AD-A050 539 ARMY RESEARCH INST OF ENVIRONMENTAL MEDICINE NATICK MASS F/G 6/19
RECURRENT HEAT EXPOSURE: EFFECTS ON HORMONAL RESPONSES IN RESTI--ETC(U)
FEB 77 R P FRANCESCONI, J T MAHER, J W MASON

UNCLASSIFIED USARIEM-M-9/77 NL

| OF |
AD
A050539

END
DATE
FILMED
3-78
DDC

RECURRENT HEAT EXPOSURE: EFFECTS ON HORMONAL RESPONSES IN RESTING AND EXERCISING MEN

R. P. Francesconi, J. T. Maher, J. W. Mason,*

and G. D. Bynum

US Army Research Institute of Environmental Medicine

Natick, MA 01760

and

*Walter Reed Army Institute of Research

Walter Reed Army Medical Center

Washington, DC

Running Head: Heat acclimatization and hormonal responses

Send proofs to: Dr. Ralph Francesconi
Heat Research Division
US Army Research Institute of Environmental Medicine
Natick, MA 01760

Abstract

Heat acclimatization was induced in a group of healthy young men by walking on a treadmill (5.6 km / hr^{-1}, 49°C/27°C dry/wet bulb, 90 min / day^{-1}, 7 days), and confirmed by recording significantly reduced final rectal temperatures and heart rates on the seventh day of exercise in the heat. A second group, paired for maximal O_2 consumption and body weight, remained sedentary under identical environmental conditions. After correcting for minor changes in hematocrit, both the sedentary and exercising groups demonstrated significant reductions in plasma cortisol on the control day, indicating a strong anticipatory response. Heat exposure reduced plasma cortisol levels in the sedentary men, but the mild exercise program neutralized these effects in the exercising group. Patterns of alteration of growth hormone indicated a significant response even to the mild exercise program described here, while heat stress, pre- and post-acclimatization, seemed to have no effect upon plasma levels. Plasma total T_4 levels demonstrated several randomized changes which, however, did not reflect decreased output under the environmental conditions described here for either the walking or sedentary group. The mild exercise program elicited significantly reduced levels of plasma insulin which were not affected by the recurrent heat exposure. Generally, while the recurrent heat exposure effected hormonal responses among both exercising and sedentary groups, these alterations were not correlated with the process of heat acclimatization.

Key Words: Heat acclimatization, cortisol, growth hormone, thyroxin, insulin

INTRODUCTION

Although the physiological responses to heat acclimatization have been long known and reviewed in several reports (1-3), relatively few investigations have addressed the hormonal changes which may be associated with or even necessary for this process to occur. Several earlier experiments were concerned with adrenocortical activation induced by acute exposure to thermal stress (4, 5), and Collins and Weiner(6) have reviewed data indicating generally reduced thyroid activity, equivocal adrenocortical involvement, and increased aldosterone and antidiuretic hormone secretion. With the advent of sensitive radioimmunoassays for constituents present in extremely small concentrations, it became possible to quantify additional hormones and neuroendocrines. For example, it was demonstrated that acute exposure to heat stress effected increases in plasma levels of growth hormone and luteinizing hormone (7), decreases in thyroid stimulating hormone with concurrent increases in prolactin (8), and increases in urinary excretion of histamine and thyroxine (9). However, nearly all of these reports described responses to acute heat exposure only. In the present studies we were primarily interested in the endocrinological responses occurring among humans during the process of heat acclimatization, particularly to determine whether any of these responses might be associated with the actual mechanism of acclimatization.

For example, Okada et al. (10) have demonstrated the inability of a heat-intolerant subject to respond to acute heat exposure with incremented growth hormone production, while remaining members of the experimental group increased plasma levels. It is unknown, however, whether the normal

response of human growth hormone or other neuroendocrines to acute heat exposure is related to the generalized stress response, to increased metabolic requirements, or possibly to fluid and electrolyte balance. Likewise, it is unknown whether the susceptibility of certain individuals to heat-induced disorders might be related to an attenuated neuroendocrinological response to the heat. Few, if any, investigations have addressed the potential relationships between the mechanisms of heat acclimatization and endocrinological response. Thus, we have designed the present experiments to relate, if possible, secretory rates, as manifested in plasma levels of several constituents, to the process of acclimatization to work in the heat.

METHODS

Eight healthy men were fully apprised of the protocol of this study, and served voluntarily for the duration of these experiments. Initially, they were divided into two groups based upon physical fitness and body weights. Levels of fitness were estimated by measuring the maximal oxygen uptake ($\dot{V}O_{2\ max}$) while running on a motor-driven treadmill by the conventional Douglas bag technique (11). The two groups, one of which would later walk in the heat (exercise) while the second remained inactive (sedentary), had mean $\dot{V}O_{2\ max}$ values of 53.4 ± 1.2 ml·kg^{-1}·min^{-1} and 53.0 ± 3.5 ml·kg^{-1}·min^{-1}, respectively. The mean age, height, and weight (±S.E.M.) of the 8 men were 20.3 ± 1 yr., 175 ± 2 cm, and 72.7 ± 4 kg.

Prior to the start of recurrent heat exposure the exercising group walked on a treadmill in a large environmental chamber for 90 min per day

for 8 days to achieve stable heart rates and rectal temperatures as well as neuroendocrinological measures. During this stabilization period chamber temperature was maintained at 21°C/10°C dry/wet bulb and the treadmill speed was constant at 5.6 $km \cdot hr^{-1}$. The sedentary group remained under identical environmental conditions in the same chamber, sitting upright on canvas-backed lounge chairs. On the final control day (day 8) brachiovenous catheters were inserted approximately one hour before the start of the experiment for serial blood sampling. The catheters were kept patent by flushing at 15-min intervals with 1 ml of sterile heparinized saline (1 U.S.P. unit $\cdot ml^{-1}$). Prior to the start of exercise in the chamber (time 0) and subsequently, at times 20, 45, and 90 min, 20 ml of blood were removed for biochemical analyses. The blood was brought to the laboratory where hematocrits were performed immediately. The plasma was then separated (7000 x g, 4°C) and frozen (-30°C) for subsequent analysis.

After completion of the 8-day stabilization period both exercise and sedentary groups were exposed to ambient conditions reported to effect heat acclimatization when combined with a mild exercise program (12). The temperature in the chamber was raised to 49°C/27°C dry/wet bulb; treadmill (5.6 $km \cdot hr^{-1}$) and wind (3.3 $km \cdot hr^{-1}$) speeds as well as length of exposure (90 min) were identical to those of the control period. On days 1 and 7 of heat exposure brachiovenous catheters were reintroduced. Heart rates were monitored electrocardiographically and rectal temperatures were measured by indwelling (7.5 cm) thermistors. Copper-constantan thermocouples affixed to the chest, medial forearm region, and mid-calf were used to

calculate mean-weighted skin temperatures although these data are not presented here. Nude weights were recorded immediately before and after each exposure; during the 90-min heat stress the exercising men received 250 ml each of tap water at 20, 40, 60, and 80 min, while subjects remaining sedentary received 175 ml at the same intervals, based on anticipated losses. Both groups of men were clad in shorts, wool socks, and athletic shoes during all chamber exposures. Test subjects remained under controlled dormitory conditions during non-experimental periods and were permitted only minimal additional physical activity. Standardized sleep-wakefulness conditions, dietary intake, and recreational activities were imposed upon the men for the duration of the experiment.

Radioimmunoassays were performed on the plasma constituents according to the following techniques: insulin by the method of Yalow and Berson (13), growth hormone after Schalch and Parker (14), cortisol after Ruder et al. (15), and the method of Murphy and Jachan (16) for total thyroxine. Results were corrected for small changes in hematocrit ratios by using the time 0 values (i.e. prior to heat exposure) as reference standards. Multiple analyses of variance were performed, and, where appropriate, statistical significance was established by the paired and non-paired t test. The null hypothesis was rejected at $p < .05$.

RESULTS

The mild intermittent exercise regimen imposed upon the walking group was effective in inducing heat acclimatization; this was reflected in significant reductions in final Tre on day 7 when compared to those of day

exercising group were significantly higher than those of the sedentary ($p<.05$).

Growth hormone data are depicted in Fig. 2, and the results indicate large interindividual variability with respect to the mild exercise program as well as the heat exposure. If one examines the data closely, there occurred a marked response to exercise culminating in a significant mean increase ($p<.05$; 0 vs 90 min) on the control day. Likewise, at the same time interval there was a significant increment in plasma levels of the walking group when compared with those of the sedentary ($p<.001$). However, the imposition of the heat stress did not significantly affect levels of growth hormone in either group. Among exercising men there was an attenuation of the growth hormone response to exercise on the first day of heat exposure which continued through the final day (e.g. day 1 vs day 7 90 min, $p<.05$, one-tailed).

Fig. 3 illustrates the effects of temperature and exercise on plasma levels of total T_4. While there was an effect of exercise on the first day of heat exposure (0 vs 90, $p<.001$), this effect had disappeared on the final heat day. Likewise, a significant ($p<0.02$) decrease (0 vs 90) on the control day among sedentary individuals may be part of a non-specific relaxation response.

Data for plasma insulin (Fig. 4) demonstrate generally reduced levels as a result of the mild exercise regimen. These reductions, while seemingly apparent on all three days, actually achieved maximal significance on the first heat day at all three sampling times after heat exposure

1 (38.25°C ± .12 vs 38.50°C ± .09, $p < .05$) with analogous results for heart rates (130.0 ± 8.9 vs 143.3 ± 7.5, $p < .025$). Sweat rate among walkers increased from 1.59 ± .23 kg to 1.73 ± .08 kg. All three parameters, commonly used to assess acclimatization levels, were unchanged between days 1 and 7 of heat exposure in the sedentary group. The relatively small, but significant, changes in the exercising group probably reflect residual summer acclimatization as these experiments were run in late October, early November.

Fig. 1 demonstrates interesting data relating to the adrenocortical response to recurrent exercise and heat exposure. Initially, if one examines the data for the control day (left side), there occurred a significant decrease between the time 0 values and those at times 20, 45, and 90 for the exercising group ($p < .025$, minimal significance). Despite less consistency among the sedentary group there again occurred a significant decrement ($p < 0.05$, 0 vs 90) in mean plasma levels of cortisol. When heat stress is introduced, marked differences between the exercising and sedentary groups emerged. That passive heat exposure led to a reduction in plasma cortisol levels was reflected in the consistent and significant decreases in plasma concentrations on both days 1 and 7 after 20, 45, and 90 min of heat exposure (e.g. day 7, 0 vs 45 min, $p < .001$). Alternatively, the mild exercise program imposed upon the walking group appeared to neutralize the repressive effects of heat exposure, and no significant differences were observed at any time intervals on day 1 or 7. For example, at 45 and 90 minutes on the first heat day values for the

($p < .05$, minimal significance). Among the sedentary group there were no significant changes at any of the time intervals selected on each of the three experimental days although heat exposure appeared to reduce levels in one individual on both days 1 and 7 of heat. Similarly, there were no significant inter-group differences in plasma insulin levels between the exercising and sedentary groups.

DISCUSSION

The extent of adrenal glucocorticoid secretion during heat stress in man has remained in question due to inadequate quantitative analyses, altered hepatic and renal clearance rates, the imposition of the added stress of work in the heat, acclimatization level, humidity, test protocol, and the degree of hyperthermia incurred. Several groups of workers have hypothesized that since exposure to high environmental temperatures decreases the need for active thermogenesis, reductions may occur in the activity of the hypophyseal-thyroidal-adrenal axis (17, 18). However, Collins et al. (4) presented data which indicated that although levels of plasma cortisol were suppressed during the first 60 min of exposure to 46°C dry bulb, 36°C wet bulb, these values returned to normal by 90 min and were significantly elevated by 120 min. They hypothesized that levels increased after a critical rectal temperature (38.3°C) was achieved. Generally, the results of the present study seem to support this hypothesis with significant decreases up to 45 min of heat exposure, and an apparent stabilization thereafter for both sedentary and exercising individuals. However, in terms of the heat acclimatization process, patterns of plasma cortisol

concentrations are remarkably similar on both days 1 and 7 of heat exposure, probably negating a potential role in the regulation of plasma volume and sodium retention.

The data present other interesting aspects related to plasma cortisol levels. For example, on the control day there were significant decreases from time 0 values in both the exercising and sedentary groups. Since no environmental stress had been imposed on this day, the relatively high time 0 levels ($\bar{X}$ = 11.9 ug%, exercising and 11.1 ug%, sedentary) would necessarily be attributable to the effects of catheterization and the novelty of the experimental situation, and the ensuing trend toward reduced levels could be explained by increasing familiarity and reduced anxiety. Several previous reports described high initial values of plasma cortisol which remained relatively stable even when additional stressors were imposed (19, 20); we later demonstrated (21) that exposure for 7 hours to a hot, wet environment with attendant mental fatigue evoked marked increases in plasma cortisol despite high initial levels.

The mild level of exercise imposed in this study would not be expected to elicit an adrenocortical response. For example, Hartley and his coworkers (22) demonstrated an adrenocortical response to graded exercise only at work loads of 98% of $\dot{V}O_{2\ max}$ with no decrease in response after physical training (23). Nonetheless, the present data, particularly those of the first day of mild exercise in combination with severe heat stress, demonstrated that the combined regimen prevented the decrements occurring among sedentary men, and this resulted in significant differences between

the two groups at 45 and 90 min.

Hartley et al. (22, 23) demonstrated a greater response of plasma growth hormone to moderate than heavy physical exercise. In the present experiments even the mild exercise of the control day induced a significant increase in growth hormone levels after 90 minutes. Although the ambient conditions in this study were similar to those used by Okada et al. (10), we were unable to observe significant increases among sedentary individuals in plasma levels of growth hormone. However, it should be noted that in several experiments utilizing animals the stress of a novel environment (24) or acute heat exposure (8) not only failed to induce increases in plasma growth hormone levels, but significant decrements were observed. Results of the present experiments confirmed the greater sensitivity of human growth hormone secretion to even mild exercise when compared to severe heat stress.

Although Collins and Weiner (6) reviewed data suggesting decreased thyroidal activity and oxygen consumption concurrent with heat exposure, most of the studies were done with animals and those involving humans were directed toward long-term exposures and natural acclimatization. The intermittent hyperthyreosis described by Sulman et al. (9), which was characterized by extremely high excretion rates of T_4 and a myriad of physiological disturbances, seemed to be exacerbated by heat stress (28 - 40^{o}C), but only in individuals with prodromal thyroid disorders. Yoshimura et al. (25) postulated that the seasonal variation of basal metabolic rate among Japanese may be related to decreased thyroid activity during the

summer season. Data from the present experiments confirmed minimal thyroid involvement during acute exposure to recurrent heat stress. In fact, the significant decrease among sedentary individuals on the control day (90 min) and on the first heat day (20 min) probably are more reflective of the quiescent metabolic state and low 0_2 consumption. On the first day of heat exposure there did occur a significant ($p<.001$) increase in total T_4 (0 vs 90 min); however, the mild exercise levels imposed upon these test subjects was evidently not sufficient to elicit consistently significant increases in plasma thyroxin at any other sampling times.

Although the response of plasma insulin levels to physical exercise and physical conditioning have been well established (22, 23, 26), to the best of our knowledge no research has addressed the potential role of insulin in the heat acclimatization process. In the present study, we have found that recurrent heat exposure among the sedentary group had no effect on levels of plasma insulin. The mild exercise regimen significantly reduced plasma insulin levels at each sampling time on the first heat day while on the final heat day an extremely high time 0 value in one test subject led to variances so high as to preclude significance. However, generally, the data can be interpreted as reflective of no significant changes in the insulin response to recurrent exercise in the heat.

We concluded from these studies that the combination of mild exercise and heat exposure in the case of the exercising group and passive recurrent heat exposure in the case of the sedentary group did elicit

significant responses in plasma hormone levels. However, if one examines closely the hormonal responses on day 1 (pre-acclimatization) and compares these with the alterations occurring on day 7 (post-acclimatization), one must also conclude that the patterns of response are quite similar on both days. While we are aware that plasma levels are dependent not only upon secretory rate but also upon blood flow distribution, hepatic metabolic rate, renal clearance rate, and target-organ uptake, we felt that serial sampling at relatively brief intervals would be adequate to detect changes in glandular activity. Actually, this was manifested in the changes noted in the case of several hormones when serial samples were examined. Overall, the patterns of hormone response indicated relatively little or no involvement of these hormones in the acclimatization process.

Acknowledgement

The technical, administrative, and logistical support of Ms's G. Farese, J. Cardillo, L. Elliott, E. Munro, and S. Beach and Mr.'s R. Byrom, S. O'Connor, M. Marsili, and D. Schoon and Army SP's Lussier, Criss, and Caleb is gratefully acknowledged. We gratefully acknowledge Mrs. A. Debra Caruso for typing the manuscript. We would especially like to thank the test volunteers for their cooperation and willingness to undergo the discomforts encountered.

The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

This human research study, in protocol form, was reviewed and approved by the Office of the Surgeon General for the Department of the Army in accordance with Army Regulation 70-25.

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Reg. 70-25 on Use of Volunteers in Research.

Figure Legend

Fig. 1 Effects of heat (49°C/27°C dry/wet bulb) and work (treadmill, 5.6 km/hr, 1.5 hr) on the plasma cortisol response of both the exercising (upper) and sedentary (lower) groups. The left panels display data for the control day, (21°C/10°C dry/wet bulb), center for day 1 of heat exposure, and right panels for day 7. Time 0 values refer to samples taken immediately prior to entrance into the chamber. Individual data for four men in each group are presented.

Fig. 2 Effects of heat and work on plasma growth hormone response for the exercising and sedentary groups. All conditions are identical to those in Fig 1.

Fig. 3 Effects of heat and work on plasma total T_4 concentrations in exercising and sedentary men. All conditions are the same as those in Fig 1.

Fig. 4 Effects of heat and work on plasma insulin response in exercising and sedentary men. For conditions, see Fig 1.

REFERENCES

1. Bass, D. E., Thermoregulatory and circulatory adjustments during acclimatization to heat in man, "Temperature, Its Measurement and Control in Science and Industry," New York, Reinhold, 3: 299, 1963.

2. Lind, A. R. and D. E. Bass, Optimal exposure time for development of acclimatization to heat, Fed Proc 22: 704, 1963.

3. Wyndham, C. H., The physiology of exercise under heat stress, Ann Rev Physiol 35: 193, 1973.

4. Collins, K. J., J. D. Few, T. J. Forward, and L. H. Giec, Stimulation of adrenal glucocorticoid secretion in man by raising the body temperature, J Physiol 202: 645, 1969.

5. Hellman, K., K. J. Collins, C. H. Gray, R. M. Jones, J. B. Lunnon, and J. S. Weiner, The excretion of urinary adrenocortical steroids during heat stress, J Endocrin 14: 209, 1956.

6. Collins, K. J, and J. S. Weiner, Endocrinological aspects of exposure to high environmental temperatures, Physiol Rev 48: 785, 1968.

7. Leppaluoto, J., T. Ranta, U. Laisi, J. Partanen, P. Virkkunen, and H. Lybeck, Strong heat exposure and adenohypophyseal hormone secretion in man, Horm Metab Res 7: 439, 1975.

8. Mueller, G. P., H. T. Chen, J. A. Dibbet, H. J. Chen, and J. Meites, Effects of warm and cold temperatures on release of TSH, GH, and prolactin in rats, Proc Soc Exptl Biol Med 147: 698, 1974.

9. Sulman, F. G., E. Tal, Y. Pfeifer, and E. Superstine, Intermittent hyperthyreosis - a heat stress syndrome, Horm Metab Res 7: 424, 1975.

10. Okada, Y., T. Matsuoka, and Y. Kumahara, Human growth hormone secretion during exposure to hot air in normal adult male subjects, J Clin Endocrin Metab 34: 759, 1972.

11. Maher, J. T., L. G. Jones, and L. H. Hartley, Effects of high altitude exposure on submaximal endurance capacity of men, J Appl Physiol 37: 895, 1974.

12. Maher, J. T., D. E. Bass, D. D. Heistad, E. T. Angelakos, and L. H. Hartley, Effect of posture on heat acclimatization in man, J Appl Physiol 33: 8, 1972.

13. Yalow, R. S., and S. A. Berson, Immunoassay of endogenous plasma insulin in man, J Clin Invest 39: 1157, 1960.

14. Schalch, D. S., and M. L. Parker, A sensitive double antibody immunoassay for human growth hormone in plasma, Nature, 203: 1141, 1964.

15. Ruder, H. J., R. L. Guy and M. D. Lipsett, A radioimmunoassay for cortisol in plasma and urine, J Clin Endocrin Metab 35: 219, 1972.

16. Murphy, B. E., and C. Jachan, The determination of thyroxine by competitive protein-binding analysis employing an anion exchange resin and radiothyroxine, J Lab Clin Med 66: 161, 1965.

17. MacFarlane, W. V., Endocrine functions in hot environments, Environmental Physiology and Psychology in Arid Conditions, Reviews of Research, Paris: UNESCO, 22: p 153, 1963.

18. Robinson, K. W., and W. V. MacFarlane, Urinary excretion of adrenal steroids during exercise in hot atmospheres, J Appl Physiol 12: 13, 1958.

19. Haibach, H., R. J. Underwood, M. A. Greer, and V. P. Vanderlaan, Failure to stimulate significant cortisol or growth hormone secretion in man by ether infusion, Experientia 26: 1146, 1970.

20. Rahe, R. H., Biochemical correlates of human behavior: Serum uric acid, cortisol, and cholesterol, Proc V World Cong Psychiat 1: 400, 1971.

21. Francesconi, R. P., B. J. Fine, and J. L. Kobrick, Heat and simulated high altitude: Effects on biochemical indices of stress and performance, Aviat Space Environ Med 47: 548, 1976.

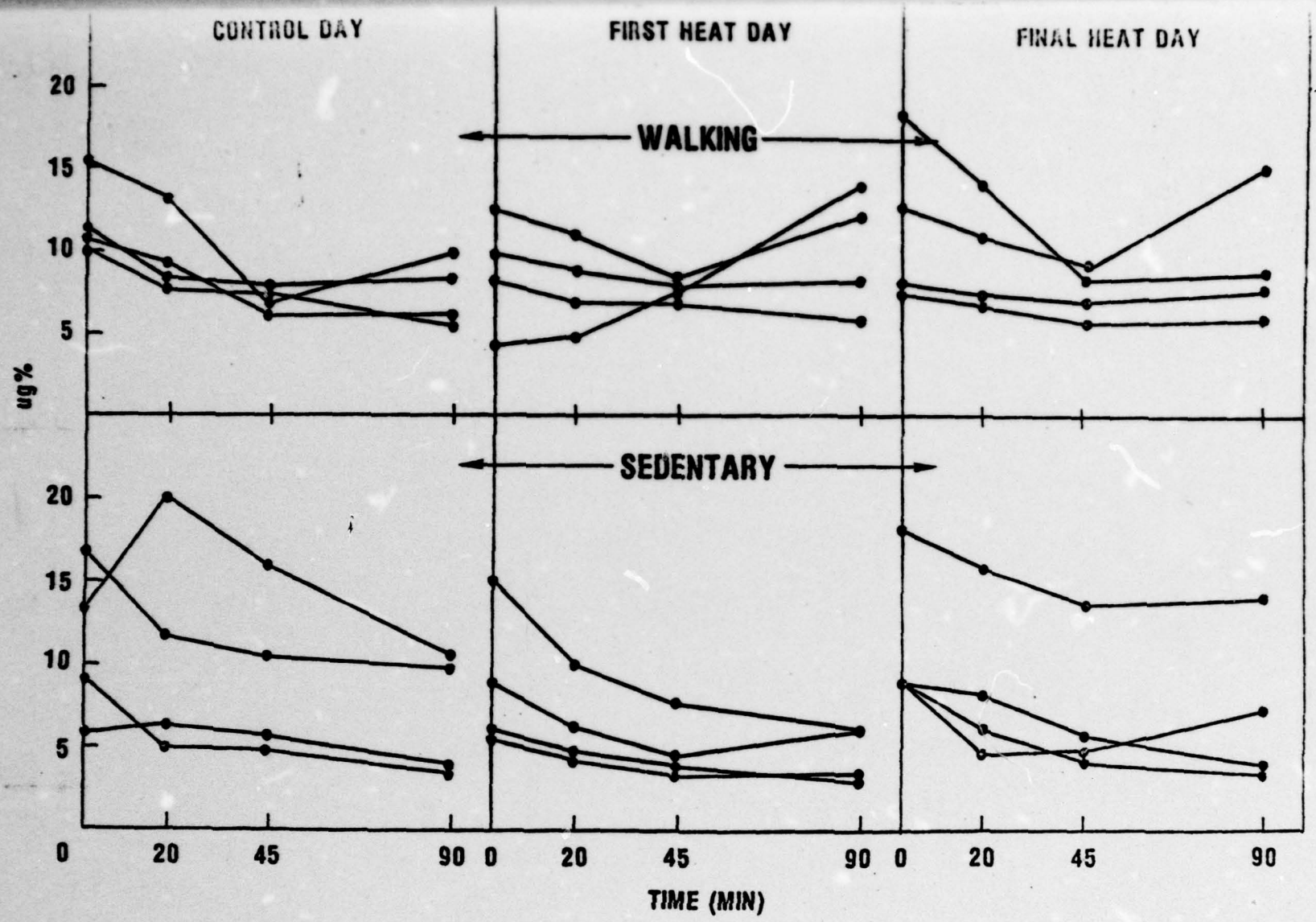
22. Hartley, L. H., J. W. Mason, R. P. Hogan, L. G. Jones, T. A. Kotchen, E. H. Mougey, F. E. Wherry, L. L. Pennington, and P. T. Ricketts, Multiple hormonal responses to graded exercise in relation to physical training, J Appl Physiol 33: 602, 1972.

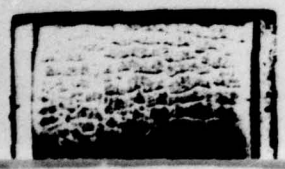
23. Hartley, L. H., J. W. Mason, R. P. Hogan, L. G. Jones, T. A. Kotchen, E. H. Mougey, F. E. Wherry, L. L. Pennington, and P. T. Ricketts, Multiple hormonal responsss to prolonged exercise in relation to physical training, J Appl Physiol 33: 607, 1972.

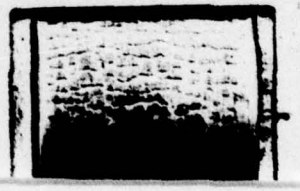
24. Seggie, J. A., and G. M. Brown, Stress response patterns of plasma corticosterone, prolactin, and growth hormone in the rat, following handling or exposure to novel environment, Can J Physiol Pharmocol 53: 629, 1975.

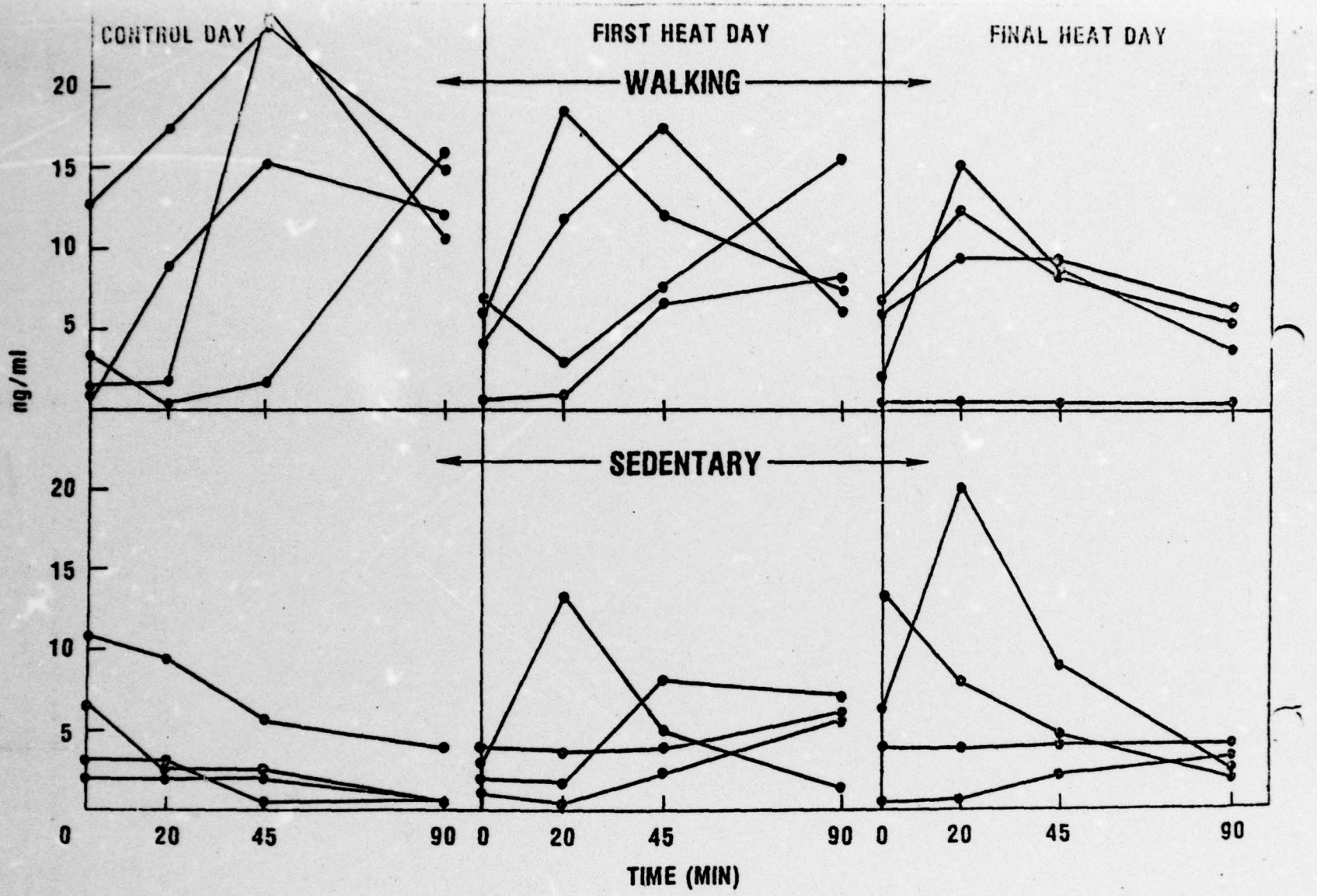
25. Yoshimura, M., S. Hori, and H. Yoshimura, Effect of high-fat diet on thermal acclimatization with special reference to thyroid activity, Jap J Physiol 22: 517, 1972.

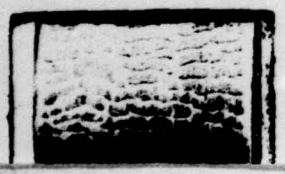
26. Hunter, W. M., and M. Y. Sukkar, Changes in plasma insulin levels during muscular exercise, J Physiol 196: 110P, 1968.

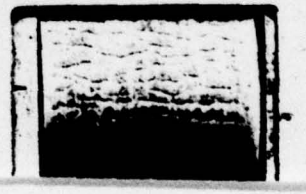

CONTROL DAY
FIRST HEAT DAY
FINAL HEAT DAY
WALKING
SEDENTARY
ug%
20
15
10
5
0
20
45
90
TIME (MIN)

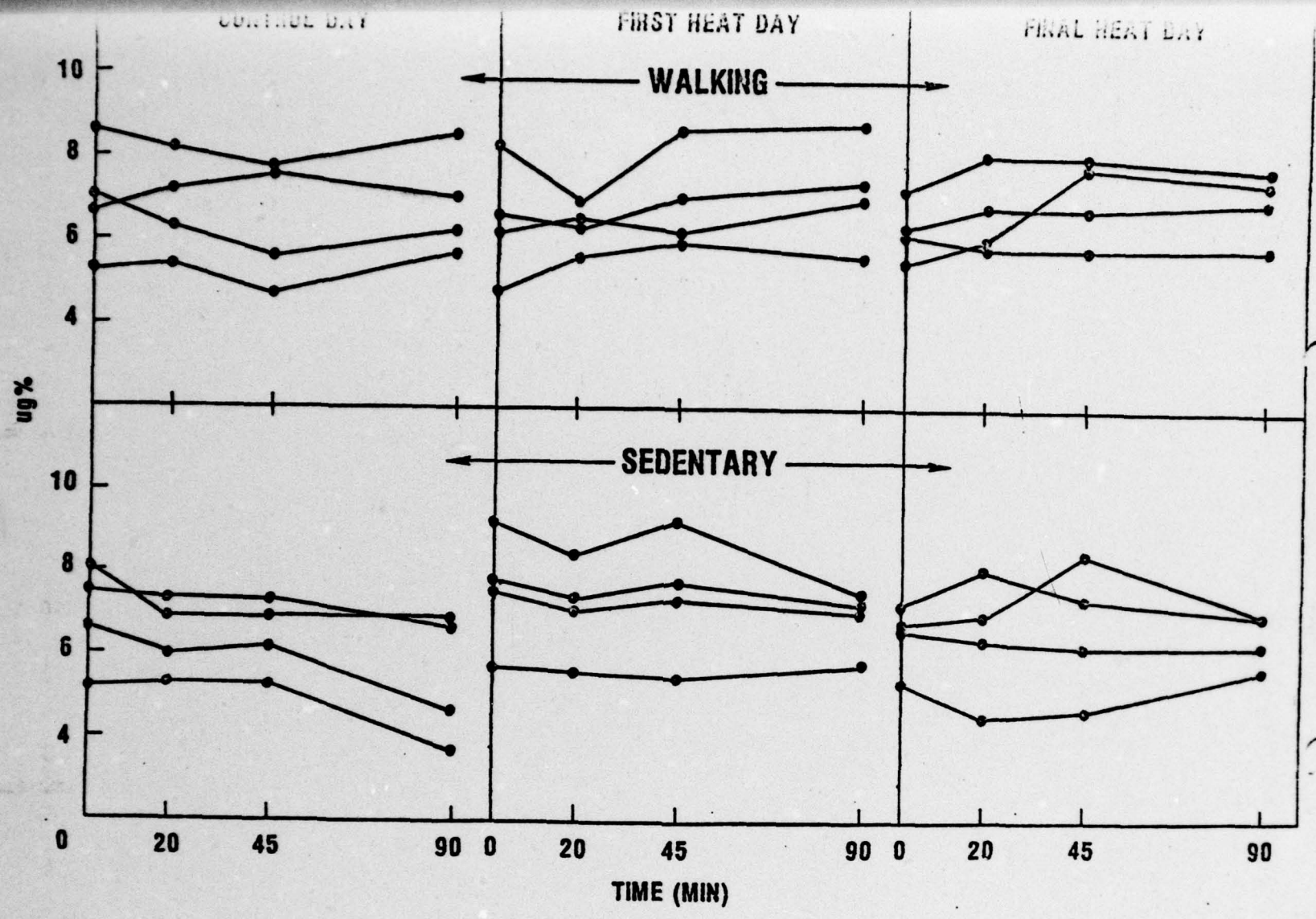


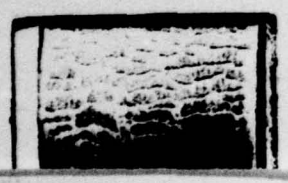


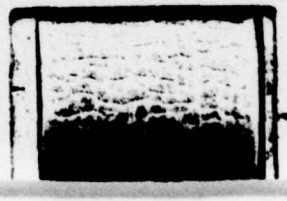

CONTROL DAY
FIRST HEAT DAY
FINAL HEAT DAY
WALKING
SEDENTARY
ng/ml
20
15
10
5
0
20
45
90
TIME (MIN)

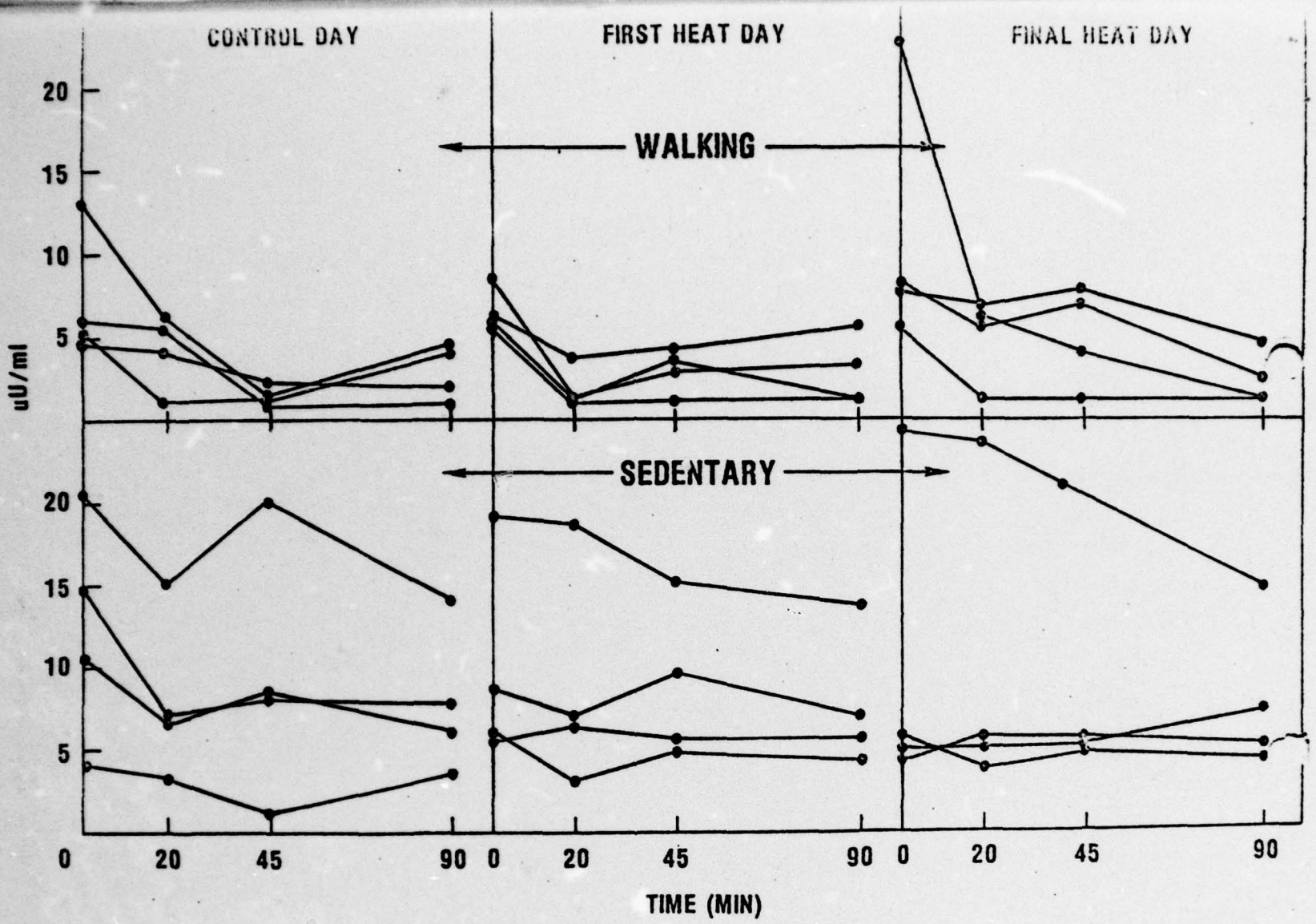


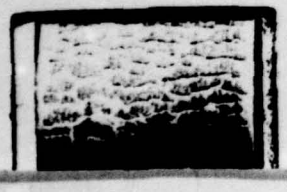


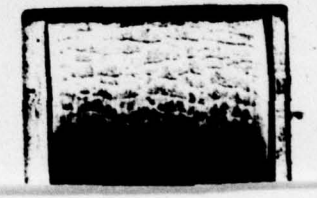

CONTROL DAY
FIRST HEAT DAY
FINAL HEAT DAY
WALKING
SEDENTARY
%g
10
8
6
4
0
20
45
90
TIME (MIN)






CONTROL DAY
FIRST HEAT DAY
FINAL HEAT DAY
WALKING
SEDENTARY
uU/ml
20
15
10
5
0
20
45
90
TIME (MIN)





UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE	READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER: M 9/77 (14) USARIEM-M-9/77	2. GOVT ACCESSION NO. / 3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle): (6) Recurrent Heat Exposure: Effects on Hormonal Responses in Resting and Exercising Men	5. TYPE OF REPORT & PERIOD COVERED
	6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s): (10) R. P. Francesconi, J. Maher, J. Mason, G. Bynum	8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS: U. S. Army Research Institute of Environmental Medicine, Natick, MA 01760	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS: 3E161102BS08 / 74182005 (16) (19)
11. CONTROLLING OFFICE NAME AND ADDRESS: U. S. Army Medical Research & Development Command, Washington, DC 20314 (11)	12. REPORT DATE: 14 Feb 77
	13. NUMBER OF PAGES: 15 (12) 25p.
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office): Same	15. SECURITY CLASS. (of this report): Unclassified
	15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Distribution of this document is unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

NA

18. SUPPLEMENTARY NOTES

NA

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Heat acclimatization, cortisol, growth hormone, thyroxin, insulin

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

Heat acclimatization was induced in a group of healthy young men by walking on a treadmill (5.6 km / hr^{-1}, 49°C/27°C dry/wet bulb, 90 min, day^{-1}, 7 days), and confirmed by recording significantly reduced final rectal temperatures and heart rates on the seventh day of exercise in the heat. A second group, paired for maximal O_2 consumption and body weight, remained sedentary under identical environmental conditions. After correcting for minor changes in hematocrit, both the sedentary and exercising groups demonstrated

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

040850

significant reductions in plasma cortisol on the control day indicating a strong anticipatory response. Heat exposure reduced plasma cortisol levels in the sedentary men, but the mild exercise program neutralized these effects in the exercising group. Patterns of alteration of growth hormone indicated a significant response even to the mild exercise program described here, while heat stress, pre- and post-acclimatization, seemed to have no effect upon plasma levels. Plasma total T_4 levels demonstrated several randomized changes which, however, did not reflect decreased output under the environmental conditions described here for either the walking or sedentary group. The mild exercise program elicited significantly reduced levels of plasma insulin which were not affected by the recurrent heat exposure. Generally, while the recurrent heat exposure effected hormonal responses among both exercising and sedentary groups, these alterations were not correlated with the process of heat acclimatization.