

AD-A273 292



AD \_\_\_\_\_



REPORT NO T94-6

RESPONSES TO MODERATE AND LOW SODIUM DIETS  
DURING EXERCISE-HEAT ACCLIMATION

**U S ARMY RESEARCH INSTITUTE  
OF  
ENVIRONMENTAL MEDICINE  
Natick, Massachusetts**

NOVEMBER 1993

**DTIC  
SELECT  
NOV 30 1993  
S B D**



Approved for public release; distribution unlimited.

**UNITED STATES ARMY  
MEDICAL RESEARCH & DEVELOPMENT COMMAND**

**93-29097**



**93 11 23 1 25**

# REPORT DOCUMENTATION PAGE

Form Approved  
OMB No 0704-0188

Public reporting burden for this collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to Washington Headquarters Services, Directorate for Information Operations and Reports, 1215 Jefferson Davis Highway, Suite 1204, Arlington, VA 22202-4302, and to the Office of Management and Budget, Paperwork Reduction Project (0704-0188), Washington, DC 20503.

<b>1. AGENCY USE ONLY (Leave blank)</b>		<b>2. REPORT DATE</b> Nov 93	<b>3. REPORT TYPE AND DATES COVERED</b> final	
<b>4. TITLE AND SUBTITLE</b> Responses to Moderate and Low Sodium Diets During Exercise-Heat Acclimation			<b>5. FUNDING NUMBERS</b>	
<b>6. AUTHOR(S)</b> Lawrence E. Armstrong, Roger W. Hubbard E. Wayne Askew, Jane P. DeLuca, Catherine Obrien, Angela Pasqualichhio, and Ralph Francesconi				
<b>7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)</b> US Army Res Inst of Env Med Kansas St. Natick, MA 01760			<b>8. PERFORMING ORGANIZATION REPORT NUMBER</b>	
<b>9. SPONSORING MONITORING AGENCY NAME(S) AND ADDRESS(ES)</b>			<b>10. SPONSORING MONITORING AGENCY REPORT NUMBER</b>	
<b>11. SUPPLEMENTARY NOTES</b>				
<b>12a. DISTRIBUTION AVAILABILITY STATEMENT</b>  Approved for public release; distribution is unlimited.			<b>12b. DISTRIBUTION CODE</b>	
<b>13. ABSTRACT (Maximum 200 words)</b>  This investigation examined whether low sodium (Na <sup>+</sup> ) (LNA; 68 mEq Na <sup>+</sup> ·d <sup>-1</sup> ) or moderate Na <sup>+</sup> (MNA; 137 mEq Na <sup>+</sup> ·d <sup>-1</sup> ) intake allowed humans to maintain health, exercise, and physiologic function during 10 days of prolonged exercise-heat acclimation (HA). Seventeen volunteers, ages 19 to 21, consumed either LNA (n=8) or MNA (n=9) during HA (41°C, 21% RH; treadmill walking for 30 min·h <sup>-1</sup> , 8 h·d <sup>-1</sup> at 5.6 km·h <sup>-1</sup> , 5% grade), which resulted in significantly reduced heart rate, rectal temperature, and urine Na <sup>+</sup> for both groups. There were few between-diet differences in any variables measured. Mean plasma volume in LNA expanded significantly less than in MNA by Days 11 and 15, but reached the MNA level on Day 17 (+12.3 vs. +12.4%). The absence of heat illness, the presence of normal physiologic responses, and the total distance walked indicated successful and similar HA with both levels of dietary Na <sup>+</sup> .				
<b>14. SUBJECT TERMS</b> fluid-electrolyte balance, rectal temperature, cardiovascular, hematology, potassium, urine			<b>15. NUMBER OF PAGES</b> 15	
			<b>16. PRICE CODE</b>	
<b>17. SECURITY CLASSIFICATION OF REPORT</b> Unclassified	<b>18. SECURITY CLASSIFICATION OF THIS PAGE</b> Unclassified	<b>19. SECURITY CLASSIFICATION OF ABSTRACT</b> Unclassified	<b>20. LIMITATION OF ABSTRACT</b> UL	

## Responses to Moderate and Low Sodium Diets During Exercise-Heat Acclimation

Lawrence E. Armstrong, Roger W. Hubbard,  
E. Wayne Askew, Jane P. De Luca, Catherine O'Brien,  
Angela Pasqualicchio, and Ralph P. Francesconi

This investigation examined whether low sodium ( $\text{Na}^+$ ) (LNA; 68 mEq  $\text{Na}^+\cdot\text{d}^{-1}$ ) or moderate  $\text{Na}^+$  (MNA; 137 mEq  $\text{Na}^+\cdot\text{d}^{-1}$ ) intake allowed humans to maintain health, exercise, and physiologic function during 10 days of prolonged exercise-heat acclimation (HA). Seventeen volunteers, ages 19 to 21, consumed either LNA ( $n=8$ ) or MNA ( $n=9$ ) during HA (41°C, 21% RH; treadmill walking for 30  $\text{min}\cdot\text{h}^{-1}$ , 8  $\text{h}\cdot\text{d}^{-1}$  at 5.6  $\text{km}\cdot\text{h}^{-1}$ , 5% grade), which resulted in significantly reduced heart rate, rectal temperature, and urine  $\text{Na}^+$  for both groups. There were few between-diet differences in any variables measured. Mean plasma volume in LNA expanded significantly less than in MNA by Days 11 and 15, but reached the MNA level on Day 17 (+12.3 vs. +12.4%). The absence of heat illness, the presence of normal physiologic responses, and the total distance walked indicated successful and similar HA with both levels of dietary  $\text{Na}^+$ .

**Key Words:** fluid-electrolyte balance, rectal temperature, cardiovascular, hematology, potassium, urine

The reported dietary  $\text{Na}^+$  intake of adults in the United States ranges from 78 to 218 mEq  $\text{Na}^+$  per day, depending on the method of assessment (21, 22). Empirical studies have demonstrated that this intake is greater than the levels consumed by the inhabitants of several tropical countries (4, 6, 15), and that the basal biologic human requirement for  $\text{Na}^+$  ranges from only 2 to 8 mEq  $\text{Na}^+$  per day (6). These facts, and previous studies that related dietary sodium chloride ( $\text{NaCl}$ ) to hypertension (27), led to a recent dietary recommendation of 104 mEq of  $\text{Na}^+$  per day (21) for U.S. residents living in temperate climates.

However, observations made in hot environments (22) suggest that this dietary recommendation may be inadequate because of increased daily  $\text{Na}^+$  losses during exercise in the heat. For example, Denton (8) reported losses of up to

---

Armstrong and Pasqualicchio are with The University of Connecticut, Human Performance Laboratory, Box U-110, 2095 Hillside Road, Storrs, CT 06269-1110. Hubbard, Askew, De Luca, O'Brien, and Francesconi are with the U.S. Army Research Institute of Environmental Medicine, Natick, MA 01760-5007.

408 mEq Na<sup>+</sup>·d<sup>-1</sup> in the sweat of unacclimatized humans in hot climates. Several laboratory studies concluded that humans who eat low Na<sup>+</sup> diets and perform strenuous exercise in the heat have an increased risk (1, 14) or incidence of heat exhaustion (26) or heat syncope (3). Other experts (5, 19, 25) used field observations to derive Na<sup>+</sup> recommendations as high as 221–816 mEq Na<sup>+</sup> per day. In addition, recent animal research has demonstrated that heat exposure following Na<sup>+</sup> depletion may have severe effects on heat tolerance (11) and on thermoregulatory, endocrinologic, and hematologic variables (7).

Human exercise-heat acclimation (HA) results in a series of physiologic changes that reduce the strain caused by heat stress and improve one's ability to live and exercise in a hot environment (10, 12, 13, 28). Laboratory studies of HA (2, 4) and dietary Na<sup>+</sup> intake (6, 22) have suggested, in contrast to the findings above, that humans function well when consuming relatively low Na<sup>+</sup> diets ranging from 33 to 103 mEq Na<sup>+</sup>·d<sup>-1</sup>. Unfortunately, those human studies often did not (a) involve prolonged exercise on successive days, (b) report changes in the classical indices of HA (e.g., heart rate, rectal temperature, plasma volume), (c) allow ample time for dietary Na<sup>+</sup> stabilization prior to HA, or (d) evaluate the effects of salt depletion apart from the effects of water depletion by controlling water intake. The inconsistencies described above may also be found in the literature because fewer than 3% of all human HA studies (1, 12) have controlled dietary Na<sup>+</sup> consumption, distinguished between the Na<sup>+</sup> requirements of non-acclimated and acclimated humans, or used low dietary Na<sup>+</sup> as a treatment (1).

Therefore the purpose of the current investigation was to evaluate the effects of moderate (137 mEq Na<sup>+</sup>; abbreviated MNA) and low (68 mEq Na<sup>+</sup>; abbreviated LNA) Na<sup>+</sup> diets on thermoregulatory, cardiovascular, hematologic, and fluid-electrolyte variables during 10 consecutive days of prolonged intermittent exercise (8 h·d<sup>-1</sup>) in a simulated desert environment.

Three features of the current study were noteworthy. First, an initial 7-day dietary stabilization period (MNA) was used for both groups. Second, subjects were confined to a climatic chamber facility during the entire 17-1/2-day course of this research and ate only the food provided. Third, the water losses in sweat and urine were replaced each hour during HA trials to reduce the likelihood of water-depletion heat exhaustion (14). The current experiment is relevant to athletic, military, and industrial populations because (a) the dietary Na<sup>+</sup> intake of the U.S. population has decreased in recent years (21, 22), (b) caloric and Na<sup>+</sup> intake typically decrease during the initial days of living in a hot environment, and (c) the maintenance of intravascular and intracellular fluid-electrolyte balance is essential to prolonged exercise in the heat.

## Methods

The subjects of this investigation were 17 males who gave their informed and voluntary consent to participate in the current investigation and underwent a medical examination. Selected physical characteristics for both treatment groups appear in Table 1. The moderate spring climatic conditions and a 30-day activity/heat exposure questionnaire verified that subjects were neither highly trained nor exposed to regular, natural climatic heat acclimatization episodes prior to this investigation.

Table 1

Day 1 Mean (±SE) Characteristics of Subjects Consuming LNA and MNA

Variable	Diet		Statistical significance
	LNA (n=8)	MNA (n=9)	
Age (yrs)	20 ± 1	20 ± 1	ns
Height (cm)	180.1 ± 2.3	178.7 ± 2.3	ns
Mass (kg)	79.80 ± 3.18	77.86 ± 3.80	ns
Estimated body fat (%)	14 ± 1	14 ± 1	ns
VO <sub>2</sub> peak (ml·kg <sup>-1</sup> ·min <sup>-1</sup> )	45.73 ± 1.69	47.09 ± 1.54	ns
HR peak (beats·min <sup>-1</sup> )	207 ± 9	211 ± 7	ns

ns = not significantly different, *p* > 0.05.

Reprinted with permission from *Nutritional Needs in Hot Environments*. Copyright 1993 by the National Academy of Sciences. Courtesy of the National Academy Press, Washington, DC.

During the week prior to the dietary equilibration period, a peak aerobic power (VO<sub>2</sub> peak) test was performed on a motorized treadmill using a multistage, continuous protocol. Expired respiratory gases were sampled continuously by a precalibrated, computerized on-line system. Exercise was terminated when the subject signaled for volitional termination. VO<sub>2</sub> peak was determined by observing two consecutive 30-sec VO<sub>2</sub> values that were less than 150 ml O<sub>2</sub>·kg<sup>-1</sup>·min<sup>-1</sup> and different. Following a 10-min rest, each subject repeated the terminal speed and grade to verify VO<sub>2</sub> peak. The peak heart rate was recorded during this test via an electrocardiographic telemetry system. Each subject's percent body fat was estimated on Days 1, 8, and 17, using skinfolds (six sites) and the body composition formula of Jackson and Pollock (16).

During this 17-1/2-day study, subjects were housed 24 hrs a day in a research building that contained sleeping, dining, and environmentally controlled chamber facilities. Upon awakening each morning (Days 1–18), the subjects were measured for the following: nude body mass (±50 g), first void urine specific gravity (refractometer), and first void urine Na<sup>+</sup> and K<sup>+</sup> concentrations (flame photometer). During the initial 7-day dietary equilibration period (Days 1–7), all 17 subjects consumed MNA (137 mEq Na<sup>+</sup>·d<sup>-1</sup> [8 g NaCl·d<sup>-1</sup>]) and were housed in an ambient temperature of 21°C. During the subsequent 10-day HA period (Days 8–17), 9 subjects continued to consume MNA and 8 subjects began to consume LNA (68 mEq Na<sup>+</sup>·d<sup>-1</sup> [4 g NaCl·d<sup>-1</sup>]). Food items were evaluated for Na<sup>+</sup> content via inductively coupled emission spectrometer (Plasma 1000 model, Perkin Elmer, Inc.). All subjects were assigned randomly to either LNA or MNA; two LNA and MNA trials were performed during the months of March and May.

Subjects ate a diet of defined sodium composition, provided by a team of nutritionists, and were instructed that if they consumed other foods they would be dismissed from all phases of this investigation. A proctor was present at all times to ensure that no subject left and that no food entered the research building.

Breakfast and dinner were consumed in the dormitory kitchen (21°C) on Days 8-17, while lunch was eaten in the environmental chamber (41°C) during the fifth rest period. Three primary meals and two snacks provided subjects with 55% carbohydrate, 13% protein, and 32% fat in both LNA and MNA. This diet was designed to provide sufficient carbohydrates and calories (3,600 Kcal·d<sup>-1</sup>) for treadmill walking (22.4 km·day<sup>-1</sup>) and sedentary living. Subjects drank assorted beverages (e.g., Kool Aid™, fruit juice; known Na<sup>+</sup> content) ad libitum when not in the climatic chamber (15.5 h·d<sup>-1</sup>).

Before entering the heat (Days 8-17), subjects had a catheter implanted into an antecubital vein. Blood samples were collected prior to exercise on Days 8, 11, 15, and 17. No blood was drawn on Day 1 because the 7-day dietary equilibration period per se was not the object of this study. Subjects entered the 41°C environment and stood quietly for 20 min before the sample was drawn. A fresh, heparinized aliquot was analyzed in triplicate for hematocrit (microhematocrit technique), hemoglobin (cyanomethemoglobin method, Hycel, Inc.), and total plasma protein (refractometer, Fisher Scientific). The percent change in plasma volume (%ΔPV) was calculated from hematocrit and hemoglobin by the method of Dill and Costill (9). The mean corpuscular hemoglobin concentration (MCHC) was calculated by using the following formula, in which hemoglobin was expressed as g% and hematocrit as %:

$$\text{MCHC} = (\text{hemoglobin} / \text{hematocrit}) \times 100$$

Serum was assayed for Na<sup>+</sup> and K<sup>+</sup> (flame photometer, Rainin Instruments). Plasma osmolality was determined via the freezing point depression technique. Aliquots of plasma were frozen for subsequent analysis of colloid oncotic pressure (COP) with a colloid osmometer (Wescor, Inc.).

Daily HA trials were conducted in ambient conditions of 41°C, 21% relative humidity (% RH), and 1.2 m·s<sup>-1</sup> air speed for 8 h·d<sup>-1</sup>. During the remainder of each day (15.5 h·d<sup>-1</sup>), subjects lived at an ambient temperature of 21°C in an effort to simulate a 24-hr desert temperature cycle. Exercise involved eight periods of alternating rest (30 min·h<sup>-1</sup>) and moderate (5.6 km·h<sup>-1</sup>, 5% grade; estimated exercise intensity 40-45% VO<sub>2max</sub>) treadmill exercise (30 min·h<sup>-1</sup>) while wearing shorts, socks, and sneakers. Rectal temperature (T<sub>re</sub>: probe inserted 8 cm beyond the anal sphincter) and heart rate (HR; electrocardiographic telemetry system, Hewlett Packard, Inc.) were recorded during every HA trial.

Exercise was terminated and subjects rested in the heat for the remainder of the trial if HR exceeded 180 beats·min<sup>-1</sup>, if T<sub>re</sub> exceeded 39.5°C, or if T<sub>re</sub> rose 0.6°C during any 5-min period. Body mass was measured (±50 g) immediately prior to exercise and at the end of each hour of exercise (corrected for water intake, urine output, fecal excretion, and food consumption) to allow the calculation of hourly evaporative water loss (i.e., sweat rate).

Subjects drank pure water (<1 mEq Na<sup>+</sup>·l<sup>-1</sup> at 10-15°C) ad libitum from canteens, during treadmill walking and rest periods. Because dehydration could have altered physiologic variables, and because hypohydration could have altered the normal course of HA, body mass was maintained each hour by requiring that subjects drink, at the end of each rest period, a volume of pure water that matched the amount of body mass not replaced by ad libitum drinking.

Statistical significance was tested by using a repeated measures ANOVA

with Tukey's post hoc analysis. The two factors in this design were diet (LNA and MNA) and days (Days 1-17, Days 8-17, and Days 8, 11, 15, 17). The null hypotheses were rejected at the  $p=0.05$  confidence level. All data were expressed as mean ±SE.

## Results

### Morning Body Mass and Urinalysis

The mean morning body mass values for LNA and MNA (Days 1-18) appear in Figure 1. There were no between-diet differences ( $p>0.05$ , ns). Between-day differences ( $p<0.001$ ) were observed in the body mass of LNA, in that Days 10-15 were significantly lower ( $p<0.001$ ) than Day 8, the initial day of heat exposure. The day-to-day body mass fluctuations in LNA and MNA may have involved changes in body fat, fat-free mass, or total body water. However, estimates of percent body fat showed no significant diet or day effects: Day 1 = 14 ±1% (LNA), 14 ±1% (MNA); Day 8 = 13 ±1% (LNA), 14 ±2% (MNA); Day 17 = 14 ±1% (LNA), 15 ±1% (MNA). Analyzing the data as the percent change from baseline values did not alter the interpretation of these results.

The mean (±SE) morning urine specific gravity values for LNA and MNA (Days 1-18) exhibited no between-diet differences ( $p>0.05$ , ns); these values ranged from 1.016 to 1.023 (LNA) and from 1.018 to 1.023 (MNA) on Days 8-18. All mean urine specific gravity values indicated normal hydration status for both LNA and MNA on all days.

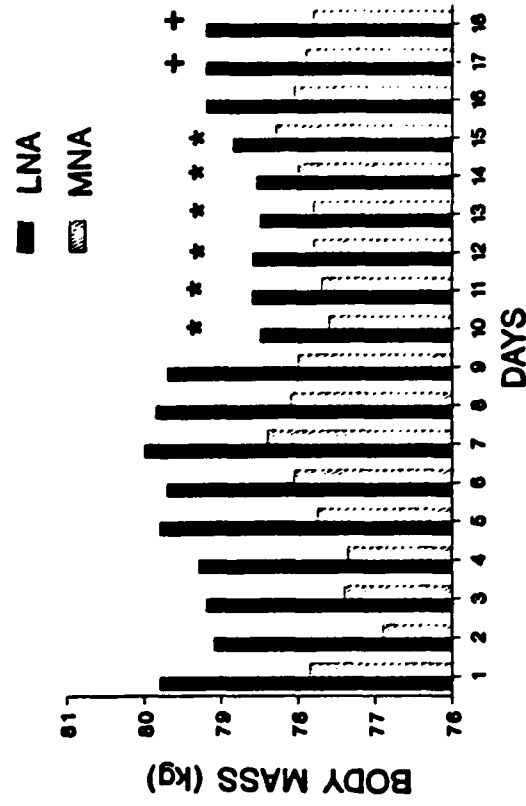


Figure 1 — Mean morning body mass values of LNA ( $n=8$ ) and MNA ( $n=9$ ) on Days 1-18. No significant between-diet differences were observed. Both LNA and MNA consumed MNA on Days 1-7. \*LNA on Days 10-15 was significantly different from Day 8. †Significant Diet × Day interaction was observed on Days 17 and 18.

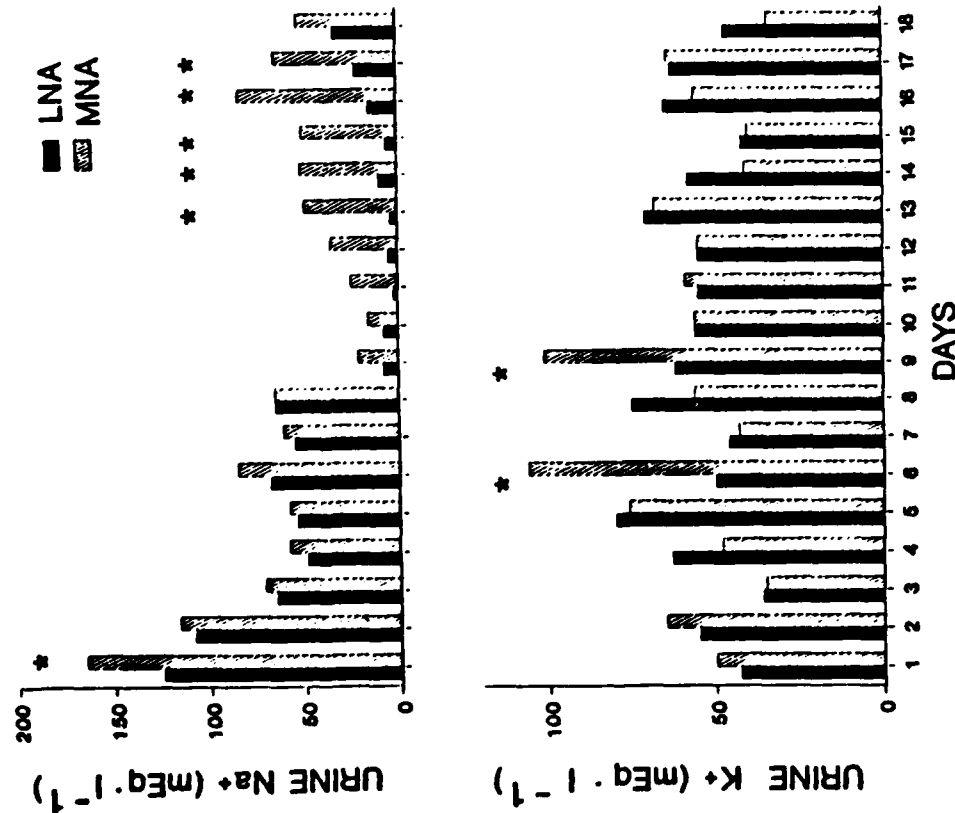


Figure 2 — Mean Na<sup>+</sup> and K<sup>+</sup> concentrations (mEq·l<sup>-1</sup>) of urine samples collected from LNA and MNA after awakening each morning. \*Represents significant between-diet (LNA vs. MNA) differences ( $p < .05$ ) in Na<sup>+</sup> and K<sup>+</sup>. Reprinted with permission from *Nutritional Needs in Hot Environments*. Copyright 1993 by the National Academy of Sciences. Courtesy of the National Academy Press, Washington, DC.

Figure 2 presents the concentrations of Na<sup>+</sup> and K<sup>+</sup> (mEq·l<sup>-1</sup>) in the initial morning urine samples. The extremely low mean Na<sup>+</sup> concentration on Days 9–15 indicated that LNA adhered to the salt-restricted dietary regimen. The significant between-diet (LNA vs. MNA) differences ( $p < .05$  to  $.001$ ) in Na<sup>+</sup> and K<sup>+</sup> are represented by asterisks. The differences in urinary Na<sup>+</sup> were attributed to differential Na<sup>+</sup> consumption and conservation, while differences in urinary K<sup>+</sup> were of unknown origin and may have involved type I statistical errors of

null hypothesis testing. Significant day-to-day differences in urinary Na<sup>+</sup> (not shown in Figure 2) were identified for LNA between Day 1 and Days 3–18 ( $p < .05$  to  $.001$ ), as well as between Day 8 and Days 9–17 ( $p < .05$  to  $.001$ ). Significant day-to-day differences were observed for urine Na<sup>+</sup> in group MNA between Day 1 and Days 2–18 ( $p < .01$  to  $.001$ ).

**Preexercise Blood Measurements**

Mean ( $\pm$ SE) values for specific circulatory variables in Table 2 represent pre-exercise samples drawn at 7:30 a.m. on Days 8, 11, 15, and 17. A noteworthy between-diet difference in % $\Delta$ PV occurred on Days 11 and 15. Although the LNA group exhibited a significantly smaller ( $p < .05$ ) expansion of PV than MNA on Days 11 and 15, both treatment groups manifested a similar % $\Delta$ PV by Day 17 (+12.3% vs. +12.4%). Similar, significant between-day decreases (Table 2) were identified for total plasma protein in LNA and MNA (Day 8 vs. Days 11, 15, 17;  $p < .01$ ), even though PV expansion exhibited significant between-group differences (LNA vs. MNA,  $p < .05$ ) on Days 11 and 15. The interaction term of the ANOVA of % $\Delta$ PV was significant between Days 8–11 and between Days

**Table 2**  
Blood Variables of LNA (n=8) and MNA (n=9) at the Beginning of HA Trials on Days 8, 11, 15, and 17

Measurement	Diet	Day 8	Day 11	Day 15	Day 17
Serum Na <sup>+</sup> (mEq·l <sup>-1</sup> )	LNA	137 $\pm$ 2	138 $\pm$ 2	140 $\pm$ 3**	137 $\pm$ 2***
	MNA	140 $\pm$ 1	140 $\pm$ 1	140 $\pm$ 1	139 $\pm$ 2
Serum K <sup>+</sup> (mEq·l <sup>-1</sup> )	LNA	4.5 $\pm$ 0.2	4.5 $\pm$ 0.3	4.5 $\pm$ 0.2	4.4 $\pm$ 0.1
	MNA	4.2 $\pm$ 0.2	4.3 $\pm$ 0.2	4.3 $\pm$ 0.2	4.3 $\pm$ 0.2
Plasma osmolality (mOsmol·kg <sup>-1</sup> )	LNA	287 $\pm$ 1	285 $\pm$ 3	286 $\pm$ 3	287 $\pm$ 4
	MNA	287 $\pm$ 2	287 $\pm$ 2	288 $\pm$ 4	289 $\pm$ 2
% $\Delta$ PV	LNA	-	2.0 $\pm$ 1.8	6.6 $\pm$ 2.0*	12.3 $\pm$ 1.7*
%	MNA	-	11.5 $\pm$ 2.8	12.8 $\pm$ 2.2	12.4 $\pm$ 1.7
MCHC (g·100 ml rbc <sup>-1</sup> )	LNA	33.90 $\pm$ 0.94	33.76 $\pm$ 1.13	34.40 $\pm$ 1.18	33.79 $\pm$ 0.68
	MNA	34.00 $\pm$ 0.90	34.34 $\pm$ 0.78	34.65 $\pm$ 1.01	33.87 $\pm$ 0.79
Total plasma protein (g·100 ml <sup>-1</sup> )	LNA	8.8 $\pm$ 0.5	7.5 $\pm$ 0.3**	7.1 $\pm$ 0.4**	7.2 $\pm$ 0.3**
	MNA	8.6 $\pm$ 0.9	7.4 $\pm$ 0.4**	7.1 $\pm$ 0.3**	7.3 $\pm$ 0.3**
COP (mm Hg)	LNA	27.4 $\pm$ 1.3	28.6 $\pm$ 1.9	27.0 $\pm$ 1.4	26.3 $\pm$ 1.7
	MNA	29.7 $\pm$ 2.5 **	28.0 $\pm$ 2.3 **	28.2 $\pm$ 1.8	29.3 $\pm$ 2.2

\*Significantly different,  $p < .05$ , .01, from Day 11; \*\* $p < .05$ , .01, from Day 8; \*\*\* $p < .05$ , from Day 15.  
 †Significant difference,  $p < .05$ , between LNA and MNA; ††significant interaction effect,  $p < .05$ , .01, from Day 8 to 11, and from Day 11 to 15.  
 Reprinted with permission from *Nutritional Needs in Hot Environments*. Copyright 1993 by the National Academy of Sciences. Courtesy of the National Academy Press, Washington, DC.

11-15. Similarly, COP exhibited significant interaction terms on those days, suggesting that LNA and MNA resulted in different patterns of fluid shifts during Days 8-15 of HA.

### Responses During HA Trials

The treadmill exercise during daily HA trials required that subjects walk a total distance of 224 km (139.1 mi), up to a 5% grade, in 10 days. Although no cases of heat exhaustion, heat syncope, heat cramps, or heatstroke (14) occurred, this prolonged exercise resulted in numerous foot blisters and minor orthopaedic injuries, which allowed only 4 subjects to complete all 80 of the 30-min exercise bouts. The total distance walked by LNA and MNA, respectively, were  $168.7 \pm 18.9$  and  $185.4 \pm 10.1$  km ( $104^{-1}$  ( $p > .05$ , ns); these distances were not significantly correlated with  $\dot{V}O_2$ -peak ( $p > .05$ , ns). Given that the physiologic responses of LNA and MNA might be differentially affected by differences in the total distance walked, a statistical ANCOVA was performed which covaried the total distance walked with each variable measured during this investigation. The ANCOVA indicated that only one factor was significantly affected: HR for both LNA and MNA. This was interpreted as a cardiovascular training response in both groups and was recognized as a minor limitation in the interpretation of the HR data only.

Figure 3 illustrates the HR values for LNA and MNA on all days of HA, during Exercise Periods 1, 2, 4, 6, and 8. There were no between-diet (LNA vs. MNA) differences in HR on any day. Both MNA and LNA resulted in similar Day 8 versus Day 17 decreases in HR (e.g., 140 beats·min<sup>-1</sup> on Day 8 vs. 121 beats·min<sup>-1</sup> on Day 17) at the end of all exercise periods.

Figure 4 illustrates  $T_{re}$  data for LNA and MNA, for the same days and periods as Figure 3. There were no between-diet differences in  $T_{re}$  on any day. Both MNA and LNA resulted in similar Day 8 versus Day 17 decreases in  $T_{re}$  (e.g., 38.3°C on Day 8 vs. 37.8°C on Day 17) at the end of all exercise periods.

Sweat rate measurements during heat exposure were analyzed each day of HA for all subjects who completed eight exercise periods. These values ranged from 2,850 to 3,000 g·m<sup>-2</sup>·8h<sup>-1</sup> for LNA, and from 2,900 to 3,050 g·m<sup>-2</sup>·8h<sup>-1</sup> for MNA, on Days 8-17 (the total volume of sweat approximated 6 kg·8h<sup>-1</sup>). There were no between-diet differences and no between-day differences ( $p > .05$ , ns) in sweat rate. Fluid intake during HA approximated 5 kg·8h<sup>-1</sup> during heat exposure on Days 8-17. The total urine volume was approximately 0.7 kg·8h<sup>-1</sup> during HA on Days 8-17.

Whole-body Na<sup>+</sup> balance was calculated by comparing dietary Na<sup>+</sup> intake to daily Na<sup>+</sup> losses in urine, feces, and sweat. Although these calculations will be published elsewhere,<sup>1</sup> they can be summarized by stating that a few LNA

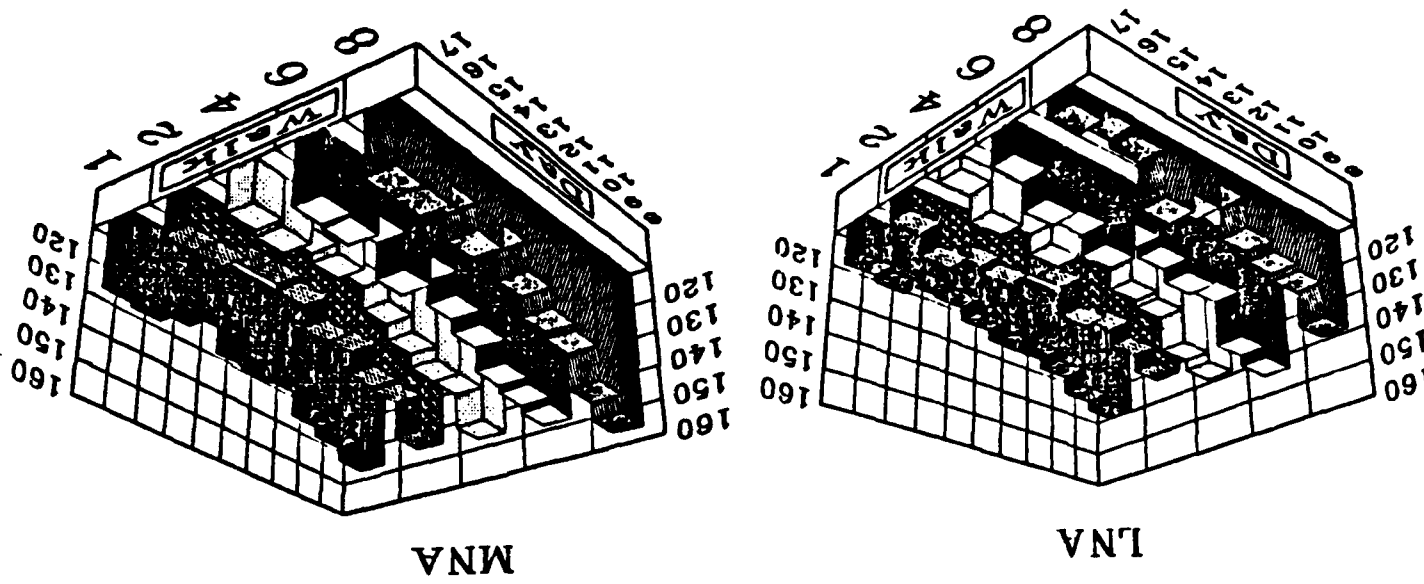


Figure 3 — Mean HR values (beats·min<sup>-1</sup>) for LNA and MNA during each exercise period (labeled "walk") on Days 8-17 of HA.

<sup>1</sup>The data on 24-hr urine Na<sup>+</sup> losses and daily whole-body Na<sup>+</sup> balance will be published elsewhere by Robert Moore, PhD. Those data indicated that a few LNA subjects experienced a mild negative Na<sup>+</sup> balance but that most LNA and MNA subjects were in neutral or positive Na<sup>+</sup> balance by Day 18. Furthermore, the 24-hr urine Na<sup>+</sup> losses followed a pattern similar to that exhibited in Figure 2 for LNA and MNA subjects.

subjects experienced a mild negative Na<sup>+</sup> balance. Most subjects were in neutral or positive Na<sup>+</sup> balance, as verified by the absence of between-diet differences in HR, T<sub>re</sub>, total distance walked, and the incidence of heat illness (see above).

### Discussion

The effects of LNA (68 mEq Na<sup>+</sup>·d<sup>-1</sup>) and MNA (137 mEq Na<sup>+</sup>·d<sup>-1</sup>) on thermoregulatory, cardiovascular, hematologic, and fluid-electrolyte responses were determined during 10 consecutive days of prolonged, intermittent exercise in a simulated desert environment. Consumption of 137 mEq Na<sup>+</sup>·d<sup>-1</sup> (MNA) is considered a moderate Na<sup>+</sup> intake prior to and during HA. For the conditions of heat and exercise imparted by this study, consumption of 68 mEq Na<sup>+</sup>·d<sup>-1</sup> (LNA) would, by most standards, be considered low consumption. Both LNA and MNA involved a large increase in the daily exercise requirement at the onset of HA.

An evaluation of physical characteristics (Table 1), morning body mass, and morning urine specific gravity (see Results) indicated no between-diet differences. With respect to HA trials, there were no between-diet differences in total distance walked, HR (Figure 3), T<sub>re</sub> (Figure 4), sweat rate, or in six out of seven blood variables (Table 2). The absence of heat cramps, heat syncope, or heat exhaustion in both LNA and MNA supported these data. It was concluded that dietary Na<sup>+</sup> restriction (LNA) resulted in HA responses that were not significantly different from those exhibited during moderate Na<sup>+</sup> intake (MNA). In fact, only three variables showed LNA versus MNA differences: urine Na<sup>+</sup> (Days 13-17), urine K<sup>+</sup> (Days 6 and 9), and %ΔPV (Days 11 and 15; see Table 2). Although an increase in sweat rate often occurs during human HA, it is not inevitable (2). A review of this topic (13) noted that 36% of HA studies (n=55) showed no changes in sweat rate, and that sweat rate remained unchanged in hot, dry environments but increased markedly in humid environments. The environmental conditions were hot and dry (41°C, 21% RH) in the current investigation. In addition, the day-to-day decline in T<sub>re</sub> during exercise (see above) reduced the central drive for sweat production.

### Plasma Volume Expansion

To date, three theories have been advanced to explain PV expansion during HA. These involve Na<sup>+</sup> and water retention (28), an interplay between osmolar control and cardiovascular baroreceptor control (23), and protein movement from the interstitial compartment into the vasculature (24). Our findings disputed the widely held concept that an increase in total plasma protein is primarily responsible for the PV expansion observed during HA (24), because the large expansion of PV (+12.3% and +12.4% by Day 17 in both MNA and LNA) was accompanied by a decrease in plasma protein concentration (-18% in LNA and -15% in MNA by Day 17) and no change in COP, in both diets.

This finding was in accord with previous research involving controlled Na<sup>+</sup> intake during HA (1). However, because this PV expansion occurred isotonicity in both LNA and MNA, concurrent with little or no change in the concentrations of serum Na<sup>+</sup> and plasma osmolality (Table 2), it is logical that an increase occurred in the total number of osmotically active particles in the extracellular fluids of both treatment groups (28). Because Na<sup>+</sup> and Cl<sup>-</sup> account for more than

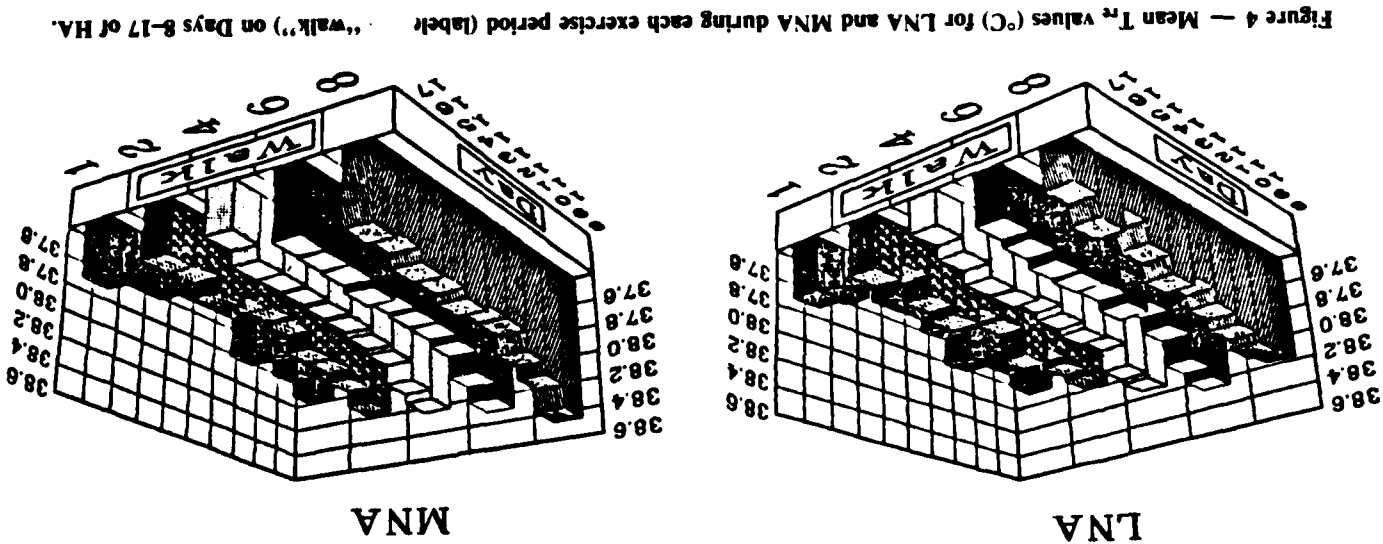


Figure 4 — Mean T<sub>re</sub> values (°C) for LNA and MNA during each exercise period (labeled "walk") on Days 8-17 of HA.



80% of such particles in the extracellular fluid of humans, it is likely that  $\text{Na}^+$  and  $\text{Cl}^-$  were conserved in sweat and urine, particularly since protein concentration decreased (Table 2).

We have reported elsewhere (10) that the plasma aldosterone levels of the present subjects were differentially altered on Days 8-17; during the HA trials, the aldosterone levels in LNA increased significantly more than those of MNA on Days 11 and 15, but subsequently decreased to the same level as MNA by Day 17. These facts strongly implicate osmotic forces in the differential expansion of PV shown by LNA and MNA. Other circulating hormones may have affected PV expansion, in that antidiuretic hormone, cortisol, and glucocorticoids have been associated with PV expansion previously (1).

A delayed PV expansion has been reported (1) for subjects consuming a low  $\text{Na}^+$  diet (98 mEq  $\text{Na}^+\cdot\text{d}^{-1}$ ) when compared to a high  $\text{Na}^+$  diet (399 mEq  $\text{Na}^+\cdot\text{d}^{-1}$ ) during a HA regimen involving 90 min of continuous daily exercise. It was suggested that this delay in PV expansion (Days 3-6 of HA) increased the risk of circulatory inadequacy or heat exhaustion (1), because several between-diet differences were observed (e.g., HR,  $T_{re}$ , % $\Delta$ PV, sweat  $\text{Na}^+$ , plasma  $\text{Na}^+$ ). However, the absence of any form of heat illness (e.g., heat exhaustion) in the current study strongly suggests that LNA did not elicit an increased risk of circulatory incompetence or heat exhaustion. Because the current investigation involved mild to moderate exercise, the results may not apply to situations involving high-intensity exercise (18).

#### **$\text{Na}^+$ Balance During HA**

The current data raise the question of whether there is a minimal or optimal range of daily salt consumption that optimally supports the acquisition and sustainment of HA. Because HA is intimately linked with adrenocortical regulation of urine/sweat  $\text{Na}^+$  losses, and because  $\text{Na}^+$  losses might be large during exercise-heat exposure (8), it has been concluded that a high salt diet is advisable prior to and during the initial days of exercise in the heat (5, 19, 25) and that excess  $\text{Na}^+$  simply would be excreted in urine without harm to health. However, an excess of whole body  $\text{Na}^+$  will typically repress plasma aldosterone levels (18). Theoretically, this is exactly opposite the hormonal status desired, especially if secondary challenges (e.g., decreased food consumption, increased work requirements) are presented; future research is required to determine whether this results in an increased incidence of heat illness (14, 15).

The mean ratios of  $\text{Na}^+:\text{K}^+$  concentration in the morning urine samples of LNA and MNA on Days 1-18 (not shown, calculated from data in Figure 2) provided an index of the relative intensity of renal  $\text{Na}^+$  conservation. Once subjects had stabilized on the 137 mEq  $\text{Na}^+\cdot\text{d}^{-1}$  diet (Days 4-8), MNA resulted in a  $\text{Na}^+:\text{K}^+$  ratio of 0.8-1.4. With the onset of daily HA trials (Day 8), this ratio fell below 0.8 for several days in both diets but rose toward the pre-HA levels on later days. This suggested that retention of  $\text{Na}^+$  at the kidney (and probably the sweat glands, see Refs. 2, 4) eventually resulted in  $\text{Na}^+$  balance in both LNA and MNA.

Urinary  $\text{Na}^+$  concentrations late in the course of HA (Figure 2) illustrated this concept, in that both LNA (Day 16) and MNA (Day 11)  $\text{Na}^+$  concentrations increased, although MNA exhibited this upward inflection of  $\text{Na}^+$  concentration earlier. These data were supported by 24-hr urine  $\text{Na}^+$  losses<sup>1</sup> which verified that

most subjects were in neutral or positive  $\text{Na}^+$  balance by Day 17. Strauss et al. (25) demonstrated that such urinary  $\text{Na}^+$  concentration increases would not have occurred if a balance of daily  $\text{Na}^+$  turnover had not been achieved by Day 17.

Therefore, the current investigation demonstrated that a typical U.S. diet (containing 78-218 mEq  $\text{Na}^+\cdot\text{d}^{-1}$ ; represented by MNA in the current investigation) allowed normal HA during 10 days of prolonged (8 h·d<sup>-1</sup>), intermittent exercise in the heat. Moreover, if  $\text{Na}^+$  consumption was reduced by 50% (LNA), HA still occurred and performance was maintained at the same level as MNA. Our requirement to replace sweat and urine losses with water, during each hour of HA, was believed to be an important factor in the induction of normal HA, as the subjects in both groups walked an average of 16.9 and 18.5 km·d<sup>-1</sup> ( $p > 0.5$ , ns), respectively, in the 41°C environment.

The current investigation also reduced concerns about the occurrence of salt depletion heat exhaustion in LNA and MNA, described by McCance in 1936 (20). This illness reportedly occurs when large losses of  $\text{Na}^+$  in sweat and urine are replaced by drinking a hypotonic fluid (e.g., pure water). Although such a scenario was simulated during each day of the current investigation, and although each subject lost approximately 60 liters of sweat during the 10-day course of HA, no subject exhibited the symptoms of salt-depletion heat exhaustion (e.g., vertigo, hypotension, tachycardia, vomiting; Ref. 14) during the 10 days of HA. Furthermore, the serum  $\text{Na}^+$ , serum  $\text{K}^+$ , plasma osmolality, MCHC, total plasma protein, and COP values (Table 2) were all within the normal range for men on all days.

The foregoing observations are in agreement with the results of Johnson et al. (17), who concluded that a well-balanced diet and a regimen of hourly water consumption adequately maintained performance and resulted in normal fluid-electrolyte measurements during strenuous physical activity (5 h·d<sup>-1</sup>) in a hot environment.

#### **References**

1. Armstrong, L.E., D.L. Costill, and W.J. Fink. Changes in body water and electrolytes during heat acclimation: Effects of dietary sodium. *Aviat. Space Environ. Med.* 58:143-148, 1987.
2. Armstrong, L.E., D.L. Costill, W.J. Fink, M. Hargreaves, I. Nishibata, D. Bassett, and D.S. King. Effects of dietary sodium intake on body and muscle potassium content in unacclimatized men during successive days of work in the heat. *Eur. J. Appl. Physiol.* 54:391-397, 1985.
3. Bean, W.B., and L.W. Eichna. Performance in relation to environmental temperature. Reactions of normal young men to a simulated desert environment. *Fed. Proc.* 2:144-158, 1943.
4. Conn, J.W. Aldosteronism in man: Some clinical and climatological aspects. Part 1. *JAMA* 183:775-781, 1963.
5. Consolazio, C.F. Nutrient requirements of troops in extreme environments. *Army Research Development Magazine* 11:24-27, 1966.
6. Dahl, L.K. Salt intake and salt need. *New Engl. J. Med.* 258:1152-1157, 1958.
7. De Garavilla, L., M.J. Durkot, T.M. Ihley, N. Leva, and R.P. Francesconi. Adverse effects of dietary and furosemide-induced sodium depletion on thermoregulation. *Aviat. Space Environ. Med.* 61:1012-1017, 1990.

8. Denton, D. *The Hunger for Salt*. New York: Springer-Verlag, 1982.
9. Dill, D.B., and D.L. Costill. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J. Appl. Physiol.* 37:247-248, 1974.
10. Francesconi, R.P., L.E. Armstrong, N. Leva, R. Moore, P.C. Szlyk, W. Matthew, W. Curtis, R.W. Hubbard, and E.W. Askew. Endocrinological responses to dietary salt restriction during heat acclimation. Washington, DC: National Academy Press, in press.
11. Francesconi, R.P., and R.W. Hubbard. Chronic low-sodium diet in rats: Responses to severe heat exposure. *J. Appl. Physiol.* 58:152-156, 1985.
12. Greenleaf, J.E., and C.J. Greenleaf. *Human Acclimation and Acclimatization to Heat: A Compendium of Research*. Moffett Field, CA: NASA Tech. Rep. No. TMX-62008, 1970.
13. Hensare, R. Acclimatization to heat in man: Giant or windmill, a critical reappraisal. In *Advances in Animal and Comparative Physiology: Proceedings of the 28th International Congress of Physiological Science*, G. Pethes and V.L. Fiengo (Eds.), Elmsford, NY: Pergamon Press, 1981, pp. 275-284.
14. Hubbard, R.W., and L.E. Armstrong. The heat illnesses: Biochemical, ultrastructural, and fluid-electrolyte considerations. In *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*, K.B. Pandolf, M.N. Sawka, and R.R. Gonzalez (Eds.), Indianapolis: Benchmark Press, 1988, pp. 305-360.
15. Hubbard, R.W., L.E. Armstrong, P.K. Evans, and J.P. De Luca. Long-term water and salt deficits—A military perspective. In *Predicting Decrements in Military Performance Due to Inadequate Nutrition*. Washington, DC: National Academy Press, 1986, pp. 29-48.
16. Jackson, A.S., and M.L. Pollock. Generalized equations for predicting body density of men. *Br. J. Nutr.* 40:497-504, 1978.
17. Johnson, H.L., R.A. Nelson, and C.F. Consolazio. Effects of electrolyte and nutrient solutions on performance and metabolic balance. *Med. Sci. Sports Exerc.* 20:26-33, 1988.
18. Ladell, W.S.S. Disorders due to heat. *Trans. Royal Soc. Tropical Med. Hygiene* 51:189-216, 1957.
19. Ladell, W.S.S., J.C. Waterlow, and M.F. Hudson. *Desert climate: Physiological and clinical observations*. *Lancet* 2:491-497, 1944.
20. McCance, R.A. Experimental sodium chloride deficiency in man. *Proc. Royal Soc. London Biol.* 119:245-268, 1936.
21. National Research Council. *Diet and Health: Implications for Reducing Chronic Disease Risk*. Washington, DC: National Academy Press, 1989.
22. National Research Council. *Recommended Dietary Allowances*. (10th ed.). Washington, DC: National Academy Press, 1989.
23. Rowell, L.B., J.R. Blackman, R.H. Martin, J.A. Mozzarella, and R.A. Bruce. Hepatic clearance of indocyanine green in man under thermal and exercise stress. *J. Appl. Physiol.* 20:384-394, 1965.
24. Senay, L.C. Effects of exercise in the heat on body fluid distribution. *Med. Sci. Sports* 11:42-48, 1979.
25. Strauss, M.B., E. Lamdin, W.P. Smith, and D.J. Bleifer. Surfeit and deficit of sodium. *Arch. Intern. Med.* 102:527-536, 1958.
26. Taylor, H.L., A. Henschel, O. Mickelsen, and A. Keys. The effect of sodium chloride intake on the work performance of man during exposure to dry heat and experimental heat exhaustion. *Am. J. Physiol.* 140:439-451, 1944.

27. Tobian, L. The relationship of salt to hypertension. *Am. J. Clin. Nutr.* 32:2739-2748, 1989.
28. Wyndham, C.H., A.J.A. Benade, C.G. Williams, N.B. Strydom, A. Golden, and A.J.A. Heynes. Changes in central circulation and body fluid spaces during acclimatization to heat. *J. Appl. Physiol.* 25:586-593, 1968.

### Acknowledgments

The authors gratefully acknowledge the many hours of dedicated assistance provided by Robert J. Moore, Natalie Leva, Patricia C. Szlyk, Ingrid V. Sils, Richard F. Johnson, William J. Tharion, Glenn J. Thomas, and Simone Adams. Dr. Richard Tulley, Pennington Biomedical Research Institute, Louisiana State University, performed analyses of dietary items and urine/sweat electrolytes via emission spectrometer. Dr. Michael Sutherland, University of Massachusetts, administered statistical analyses. The authors also acknowledge the support of Dr. Carl M. Maresch, University of Connecticut.

The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as official Department of the Army position, policy, or decision, unless so designated by official documentation. Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research. Citations of commercial organizations and trade names in this report do not constitute an official Department of the Army endorsement or approval of the products or services of these organizations.

Accession For	
NTIS GRA&I	<input checked="" type="checkbox"/>
DTIC TAB	<input type="checkbox"/>
Unannounced	<input type="checkbox"/>
Justification	
By	
Distribution/	
Availability Codes	
Dist	Avail and/or Special
A-1	20

DTIC QUALITY INSPECTED 8