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Please answer the following questions according to the original article entitled "Aerobic influence on neuromuscular function and tolerance during passive hyperthermia" by Morrison SA, Sleivert GG, and Cheung SS. (from *Medicine & Science in Sports & Exercise* 2006 Oct; 38:1754-1761)
For answering the questions, it may not be necessary to read the whole article.

1. How is the research plan developed within a reasonable theoretical framework? （20%）
2. Please draw a schematic flowchart of whole experimental interventions in this study.（15%）
3. What are the significant contributions of this investigation to add knowledge about this special area?（10%）
4. What are the strength and weakness of this study? （20%）
5. Please try to design a related topic which is inspired by this study. Describe your experimental design and possible methods you would like to use.（35%）

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Aerobic Influence on Neuromuscular Function and Tolerance during Passive Hyperthermia

SHAWNDA A. MORRISON[1], GORDON G. SLEIVERT[2], and STEPHEN S. CHEUNG[3]

[1]Human Performance Laboratory, Faculty of Kinesiology, University of New Brunswick, Fredericton, New Brunswick, CANADA; [2]PacificSport Canadian Sport Centre Victoria, Victoria, British Columbia, CANADA; and [3]Environmental Ergonomics Laboratory, School of Health and Human Performance, Dalhousie University, Halifax, Nova Scotia, CANADA

ABSTRACT

MORRISON, S. A., G. G. SLEIVERT, and S. S. CHEUNG. Aerobic Influence on Neuromuscular Function and Tolerance during Passive Hyperthermia. *Med. Sci. Sports Exerc.*, Vol. 38, No. 10, pp. 1754–1761, 2006. **Purpose:** To determine the role of aerobic fitness on central neuromuscular activation and maximal voluntary contractile force during hyperthermia. **Methods:** Thirty-seven healthy males in three distinct groups based on aerobic fitness and training history were passively heated using a liquid conditioning garment in a hot (35°C, 50% RH) environment with the intention of testing neuromuscular function with whole-body hyperthermia. Of these initial participants, 11 of the 13 highly fit (HF; $\dot{V}O_{2max}$ = 71.2 ± 5.9 mL·kg^{-1}·min^{-1}, body fat = 5.6 ± 1.9%), 11 of the 13 moderately fit (MF; 57.2 ± 4.2 mL·kg^{-1}·min^{-1}, 11 ± 3.4%), and 4 of the 11 lower-fit (LF; 49.6 ± 1.1 mL·kg^{-1}·min^{-1}, 19.4 ± 2.6%) individuals tolerated heating to 39.0°C, with the remainder terminating the experimental protocol early. Maximal force output and voluntary activation were examined during a 10-s maximal isometric knee extension. **Results:** Passive heating attenuated force production (−61.7 ± 69.6 N change from initial values) and decreased voluntary activation (8.6 (12.6), 18.1 (12.4), and 6.1 (3.1)% for HF, MF, and LF training groups, respectively). Cardiovascular strain moderately increased to 60 ± 14% ($P < 0.001$), whereas HF and MF had significantly higher MAP than LF at the end of heating (98 ± 15, 99 ± 7, and 79 ± 5 mm Hg for HF, MF, and LF, respectively; $P < 0.05$). However, the ability to tolerate passive heating to 39.0°C (and above) differed between the HF and MF compared with LF, despite no difference in their psychophysical rankings of thermal sensations and/or (dis)comfort. **Conclusion:** Low aerobic fitness and activity level are associated with a decreased tolerance to passive hyperthermia. However, at high body temperatures, maximum force production and voluntary activation were impaired to an equal level regardless of training status. **Key Words:** INTERPOLATED TWITCH, FATIGUE, LIQUID CONDITIONING GARMENTS, CRITICAL INTERNAL TEMPERATURE

High environmental temperatures (7) and internal heat storage (8) have both been demonstrated to accelerate fatigue during prolonged and voluntary submaximal exercise. Recently, support has developed for the hypothesis that a critically high internal body temperature directly precipitates voluntary exhaustion through multifactorial impairment of different systems throughout the body (2). Despite different initial core temperatures or different rates of heat storage, highly fit participants reached voluntary exhaustion at a similar 40.1–40.2°C esophageal temperature (8). Consistent endpoint core temperatures have also been reported in animals with various preheating protocols at different ambient conditions (5,25). A primary role in eliciting exhaustion has been attributed to a compromised central nervous system, based on observations of impaired neuromuscular activation (12,15,18,22,24) along with decreased cortical arousal and elevated perception of strain during hyperthermia (13,16). Recent studies in our laboratory (12,22) used a passive heating and cooling protocol and reported that voluntary neuromuscular activation (VA) during maximal isometric contractions of the leg was progressively impaired with elevated core temperatures up to 39.4°C. Activation remained decreased even with rapid skin cooling, then progressively returned to baseline values with core cooling back to thermoneutrality (12,22), demonstrating that neuromuscular function was likely centrally mediated.

Studies on voluntary exhaustion and on neuromuscular activation during exercise at high temperatures have generally used highly fit and trained subjects (8,12,13,16,22), typically to ensure the achievement of a high internal temperature or "true" exhaustion. Not only is this high level of fitness atypical of the general population, the homogeneity in subject fitness does not permit investigation of the underlying mechanisms behind improved heat tolerance in fit subjects. Although the endpoint core temperatures at voluntary exhaustion within a sample group during treadmill exercise in the heat have been consistent, a clear separation has been reported between highly and moderately fit humans (1,21) even after normalizing for body composition (21), suggesting that heat tolerance can be improved through aerobic fitness. Some potential physiological mechanisms of

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benefit arising from higher aerobic fitness include adaptations to the cardiovascular system to maintain blood flow to both the muscles and thermoregulatory system during exercise (4) and improved resistance to local tissue ischemia (19). However, the possibility of a central nervous system mechanism for differences in heat tolerance across fitness groups is suggested by the findings of Tikuisis et al. (23), who reported an attenuated perception of thermal strain compared with physiological strain throughout exercise during uncompensable heat in fit subjects but not in moderately fit individuals. Therefore, it is possible that neuromuscular activation during hyperthermia may also be impaired to a greater extent in individuals of lower fitness.

The purpose of this study was to determine the influence aerobic fitness and training status might have on maximum voluntary isometric contractions and VA during passive hyperthermia. This was achieved by recruiting highly trained cross-country runners and matching them with moderately fit individuals on such physical characteristics as body surface area (height and weight) but differing on their aerobic power scores. Additionally, lower-fit individuals were recruited who differed in both body composition and aerobic power scores compared with the other two groups. Using a similar protocol to our previous studies (12,22), participants performed 10-s maximal isometric knee extensions with simultaneous assessment of VA at ± 0.5°C core temperature (T_{re}) intervals during heating from 37.4 to 39.0°C (warm skin) and subsequent cooling from T_{re} of 39.5 to 37.5°C (cool skin). It was hypothesized that individuals with higher aerobic fitness scores would produce more power and higher VA levels at a given T_{re} compared with their untrained counterparts.

METHODS

This study was conducted following the Canadian Tri-Council Policy for the ethical treatment of human participants as part of the Helsinki Declaration II, and institutional research ethics board approval from the University of New Brunswick and Dalhousie University were obtained before any experimentation. All testing was performed in an environmental chamber at the Human Performance Lab, University of New Brunswick. Before participating in this study, participants were screened for their resting heart rate (< 100 bpm) and blood pressure (< 144/94 mm Hg) values and given a concise explanation of all procedures and potential risks before signing a written informed consent.

Definition of groups. Thirty-seven healthy adult males initially volunteered for this study. Participants were recruited to fit into three distinct groups based on aerobic fitness, anthropometrics, and training history. The highly fit (HF) participants were matched for body surface area with moderately fit (MF) individuals to investigate whether increased aerobic fitness could exclusively be linked with changes in force production and VA. HF participants were members of a varsity cross-country team who had $\dot{V}O_{2peak}$ > 65 mL·kg^{-1}·min^{-1} and who regularly trained (≥ 6 d·wk^{-1}) for more than 30 min per exercise session. MF individuals were recreationally active, had $\dot{V}O_{2peak}$ between 50 and 60 mL·kg^{-1}·min^{-1}, and exercised on some days of the week (2–4 d·wk^{-1}). Lower-fit (LF) individuals had $\dot{V}O_{2peak}$ < 50 mL·kg^{-1}·min^{-1} and were rarely active for more than 30 min, 2 d·wk^{-1}. Percent body fat was calculated using the Jackson–Pollock seven-site skinfold protocol (9), and this value was used to determine participants' body surface area (3).

Determination of aerobic fitness. Participants performed a maximal graded exercise test on a treadmill to directly assess their aerobic power. The protocol began with a submaximal running pace that the participant thought he could maintain for 20–30 min. The treadmill grade was increased by 2° every 2 min until exhaustion. Heart rate was monitored continuously by a Polar Vantage NV heart rate monitor (Polar Electro Inc., Port Washington, NY). During exercise, participants breathed into a mouthpiece to determine respiratory measures through open-circuit spirometry. Expired air was sampled every 20 s using a True Max 2400 Metabolic Measurement System (version 3.3, Parvo Medics Inc., Sandy, UT). Before the exercise test, the gas analyzers were calibrated with known oxygen (O_2) and carbon dioxide (CO_2) gas concentrations. The volume transducer was also calibrated with a 3-L syringe.

Experimental design. Each participant's ability to produce maximal force and recruit the knee extensors was examined at 0.5°C T_{re} intervals spanning the range from normothermia (~37.5°C) to hyperthermia (~39.5°C). With each increase in T_{re} of 0.5°C, the neuromuscular, cardiovascular, and psychophysical status of each participant was tested. Participants were verbally encouraged throughout the contractions in identical fashion, and visual feedback was provided of their voluntary force production via computer monitors within the chamber. Participants were gradually warmed to a temperature of approximately 39.5°C by running 52°C fluid through the tubing of a liquid conditioning garment (LCG), with early termination upon a) the participant's request or b) concern for the participant's safety. Some participants showed presyncope symptoms including headache, pallor, dizziness, nausea, or vomiting. Any of the aforementioned symptoms constituted the termination of the protocol. After attaining a T_{re} of 39.5°C, participants were then cooled back down to their initial temperature by running 8°C fluid through the LCG. Muscle force, VA, and cardiovascular and psychophysical strain were then measured in an identical fashion to the warming protocol, again at every 0.5°C decrease in T_{re} until a T_{re} of approximately 37.5°C was reached.

Thermal control. The heating protocol was conducted in an environmental chamber set at 35°C (50% RH). During cooling, the chamber was immediately turned off and the door was opened to circulate room air into the chamber (~22°C). Body temperature was also controlled using a LCG (Med-Eng, Ottawa, Canada) consisting of a three-piece suit made of black elastic cotton that covered the head, neck, trunk, and arms, along with an impermeable rain suit worn over top of the LCG. Small polyvinyl chloride tubing (41.15 m × 2.5-mm internal diameter × 4.2-mm outside diameter) was sewn to the inside of the LCG. To maximize heat exchange between the LCG and

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the participant's skin surface, a form- fitting bathing suit or workout shorts (i.e., cycling spandex) were required for the testing session, and no shirts were worn under the LCG. Water was pumped through the tube network during both heating and cooling using a pneumatic pump (DTI, Ottawa, Canada) at a flow rate of 800 $mL \cdot min^{-1}$. Water temperature was controlled using a water heater with an accuracy of approximately 0.5°C.

Instrumentation. Heart rate was monitored throughout the experimental session using a 12-lead electrocardiograph (Quinton Instrument Co, Bothell, WA), and heart rate is expressed as a relative percentage of the range between resting and known maximum heart rate (heart rate reserve, HRR). Blood pressure was measured manually with a stethoscope and sphygmomanometer at each 0.5°C T_{re} increment. Both T_{re} and T_{sk} were monitored continuously using an eight-channel data logger (SmartReader 8 Plus, ACR Systems, Surrey, BC, Canada) that sampled data every 8 s.

Rectal temperature (T_{re}) was monitored using disposable temperature probes (Mon-a-Therm General Purpose, Mallinckrodt, St. Louis, MO) inserted 10 cm beyond the anal sphincter. Skin temperature (T_{sk}) was monitored at four sites (chest, upper arm, thigh, calf) using biomedical ceramic chip thermistors (MA 100, 10 Kohm negative temperature coefficient, Thermometrics, NJ) for calculation of area-weighted mean T_{sk} (17). Mean body temperature (T_b) was calculated using a T_{re} and T_{sk} weighted formula of $T_b = (T_{re} \times 0.65) + (T_{sk} \times 0.35)$ (17).

To get a more accurate indication of the magnitude of physiological strain experienced by each of the participants during the heating and cooling protocol, the physiological strain index (PSI) was calculated (11). This index runs from 0 (no/little strain) to 10 (high) physiological strain. The PSI did not take into account changes in T_{sk}, but it was seen as a reasonable indication of overall strain, combining heart rate and T_{re} measures. Additionally, the subjective comfort levels of the individual being tested were assessed. A rating of thermal comfort and a measure of thermal sensation were collected at every T_{re} stage throughout the testing protocol (6).

Force and VA measurement. The testing protocol involved systematically measuring the participant's maximum sustained (10 s) voluntary contraction (MVC) for unilateral knee extension with every T_{re} change of 0.5°C. MVC was tested on a custom-made isometric knee extensor myograph. A sustained contraction of 10 s was used rather than the more usual 3- to 5-s MVC because it has been previously demonstrated that VA is not impaired in the first 5 s of a sustained MVC with exercise-induced hyperthermia (15). The right leg was attached to a metal plate lined with high-density foam coupled to a force transducer (ST-250, Precision Transducers Ltd., Castle Hill, Australia), upon which the participant exerted their maximal isometric knee extension. Straps were also placed around the chest and right thigh at the time of contraction to ensure that any extraneous movements were restricted. Force signals were amplified, sent through an A/D board, and sampled at a frequency of 2 kHz by data acquisition hardware (DI-720, DATAQ Instruments Inc., Akron, OH) and software (Windaq, DATAQ Instruments Inc., Akron, OH). The average MVC force was calculated as the average force between the first and ninth second of the contraction, excluding the interpolated twitches and the corresponding antidromic effect that the stimulation procedure elicited in some participants.

VA of the knee extensors was determined using percutaneous muscle stimulation and the interpolated twitch technique. The muscle-stimulation protocol used a supramaximal 200-s duration square-shaped pulse delivered to the knee extensors using a modified constant-current (maximum 1 A) electrical stimulator (Digitimer DS7A, Hertfordshire, UK). Carbon rubber electrodes (5.08 × 10.2 cm) were placed both proximally (anode) and distally (cathode) on the thigh. The intensity of the supramaximal pulse was determined at the start of the experiment by increasing the current of the stimulus in 25 mA increments (beginning at 25 mA) until no further increase in twitch force was observed. To determine VA, a supramaximal stimulus was delivered twice during each MVC (coincident with visually identified peak force levels and usually between 3 and 7 s) and immediately after each MVC. VA was calculated in the standard fashion (10) as

$$[1 - (\text{superimposed twitch/control twitch})] \times 100.$$

The superimposed twitch used was the smaller of two twitches elicited over the MVC so as not to underestimate central activation. The control twitch was the twitch elicited immediately (5 s) after the contraction.

Hydration status. Nearly nude body weight was determined at the start and end of the experimental testing session to the nearest 10 g with participants only wearing their form-fitting garment (i.e., cycling shorts). Participants emptied their bladders before the testing session began, and the amount of water consumed during the trial was monitored with the intention of all participants remaining normally hydrated. The amount of water available for each participant was unlimited; only the amount of fluid ingested and excreted was recorded. Sweat loss was not measured directly in this study; consequently, only changes in body weight are reported.

Data analysis. Mean and standard deviation were used to describe all data. A two-way analysis of variance (ANOVA: fitness level × T_{re}) with repeated measures on one factor (temperature) was used to determine whether body temperature influenced the ability to maximally produce force and activate the knee extensors and whether aerobic fitness level influenced this response. Bonferroni *post hoc* comparisons were used to determine where specific differences occurred when a significant main or interaction effect was present. The analysis was limited to contrasting four thermal states: 1) the initial T_{re} and T_{sk}, 2) the hot T_{re} and hot T_{sk} at the end of heating, 3) hot T_{re}, cool T_{sk} with the introduction of cool water circulation, and 4) end of the protocol, when T_{re} returned to normal and T_{sk} was cool. All statistics were protected from type I error at the $P < 0.05$ level of significance. Statistical analysis was performed using the

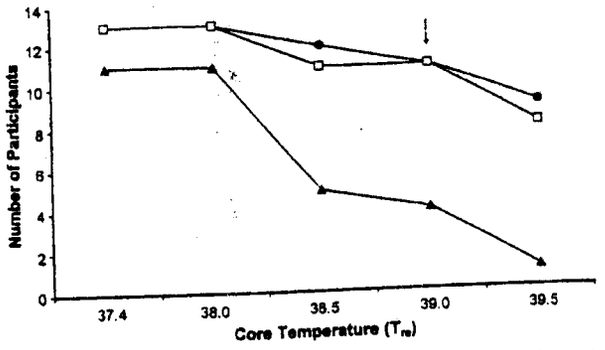

FIGURE 1—Participant survival graph. Sample size of participants from each training status throughout passive heating protocol. The highly fit (*circles*), moderately fit (*squares*), and lower-fit groups (*triangles*) began with similar sample sizes, although the majority of untrained participants were not able to complete the protocol. The *arrow* denotes the core temperature at which data were analyzed.

statistical software package SPSS version 11.5 for Windows (SPSS Inc. Chicago, IL).

RESULTS

Based on our previous studies using this passive heating and cooling model (12,22), our experimental design initially aimed to raise T_{re} to 39.5°C in all subjects across the three fitness groups. However, only 26 participants (11 of the 13 HF, 11 of the 13 MF, and 4 of the 11 LF) tolerated passive heating to a rectal temperature of 39.0°C, with the remaining 11 participants voluntarily terminating the protocol before 39.0°C because of discomfort. This constituted a significant dropoff in the ability of participants to complete the protocol across the three groups (Fig. 1). Thus, it was decided that all subsequent analyses would be performed at 39.0°C across the three groups because it was doubtful that T_{re} of 38.0–38.5°C would provide a sufficient heat load to adequately test our hypotheses, although retention at 39.5°C clearly became an issue during passive heating. Physical characteristics of the participants attaining 39.0°C heating are described in Table 1. Participants were of similar age, height, and body surface area, but differed from each other on aerobic fitness and percent body fat.

Thermal strain. A consistent thermal protocol was successfully maintained, ultimately achieving similar thermal profiles of heating and cooling across each of the groups. Mean testing duration in the chamber across all training groups was 162.7 (28.2) min, with 80.1 (17.6) min of passive heating and 82.6 (29.5) min of cooling. Heating duration was significantly different between the HF and LF groups (85 ± 18 vs 62 ± 18 min) ($P < 0.05$). There were no differences in cooling duration between training status groups. T_{sk} increased rapidly for all groups ($P < 0.001$) with the heating protocol from its initial temperature of 35.3 (0.7) to 39.9 (0.5)°C at a T_{re} of 39.0 (0.1)°C. Passive cooling reduced both T_{sk} and T_{re} ($P < 0.001$) for all groups. T_{re} returned to initial values for all fitness groups on completion of the cooling protocol, whereas T_{sk} decreased significantly ($P < 0.001$) to 26.0 (2.1)°C on completion of the experiment (Fig. 2).

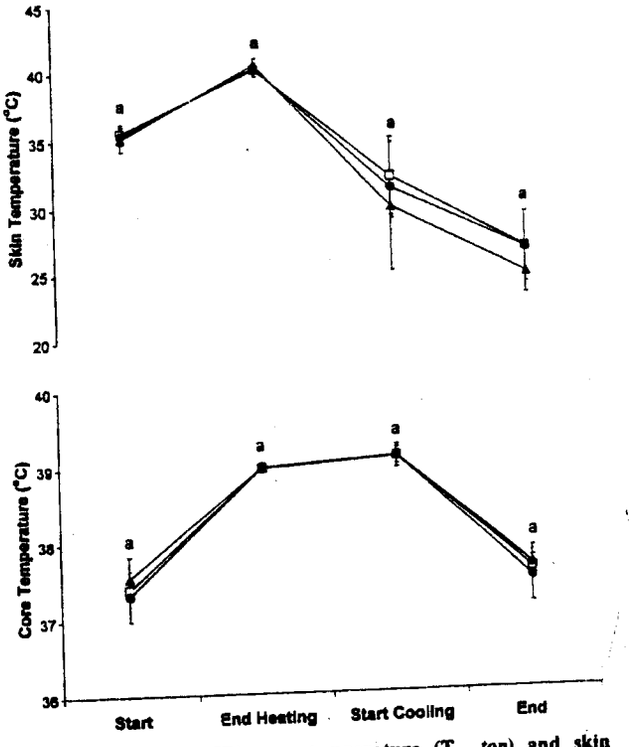

FIGURE 2—Mean (SD) core temperature (T_{cr}, *top*) and skin temperature (T_{sk}, *bottom*) during passive heating and cooling for the highly fit (*circles*), moderately fit (*squares*), and lower-fit (*triangles*) groups. *Matching letters* indicate significant main effect differences between time points ($P < 0.05$).

Neuromuscular function. Passive heating decreased MVC levels ($P = 0.0001$, $F = 151.5$; Fig. 3, top), with no significant differences between groups ($P = 0.193$, $F = 1.772$). Change scores were decreased by 16 (61), 48 (92), and 122 (82) N (95% CI: HF LL = −24.98, UL = 56.98; MF LL = −13.80, UL = 109.80; LF LL = −28.64, UL = 272.64) for each of the HF, MF and LF groups, respectively,

TABLE 1. Participant characteristics divided by training status for those who completed the passive heating protocol to a core temperature of > 39.0°C.

Training Group	Age	Height	Weight	BMI	%BF	BSA (m^2)	$\dot{V}O_{2peak}$	HR Rest
Highly fit (N = 11/13)	21 (2)	1.8 (0.1)	71.5 (8.8)*	22 (2)*	5.6 (1.9)*	1.89 (0.14)	71 (6)*	58 (8)
Moderately fit (N = 11/13)	23 (3)	1.8 (0.1)	74.0 (9.3)	23 (3)	11.0 (3.4)*	1.92 (0.15)	57 (4)*	67 (11)
Lower fit (N = 4/11)	22 (4)	1.8 (0.0)	84.6 (11.3)*	27 (3)*	19.4 (2.6)*	2.01 (0.14)	50 (1)*	70 (11)

Matching letters (*) indicate significant differences between those characteristics within each column of data ($P < 0.05$).
Participant characteristics for the seven lower-fit subjects who terminated the experiment before attaining a rectal temperature of 39.0°C were similar in all characteristics to the four lower-fit subjects who did attain 39.0°C.

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with elevations in T_{re} (~39.0°C) compared with endpoint measures (T_{re} ≈ 37°C). Passive heating decreased VA (P = 0.0001, F = 5.325) for all groups (Fig. 3, bottom), with no significant difference found between training groups (P = 0.071, F = 2.978). VA was reduced by 8.6 (12.6), 18.1 (12.4), and 6.1 (3.1)% (95% CI: HF LL = −0.06, UL = 17.26; MF LL = 9.77, UL = 26.43; LF LL = 0.59, UL = 11.61) for HF, MF, and LF, respectively, at the end of the heating protocol, but returned to baseline values by the end of the testing session after cooling (Fig. 3, bottom).

Cardiovascular strain. Percent HRR increased for all groups during passive heating to 60 ± 14% (P < 0.001) before returning to baseline values (5 ± 14%). The heating and cooling protocol elicited no significant main effect on mean arterial pressure (Fig. 4, bottom), but there were differences observed among groups (P < 0.05). Both HF and MF had significantly higher MAP than LF at the end of heating (98 ± 15, 99 ± 7, and 79 ± 5 mm Hg for HF, MF, and LF, respectively; P < 0.05). There were no differences across groups at the end of the cooling protocol (95 ± 8, 101 ± 10, and 96 ± 9 mm Hg for HF, MF, and LF, respectively), although an interaction effect was observed (P = 0.017). Diastolic blood pressure was significantly elevated with skin cooling from passive heating (76 ± 12 to 86 ± 10 mm Hg) in all training groups. There were no significant training status differences in changes with systolic blood pressure.

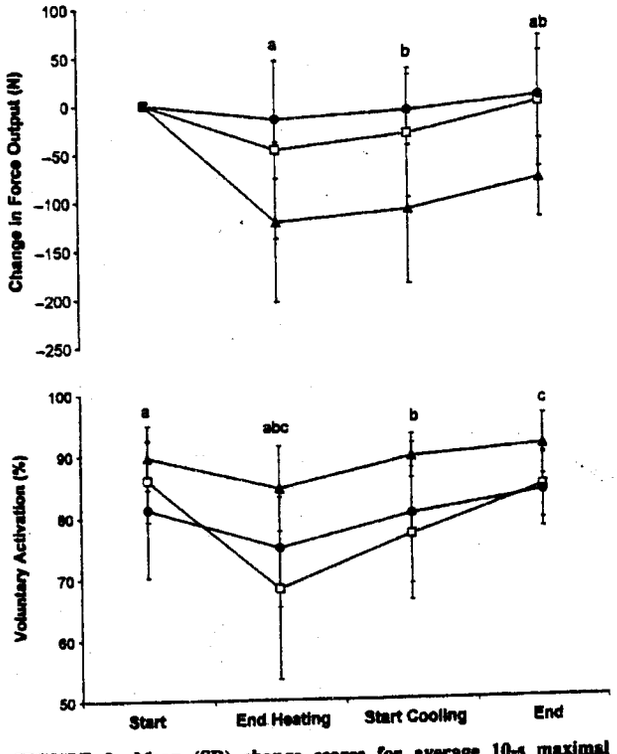

FIGURE 3—Mean (SD) change scores for average 10-s maximal voluntary isometric contraction force (aMVC, *top*) and percent voluntary activation (VA, *bottom*) during passive heating and cooling for highly fit (*circles*), moderately fit (*squares*), and lower-fit (*triangles*) groups. *Matching letters* indicate significant main effect differences between time points (P < 0.05).

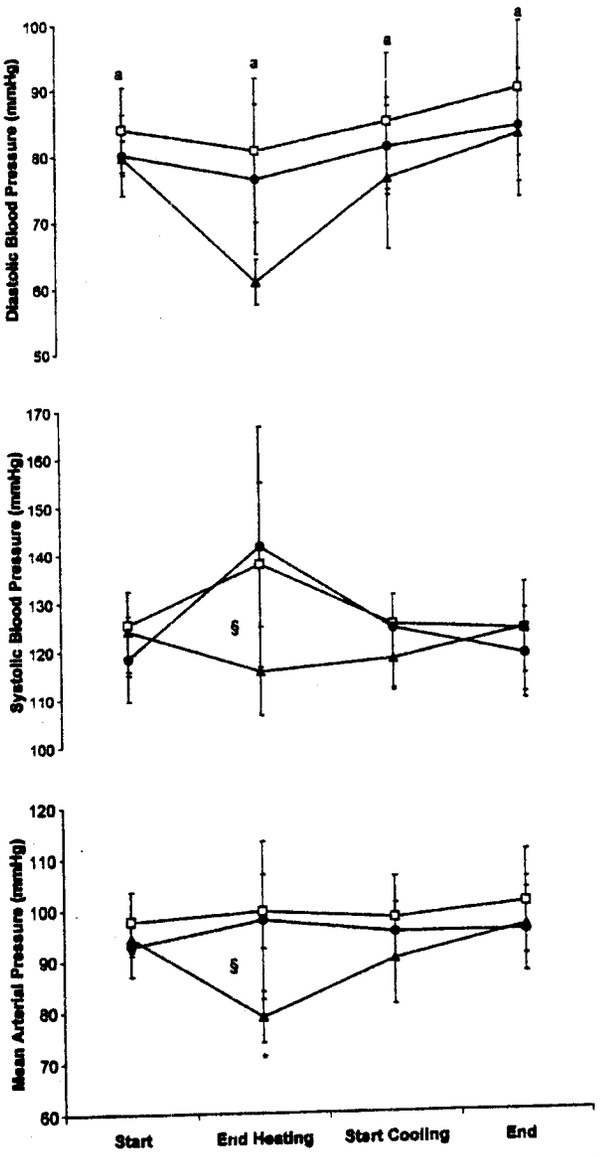

FIGURE 4—Mean (SD) systolic blood pressure (*top*), diastolic blood pressure (*middle*), and mean arterial pressure (MAP, *bottom*) during passive heating and cooling for highly fit (*circles*), moderately fit (*squares*), and lower-fit (*triangles*) groups. *Matching letters*, significant main effect differences between time points; § significant interaction between variables (P < 0.05).

Psychophysical. Passive heating increased physiological strain moderately to 7.0 (0.8), with no differences observed across fitness groups. Thermal comfort and thermal sensation for all groups were also significantly influenced by passive heating and cooling (P < 0.001), with no differences observed between groups. Specifically, thermal comfort decreased with passive heating such that all individuals rated themselves at a 5 ("extremely uncomfortable") but returned towards baseline to 1 ("comfortable") with the initiation of cooling. Thermal sensation generally mirrored the changes in T_{sk} and did not necessarily reflect changes in T_{re}. Participants in each training group ranked their thermal sensation differently with the introduction of cool-water circulation (from 9,

"very hot"; to 3, "cool") regardless of still having a significantly elevated T_{re} of 39.1 ± 0.1°C ($P < 0.001$).

DISCUSSION

Cheung and McLellan (1) and Selkirk and McLellan (21) reported a significantly higher T_{re} at the point of voluntary exhaustion in highly fit individuals during exercise in uncompensable heat-stress environments, whereas decrements in neuromuscular activation were reported at the point of voluntary exhaustion in the heat (15,18). Previous studies on neuromuscular function with hyperthermia have mostly employed active exercise-induced hyperthermia (15,18) with a fairly homogenous cohort of highly fit individuals (12,15,18,22). Therefore, the present study used a cross-sectional design to investigate the impact of aerobic fitness and training status on neuromuscular function and central activation during progressive elevations in T_{re}. We passively elevated T_{re} in HF, MF, and LF individuals and observed progressive decrements in neuromuscular force and central activation with rising T_{re} in all groups, supporting the findings of Morrison et al. (12) and Thomas et al. (22), but we did not observe differences in the pattern or magnitude of impairment across groups. Interestingly, the primary observed difference was in the "survival" pattern across the different groups, with a far greater dropout rate in the LF group.

The neuromuscular response to elevated T_{re} were similar in pattern and magnitude across fitness groups, suggesting that fitness-related differences in exercise-heat tolerance (1,21) are not attributable to differences in the capacity to voluntarily recruit muscular force. However, these data should be approached with caution because there were so many participants in the LF group who did not complete the entire passive heating protocol, thereby potentially decreasing statistical power for comparison across all three fitness groups. With force and VA P values of 0.19 and 0.07, respectively, we cannot guarantee the possibility of a type 2 error occurring, especially between the LF group compared with the MF and HF groups. Nevertheless, there were no significant differences observed between the HF and MF groups, both of whom had equal numbers of participants who completed the heating protocol ($N = 11$ for each). One potential explanation for the lack of difference across the MF and HF groups may be the removal of exercise as an intervention, such that the subsequent decrease in cardiovascular strain and competition between metabolic and thermoregulatory blood flow may have minimized the cardiovascular benefits of higher stroke volume and maximal cardiac output with aerobic fitness. Although no vascular volumes were measured in our study, Sawka et al. (20) have reported no correlation between aerobic fitness and various indices of vascular volume, with the biggest predictor being lean body mass, which was similar across our fitness groups. Cardiovascular strain also was similar across fitness groups and only reached 60% of HRR in our protocol, much lower than the 90–100% HRR observed in other studies employing active exercise in the heat with different fitness groups (1,21). The decrement in VA at low levels of cardiovascular strain in our study and in Todd et al. (24) and also at higher T_{re} and cardiovascular strain with active exercise (15) would suggest a minor role of cardiovascular capacity in the observed neuromuscular activation impairment in the heat.

Thermal sensation and thermal comfort ratings were similar across fitness groups at 39.0°C, suggesting that perceptual responses to heat stress did not differ across fitness groups in the participants who could tolerate hyperthermia. However, the 6 out of 11 LF participants who terminated the heating protocol almost immediately (upon attainment of 38.0°C and before we could perform any neuromuscular testing) did so because extreme personal discomfort, as did the two MF and two HF participants; thus, it is evident that a large variability exists in interindividual perceptual tolerance to hyperthermia. Tikuisis et al. (23) modeled physiological (T_{re} and heart rate) and perceptual (thermal comfort and perceived exertion) heat-strain indices for both fit and nonfit participants in an uncompensable heat-stress environment. Nonfit participants had similar values and patterns for the two indices, but the perception of heat strain was significantly lower than the PSI for fit participants (23). High T_{sk}, which can cause extreme discomfort, were likely the primary reason for early termination in our subjects. Although high T_{sk} and its convergence with T_{re} was demonstrated to be a poor predictor of heat tolerance (14), it remains possible that there is a different perception of high T_{sk} across individuals, with an attenuated discomfort attributable to elevated T_{sk} in the 26/37 subjects who tolerated heating to 39.0°C T_{re}, and intolerance to high T_{sk} may have been the primary reason for the high dropout rate in the LF group.

Although T_{re} was clamped in this study, it appears that both the HF and MF groups were better able to tolerate passive heating as observed by the significant decrease in LF participant numbers at elevated T_{re}. However, there were no differences between groups in reasons for terminating the heating protocol. All participants who terminated the experimental session early reported extreme thermal discomfort, headache, and nausea as their primary reasons for stopping. Conversely, Selkirk and McLellan (21) reported differences in terminal endpoint criteria with active hyperthermia between their four training status groups, which included both fitness and fatness components. By far, all their trained, low-fat participants terminated exercise because they reached the ethical cutoff for elevated T_{re}, compared with their untrained, low-fat counterparts, who terminated exercise because of exhaustion, discomfort, or nausea. It is important to note that the similar physiological outcomes of this study between our HF and MF groups may be attributed to the fact that compared with previously cited research (1,21), our MF group would have been classified as their highly trained groups. Indeed, our MF group had an aerobic fitness score of 57 ± 4 $mL \cdot kg^{-1} \cdot min^{-1}$ and a percent body fat of 11 ± 3%, although the participants reported only being aerobically active on some days of the week (> 30 min, between 2 and 4 d).

(背面仍有題目,請繼續作答)

The other paradigm by which to view the present data is "what is unique about the LF survivors?". A dramatic dropout occurred almost immediately on initiation of heating in the LF group, with 6 out of 11 terminating passive heating at 38.0°C with such discomfort that we were not able to collect any neuromuscular data. However, 4 out of 11 tolerated passive heating to 39.0°C despite no differences in any measured anthropometric measure or in the pattern of fitness activity with the other LF individuals, and they also had no difference in neuromuscular response from the MF and HF individuals. It is important to note that those individuals assigned to each training group were required to fit specific fitness, training, and anthropometric criteria. Therefore, the four LF individuals who were able to complete the passive heating and cooling protocol were not significantly different than the other LF individuals in terms of age, height, weight, body composition, aerobic fitness, or activity levels. It is therefore plausible that the four LF individuals who were able to attain higher T_{re} differed from the others in terms of their motivation to succeed, or they may have differed in their perceived anticipation of a physiological "catastrophe" with the increased thermal heat load.

One limitation of the data analysis is the relatively low number of LF attaining hyperthermia of 39.0°C T_{re}. Previous research on passive heating in fit individuals demonstrated no problems with subject dropout (12,22), so the high level of LF dropout was a surprising result. We deemed it unrealistic to continue recruiting participants indefinitely for the LF group specifically to achieve a high sample size attaining 39.0°C. This was partly attributed to the difficulty in recruiting LF individuals to undertake the strenuous protocol, and also because disproportionately increasing the total subject numbers in the LF group would have biased the design towards those who were tolerant to hyperthermia. Nevertheless, although our statistical power may have been lower than desirable for the comparison at 39.0°C, the high dropout rate in the LF group suggests that the primary difference in heat tolerance across fitness groups was likely not based on issues of neuromuscular drive.

In conclusion, we sought to isolate the direct contribution of T_{re}, via passive whole-body hyperthermia, on the capacity for maximal neuromuscular activation across three distinct groups of individuals categorized according to aerobic capacity, anthropometric characteristics, and activity level. In support of previous work, a significant decrement in central activation was observed with passive hyperthermia to 39.0°C despite relatively low cardiovascular strain. However, although a large decrement in "survivability" to hyperthermia was observed, with only a small proportion of LF individuals able to tolerate passive hyperthermia of 39.0°C, the decrement in central activation was similar at 39.0°C across the three groups. Therefore, we conclude that there do not appear to be differences in capacity for neuromuscular activation during whole-body hyperthermia across fitness levels, although more extensive research targeting lower-fit individuals is needed to address these hypotheses further.

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