Accepted for publication in International Journal of Biometeorology on 12th July 2016

The biophysical and physiological basis for mitigated elevations in heart rate with electric fan use in extreme heat and humidity

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Running Title: *Electric fan use during heat waves*

Keywords: heat waves, cardiovascular strain, thermoregulation, sweating

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ABSTRACT

Electric fan use in extreme heat wave conditions has been thought to be disadvantageous because it might accelerate heat gain to the body via convection. However, it has been recently shown that fan use delays increases in heart rate even at high temperatures (42°C) in young adults. We here assess the biophysical and physiological mechanisms underlying the apparently beneficial effects of fan use. Eight males (24±3 y; 80.7±11.7 kg; 2.0±0.1 m^2) rested at either 36°C or 42°C, with (F) or without (NF) electric fan use (4.2 m/s) for 120 min while humidity increased every 7.5 min by 0.3 kPa from a baseline value of 1.6 kPa. Heart rate (HR), local sweat rate (LSR), cutaneous vascular conductance (CVC), core and mean skin temperatures, and the combined convective/radiative heat loss (C+R), evaporative heat balance requirements (E_{req}) and maximum evaporative potential (E_{max}) were assessed. C+R was greater with fan use at 36°C (F: 8±6, NF: 2±2 $W \cdot m^{-2}$; $P=0.04$) and more negative (greater dry heat gain) with fan use at 42°C (F: -78±4, NF: -27±2 $W \cdot m^{-2}$; $P<0.01$). Consequently E_{req} was lower at 36°C (F: 38±16, NF: 45±3 $W \cdot m^{-2}$; $P=0.04$) and greater at 42°C (F: 125±1, NF: 74±3 $W \cdot m^{-2}$; $P<0.01$) with fan use. However, fan use resulted in a greater E_{max} at baseline humidity at both 36°C (F: 343±10, NF: 153±5 $W \cdot m^{-2}$; $P<0.01$) and 42°C (F: 376±13, NF: 161±4 $W \cdot m^{-2}$; $P<0.01$) and throughout the incremental increases in humidity. Within the humidity range that a rise in HR was prevented by fan use but not without a fan, LSR was higher in NF at both 36°C ($P=0.04$) and 42°C ($P=0.05$), and skin temperature was higher in NF at 42°C ($P=0.05$), but no differences in CVC or core temperatures were observed (all $P>0.05$). These results suggest that the delayed increase in heart rate with fan use during extreme heat and humidity is associated with improved evaporative efficiency.

INTRODUCTION

Over the past 20 years, heat waves - characterized by extended bouts of extreme heat and humidity - have led to high levels of excess morbidity and mortality in the United States (Whitman et al. 1997), Europe (Fouillet et al. 2006), Australia (Nitschke et al. 2011), and most recently India and Pakistan (Lancet 2015). Cardiovascular events are consistently identified as an underlying cause of heat-related mortality and morbidity (Bouchama et al. 2007; Hajat et al. 2010), with those who do not have access to air conditioning being particularly vulnerable. Moreover, the high electricity requirements associated with widespread air conditioning use by the majority of households in urban areas during heat waves have in some cases led to massive power failures (Luber and McGeehin 2008), and a consequent surge in morbidity and mortality rates (Schuman 1972; Hartz et al. 2012). It is therefore evident that affordable and energy efficient cooling strategies (Kravchenko et al. 2013) are urgently needed to mitigate cardiovascular strain during heat waves.

Electric fans provide a simple cooling intervention at a fraction of the price and energy requirement of modern air conditioning (Gupta et al. 2012; Salamanca et al. 2014). However, current heat management guidelines from public health agencies such as the World Health Organization, United States Environmental Protection Agency, and The Centers for Disease Control and Prevention typically advise against fan use at air temperatures above 35 to 37°C as they are thought to, at best, be ineffective (Wolfe 2003; CDC 2004), and at worst, exacerbate physiological strain and the risk of heat illness and dehydration (Wolfe 2003; Matthies et al. 2008; Victorian Government Department of Health 2013). We recently demonstrated that electric fan use at air temperatures up to 42°C delays heat-induced elevations in heart rate in young healthy males (Ravanelli et al. 2015). However, the underlying physiological and biophysical mechanisms for the protective effect of electric fans at high air temperatures and humidity were not determined.

When ambient temperature exceeds skin temperature, which in a hot environment will typically be ~35°C (Gagge et al. 1937), heat will be gained via convection. With fan use, this environmental heat load will be added to the body at a faster rate. However, fan use favours elevated rates of sweat evaporation. Importantly, increased levels of sweat evaporation with fan use can be achieved without the need for greater sweat production through improvements in evaporative efficiency - the amount of sweat that evaporates relative to the amount produced (Adams et al. 1992). In contrast, not using a fan would lead to decrements in evaporative efficiency and therefore greater sweat rates to overcome compromised sweat evaporation (Candas et al. 1979b). Since greater sweat rates are generally accompanied by greater cutaneous vasodilation (Wingo et al. 2010; Smith et al. 2013), it is possible that the delayed increase in heart rate with fan use during passive heat exposure is associated with less peripheral vasodilation and therefore less of a need for cardiac output to increase in order to maintain blood pressure.

The purpose of the present study was to i) evaluate how changes in physiological heat loss responses and human heat balance are altered by electric fan use during simulated extreme heat wave conditions; and ii) identify how fan use previously resulted in a lower heart rate (Ravanelli et al. 2015) at air temperatures equal to (36°C), and far exceeding (42°C) the limits for fan use presently stated in public health recommendations (CDC 2004; WHO 2009). It was hypothesized that improved sweat evaporation with fan use outweighs greater convective heat gain, leading to a lower requirement for skin blood flow and sweat production.

METHODS

The data presented in the current manuscript were collected as part for a larger study examining humidity inflection points for heart rate and core temperature with and without fan use (Ravanelli et al. 2015). Eight healthy, normotensive, non-smoking young males, with no pre-existing cardiovascular, metabolic, or neurological issues participated in the study (age: 24±3 y; mass: 80.7±11.7 kg; height: 1.77±0.05 m; BSA: 1.98±0.14 m^2). All participants completed one preliminary visit and four experimental trials. The experimental protocol was approved by the University of Ottawa Research Ethics Board, and conformed to the guidelines set forth in the 1964 Declaration of Helsinki. All participants provided written informed consent prior to their participation in the study. Participants were instructed to avoid vigorous exercise or physical activity 24 hours prior, refrain from alcohol 12 hours prior, eat a light meal, and avoid any caffeinated beverages at least 6 hours prior to testing. The preliminary visit consisted of providing informed consent and anthropometric measurements (weight and height) to estimate body surface area (DuBois and Dubois 1916).

Instrumentation

Rectal temperature was measured using a thermistor probe (Mon-a-therm®, Mallinckrodt Medical, St. Louis, MO) inserted to a depth of 20 cm past the anal sphincter. Esophageal temperature was measured using a thermistor probe (Mon-a-therm®, Mallinckrodt Medical, St. Louis, MO) inserted through the nasal cavity into the esophagus. The end of the thermistor probe was estimated to be located at a region nearest the left ventricle (Mekjavic and Rempel 1990). Skin temperature was measured using four thermistors (Concept Engineering, Old Saybrook, CT, USA) which were secured to the skin using surgical tape (Transpore®, 3M, London, ON). Mean skin temperature was calculated as the weighted average of four sites using the formula reported by Ramanathan (1964): chest 30%, triceps 30%, thigh 20%, and calf 20%. Temperature measurements were sampled every 5 s (NI cDAQ-91722 module, National Instruments, Austin, TX) and displayed in real-time on a desktop computer using customized LabView software (v7.0, National Instruments, Austin, TX).

Heart rate was measured using cardio-recorder (Polar RS 800, Polar electro Oy, Kempele, Finland) and coded transmitter (Polar wearlink T31 coded, Polar electro Oy, Kempele, Finland) which recorded every 5 seconds. The recording was downloaded to a desktop computer using the manufacturer's software (Polar ProTrainer Versions 5.40.172, Kempele, Finland) and averaged every minute. Systolic and diastolic blood pressures were measured using an automated cuff (E-Sphyg II 9002, American Diagnostic Corporation, Hauppauge, NY, USA) at baseline, and at the end of each humidity stage during the ramp protocol. Mean arterial pressure was subsequently calculated as:

$$(1/3 \times \text{systolic blood pressure}) + (2/3 \times \text{diastolic blood pressure})\ [\text{mmHg}] \qquad (1)$$

Skin blood flow was measured using Laser Doppler Flowmetry probes (Small Angled Thermostatic Probe #457, Perimed, Järfälla, Sweden) placed on the chest and forearm. Skin blood flow perfusion units were displayed by the Laser Doppler Perfusion Monitor (Periflux System 5000, Perimed, Järfälla, Sweden) and simultaneously recorded at a sampling rate of 5 seconds by the manufacturers software (Perisoft for Windows Version 2.5.5, Perimed, Järfälla, Sweden). Skin blood flow was averaged between recordings from the chest and forearm and expressed as i) absolute values and ii) cutaneous vascular conductance, which was derived as the quotient of perfusion units and mean arterial pressure.

Local sweat rates of the chest and forearm were measured using ventilated sweat capsules. Anhydrous air was supplied through each 4.1-cm^2 capsule at a rate of 1.2 L/min (chest) and 1.4 L/min (forearm). Capsules were secured to the skin using surgical tape. The temperature and humidity of the air leaving both capsules were measured by individually factory calibrated capacitance hygrometers (HMT333, Vaisala, Vantaa, Finland). Local sweat rates were calculated as the product of flow rate and effluent absolute humidity, and expressed relative to the amount of skin surface covered by the capsule (mg/cm^2/min). Local sweat rate was expressed as the average between chest and forearm.

Experimental protocol

All trials were performed in a climatic chamber that precisely regulated ambient air temperature and absolute humidity, situated at the Thermal Ergonomics Laboratory at the University of Ottawa in Canada. During the fan trials, an 18” diameter mechanical fan (Whirlpool, Benton Harbor, MI, USA) was set at full speed and placed 1.0 m directly in front of the participant. The mean whole body air velocity (4.2 m/s) generated by the fan was derived from calculations of the convective heat transfer coefficient (h_c – see equation 4) using measurements of convective heat loss (see equation 3) in a 15°C environment using a 34 zone thermal manikin (NEWTON; Measurement Technology Northwest, Seattle, USA) at the Environmental Ergonomics Centre at Loughborough University, UK. The four experimental trials were i) 36˚C with fan (36F); ii) 36˚C with no fan (36NF); iii) 42˚C with fan (42F); and iv) 42˚C with no fan (42NF). The experimental trials were presented in a balanced order determined using a Latin square design. All trials were separated by at least 48 h. Upon arrival at the laboratory, participants provided a urine sample to ensure euhydration and similar hydration states between trials by measuring urine specific gravity with a refractometer (Reichert TS 400, Depew, NY). All urine specific gravity measurements were lower than 1.025 (Kenefick and Cheuvront 2012) and were similar between experimental trials for each person (±0.002). Each participant wore a standardized t-shirt and shorts and sat on a plastic chair that covered part of their back and upper rear thigh. The dry insulation (with fan: 0.04 clo; without fan: 0.10 clo) and evaporative resistance (0.01 m^2kPa/W) of this standardized ensemble was measured using a thermal manikin at Loughborough University, UK. During the 36NF and 42NF trials, participants sat behind a 122 cm high barrier to ensure still (<0.1 m/s) air flow around them. Throughout all trials, ambient air velocity was measured using a hot wire anemometer (VelociCalc 9535, TSI Inc, Shoreview MN, USA) positioned ~20 cm anterior to the participants torso.

Each trial began with the participant entering the climatic chamber regulated at a temperature of either 36°C or 42°C, and an ambient vapor pressure of 1.6 kPa, and sitting quietly for 45 min. An initial body mass measurement was then taken using a platform scale (Combics 2, Sartorius, Mississauga, ON, Canada). Following a further 20 min at a vapor pressure of 1.6 kPa, vapor pressure was increased in a step-wise fashion by 0.3 kPa every 7.5 minutes (Kenney et al. 1993) until 5.6 kPa, at which point the participant's body mass was once again measured and a urine sample was obtained. The duration of each trial (excluding the 45-min baseline rest) was 120 min. Table 1 illustrates the ambient temperature and absolute humidity for each stepwise increase in humidity.

Partitional Calorimetry

Heat balance was estimated using partitional calorimetry and parameters are presented as the mean values for each condition. Metabolic heat production (H_{prod}) was not measured and was assumed to be 1.2 W/kg of total body based on the following equation:

$$H_{prod} = VO_2 \cdot \frac{\left(\left(\frac{RER - 0.7}{0.3}\right)e_c\right)+\left(\left(\frac{1.0 - RER}{0.3}\right)e_f\right)}{60 \cdot A_D} \cdot 1000 \text{ [W/m}^2\text{]} \quad (2)$$

Where oxygen consumption (VO_2) was estimated as 3.5 ml/kg/min, the respiratory exchange ratio (RER) was assumed to be 0.85, e_c is the caloric equivalent per liter of oxygen for the oxidation of carbohydrates (21.13 kJ per L of O_2 consumed), e_f is the caloric equivalent per liter of oxygen for the oxidation of lipids (19.62 kJ per L of O_2 consumed).

Convective heat exchange from the skin, *C*, was calculated as (Kerslake, 1972):

$$C = h_c \cdot (T_{sk} - T_a) \text{ [W/m}^2\text{]} \quad (3)$$

Where: h_c is the convective heat transfer coefficient for an individual facing an air velocity (Mitchell 1974):

$$h_c = 8.3 \cdot v^{0.6} \text{ [W/m}^2\text{/K]} \quad (4)$$

Where: v is mean air velocity derived using a thermal manikin (4.2 m/s). During the 36NF and 42NF trials air velocity was less than 0.2 m/s and h_c was assumed to be 3.1 $W/m^2/K$ (Parsons 2002).

Radiant heat transfer (R) was estimated by:

$$R = h_r \cdot (T_{sk} - T_r) \text{ [W/m}^2\text{]} \quad (5)$$

Where: h_r (radiant heat transfer coefficient) in $W \cdot m^{-2} \cdot K^{-1}$ is estimated using the following:

$$h_r = \varepsilon \cdot 4\sigma \cdot (A_r/A_D) \cdot ((T_{sk} + T_r)/2 + 273.15)^3 \text{ [W/m}^2\text{/K]} \quad (6)$$

Where: ε is the area weighted emissivity of the body surface (0.95), σ is the Stefan-Boltzmann constant ($5.67\text{x}10^{-8}$ W/m^2/K^4), A_r/A_D is the effective radiative surface area (ND) which can be estimated as 0.70 for a seated person (Fanger 1967), and $T_{sk} + T_r$ is the sum of the mean skin temperature and mean radiant temperature (°C), assumed to be equivalent to T_a (°C).

Respiratory heat loss was estimated using the following:

$$E_{res} + C_{res} = 0.0173 \cdot (H_{prod}) \cdot (5.87 - P_a) + 0.0014 \cdot (H_{prod}) \cdot (34 - T_a) \text{ [W/m}^2\text{]} \quad (7)$$

The evaporative requirement to maintain heat balance (E_{req}) in W/m^2 was estimated by rearranging the conceptual heat balance equation:

$$E_{req} = H_{prod} - (C + R + C_{res} + E_{res}) \text{ [W/m}^2\text{]} \quad (8)$$

Required skin wettedness (ω_{req}), defined by Gagge (1937), was estimated as:

$$\omega_{req} = E_{req} / E_{max} \text{ [ND]} \quad (9)$$

Where: E_{max} is the theoretical maximum rate of evaporation in the prevailing climate when 100% of the skin surface is saturated in sweat, and can be calculated using:

$$E_{max} = (P_{sk,sat} - P_a) / (R_{e,cl} + [1/(f_{cl} \cdot h_e)] \qquad \text{[W/m}^2\text{]} \quad (10)$$

Where: $R_{e,cl}$ is the evaporative heat transfer resistance of the clothing layer in m^{2}kPa/W, f_{cl} is the clothing area factor (surface area of the clothed body divided by the surface area of the nude body; ND), and h_e is the evaporative heat transfer coefficient in W/m^2/kPa. $P_{sk,sat} - P_a$ is the difference in water vapor pressure between the skin and air in kPa. While P_a is a measured in absolute terms (in kPa), $P_{sk,sat}$ can be derived from Antoine's equation:

$$P_{sk,sat} = (\exp (18.956 - [4030.18/(T_{sk} + 235)]))/10 \text{ [kPa]} \quad (11)$$

Where: T_{sk} is mean skin temperature (°C).

For equation 10, the evaporative heat transfer coefficient (h_e) in W/m^2/kPa can be estimated using the product of the Lewis number (16.5 ND) and h_c:

$$h_e = 16.5h_c \qquad (12)$$

Statistical Analysis

Based on a power calculation (G*Power 3.1.9.2) with β- and α-values equal to 0.95 and 0.05 respectively, a minimum sample size of 5 participants was required based on evidence from critical vapor pressures of 4.16±0.19 kPa and 4.60±0.13 kPa for unacclimated (Kenney and Zeman 2002) and heat acclimated (Kamon and Avellini 1976) women, respectively. All thermometric, cardiovascular, and heat loss measurements were averaged over the last minute of each humidity stage and expressed as means (± standard deviation).

As reported previously (Ravanelli et al. 2015), the critical humidity at which elevations in heart rate were observed was higher with fan use at both 36°C (F: 4.9±0.4 kPa, NF: 3.7±0.5 kPa; $P<0.001$) and 42°C (F: 3.8±0.6 kPa, NF: 3.1±0.6; $P=0.01$). The temperature and humidity ranges for stages during which an elevation in heart rate was observed are presented in Table 1. At the stage corresponding to the upward rise in heart rate, paired t-tests were used to assess differences between groups (36F vs 36NF; 42F vs 42NF) for C + R, E_{req}, E_{max}, and ω_{req}. Moreover, paired t-tests were used to assess the change from baseline to the end of the humidity ramp protocol between fan conditions (i.e. 36F vs 36NF; 42F vs 42NF) for heart rate, and esophageal, rectal, and mean skin temperatures.

To compare physiological variables across humidity levels during which elevations in heart rate were observed during the NF condition but not the F condition, three separate humidity "zones" were identified for each participant (Figure 1) at 36°C and 42°C. These zones were defined as: Zone 1 (Z1): heart rate not elevated from baseline during both fan conditions; Zone 2 (Z2): heart rate elevated during NF, but not during the F condition; Zone 3 (Z3): heart rate elevated during both fan conditions. A two-way repeated measures ANOVA was used to analyze the data using the repeated factor of humidity "zone" (Rest, Z1, Z2, Z3, and End-Trial) and the non-repeated factor of fan use (Levels: F and NF) to compare heart rate, skin, esophageal and rectal temperatures, as well as local sweat rate and cutaneous vascular conductance. When significance was found, individual differences were assessed using a Student's t-test. For all multiple comparisons a fixed probability (5%) of making a type I error was maintained throughout using a Holm-Bonferroni correction. All analysis was conducted using Graphpad Prism 6 for Windows statistical software (Version 6.01, La Jolla, CA, USA).

RESULTS

Alterations in human heat balance with fan use

At 36°C, dry heat loss was greater with fan use ($P=0.04$), which led to a lower E_{req} (Figure 2A). E_{max} at baseline was increased more than 2-fold during fan use compared to no fan ($P<0.01$). During the subsequent step-wise increases in humidity, E_{max} declined to a greater ($P<0.01$) extent with fan use before an upward inflection in heart rate was observed (Figure 2A). The ω_{req} at baseline was lower ($P<0.001$) with fan use (0.13 ± 0.02) compared to the no fan condition (0.28 ± 0.04). At the critical humidity at which an upward inflection in heart rate was observed, ω_{req} remained lower ($P<0.01$) with a fan (0.38 ± 0.13) than without a fan (0.52 ± 0.11).

At 42°C, dry heat gain was ~70 W/m^2 greater ($P<0.01$) with fan use (Figure 2B), which resulted in a greater E_{req} ($P<0.01$). However, E_{max} was 2-fold greater with fan use at baseline, and declined a greater extent compared to the no fan condition ($P<0.01$) before an upward rise in heart rate was observed (Figure 2B). At baseline, ω_{req} was lower ($P<0.01$) with fan use (0.35±0.02) compared to the no fan condition (0.45±0.03). The ω_{req} at the critical humidity at which an upward inflection in heart rate was observed was similar ($P=0.34$) with a fan (0.56±0.14) compared to the no fan condition (0.61±0.07).

Heart Rate

The range of humidity which captured Z1, Z2, and Z3 is outlined in Tables 2 and 3 for 36°C and 42°C, respectively. While Z2 demonstrates overlap in humidity due to individual variability for the critical humidity at which an inflection in heart rate occurred, no overlap is present between Z1 and Z3 for 36°C and 42°C. Prior to beginning the humidity–ramp protocol, heart rate was similar between conditions at 36°C ($P=0.60$) and 42°C ($P=0.35$). In humidity zone 1 (Z1), heart rate remained similar between conditions at both 36°C ($P = 0.27$) and 42°C ($P = 0.20$). By definition, heart rate was greater during the no fan condition at both 36°C ($P = 0.002$) and 42°C ($P = 0.05$) in Z2. In Z3, heart rate was elevated from baseline during both conditions, but was greater during the no fan condition at both 36°C ($P = 0.003$) and 42°C ($P = 0.01$). At the end of the humidity-ramp protocol, heart rate was greater during the no fan condition at both 36°C ($P = 0.02$) and 42°C ($P < 0.001$).

Core and mean skin temperatures

At 36°C, esophageal and rectal temperatures were similar ($p>0.05$) between conditions at baseline. Core temperatures were also similar between conditions across the 3 zones (Table 2). At the end of the humidity-ramp protocol, esophageal temperature was greater without fan use

(P=0.01), but rectal temperature was similar (P=0.08) to when a fan was used (Table 2). Similarly, at 42°C, esophageal and rectal temperatures were similar (P>0.05) between conditions at baseline, and at each zone (Table 3). At the end of the humidity-ramp protocol, esophageal temperature was greater without a fan (P=0.03), but rectal temperature was similar (P=0.21) between conditions (Table 3).

At 36°C, mean skin temperature was similar (P>0.05) between conditions at baseline, heart rate zones 1 and 2 (Table 2), but became greater during the no fan condition during heart rate zone 3 (P=0.01) and at the end of the humidity-ramp protocol (P=0.007). At 42°C, mean skin temperature was greater (P<0.05) with fan use at baseline, and during all three zones (Table 3). By the end of the humidity-ramp protocol, mean skin temperature was similar between conditions (P=0.14) due to a greater (P=0.04) increase in mean skin temperature from baseline during the no fan condition (Table 3).

Mean Arterial Pressure

Mean arterial pressure was similar (P>0.05) between conditions throughout the humidity-ramp protocol at both 36°C (Table 2) and 42°C (Table 3).

Sweating

At 36°C, local sweat rate was similar between conditions at baseline and during Z1, but was greater (P<0.05) without fan use during Z2 and Z3, as well as at the end of the humidity-ramp protocol (Table 2). At 42°C, local sweat rate was also similar between conditions at baseline and during Z1, but became greater (P<0.05) without fan use during Z2 and Z3 as well as at the end of the humidity-ramp protocol (Table 3). As previously reported (Ravanelli et al. 2015), whole body sweat rate was greater at 36°C with a fan (180±10 g/hr) than without (153±18 g/hr; P=0.01). Similarly at 42°C, whole body sweat rate was greater with a fan (399±26 g/hr) than without (241±46 g/hr; P<0.001).

Skin blood flow

At 36°C, skin blood flow (absolute values) and cutaneous vascular conductance were similar between conditions at baseline and during Z1 and Z2, but became greater without a fan during Z3 and at the end of the protocol (Table 2). At 42°C, skin blood flow (absolute units) and cutaneous vascular conductance was similar between conditions throughout the humidity-ramp protocol (Table 3).

DISCUSSION

The current study examined potential biophysical and physiological factors associated with the delayed increase in heart rate with fan use during extreme heat and humidity conditions. Biophysically, a greater air velocity across the skin surface with fan use led to negligible changes in dry heat exchange at 36°C, whereas ~70 W/m^2 of additional dry heat was gained via convection at 42°C (Figure 2). However, at both ambient temperatures the greater potential for evaporation with fan use increased evaporative efficiency. While evaporative efficiency was not directly quantified, the additional ~70 W/m^2 of dry heat gain with fan use at 42°C must have been offset by at least an equally greater evaporative heat loss as the increase in core temperature was delayed relative to the no fan condition (Ravanelli et al. 2015). From a physiological perspective, the different inflection points for increases in heart rate between the fan and no fan conditions seemed to coincide with elevations in sudomotor output at both 36°C and 42°C. Collectively, the delayed increase in heart rate with electric fan use was associated with increased evaporative efficiency and lower sudomotor output.

At 36°C, air temperature was similar to mean skin temperature. Therefore, differences in dry heat loss and thus the evaporative requirement for heat balance were trivial (<5 W/m^2) between conditions. The influence of fan use on the potential for evaporative heat loss however was profound (i.e. ~250 W/m^2 greater with a fan; Figure 2A) due to a greater convective and therefore evaporative heat transfer coefficient (Nelson et al. 1948; Clifford et al. 1959). As ambient humidity progressively increased during the humidity-ramp protocol, E_{max} naturally declined due to a shrinking humidity gradient between the skin and air. The E_{max} value at which elevations in heart rate occurred was slightly greater with fan use. However, because E_{max} started at a much greater level with fan use it look longer, and therefore a greater relative humidity (i.e. 83±6% RH; Ravanelli et al. 2015), for E_{max} to reach a similar level as that observed during the no fan condition. This greater "buffer" for increases in humidity with fan use at 36°C can be explained in terms of greater evaporative efficiency. The work of Candas et al. (1979ab) and Alber–Wallstrom et al. (1985) demonstrate that if E_{req} is small relative to E_{max}, evaporative efficiency is greater, but as E_{req} approaches E_{max} evaporative efficiency rapidly declines. Prior to the start of the humidity ramp protocol at 36°C, E_{req} was ~10% of E_{max} with fan use but ~30% of E_{max} without fan use (Figure 2A). Decrements in evaporative efficiency would have therefore occurred at a lower relative humidity (i.e. earlier during the ramp protocol) during the no fan condition. In order to maintain heat balance during heat stress, E_{req} must be sustained. With reductions in evaporative efficiency, a concomitant rise in sweating must occur to sustain E_{req} which was reflected by greater local sweat rate values (Table 2).

The main argument proposed by public health agencies for not using a fan during heat waves is that additional air flow across the skin will accelerate dry heat gain (Wolfe 2003; CDC 2004; WHO 2009). Indeed, this was observed during the 42°C trial as fan use resulted in ~70

W/m^2 greater dry heat gain. However, an often-neglected advantage of fan use in the public health literature is that it also promotes evaporation by increasing evaporative efficiency. In this study, fan use resulted in an evaporative heat loss potential that was 160 W/m^2 greater relative to no fan use. As such, the required evaporation for heat balance only accounted for ~35% of the maximum evaporative potential during fan use, relative to ~45% with no fan. According to the findings of Alber-Wallstrom et al. (1985), decrements in evaporative efficiency would have occurred even before the humidity ramp protocol started during the no-fan condition, whereas fan use would have maintained sweat evaporation at ~100%. Moreover, the critical skin wettedness (ω_{crit}; E_{req}/E_{max}) at which elevations in heart rate were observed by Berglund and Gonzalez (1977) was lower with air movement relative to still air. The present results partially concur with these findings with lower ω_{crit} when the inflection in heart rate occurred at 36°C (F: 0.38±0.13; NF: 0.52±0.11), but not 42°C (F: 0.56±0.14; NF: 0.61±0.07). The reason for differences in ω_{crit} between fan conditions at 36°C but not 42°C is unclear, however it must be acknowledged that partitional calorimetric estimates of heat transfer values are based on several assumptions and subject to variability. Despite these limitations, we propose that fan use facilitated a greater evaporative efficiency during the humidity-ramp protocol, which is further supported by the lower local sweat rate values (Table 3).

The underlying physiological mechanisms responsible for the delayed increase in heart rate with fan use are difficult to determine from the present data. It was hypothesized that earlier heart rate elevations without fan use would be preceded by greater peripheral vasodilation, leading to a greater heart rate requirement for the maintenance of blood pressure. Indirect evidence suggests this may be the case. Assuming stroke volume was similar between fan and no fan conditions, greater heart rate during the no fan conditions presumably lead to greater cardiac output. Given that blood pressure was similar between fan and no fan conditions, it is possible that a greater cardiac output would be associated with greater peripheral vasodilation during the no fan conditions. In theory, this could be due to greater cutaneous vasodilation, although we cannot rule out the possibility of greater vasodilation within other vascular beds. While this hypothesis is supported by greater cutaneous vasodilation during the no fan condition when elevations in heart rate were observed at 36°C, a separation in heart rate between fan conditions (Z2) was observed without any preceding differences in cutaneous vasodilation at 42°C (Table 3). Alternatively, a higher mean skin temperature at 42°C with fan use could have theoretically led to greater cutaneous vasodilation (Rowell et al. 1970; Wyss et al. 1975; Wingo et al. 2010) and heart rate via stimulation of cutaneous thermoreceptors (Shibasaki et al. 2015); but this was not observed. In fact, heart rate was lower with fan use at 42°C.

It should be noted that cutaneous vasodilation was only measured at two local sites and it is therefore possible that differences in other body regions were not detected. Rowell et al. (1970) reported that elevations in heart rate during aggressive passive heating (47.5°C water perfused suit) were not lowered following the restoration of mean arterial pressure to normothermic levels, suggesting that blood pressure maintenance is not necessarily the primary driver of heat-related elevations in heart rate. This notion was further supported by Cui et al. (2002) who observed only a minor decrease in heart rate during passive heating (46°C water perfused suit) following the reestablishment of normothermic blood pressure with phenylephrine infusion. Collectively, these and other studies (Kamon and Belding 1971; Wyss et al. 1974; Gorman and Proppe 1982) suggest that heart rate elevations during passive heating are partially driven by direct effects of temperature upon the heart (Jose et al. 1970; Gorman and Proppe 1982). However, core temperatures were similar between fan and no fan conditions, and were actually unchanged from baseline when the elevations in heart rate occurred at both ambient temperatures. Differences in core temperature therefore cannot explain the earlier elevations in heart rate observed without fan use. The only physiological response measured that differed between fan conditions at both ambient temperatures when the elevation in heart rate occurred without fan use was local sweat rate (Table 2 & 3).

Perspectives

The present results suggest that the different critical humidities at which elevations in heart rate are observed with and without fan use are potentially associated with an elevated sudomotor drive, secondary to decrements in evaporative efficiency. While future studies are required to examine whether this is a direct cause-and-effect or indirect link, cooling interventions during extreme heat exposure (i.e. heat waves) that strive to mitigate elevations in heart rate could possibly focus on reducing the heat balance requirement for sweat production. Under circumstances that air conditioning is not available, which is commonplace for most vulnerable populations during heat waves (Bouchama et al. 2007; Basu and Ostro 2008; Kravchenko et al. 2013), the propagation of convective flow across the skin coupled with external moistening of the skin may suppress the need for sweating. Empirical evidence supporting this notion however is needed. If supplemental air flow is not available, a combination of external skin wetting and conductive cooling (e.g. cold water forearm or foot immersion) that provides ~80-90 W/m^2 of heat loss would minimize the evaporative requirement for heat balance and therefore reduce the necessity for sweating at 42°C (Figure 2B).

Limitations and Future Studies

The present data pertain only to young, healthy males; they therefore do not account for age-related decrements in sweating capacity of older individuals (Kenney and Hodgson 1987;

Inoue et al. 1991), nor the lower maximum evaporative capacity of females (Gagnon and Kenny 2011). The potential benefit of fan use has also only been demonstrated in hot/humid conditions. Inhabitants of some geographical regions (e.g. South Australia) often experience very hot (>45°C) and dry (RH<10%) heat waves. In such environments, most secreted sweat would readily evaporate in relatively still air and fan use may therefore not increase evaporative efficiency while creating additional dry heat gain. The efficacy of fan use under hot/dry versus hot/humid conditions must therefore be evaluated. Moreover, metabolic heat production was not measured directly and assumed to be constant. While this assumption may be limited, Hardy & Stolwijk (1966) observed very minor differences in metabolic rate between the ambient temperatures tested in the present study. It is also difficult to provide a comprehensive explanation for the different heart rate responses between fan conditions without measurements of cardiac output, therefore further research incorporating this measure is warranted. Finally, only one fan speed, diameter, orientation and distance from the participant was tested and further research is required to assess the influence of these variables on thermal and cardiovascular strain.

Conclusion

In conclusion, delayed elevations in heart rate with fan use during extreme heat and humidity conditions are associated with i) a greater increase in evaporative efficiency relative to the increase in convective heat gain; and ii) a lower sudomotor output.

ACKNOWLEDGEMENTS

The authors thank the volunteers for their participation in the present study.

GRANTS

This research was supported by the Natural Sciences and Engineering Research Council of Canada (NSERC) Discovery Grant (no.386143-2010, O. Jay). N.M. Ravanelli is supported by a University of Ottawa Excellence Scholarship and a NSERC Postgraduate Scholarship (PGS-D).

AUTHOR CONTRIBUTIONS

N.M.R., O.J., S.H., & G.H. were involved in the concept and design of the research question and methodology; N.M.R. performed all data collection; N.M.R. analyzed the data; N.M.R., O.J., S.H., G.H., & D.G. interpreted the results; N.M.R. prepared figures; N.M.R. & O.J. drafted the manuscript; N.M.R. & O.J. edited the manuscript; N.M.R., O.J., D.G., S.H., & G.H. approved the final version of manuscript.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by any of the authors.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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Table 1 Ambient temperature (T_a) and absolute humidity (AH) for each stepwise increase in humidity at 36°C and 42°C expressed as mean ± standard deviation. Shaded rows denote the stages coinciding with an upward rise in heart rate.

Stage	**36°C - Fan**		**36°C – No fan**		**42°C - Fan**		**42°C – No fan**	
	T_a (°C)	**AH (kPa)**	**T_a (°C)**	**AH (kPa)**	**T_a (°C)**	**AH (kPa)**	**T_a (°C)**	**AH (kPa)**
1	36.6 ± 0.2	1.7 ± 0.2	36.2 ± 0.3	1.7 ± 0.1	42.8 ± 0.1	1.8 ± 0.1	41.3 ± 0.4	1.9 ± 0.1
2	36.7 ± 0.3	2.0 ± 0.0	36.3 ± 0.3	2.0 ± 0.0	42.8 ± 0.1	2.0 ± 0.1	41.4 ± 0.3	2.0 ± 0.1
3	36.6 ± 0.4	2.4 ± 0.1	36.4 ± 0.2	2.4 ± 0.1	42.8 ± 0.1	2.3 ± 0.2	41.5 ± 0.3	2.3 ± 0.2
4	36.6 ± 0.4	2.7 ± 0.1	36.4 ± 0.3	2.7 ± 0.1	42.8 ± 0.1	2.6 ± 0.3	41.5 ± 0.3	2.5 ± 0.3
5	36.6 ± 0.4	3.0 ± 0.1	36.4 ± 0.2	3.0 ± 0.1	42.8 ± 0.0	2.9 ± 0.1	41.6 ± 0.2	2.9 ± 0.2
6	36.6 ± 0.4	3.3 ± 0.0	36.5 ± 0.3	3.3 ± 0.1	42.8 ± 0.1	3.2 ± 0.1	41.6 ± 0.2	3.3 ± 0.1
7	36.6 ± 0.4	3.5 ± 0.0	36.5 ± 0.3	3.5 ± 0.1	42.8 ± 0.1	3.4 ± 0.1	41.6 ± 0.2	3.5 ± 0.1
8	36.7 ± 0.3	3.8 ± 0.0	36.6 ± 0.2	3.8 ± 0.0	42.8 ± 0.1	3.7 ± 0.1	41.6 ± 0.2	3.7 ± 0.1
9	36.7 ± 0.3	4.1 ± 0.0	36.6 ± 0.2	4.1 ± 0.0	42.8 ± 0.1	3.9 ± 0.1	41.6 ± 0.2	4.0 ± 0.1
10	36.7 ± 0.3	4.4 ± 0.0	36.6 ± 0.2	4.4 ± 0.1	42.8 ± 0.1	4.1 ± 0.0	41.7 ± 0.3	4.2 ± 0.1
11	36.7 ± 0.3	4.6 ± 0.0	36.7 ± 0.2	4.7 ± 0.1	42.8 ± 0.1	4.3 ± 0.0	41.6 ± 0.3	4.4 ± 0.1
12	36.7 ± 0.2	4.9 ± 0.0	36.7 ± 0.2	5.0 ± 0.1	42.8 ± 0.1	4.6 ± 0.1	41.7 ± 0.3	4.7 ± 0.1
13	36.7 ± 0.2	5.2 ± 0.1	36.7 ± 0.2	5.2 ± 0.1	42.8 ± 0.1	4.8 ± 0.0	41.7 ± 0.3	4.9 ± 0.1
14	36.7 ± 0.2	5.4 ± 0.1	36.8 ± 0.2	5.4 ± 0.1	42.8 ± 0.1	5.1 ± 0.1	41.8 ± 0.3	5.2 ± 0.1
15	36.7 ± 0.2	5.6 ± 0.1	36.8 ± 0.2	5.6 ± 0.1	42.8 ± 0.1	5.3 ± 0.0	41.7 ± 0.3	5.4 ± 0.1
16	36.7 ± 0.2	5.6 ± 0.2	36.9 ± 0.2	5.7 ± 0.1	42.7 ± 0.1	5.6 ± 0.1	41.7 ± 0.4	5.6 ± 0.1