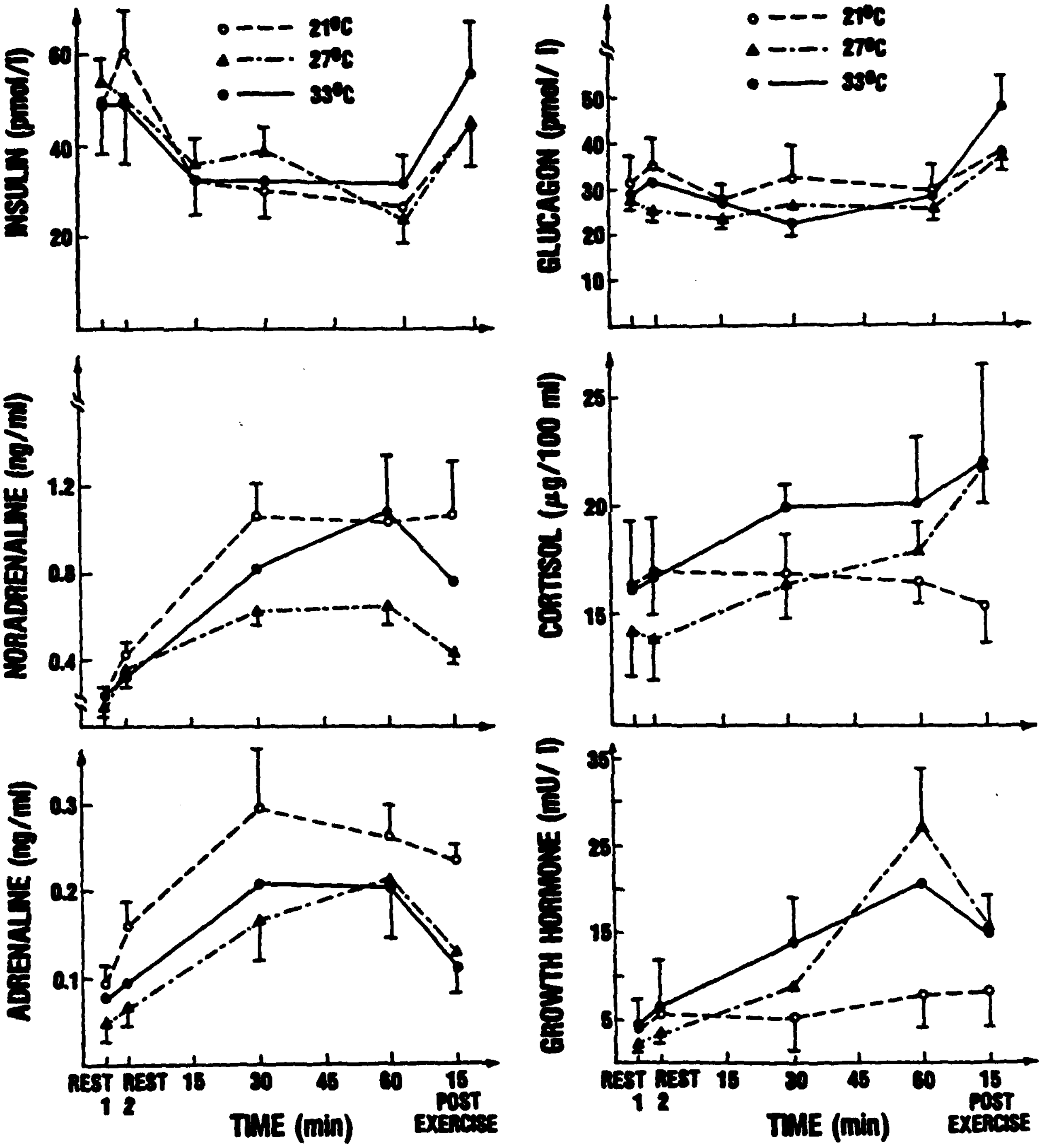

Francesconi (5)

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