



Review

Temperature regulation during exercise in the heat: Insights for the aging athlete

W. Larry Kenney^{a,b}, S. Tony Wolf^{a,*}, Gabrielle A. Dillon^a, Craig W. Berry^a, Lacy M. Alexander^a

^a Department of Kinesiology, The Pennsylvania State University, United States

^b The Graduate Program in Physiology, The Pennsylvania State University, United States

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ABSTRACT

Objective: The purpose of this review is to evaluate the currently-available literature regarding the impact of both primary aging and age-related fitness on thermoregulatory function during exercise in the heat. In so doing, we aim to (1) characterize the influence of fitness in mitigating age-related declines in thermoregulation, (2) address the limitations of prior experimental approaches for investigating age-related thermoregulatory impairments, (3) examine to what extent aerobic fitness can be maintained in the aging athlete, and (4) begin to address the specific environmental conditions in which age-related impairments in thermoregulatory function may place highly active older adults at increased risk for heat-related illness and injury and/or limited performance.

Design: Mini-review.

Methods: Review and synthesis of available information.

Results: The earth's climate is warming, accompanied by a consequently greater frequency and severity of extreme heat events. At the same time, lifespan is increasing and people of all ages are staying increasingly active. Age-related impairments in thermoregulatory function are well-documented, leading to increased heat-related health risks and reduced exercise/athletic performance for older adults in hot environmental conditions. High aerobic fitness improves body temperature regulation during exercise via augmented sweating and improved cardiovascular function, including cardiac output and skin blood flow, in humans of all ages.

Conclusions: The masters athlete is better suited for exercise/heat-stress compared to his or her less fit peers. However, while age and thermoregulation in general has been studied extensively, research on the most fit older adults, including highly competitive athletes, is generally lacking.

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1. Introduction

The world's aging population is growing rapidly, with one in six people in the world predicted to be over the age of 65 by 2050. In developed countries, a quarter of the adult life span is spent beyond the age of 65.¹ In order to preserve the *health* span along the aging continuum, studies have focused on understanding the health-preserving effects of habitual physical activity, exercise training, and the successful phenotype of the older athlete.

Along with an aging population, the earth's climate is warming, with an increase in frequency and severity of severe weather events, including heat waves.² There is a vast literature demon-

strating impaired thermoregulatory and cardiovascular responses during heat exposure in aged adults, changes that contribute to increased morbidity and mortality of older adults during extreme heat exposure.³ Although the health benefits of regular exercise training are known, there is a dearth of studies investigating thermoregulatory responses and the impact of extreme heat on the fittest of the aged – the older athlete.

This review will focus on characterizing thermoregulatory responses of older adults exercising in warm environments to give insights into thermoregulation in the older athlete in the context of healthy aging vs. the inevitable age-related decline in maximal aerobic capacity.⁴ Accordingly, for the purpose of this review, age categories are broadly considered as young (18–35 years), middle aged (36–60 years), and older adulthood (>60 year). We rely primarily on three types of studies: (1) cross-sectional comparisons between young and older adults, (2) endurance training stud-

* Corresponding author.

E-mail address: saw85@psu.edu (S.T. Wolf).

ies on older adults, and (3) multiple regression analyses to gain insights and draw our conclusions on a topic that has received relatively sparse attention over the years. Each of these experimental approaches has unique limitations with respect to the older athlete. Therefore, we carefully consider the impact of matching young and older exercising subjects for exercise workload (i.e., absolute, relative, or fixed rate of metabolic heat production), aerobic fitness and training status, biological sex, and anthropometric differences when evaluating cardiovascular responses and thermoregulatory effector mechanisms. We do so in sections related to sweating responses, cardiovascular responses, and body temperature responses to exercise in hot environments. Finally, we consider the question, “In what specific environments might these age-related impairments effect thermoregulatory function and put humans at a greater risk for heat-related illness and injury?”

2. Sweating

The capacity for humans to thermoregulate during exercise-heat stress conditions relies primarily on the ability to activate eccrine sweat glands via sympathetic cholinergic mechanisms. It is well-established that the sweating rate for a given increase in core temperature (T_c) decreases with advancing age.^{5–8} This occurs in conjunction with a delayed onset threshold temperature and a reduced thermosensitivity of the sweating response.^{9,10} Attenuated sweating capacity in older adults appears to be attributable to reduced sweat output per activated gland, rather than a reduced number of heat activated sweat glands.^{11–13} Following intradermal injection of varying concentrations of the cholinergic analogue methylcholine (MCh) into the thighs of young and older men matched for aerobic fitness, there were no age-related differences in active gland density. However, there was a markedly lower sweat output per activated gland in the older group. A middle-aged group exhibited glandular sweat outputs that were intermediate to the young and older groups,¹³ suggesting a progressive and continuous decline in sweat output per gland throughout adulthood.

Although as a population, older adults have an impaired sweat capacity compared to their younger counterparts, at least part of this reduction is due to declines in physical fitness and/or training status rather than chronological age. The sweating rate during exercise is strongly determined by both acclimation status and $\dot{V}O_{2\max}$. Sweating rates are preserved in highly fit older men and women, albeit with a higher degree of variability.^{10,14–17} In older (58 years) and young (38 years) women matched for body surface area, sweating was correlated with $\dot{V}O_{2\max}$, but not age, when resting in hot, dry conditions (40 °C, 22.2 Torr).¹⁵ These findings were extended in a study examining thermal transients, where again sweating rate was strongly determined by $\dot{V}O_{2\max}$ rather than age.¹⁶ Further, multiple regression analysis of exercise (60 W for 1 h) in a warm environment (35 °C, 80% relative humidity (RH)) in a wide range of subject ages (20–73 years) indicated a significant relation between sweat loss and $\dot{V}O_{2\max}$, but not age (importantly, age and $\dot{V}O_{2\max}$ were not interrelated in this analysis¹⁷).

The relation between $\dot{V}O_{2\max}$ and sweating rate has recently been investigated comparing older highly trained cyclists (OHT; 56 years) with two younger groups. In this investigation one younger group was matched for training status (YHT; 26 years) and the other matched for $\dot{V}O_{2\max}$ (YMT; 27 years). All subjects exercise at workloads equivalent to 70% of $\dot{V}O_{2\max}$ in both hot (35 °C, 40% RH) and thermoneutral (20 °C, 40% RH) conditions. Despite an elevated metabolic heat production in YHT, sweat loss (kg) relative to body surface area (m²) was similar to OHT. Combined data from the three groups yielded a significant correlation between sweat loss and $\dot{V}O_{2\max}$, but not age.¹⁸ Therefore, strategies aimed at improving

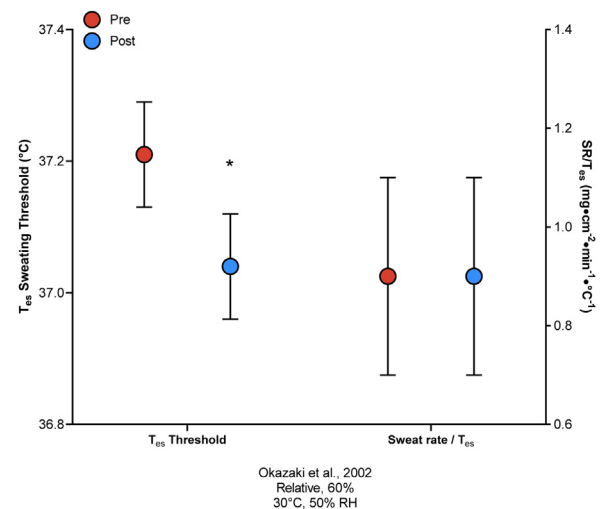


Fig. 1. Esophageal temperature (T_{es}) onset threshold for sweating responses (left y-axis) and the sweat rate (SR) thermosensitivity (i.e. slope of the increase in SR per °C rise in T_{es} ; right y-axis) in older sedentary adults before (Pre) and after (Post) 18 weeks of exercise training (from Okazaki et al.²¹). * $P < 0.05$ compared to pre-training.

aerobic fitness appear to augment sweating capacity, and thereby improve heat tolerance, in both young and older athletes.¹⁹

Exercise thermoregulatory responses in both moderate and hot conditions – including sweat onset threshold, sweat gland output, and sweat rate – can be improved in previously sedentary adults following aerobic exercise training.^{20–23} In young adults, 10 days of aerobic training for 1 h day⁻¹ in a thermoneutral environment (25 °C) at 75% $\dot{V}O_{2\max}$ increased sweat rate and shifted the sweating onset threshold to a lower T_c .²³ Similarly, following 18 weeks of aerobic exercise training on a cycle ergometer at 50–80% $\dot{V}O_{2\max}$ (resulting in an approximate 20% increase in $\dot{V}O_{2\max}$), the core temperature onset threshold for increased sweat rate, but not the thermosensitivity (i.e., the slope of the increase in SR per °C rise in T_{es}), improved in previously sedentary older (64 years) men (Fig. 1).²¹

Habitual aerobic exercise also prevents age-related decrements in sweat production.²⁴ Best et al. observed no significant differences in sweating rates relative to either body surface area or power output between young (26 years) and middle-aged (56 years) highly trained male cyclists.¹⁸ Similarly, others have reported that evaporative heat loss through sweating was impaired in untrained middle-aged and older men compared to both well-trained middle-aged and younger men during intermittent aerobic exercise in the heat, despite a similar sweating onset threshold.²⁵ Young (28 years) and middle-aged (54 years) competitive male runners matched for $\dot{V}O_{2\max}$ (Y: 58 mL kg⁻¹ min⁻¹; MA: 61 mL kg⁻¹ min⁻¹) displayed similar sweat rates and sweat sensitivity when exercising at a fixed workload (90% of 10-km race time) in the heat (40 °C, 30% rh). While a lower sweat gland output was observed in the middle-aged men, this was compensated by an increased number of heat-activated sweat glands,²⁶ suggesting a possible compensatory mechanism by which total sweat capacity may be maintained in older athletes via continued training.

It is important to note that differences in regional sweat gland function may exist between young and older adults. Following MCh injection, older adults displayed a significantly blunted sweating rate compared to young adults, an impairment observed to a greater extent on the forehead and limbs compared to the trunk.²⁷ Furthermore, the reduced sweating response was more pronounced in older women compared to age-matched men,²⁷ indicating that sex may play a role in modulating regional sweat gland function and sweat rate. Smith and Havenith explored this possibility by detailed

sweat mapping of the regional and local sweat patterns in elite young male and female runners exercising at two different intensities (60% and 75% $\dot{V}O_{2\max}$) in moderately warm (25 °C, 45% RH) conditions.^{28,29} Though men experienced greater total sweat loss at both exercise intensities,²⁸ sweating rates were highest for both sexes on the central upper back and lowest toward the limbs; however, sweat distribution was skewed toward the arms and hands in women compared to men.²⁹

With primary aging there is an attenuation in both regional sweating onset threshold and sweat rates during passive whole-body heating in older (64 years) versus young (23 years) adults.³⁰ However, in this same cohort, pharmacologically-induced regional sweating rates were similar between older and young subjects, suggesting an age-related decline in heat-activated sweat gland function but not cholinergic sensitivity.³⁰ George Havenith's laboratory at Loughborough University has recently published detailed sweat maps of older adults during both rest and exercise in a hot environment (32 °C, 50% RH).³¹ At rest, there were lower gross sweat losses and regional sweat rates in a majority of regions in older men (68 years), despite similar absolute heat production, compared to younger men (24 years). During exercise at a fixed heat production (200 W m⁻²), reductions in gross sweat loss persisted in the older group, though reductions in regional sweat rate in the older group were relegated toward the extremities (hands, legs, ankles, and feet). However, no women were included in that study. It is possible that, as indicated previously,²⁷ the reduction in gross sweat loss and regional sweat rate, as well as the greater distribution of sweat toward the extremities, observed in older men during exercise at a fixed heat production may be exacerbated in older women. Future investigation is warranted to determine whether potential sex differences in regional sweat rate and onset threshold persist with age and are modified by sex.

3. Cardiovascular responses to heat stress

In addition to evaporative heat loss, increases in cardiac output and blood flow redistribution from inactive visceral tissue to the cutaneous circulation allow for increased convective heat transfer.³² Healthy human aging is associated with attenuated central and peripheral cardiovascular responses during exercise, including reduced cardiac output and less visceral blood flow redistribution to the skin. In thermoneutral conditions, age-related attenuations in sedentary older adults are attributable to reductions in stroke volume and maximal heart rate.^{33,34} Studies specifically investigating the impact of aging on cardiovascular responses during exercise in hyperthermic conditions are contradictory. Endurance trained older adults who exercised in the heat ($\geq 60\%$ $\dot{V}O_{2\max}$; 36 °C, 20% RH) exhibited greater stroke volume, but attenuated cardiac output compared to young adults matched for $\dot{V}O_{2\max}$.³³ Cross-sectional studies differ on whether cardiac output is lower in aerobically fit older compared to young adults.^{20,35,36} Interestingly, Ho et al.²⁰ found that fit and sedentary older adults (age = 65 years, $\dot{V}O_{2\max} = 42$ vs 28 ml kg⁻¹ min⁻¹, respectively) did not differ in cardiac output when cycling at 60% $\dot{V}O_{2\text{peak}}$ (60 revolutions/min) in 36 °C and 20% rh; however, four weeks of endurance exercise training in older sedentary adults sufficient to increase $\dot{V}O_{2\text{peak}}$ by 27% yielded a significant increase in cardiac output when exercising at a relative (60% $\dot{V}O_{2\text{peak}}$), but not absolute (60% pre-training $\dot{V}O_{2\text{peak}}$), exercise intensity in 36 °C heat.

Increased cardiac output following training is likely due, at least in part, to a training-induced plasma volume expansion, although exercise studies are conflicting on whether the training-induced plasma volume expansion is attenuated with aging.^{37–40} Importantly, most training studies lack a direct within-study age comparison, and cross-sectional comparisons between fit and

sedentary older adults are equivocal regarding the role of fitness in the age-related declines in plasma volume.^{41–43} Future studies should directly compare young and older adults and the impacts of aerobic fitness and/or exercise training on plasma volume expansion.

Redistribution of blood flow during exercise in the heat from the renal and splanchnic circulations to the skin is also attenuated in older adults.³⁵ When exercising in hyperthermic conditions (30–36 °C, 20–60% RH), greater renal and splanchnic blood flow is observed in fitness-matched young vs. older adults.^{20,35,44} For example, when exercising at 60% $\dot{V}O_{2\max}$ in 36 °C, fit young adults (age = 26 years, $\dot{V}O_{2\max} = 43$ ml kg⁻¹ min⁻¹) redistributed ~37% more total blood flow away from the renal (Y: 1095 vs. 825 ml min⁻¹, O: 940 vs. 739 ml min⁻¹) and splanchnic (Y: 1139 vs 630 ml min⁻¹, O: 1137 vs. 770 ml min⁻¹) regions compared to fit older adults (age = 64 years, $\dot{V}O_{2\max} = 41.8$ ml kg⁻¹ min⁻¹).³⁵ The degree to which fitness plays a role in mediating this age-related decrement is unknown. Ho et al. found that total redistribution of splanchnic plus renal blood flow (but neither independently) was greater in fit older adults compared to their sedentary counterparts ($\Delta 568$ vs $\Delta 427$ ml min⁻¹). Four weeks of endurance training in previously sedentary older adults had no impact on either total or regional blood flow redistribution.²⁰ Although speculative, fitness-mediated differences in blood flow redistribution may be due to increased sympathetic nerve activity, and/or increased end-organ responsiveness to a given rise in sympathetic nerve activity, in fit individuals. However, plasma norepinephrine concentrations were not different between fit and sedentary older adults, or after 4 weeks of training in previously sedentary older adults,²⁰ suggesting that the latter may be a more plausible explanation.

Adequate perfusion of the cutaneous circulation is critical for temperature regulation during exercise in the heat. Increases in skin blood flow are mediated via a sympathetic cholinergic active vasodilator system. During passive, supine heat stress, skin blood flow can rise as much as 8 L/min in young, healthy adults.⁴⁵ However, healthy aging is associated with impairments in heat- and exercise-induced increases in skin blood flow of, on average, 25–50% compared to young adults.^{21,35,46–50} These impairments are largely mediated by an overall decreased sensitivity of the active vasodilator system, including decreased co-transmitter release and reduced nitric oxide bioavailability and signaling.^{48,51–53} Healthy aging is also associated with reductions in thermoregulation-induced skin sympathetic nerve activity (SSNA) responses. Older individuals exhibit reduced SSNA with heat stress and have a blunted vasodilator response for a given increase in SSNA compared to young adults, suggesting that age-related impairments in reflex cutaneous vasodilation are partially mediated by blunted efferent SSNA and/or end-organ responsiveness to SSNA during heat stress.⁵⁴ However, it is important to note these mechanistic skin blood flow and SSNA studies have been performed in non-exercising conditions due to experimental limitations with exercise. To date, no studies have investigated the impacts of fitness on these age-related declines future cross-sectional studies involving highly trained older adults and/or training studies are needed.

It is well-accepted that exercise training can improve central and peripheral cardiovascular function across all ages.³⁴ Thus, exercise training may be an effective strategy to mitigate age-related thermoregulatory cardiovascular impairments. However, the literature is equivocal regarding the effects of fitness on age-related cardiovascular function/impairment during exercise in the heat. Cross-sectional comparisons of young and older adults demonstrate age-related impairments in skin blood flow and other cardiac responses to exercise heat-stress even when subjects are matched for $\dot{V}O_{2\max}$.^{47,49,50} Fitness status (as measured by $\dot{V}O_{2\max}$) and age are independently predictive of skin blood flow responses to exer-

cise in warm humid environments (60 W for 1 h in 35 °C, 80% rh).¹⁷ In cross-sectional studies, fitness status in older cohorts is related to improved cardiovascular responses to exercise-heat stress.^{10,20} However, these responses are still generally lower than those of their younger cohorts. Thus, older elite athletes may still have decrements in skin blood flow responses to exercise-heat stress despite being highly trained.

To our knowledge, only three studies have investigated the effects of exercise training on thermoregulatory cardiovascular responses in older adults, all finding improvements in skin blood flow during heat stress.^{20–22} However, the mechanisms by which these improvements occur differ among the studies. Two investigations observed changes in the T_c onset threshold for cutaneous vasodilation, while the other saw improved thermosensitivity (i.e., increased slope of the relation between T_c and skin blood flow) (Fig. 2). Ho et al. observed a leftward shift in the mean body temperature (T_b) onset threshold for increased skin blood flow in young adults after endurance training; however, older adults who underwent the same exercise training protocol had only an improved thermosensitivity, but not onset threshold.²⁰ In contrast, Thomas et al. and Okazaki et al. both observed significant leftward shifts in onset threshold in older adults after endurance training.^{21,22} Putative underlying mechanisms mediating these changes include improved sensitivity of the active vasodilator systems (i.e. increased SSNA)²² and increased endothelium-dependent cutaneous vasodilation.⁵⁵

4. Core and skin temperature responses during exercise in the heat

Age-related impairments in sweating and skin blood flow responses can result in performance decrements during prolonged exercise-heat stress.² When comparing two groups exercising at the same absolute intensity, T_c responses are exaggerated in those with a lower $\dot{V}O_{2max}$.⁵⁶ This has led researchers conducting cross-sectional comparisons between different age cohorts to match groups for *relative* exercise intensity (i.e., a percentage of $\dot{V}O_{2max}$), often resulting in similar T_c and skin temperature (T_{sk}) responses^{10,14,28} despite disparate thermoeffector responses. Similar T_c and T_{sk} responses between young and older adults at different *absolute* exercise intensities suggest an impairment in the control of body temperature during exercise that may be at least partially mitigated with maintenance of fitness throughout aging (Table 1).

The mitigation of age-related declines in thermoeffector responsiveness with maintenance of aerobic fitness results in improved temperature regulation in the heat. However, the extent to which aerobic fitness independently modulates the maintenance of body temperature during exercise-heat stress in older adults is difficult to fully elucidate. Studies in which young, middle-aged, and/or older adults are matched for $\dot{V}O_{2max}$ most often demonstrate no differences in T_c or T_{sk} responses between age groups.^{14,25,26,33} In a cohort of 56 subjects ranging in age and $\dot{V}O_{2max}$, Havenith et al. determined the relative influence of age on cardiovascular and body temperature responses to low-intensity cycling exercise in 35 °C, 80% RH conditions using multiple regression analysis.¹⁷ The results of that study suggested that rectal temperature (T_{re}) responses were directly related to $\dot{V}O_{2max}$, but not age. However, due to age-related declines in $\dot{V}O_{2max}$ even in trained individuals, cross-sectional studies generally compare highly trained older adults to recreationally active young adults in an effort to match for $\dot{V}O_{2max}$.

Direct comparisons between age groups become more difficult when comparing highly fit middle-aged or older adults to highly fit young adults. Age-related reductions in $\dot{V}O_{2max}$ occur regardless of training state,⁴ resulting in difficulties matching groups

for metabolic heat production during exercise heat-stress. When thermoregulatory responses were compared during exercise heat-stress (30 °C, 55% rh) at ~65% $\dot{V}O_{2max}$ between 7 normally fit young (age = 29 years, $\dot{V}O_{2max}$ = 44 ml kg⁻¹ min⁻¹), 7 highly fit older (age = 64 years, $\dot{V}O_{2max}$ = 46 ml kg⁻¹ min⁻¹), and 6 normally fit older adults (age = 66 years, $\dot{V}O_{2max}$ = 33 ml kg⁻¹ min⁻¹), there were no differences in esophageal temperature (T_{es}) or T_{sk} between groups during exercise.¹⁰ Importantly, absolute exercise intensity, and thus metabolic heat production, was similar between the normally fit young and highly fit older groups, but was lower in the normally fit older group. Similarly, Best et al.¹⁸ compared young highly-trained (YHT; 26 years), young moderately-trained (YMT; 27 years), and older highly-trained (OHT; 56 years) subjects during exercise at 70% of $\dot{V}O_{2max}$ in 35 °C, 40% RH conditions. Absolute and relative $\dot{V}O_{2max}$, and power output at 70% $\dot{V}O_{2max}$, were significantly higher in YHT compared to YMT and OHT, resulting in significantly higher metabolic heat production during exercise. Despite differences in heat production, however, neither T_{re} nor T_{sk} were different among the three groups. In another study, fit middle-aged (54 years; $\dot{V}O_{2max}$, 58 ml kg⁻¹ min⁻¹) and young competitive male runners (28 years; $\dot{V}O_{2max}$, 61 ml kg⁻¹ min⁻¹) demonstrated no differences in run performance or T_{re} responses in 40 °C, 30% RH conditions.²⁶ Together, these studies provide strong evidence that age-related declines in body temperature regulation are primarily related to decrements in aerobic fitness, rather than age per se.

Training studies further indicate preserved body temperature control with improved aerobic fitness. In older (65 years), sedentary adults who exercised in the heat (36 °C, 20% rh) at the same relative intensity (60% $\dot{V}O_{2max}$) before and after 4 weeks of high-intensity aerobic training, there were no differences in T_{es} and T_{sk} responses.²⁰ When exercising at the same absolute intensity, however, both T_{es} and T_{sk} were lower at post- compared to pre-training. Similarly, 8 weeks and 18 weeks of exercise training resulted in progressive reductions in T_{es} and T_{sk} during exercise heat-stress.²¹ In another study, older adults whose $\dot{V}O_{2max}$ improved by a minimum of 5% after 16 weeks of aerobic training demonstrated a reduction in baseline and exercise T_b compared to pre-training.²² In sum, these studies suggest that exercise training-induced improvements in heat dissipation in older adults (1) reduces the T_c responses at the same absolute workload, and (2) allows older adults to perform higher workloads compared to pre-training without further increases in T_c (Fig. 3).

5. Implications for older athletes

The mechanisms mediating age-related declines in thermoregulatory capacity are multidimensional, manifesting as impairments in sweat gland output and skin blood flow. Higher $\dot{V}O_{2max}$ in older adults is related to improved sweating responses^{11,15–18} and cardiovascular parameters including cardiac output and skin blood flow,^{10,20,34} resulting in relatively preserved thermoregulatory function and better performance in the heat (Fig. 4).

Rates of decline in relative $\dot{V}O_{2max}$ (ml kg⁻¹ min⁻¹) appear to be greater in endurance-trained men and women compared to their healthy, sedentary counterparts.⁵⁷ It's unclear, however, how much of this decline in $\dot{V}O_{2max}$ are directly related to primary aging vs. secondary to reduced training volume. Training volume (i.e. intensity and duration) is reduced with advancing age,^{57,58} thereby reducing the stimulus for maintenance of aerobic capacity. In a 30-year follow-up of 5 previously-trained men, $\dot{V}O_{2max}$ remained lower in all subjects after a 6-month endurance training program compared to 30 years prior.⁵⁸ However, although the training dose in that study was similar to that which they completed 30 years earlier, in order to reduce risk of injury, the participants did not perform the same high-intensity training. Thus, although greater

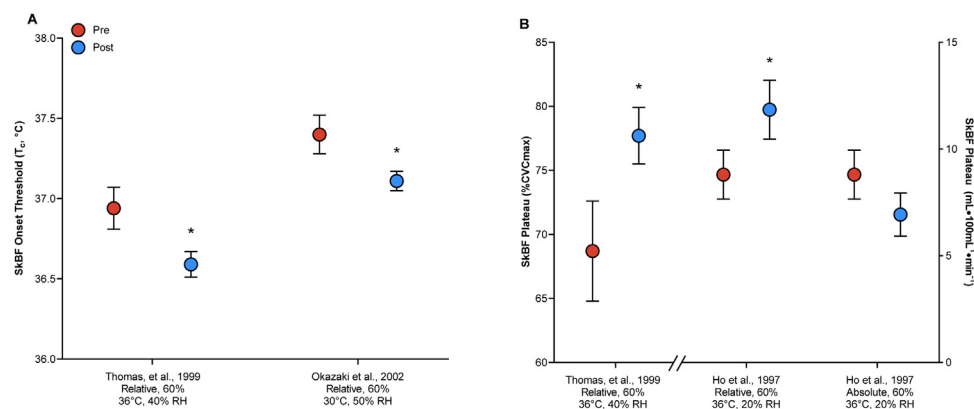


Fig. 2. The onset threshold for increased skin blood flow (SkBF; Panel A^{21,22}) and the magnitude of the SkBF response (Panel B^{20,22}) before (Pre) and after (Post) exercise training. For each study, the comparative workload and environmental conditions are provided on the x-axis. Relative = participants exercised at the same relative intensity before and after exercise training; absolute = participants exercised at the same absolute intensity before and after exercise training; RH = relative humidity; T_c = core temperature; %CVCmax = percentage of maximal cutaneous vascular conductance. * P < 0.05 compared to pre-training.

Table 1

Summary of studies investigating age-related changes in thermoregulatory responses to exercise-heat stress.

	Comparative workload	Environmental Conditions	Participants	SR	SkBF	T _{sk}	T _c
Tankersley et al., 1991	Relative 65% $\dot{V}O_{2max}$	30 °C, 55% RH	Vs. young normally fit Older highly fit Older normally fit	↔ ↓ ↓	↓ ↓ ↓	↔ ↔ ↔	↔ ↔ ↔
Best et al., 2011	Relative 70% $\dot{V}O_{2max}$	35 °C, 40% RH	Vs. young highly-trained Young moderately-trained Older highly-trained	↔ ↔ ↔	↔ ↔ ↔	↔ ↔ ↔	↔ ↔ ↔
Ho et al. 1997	Relative 60% $\dot{V}O_{2max}$	36 °C, 20% RH	Vs. young fit Young sedentary Older fit Older sedentary	↔ ↔ ↔ ↔	↓ ↓ ↓ ↓	↔ ↔ ↔ ↔	↔ ↔ ↔ ↔
de Paulo Viveiros 2011	Relative/absolute 90% 10-km time Absolute	40 °C, 30% RH 40 °C, 15% RH	Vs. young fit Middle-aged fit Vs. young normally fit Middle-aged trained	↔ ↔ ↔ ↔	↔ ↔ ↔ ↔	↔ ↔ ↔ ↔	↔ ↔ ↔ ↔
Stapleton et al. 2015	300 W 400 W 500 W Absolute 400 W	35 °C, 20% RH	Middle-aged untrained Older untrained Vs. 20–31 years 40–44 years 45–49 years 50–55 years 56–70 years	↓ ↓ ↔ ↓ ↓ ↓ ↓	↔ ↔ ↔ ↔ ↔ ↔ ↔	↔ ↔ ↔ ↔ ↔ ↔ ↔	↔ ↔ ↔ ↔ ↔ ↔ ↔
Larose et al. 2013			Vs. young Older	↔ ↔	↔ ↔	↔ ↔	↔ ↔
Kenney, 1988	Relative ~40% $\dot{V}O_{2max}$	37 °C, 60% RH	Vs. young Older	↔ ↔	↓ ↓	↔ ↔	↔ ↔
Kenney and Ho, 1995	Relative/absolute 60% $\dot{V}O_{2max}$	36 °C	Vs. young fit Older fit	↔ ↔	↓ ↓	↓ ↓	↓ ↓

Arrows indicate direction of the difference compared to the reference group (Vs.). More than one arrow suggests a greater difference. Relative/absolute suggests that fitness-matched groups resulted in the same relative and absolute exercise intensity. SR = sweat rate; SkBF = skin blood flow; T_{sk} = skin temperature; T_c = core temperature; RH = relative humidity.

aerobic fitness is clearly associated with improved thermoregulation in older adults, it remains unclear whether maintaining a similar training volume and intensity throughout aging would effectively sustain $\dot{V}O_{2max}$, or to what extent that would translate to preventing age-related thermoregulatory impairments in older athletes.

6. When does an age difference matter?

There is a vast literature of cross-sectional studies published by ourselves^{9,21,24,28,45,52} and others^{9,10,12,13,31} detailing age-related declines in physiological responses to environmental heat stress. However, these studies typically assess discrete physiological variables such as sweating rate, skin blood flow, hydration, cardiac responses, etc. Although it is important to understand age-related changes in those physiological variables, such changes may not always have meaningful impacts on tolerance to exercise in the heat

(i.e., compensable vs. uncompensable exercise heat-stress). Indeed, greater whole-body heat storage in older compared to young adults does not always result in divergent T_c responses.^{8,59,60} It is important, therefore, to consider the specific exercise intensities and environmental conditions in which age-related declines in thermoeffector responses may result in exaggerated exercise heat-stress in older adults.

6.1. Intensity

Given its role in metabolic heat production, exercise intensity is an important driver of the overall heat load independent of environmental conditions. Direct and indirect calorimetry studies from Glen Kenny's lab at the University of Ottawa have examined the exercise intensities at which aging impairs heat loss under fixed environmental conditions.^{25,60} Those investigations showed that age differences in evaporative heat loss became greater with

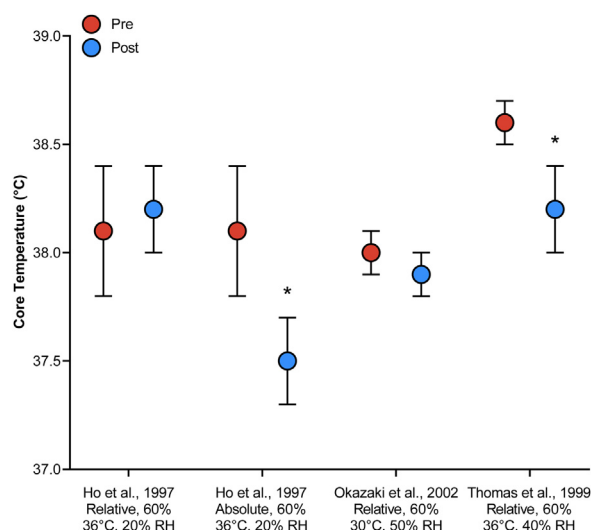


Fig. 3. Comparisons in core temperature responses during exercise-heat stress in older adults before (Pre) and after (Post) exercise training across studies.^{20–22} For each study, the comparative workload and environmental conditions are provided on the x-axis. Relative = participants exercised at the same relative intensity before and after exercise training; absolute = participants exercised at the same absolute intensity before and after exercise training. * $P < 0.05$ compared to pre-training.

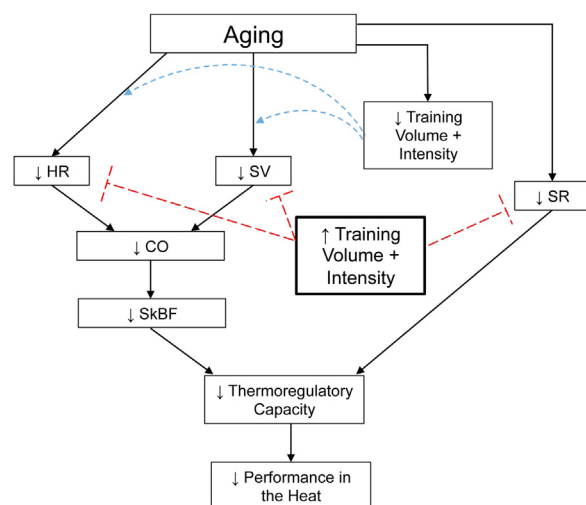


Fig. 4. The influences of aging and/or training volume and intensity on cardiovascular and thermoregulatory responses during exercise-heat stress. Aging may independently reduce heart rate (HR) and stroke volume (SV), and therefore cardiac output (CO), during exercise in athletes, resulting in impaired redistribution of blood to the cutaneous circulation during exercise-heat stress. Similarly, primary aging is associated with reduced sweat rates (SR). Reduced training volume and intensity in aging athletes likely contribute to such declines, although it is unclear to what magnitude. Although speculative, maintaining and/or increasing training volume and intensity throughout the aging process may prevent or slow the age-related declines in these responses in lifelong athletes, thereby improving thermoregulatory capacity and performance during exercise-heat stress.

increasing exercise intensities and concomitant increased heat loads. During exercise at incrementally increasing intensities in hot, dry conditions (40 °C, 15% RH), older (58 ± 5 years) women demonstrated an impaired capacity to dissipate heat compared to young, $\dot{V}O_{2\max}$ -matched women only at exercise-induced heat loads of ≥ 325 W.⁶⁰

6.2. Environment

While there is compelling evidence that aging is associated with decreased heat dissipation and elevated risk of heat illness in hot

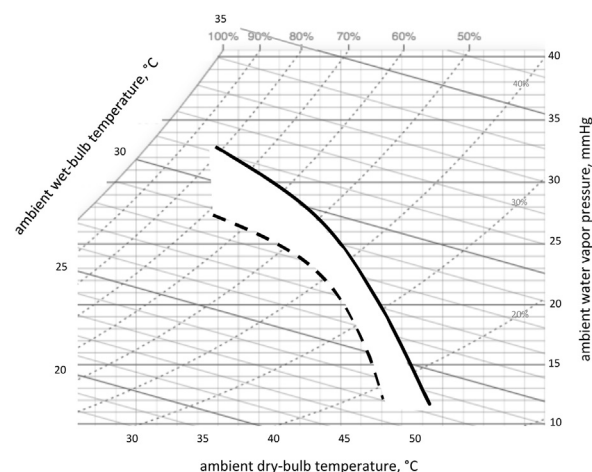


Fig. 5. Adapted from Kenney 2020,⁶³ depicts psychrometric limit lines for unacclimated young (solid line) and older (dashed line) women exercising at 30% $\dot{V}O_{2\max}$. The white area between lines theoretically describes environments in which the older women are differentially adversely affected.

ambient conditions, the significant question remains: In what specific environments does this age disparity begin to occur? Larose et al.⁵⁹ demonstrated that heat dissipation is impaired in older men in both hot, dry (35 °C, 20% rh) and warm, humid (35 °C, 60% rh) environments due to reduced rates of evaporative heat loss. In that study, exercise was performed in four 15-min bouts, separated by 15-min recovery, at a rate of metabolic heat production of 400 W and external work output of ~ 80 W.

Our laboratory originally developed, and has a long history of using, a unique, rigorous, and reproducible approach to identify critical environmental limits to prolonged heat exposure.^{61–63} This research paradigm combines physiological and biophysical approaches to delineate safe (compensable) from potentially unsafe (uncompensable) environmental conditions from a human heat balance standpoint. Our overarching goal is to identify those environments (combinations of ambient temperature and humidity) in which thermoregulatory health is compromised in healthy older adults.

Toward that end, we recently described – graphically and quantitatively – the critical environmental limits for exercising women between the ages of 62 and 80 years.⁶³ Rather than choosing a single environment (i.e., dry, humid) these psychrometric limit lines define the myriad combinations of ambient temperature and humidity above which human heat balance cannot be maintained for a given metabolic heat production. Fig. 5 depicts environmental limit lines on a standard psychrometric chart for young and older women exercising at 30% $\dot{V}O_{2\max}$. The white area between lines theoretically describes environments in which age makes a difference, i.e., older women are differentially adversely affected.

These limits, and mathematically-derived critical evaporative coefficients (K_e), can then be used to model low- to moderate-intensity exercise responses in hot environments but higher intensity exercise intensities have not been examined. In the studies performed to date, this intensity has historically been chosen because 1) it reasonably reflects light-to-moderate daily workloads typical of a healthy, normally active population; 2) it is the intensity associated with an 8-h work day in many industrial settings⁶⁴, and 3) it reflects the intensity of many self-paced recreational activities. Values for older athletes exercising at higher intensities have not been determined using this protocol. Future studies should test the global hypothesis that aging will shift critical environmental limits to a narrower range of safe environments across the psychrometric spectrum (encompassing warm-humid to hot-dry environments) in athletes exercising at high intensities.

7. Perspectives

Primary aging is associated with impaired thermoregulatory function, resulting in increased risk of heat-related illness and decreased athletic performance in the heat. Exercise training that increases or maintains $\dot{V}O_{2\max}$ throughout aging improves thermoregulatory function, including enhanced sweating and cardiovascular responses to exercise heat-stress. However, the extent to which $\dot{V}O_{2\max}$ can be maintained throughout the aging process, and how much training is requisite for maintained thermoregulatory function, remain unclear. Further, it is not evident to what degree age-related decrements in thermoregulatory function are mitigated by $\dot{V}O_{2\max}$, per se, rather than lifelong engagement in exercise training independent of improvements in $\dot{V}O_{2\max}$. Finally, although a great deal is known about the impact of aging and fitness on discrete physiological variables (such as sweating and skin blood flow responses) during heat stress, studies are only beginning to emerge detailing the specific exercise intensities and environmental conditions under which age-related changes likely result in greater body temperature responses in older compared to young adults. Future work is needed to identify those exercise intensities and/or environmental conditions under which age-related impairments in thermoregulatory function result in greater heat strain, risk of heat-related illness, and decrements in athletic performance in highly-fit and normally-fit older adults.

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