

HYPOHYDRATION AND HEAT ACCLIMATION: PLASMA RENIN AND
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PRA and ALD were greater when hypohydrated, and PRA effects were significantly moderated by heat acclimation in both the euhydration and hypohydration experiments. While PRA and ALD responses were generally correlated, acclimation did not consistently attenuate ALD increments. We concluded that hydration state, acclimation level, and environmental conditions all affected the responses of these hormones to light exercise.

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Hypohydration and heat acclimation: plasma renin and aldosterone during exercise

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FRANCESCONI, RALPH P., MICHAEL N. SAWKA, AND KENT B. PANDOLF. *Hypohydration and heat acclimation: plasma renin and aldosterone during exercise*. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 55(6): 1790-1794, 1983.—This study was designed to assess the effects of hydration, acclimation, environment, and exercise on plasma levels of renin and aldosterone. Sixteen subjects exercised ($1.34 \text{ m} \cdot \text{s}^{-1}$), both pre- and postacclimation, when euhydrated or hypohydrated ($\sim 5\%$ of body wt) in a comfortable (20°C , $\text{rh} = 40\%$), hot-wet (35°C , $\text{rh} = 79\%$), or hot-dry (49°C , $\text{rh} = 20\%$) environment. Although light exercise in a thermoneutral environment had no effects on plasma levels of renin activity (PRA) or aldosterone (ALD), exercise in both hot environments resulted in significantly increased levels of both. Increments in both PRA and ALD were greater when hypohydrated, and PRA effects were significantly moderated by heat acclimation in both the euhydration and hypohydration experiments. Although PRA and ALD responses were generally correlated, acclimation did not consistently attenuate ALD increments. We concluded that hydration state, acclimation level, and environmental conditions all affected the responses of PRA and ALD to light exercise.

hot-dry environment; hot-wet environment; temperate; dehydration

THE ROLE of fluid regulatory hormones in the acquisition of heat acclimation has been investigated for a number of years. As early as 1967 Braun et al. (1) attempted to accelerate the acclimation process in humans by the administration of *d*-aldosterone (ALD). Although their subjects demonstrated several beneficial effects of hormone treatment (1), no reduction in total acclimation time was achieved. Finberg et al. (6) reported that increments in plasma renin activity (PRA) induced by exercise and heat stress were attenuated when the subjects were heat acclimatized and euhydration was maintained. In a subsequent experiment these investigators (5) demonstrated that a 7-day heat acclimation program significantly reduced the increments in PRA during exercise in the heat, but plasma ALD was unaffected. However, Davies et al. (3) reported that increases in PRA and ALD during exercise in the heat were unaffected by heat acclimation; saline consumption reduced, but did not prevent, these increments. Convertino et al. (2) found that both sedentary heat exposure and cycle ergometry at a moderate temperature induced significant increases in PRA; consecutive exposures to either condition did not modify the increments in PRA.

Recently, Gaebelein and Senay (9) have suggested that

exercise-induced changes in serum osmolality may be dependent not only on the mode of exercise but also the initial hydration level of the test subjects. Additionally, Senay (18) had earlier reviewed data indicating that the training level of test subjects might also be expected to affect body fluid responses to exercise in a hot environment. Thus it is evident that state of acclimation, hydration level, exercise mode, and physical condition may independently or collectively affect the direction and intensity of body fluid shifts or hormonal responses during exercise in a hot environment. In addition, we cannot rule out the possibility that other factors might influence such responses. In the current experiment we have investigated several interactive variables reported or hypothesized to alter hormonal responses to exercise in the heat.

Since PRA has been correlated with plasma ALD levels (10, 14) under a variety of conditions including exercise (8), heat acclimation (3), and heat stress (11), we have examined the responses of both to exercise in the heat. Because hypohydration might be expected to affect these alterations, we have also examined the impact of reduced fluid reserves on these responses. Further, the effects of heat acclimation and environmental conditions were simultaneously investigated to obtain a comprehensive profile on the control of these adaptive responses.

METHODS

Eight male and eight female test volunteers participated in this study; male test subjects had a mean age ($\pm$ SD) of 23.6 ± 2.8 yr, height of 170.8 ± 7.2 cm, weight of 75.4 ± 7.4 kg, and maximal O_2 uptake ($\dot{V}\text{O}_{2\text{max}}$) of $48.69 \pm 3.82 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$. Respective data for the women were 25 ± 4.2 yr, 163.0 ± 6.6 cm, 62.2 ± 11.3 kg, and $42.85 \text{ ml} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$. Before initiation of the study subjects were fully apprised of the rationale, methods, procedures, and potential risks of the study. Each volunteer reserved the right to withdraw at any time without retribution.

Each subject participated in a total of 12 tests, 6 before and 6 after completion of a heat acclimation program. Before and after acclimation the subjects completed two experimental tests in each of three environments: thermoneutral ($T_a = 20^\circ\text{C}$, $\text{rh} = 40\%$), hot-wet ($T_a = 35^\circ\text{C}$, $\text{rh} = 79\%$), and hot-dry ($T_a = 49^\circ\text{C}$, $\text{rh} = 20\%$) to simulate moderate, jungle, and desert conditions. The tests were

repeated twice in each environment, once when each volunteer was euhydrated and once when hypohydrated. The sequence of environmental conditions and hydration states were systematically varied so that test repetition did not affect results. These tests were conducted on consecutive days. Each experimental test comprised a total of 140 min (4 repeated intervals of 10 min of rest and 25 min of exercise). Exercise was performed on a level treadmill at $1.34 \text{ m} \cdot \text{s}^{-1}$ (approx 28–35% $\dot{V}\text{O}_{2\text{max}}$); during each rest period the subjects were weighed and rehydrated with cool tap water to maintain either baseline weight or -5% from base line, depending on the euhydrated or hypohydrated condition. During all hypohydration tests, both pre- and postacclimation, body weights were maintained from -4.8 to -5.1% of base line.

Hypohydration was accomplished by voluntary restraint from fluid consumption for 24 h before a test and also by performing mild exercise in a hot ($T_a = 38^\circ\text{C}$, $\text{rh} = 20\%$) environment until 5% of initial body weight was lost. Having achieved the appropriate weight loss, the subjects were removed to a comfortable environment and spent the night under supervision. The subjects were awakened at 0600 h, weighed, provided a very light breakfast if their weight was sufficiently low, and tested at approximately 0800 h. The breakfast consisted of only 1–2 crackers and a small volume of juice during the hypohydration test. During the euhydration test subjects ingested cold cereal, milk, and juice. Euhydration was ascertained by daily weighing of each test subject for at least 3 wk before the first test. During experimentation euhydration was defined by a body weight $\pm 1\%$ of the measured base-line body weight.

The acclimation program consisted of 10 consecutive days of walking on a level treadmill at $1.34 \text{ m} \cdot \text{s}^{-1}$ for two 50-min exercise bouts interrupted by a 10-min rest period. Environmental conditions were alternated (e.g., 1st day, hot-dry as above; 2nd day, hot-wet). During the acclimation and the exercise test intervals, the subjects wore shorts, T-shirts, and tennis shoes; ad libitum water consumption was encouraged during the acclimation period. Acclimation was verified by significant reductions in mean final heart rates and rectal temperatures on day 10 vs. day 1.

On each of the test days venous blood (5 ml) was obtained by a catheter placed in a superficial arm vein. After implantation of the catheter no further drinking was permitted until the first rest period during either hydration condition. Usually, about 1 h elapsed between catheterization and the start of the experiment. The first blood sample (time 0) was taken after the subjects stood for 20 min in a moderate environment ($T_a = 20^\circ\text{C}$, $\text{rh} = 30\%$); the second (time 1) and third (time 2) samples were removed at approximately 15–20 min during the first and second exercise bouts in the appropriate environment. The final (time 4) blood sample was obtained at the completion of the fourth exercise bout or at the completion of exercise if the subject was unable to complete the entire protocol. Blood was collected without stasis, transferred to iced heparinized tubes, centrifuged ($10,000 \text{ g}$, 4°C), and the plasma was removed and frozen (-20°C) for subsequent assay.

Plasma samples were thawed, and PRA (normal range in adult normotensive subjects is $0.5\text{--}4.0 \text{ ng} \cdot \text{ml}^{-1} \cdot \text{h}^{-1}$) and ALD (range for normal, upright subjects is $7\text{--}29.5 \text{ ng} \cdot 100 \text{ ml}^{-1}$) levels were quantitated by radioimmunoassay using commercially available test kits manufactured by International CIS (Saluggia, Italy) and distributed by Damon Diagnostics (Needham, MA). Angiotensin I in the PRA assay procedure was generated at pH 6.0 (37°C) for 1 h. Both assays were performed according to standard techniques described in the respective technical bulletins.

The design of the present investigation permitted each test subject to serve as his or her own control for the euhydration vs. hypohydration trial as well as the pre- and postacclimation tests. For statistical evaluation of the effects of hypohydration and acclimation, Student's dependent *t* test for paired data was utilized. Since the effects of exercise under each experimental condition required repeated comparisons, analyses of variance were performed followed by the application of Tukey's test to permit all pair comparisons (12, 13). The null hypothesis was rejected at $P < 0.05$.

RESULTS

Results indicated that gender had no effect; thus data were combined under all conditions. Figure 1 demonstrates the effects of hypohydration and acclimation on PRA during exercise in the thermoneutral environment. It is important to note in both Figs. 1 and 4 (thermoneutral environment) that the ordinates are contracted when compared with remaining figures because of the reduced response in this environment. In the preacclimation samples, hypohydration values were significantly ($P < 0.05$) greater than euhydration values at each observation; however, in the same samples no effects of exercise were noted in this comfortable environment under either hydration condition. After acclimation, PRA in hypohydrated subjects was significantly greater than that in the euhydrated state at time 0 ($P < 0.01$) and time 4 ($P <$

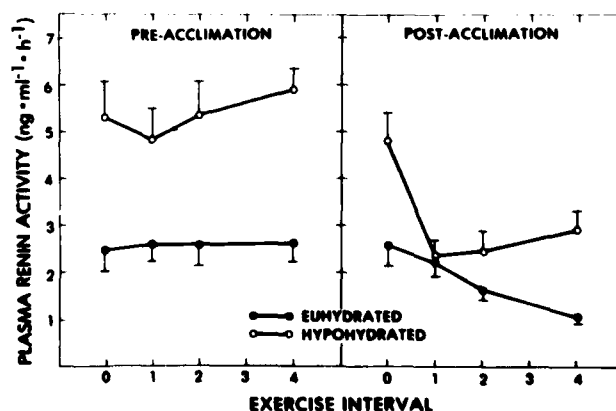


FIG. 1. Effects of hypohydration and acclimation on plasma renin activity during exercise in a thermoneutral environment. Means $\pm$ SE are shown for $n = 16$ in all cases except $n = 15$, preacclimation, hypohydration times 2 and 4. Blood samples were removed after standing for 20 min (time 0), 15–20 min of first and second exercise bouts (times 1 and 2), and at end of exercise (time 4). Exercise was performed at $1.34 \text{ m} \cdot \text{s}^{-1}$ on a level treadmill at $T_a = 20^\circ\text{C}$ and $\text{rh} = 40\%$.

0.001). It is also interesting to note that after acclimation exercise in this thermoneutral environment elicited a significant decrement ($P < 0.05$) in PRA in the time 3 and 4 (vs. time 0) samples during euhydration and significant ($P < 0.01$) decrements at times 1, 2, and 4 (vs. time 0) during hypohydration. Heat acclimation resulted in reduced PRA in both the euhydrated (time 4) and hypohydrated (times 1, 2, and 4) states.

Figure 2 shows the effects of hypohydration and acclimation on PRA during exercise in a hot-wet environment. Once again, in the euhydrated state and preacclimation, PRA was significantly ($P < 0.05$) less than the corresponding value in the hypohydrated condition. However, in this hot-wet environment it is apparent that exercise resulted in significant ($P < 0.05$) increments at times 2 and 4 (vs. time 0) in the euhydrated state. In the hypohydrated condition, a significant ($P < 0.05$) increment is observed at the time 4 (vs. time 0) interval only. Heat acclimation greatly modified the response of PRA to exercise in the hot-wet environment. For example, after acclimation in the euhydration trial, exercise elicited no significant differences (time 0 vs. time 1, 2, or 4), whereas during hypohydration only at times 2 and 4 (vs. time 0) were the increments significant ($P < 0.05$). During the hypohydration test acclimation caused a significant decrement at each sampling interval (pre- vs. postacclimation).

Figure 3 illustrates the effects of hypohydration and acclimation on PRA during exercise in a hot-dry environment. Once again, preacclimation, hypohydration elicited significantly ($P < 0.05$) increased levels of PRA at each sampling interval. Preacclimation, exercise elicited significant ($P < 0.05$) increments at times 2 and 4 (vs. time 0) in the euhydrated trial and at time 4 in the hypohydrated state. After acclimation, exercise effected significant ($P < 0.05$) increments (time 0 vs. time 1, 2, or 4) in the euhydrated state but not in the hypohydrated state. The effects of acclimation were most apparent in the hypohydrated condition with significant ($P < 0.01$) reductions noted in exercise responses at each sampling time.

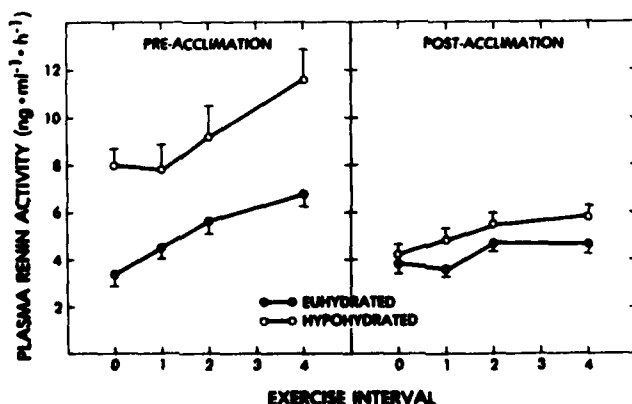


FIG. 2. Effects of hypohydration and acclimation on plasma renin activity during exercise in a hot-wet environment. All conditions are as noted under Fig. 1 except $T_a = 35^\circ\text{C}$, $rh = 79\%$, and $n = 15$, preacclimation, euhydrated time 4 and $n = 14$, postacclimation, hypohydrated time 4.

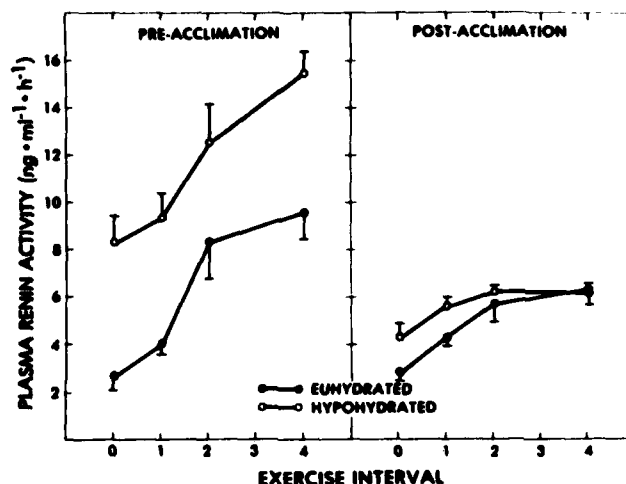


FIG. 3. Effects of hypohydration and acclimation on plasma renin activity during exercise in a hot-dry environment. All conditions are as noted under Fig. 1 except $T_a = 49^\circ\text{C}$, $rh = 20\%$, and $n = 14$, preacclimation, hypohydrated time 4.

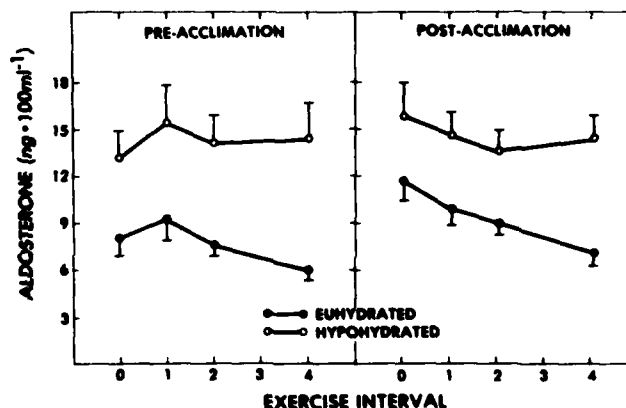


FIG. 4. Effects of hypohydration and acclimation on plasma aldosterone levels during exercise in a thermoneutral environment. All conditions are as noted under Fig. 1.

Figures 4–6 summarize the effects of hypohydration, acclimation, and heat stress on plasma ALD during exercise. In the thermoneutral environment, preacclimation (Fig. 4), hypohydration effected a significant ($P < 0.05$) increment in plasma ALD levels. However, mild exercise in this environment had no effect on ALD levels, whether the subjects were euhydrated or hypohydrated. After acclimation, hypohydration elicited significant (e.g., time 4, postacclimation, hypohydrated vs. euhydrated, $P < 0.005$) increments in plasma ALD levels. Of considerable interest is the observation that, after acclimation during euhydration, exercise in the thermoneutral environment elicited a pattern of decreasing levels of ALD culminating in significant ($P < 0.05$) decrements in the times 2 and 4 samples compared with the preexercise value (time 0). This did not occur in the hypohydration test nor were any further significant effects of acclimation noted.

Figure 5 shows the effects of hypohydration and acclimation on plasma ALD responses during exercise in a

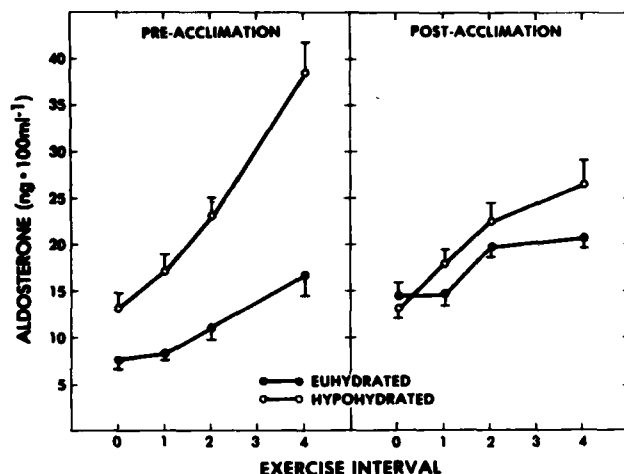


FIG. 5. Effects of hypohydration and acclimation on plasma aldosterone levels during exercise in a hot-wet environment. All conditions are as noted under Figs. 1 and 2.

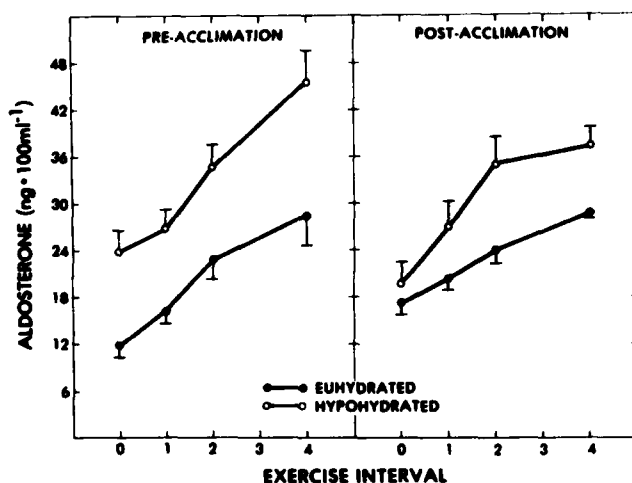


FIG. 6. Effects of hypohydration and acclimation on plasma aldosterone levels during exercise in a hot-dry environment. All conditions are as noted under Figs. 1 and 3.

hot-wet environment. Once again, in the preacclimation samples at each time interval, hypohydration effected significant ($P < 0.005$) increments in levels of ALD. Exercise in the hot-wet environment elicited significant increments ($P < 0.05$) in ALD levels in both the euhydrated (times 0 vs. times 2 and 4) and the hypohydrated (time 0 vs. times 2 and 4) conditions. After acclimation the effects of hypohydration on plasma ALD were negated, although exercise again elicited significant increments in the euhydrated (time 0 vs. times 2 and 4) and hypohydrated (time 0 vs. times 2 and 4) states. In the euhydrated condition after acclimation there occurred significant ($P < 0.001$) increments in plasma ALD (times 0, 1, and 2-preacclimation vs. postacclimation) levels.

Finally, Fig. 6 demonstrates the effects of hypohydration and acclimation on plasma ALD alterations during exercise in a hot-dry environment. Preacclimation, hypohydration elicited significant ($P < 0.005$) increments at each sampling time. Furthermore, exercise in the hot-

dry environment preacclimation, evoked significant ($P < 0.05$) increment at all times (time 0 vs. times 1, 2, and 4) in the euhydrated state and at times 2 and 4 (vs. time 0, $P < 0.01$) in the hypohydrated. After acclimation, hypohydration again effected significant increments in plasma ALD levels ($P < 0.05$, times 1, 2, and 4), and exercise again produced significant effects in both the euhydrated (times 0 vs. times 2 and 4) and hypohydrated (time 0 vs. times 1, 2, and 4) conditions. In this hot-dry environment acclimation had no effects on plasma ALD levels in either euhydration or hypohydration.

We also performed a least-squares regression analysis to demonstrate that levels of PRA and ALD were significantly correlated under all conditions. The correlation coefficient was calculated to be 0.80 and the significance of the correlation was $P < 0.005$.

DISCUSSION

The results of this investigation demonstrated that 1) hypohydration elicited increased levels of PRA and ALD with emphatic differences noted preacclimation; 2) the light exercise intensity employed in this study effected increments in PRA and ALD in conjunction with both hot environments; 3) acclimation to heat reduced levels of PRA during exercise in the heat, particularly during hypohydration; 4) acclimation to heat did not consistently affect responses of plasma ALD, particularly in euhydrated volunteers; 5) mean PRA and ALD levels were highly correlated; and 6) gender did not affect PRA and ALD responses to heat and exercise stress.

To our knowledge this is the first study to evaluate the effects of hypohydration, as well as acclimation, on the response of fluid regulatory hormones of female and male test volunteers to exercise in the heat. The large number of samples processed enabled us to evaluate the effects of these variables and draw conclusions as to their role in these processes and under these conditions. It is interesting to note that before acclimation, and before exercise (i.e., time 0 samples), hypohydration had marked incremental effects on circulating levels of PRA (112, 135, and 214%) and ALD (61, 72, and 101%), respectively, in the temperate, hot-wet, and hot-dry experiments. After acclimation these elevations were reduced to 87.5, 8.6, and 51% for PRA, and 35, 0, and 15% for ALD. These results are consonant with the hypothesis that hypohydration after acclimation may have less impact on fluid regulatory responses owing to the well-documented increased fluid volume associated with heat acclimation (2, 9, 17). Also, before heat acclimation, exercise in either the hot-wet or hot-dry environment elicited significant increments in levels of both PRA and ALD when hypohydrated or euhydrated. Acclimation moderated the responses of PRA to exercise in a hot environment, most notably, during hypohydration. Although fluid was replaced during the time of the actual exercise protocol in the current experiments, Myhre and Robinson (15) had demonstrated (during sedentary heat exposure) that plasma volume was decreased by 2.9% in six subjects at 50°C even when euhydration was maintained. During mild dehydration (2.6%) the same workers (15) demonstrated a 7.8% reduction in plasma vol-

ume. In our experiments the degree of hypohydration was even greater, and this was clearly manifested in the hormonal responses attained preacclimation during hypohydration.

Generally, some of our findings are similar to those reported by Finberg and Berlyne (5). Although conditions between the two studies differed considerably, we have confirmed that heat acclimation attenuated the response of PRA to exercise in the heat while plasma ALD responses were less affected by acclimation. Despite several specific differences in PRA and ALD responses to exercise in the heat, PRA and ALD levels were generally correlated. The reduced response of PRA after heat acclimation may be partially attributed to the increased plasma volume accompanying acclimation (10, 17), attenuated renal vasoconstriction subsequent to acclimation (16), or an overall decrease in sympathetic response to exercise in the heat after acclimation (4).

While significant effects of exercise were noted in both the hot-wet and hot-dry environments, the results clearly indicated that light exercise in the thermoneutral environment did not evoke responses in these variables. Usually, the intensity of the response appears to be related to the severity of the heat stress. For example, Follenius et al. (7) demonstrated minor increments in levels of PRA and ALD when salt-repleted subjects were exposed for 90 min to a heat stress of 46°C. Kosunen et al. (11) reported two- and fivefold elevations in PRA and ALD, respectively, upon sedentary exposure for 20 min to 85–90°C. From the current experiments we concluded that light exercise in the thermoneutral environment created neither an exercise nor an environmental stress of sufficient magnitude to elicit a response. Further, since the subjects were acclimated to both the hot-wet and hot-dry environments, it is not surprising that PRA and

ALD responses were similar under both conditions during exercise in the heat. Similarly, the effects of heat acclimation in modulating the heat and exercise response of PRA were comparable under both heat conditions.

We have concluded from these experiments that although hypohydration is extremely effective in elevating plasma PRA and ALD levels, heat acclimation moderates PRA responses but has a smaller effect on ALD alterations. Further, mild exercise in a thermoneutral environment had no effects on PRA or ALD, whereas imposition of a hot-wet or hot-dry environment was effective in inducing elevated levels. Finally, even though PRA and ALD alterations were generally correlated, they were clearly not identical among all conditions of the present experiment. All three variables, hypohydration, acclimation, and environment, affected these responses. It would be useful to extend these studies to include other heat intensities and levels of hypohydration to determine whether such responses are modulated by varying conditions. Interindividual differences in response patterns might ultimately be related to the level of acclimation achieved or the ability to withstand heat, exercise, and hypohydration stress.

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The views of the authors do not purport to reflect the positions of the Department of the Army or the Department of Defense. Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and U.S.A.M.R.D.C. Regulation 70-25 on Use of Volunteers in Research.

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