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PRIORITY REPORT



# Type 2 diabetes does not exacerbate body heat storage in older adults during brief, extreme passive heat exposure

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**ABSTRACT**

Aging exacerbates hyperthermia and cardiovascular strain during passive heat exposure, but it remains unclear whether those effects worsen in older adults with type 2 diabetes (T2D). We examined these responses in unacclimatized, physically active, older individuals with (n = 13, mean ± SD age: 60 ± 8 years, HbA1c: 7.0 ± 1.0%) and without (Control, n = 30, 62 ± 6 years) well-controlled T2D during a brief, 3-h passive exposure to extreme heat (44°C, 30% relative humidity). Metabolic heat production, dry heat gain, total heat gain (metabolic heat production + dry heat gain), evaporative heat loss, body heat storage (summation of heat gain/loss), rectal and mean skin temperatures as well as heart rate were measured continuously. No between-group differences were observed for metabolic heat production (T2D vs. Control; 53 ± 5 vs. 55 ± 7 W/m$^2$), dry heat gain (48 ± 9 vs. 47 ± 11 W/m$^2$), total heat gain (101 ± 10 vs. 102 ± 14 W/m$^2$) and evaporative heat loss (83 ± 10 vs. 85 ± 12 W/m$^2$) over the 3 h (all P > 0.05). Consequently, the changes in body heat storage (380 ± 93 vs. 358 ± 172 kJ, P = 0.67) were similar between groups. Moreover, no between-group differences in rectal and mean skin temperatures or heart rate were measured. We conclude that unacclimatized, physically active, older adults with well-controlled T2D do not experience greater hyperthermia and cardiovascular strain compared to their healthy counterparts while resting in extreme heat for a brief, 3-h period.

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Aging; body temperature; calorimetry; chronic disease; climate change; heat loss

## Introduction

Extreme heat events are considered one of the largest health threats of the 21$^{st}$ century [1]. This threat may be greater among older adults (>55 years), who demonstrate impairments in whole-body heat loss relative to young adults (primarily due to reduced sweat evaporation), which increase body heat storage and exacerbate cardiovascular demands while resting in extreme heat [2]. This impairment may be worse in older adults with type 2 diabetes (T2D), who display regional impairments in the thermoeffector responses that facilitate heat loss (cutaneous vasodilation and sweating) relative to their healthy counterparts during local heating of the skin [3–5], pharmacological stimulation [6,7], and passive heat stress [8–11]. In the context of moderate-to-vigorous exercise, these impairments have translated to reductions in whole-body heat loss in habitually-physically active, older adults with relatively well-controlled T2D ( HbA1c of ~7.0%) in mild (30°C) and high (40°C) heat stress conditions relative to age-matched healthy controls [12,13]. However, while T2D is associated with higher rates of heat-related illness and death during extreme heat events compared to the general population [14–16], it remains unclear if this may be a consequence of reductions in whole-body heat loss caused by the impairments in thermoeffector function that may exacerbate elevations in hyperthermia and cardiovascular strain, specifically under resting conditions. This is a critical knowledge gap given the importance of this information for establishing evidence-based guidelines to protect the health of individuals with

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T2D against the harmful effects of extreme heat events, which are expected to become more frequent and intense [1].

We therefore used a direct air calorimeter to measure minute-by-minute changes in whole-body evaporative and dry heat exchange and the resulting changes in body heat storage and core temperature as well as cardiovascular strain in older adults with and without T2D during a brief (3 h), extreme passive heat exposure (44°C, 30% relative humidity); conditions reflective of deadly extreme heat events, especially in temperate continental climates [17]. We hypothesized that older adults with T2D would gain more heat from the environment paralleled by reductions in evaporative heat loss relative to their healthy counterparts, which would worsen elevations in body heat storage and core temperature during the 3-h exposure. This would be paralleled by elevated cardiovascular strain.

## Materials and methods

The experimental protocol was approved by the University of Ottawa Health Sciences and Science Research Ethics Board and agrees with the *Declaration of Helsinki*. All volunteers provided written informed consent before participating.

Forty-three older adults with (n = 13, T2D) and without (n = 30; Control) T2D participated in this study (Table 1). Results from the control group were previously reported [2], whereas 11 of the 13 adults with T2D were previously included in a study that examined age- and T2D-related differences in heart rate variability during a brief, extreme passive heat exposure [18]. Only patients with relatively-well controlled T2D who had been diagnosed for ≥5 years, had HbA1c of 5.5–8.5%, had no diagnosed T2D-related complications (e.g. gastroparesis, renal disease, uncontrolled hypertension, neuropathies), were not on insulin therapy and were performing the recommended 150 min/week of moderate-to-vigorous aerobic exercise were included [19]. Participants were considered unacclimatized as all testing was completed in the late fall and winter months in Ottawa, Canada.

On separate days, participants completed one preliminary session where physical characteristics were determined and one experimental session. For both sessions, participants were instructed to arrive hydrated and having refrained from exercise, alcohol, caffeine, and anti–inflammatory drugs for 24 h. After confirming euhydration (urine specific gravity: <1.025 [20]) and completing instrumentation (~26°C), participants completed a 3-h seated passive exposure wearing shorts, sleeveless-top (females), and sandals in extreme heat conditions (44°C, 30% relative humidity).

The modified Snellen direct air calorimeter was used to perform continuous measurements of whole-body evaporative heat loss and dry heat gain ($W/m^2$) [21]. While the calorimeter is designed to ensure the evaporation of all sweat produced by the participant, it is important to note that the resultant airflow around the participant is relatively low (<0.3 m/s) [22–24]. Metabolic rate ($W/m^2$) was estimated continuously from measures of oxygen uptake and carbon dioxide production obtained using indirect calorimetry (AEI Technologies, Bastrop, TX, USA). Since sweat evaporation forms the only avenue for heat loss during exposure to ambient conditions that exceed skin temperature and therefore cause dry heat gain, body heat storage ($W/m^2$) was calculated as the sum of the total heat gain (metabolic heat production + dry heat gain) and evaporative heat loss. Core temperature was measured with a temperature probe (Mallinckrodt Medical, St-Louis, MO, USA) inserted 12 cm past the anal sphincter. Mean skin temperature was estimated from measurements on the biceps (30%), chest (30%), thigh (20%), and calf (20%) using T-type thermocouples (Concept Engineering, Old Saybrook, CT, USA) [25]. Heart rate (beats/min) was continuously recorded (Polar Electro, Oy, Finland) and expressed as absolute values and as a percentage of heart rate reserve. Heart rate reserve was calculated as the difference between using each participant's maximal age-predicted heart rate [26,27] and resting heart rate prior to heat exposure.

Calorimetry data were averaged over the first (0–30 min) and final 30-min (151–180 min) of exposure whereas thermometric and cardiovascular data were averaged over the first (0–5 min) and last (175–180 min) 5-min of exposure. Data were then compared using a two-way ANOVA with factors of group (Control and T2D) and time (Start, End). When a significant interaction was detected, between-group differences were examined using

unpaired, 2-tailed *t*-tests. Cumulative body heat storage (kJ) was calculated as the sum of the total heat gain and whole-body evaporative heat loss (as indicated by the shaded area in Figure 1). Mean body temperature change was calculated from cumulative body heat storage (kJ) divided by body mass (kg) and the average specific heat capacity of the body (i.e. 3.47 $kJ \cdot kg^{-1} \cdot °C^{-1}$) [28]. Both variables, as well as physical characteristics, were compared between groups using an unpaired, 2-tailed *t*-test. Based on the effect size (Cohen's $d = 1.12$) for a 156 (SD 139) kJ difference in cumulative body heat storage between groups with an average body mass of 85 kg (equivalent to ~0.5°C difference in body temperature for an individual weighing 85 kg, which is clinically important as such a rise in body temperature would place an individual in a mildly hyperthermic state) [2], ≥11 participants per group were required to detect between-group differences of this effect size with 80% statistical power. All data are reported as mean (SD) unless stated otherwise. Alpha was 0.05 for all comparisons. Analyses were performed using Prism 8 (GraphPad, La Jolla, CA, USA).

## Results

While there were no between-group differences in age, height, body mass, body surface area, physical activity level, or systolic and diastolic blood pressures (all $P > 0.05$), the T2D group had greater mean body mass index and percent body fat relative to their healthy counterparts (both $P < 0.05$, Table 1).

The changes in total heat gain and evaporative heat loss, as well as cumulative body heat storage, are presented in Figure 1. Data obtained for all physiological variables are presented in Table 2. Throughout the exposure, metabolic heat production increased, while dry heat gain and the resulting total heat gain decreased (main effect of time: all $P < 0.01$). Evaporative heat loss also increased over time, whereas body heat storage decreased (main effect of time: both $P < 0.001$). Furthermore, core

**Table 1.** Participant characteristics.

| | T2D | CON | |
|---|---|---|---|
| Variable | Mean (SD) | Mean (SD) | *P*-Values |
| Males/Females (#) | 11/2 | 24/6 | - |
| Age (years) | 60 (8) | 62 (6) | 0.27 |
| Height (m) | 1.74 (0.06) | 1.74 (0.08) | 0.97 |
| Mass (kg) | 90.0 (15.3) | 79.7 (16.3) | 0.06 |
| Body surface area ($m^2$)[a] | 2.1 (0.2) | 1.9 (0.2) | 0.10 |
| Body mass index ($kg/m^{-2}$)[b] | 29.7 (4.9) | 26.2 (4.1) | 0.02 |
| Body fat (%)[c] | 31.3 (7.8) | 25.6 (7.1) | 0.02 |
| Systolic blood pressure (mm Hg)[d] | 128 (10) | 125 (8) | 0.33 |
| Diastolic blood pressure (mm Hg)[d] | 80 (8) | 78 (7) | 0.44 |
| Physical activity (min/week)[e] | 331 (229) | 391 (215) | 0.42 |
| HbA1c (%) | 7.0 (1.0) | - | - |
| Duration of diabetes (years) | 12 (6) | - | - |
| Diabetes medication class | | | |
| Metformin | 13 | - | - |
| DPP-4 inhibitors | 2 | - | - |
| SGLT-2 inhibitors | 1 | - | - |
| Meglitinides | 1 | - | - |
| Cardiovascular medication class | | | |
| Statins | 8 | - | - |
| Angiotensin converting enzyme inhibitors | 5 | - | - |
| Acetylsalicylic acid | 3 | - | - |
| Angiotensin receptor blocker | 2 | - | - |
| Calcium channel blocker | 1 | - | - |
| Diuretic | 1 | - | - |

Abbreviations: T2D, participants with type 2 diabetes; CON, participants without type 2 diabetes; HbA1c, glycated hemoglobin. [a]Body surface area was calculated as 0.20247 x height $(m)^{0.725}$ x mass $(kg)^{0.425}$. [b]Body mass index was calculated as mass kg/height $(m)^{2}$. [c]Body fat was determined using the hydrostatic-weighing technique using the Siri equation [33].[d] Systolic and diastolic blood pressures were assessed by manual auscultation. [e]Physical activity was quantified as the average weekly duration spent performing structured physical activity of moderate-to-vigorous intensity using a questionnaire.

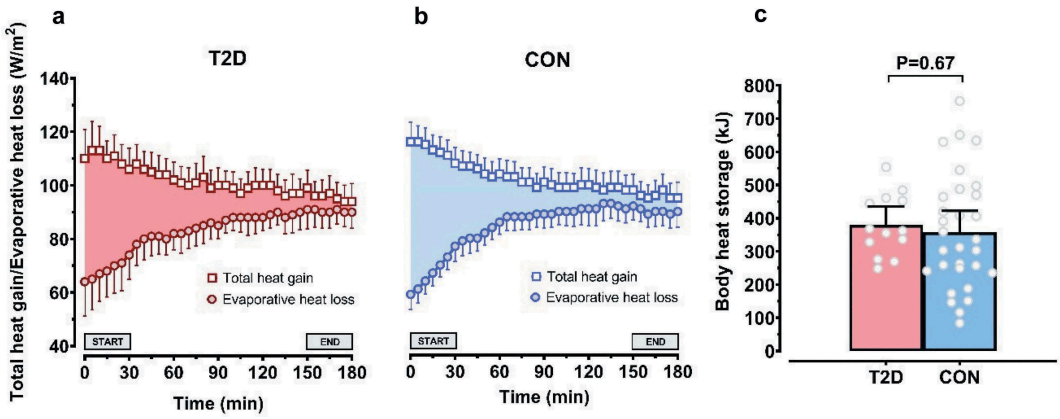


**Figure 1.** The changes (5 min averages) in total heat gain and evaporative heat loss over the course of a 3-h extreme passive heat exposure in adults with (T2D, panel A) and without (CON, panel B) well-controlled T2D as well as the subsequent cumulative change in body heat storage (panel C). Total heat gain represents the combination of metabolic heat production and dry heat gain. The shaded area within panel A and B represents body heat storage over time, which is calculated from the difference between the total heat gain and evaporative heat loss. The averages of the first (start) and last 30-min of exposure (end) were used for statistical analyses (presented in Table 2) whereas cumulative heat storage was compared between-groups using an unpaired, 2-tailed t-test. Significance was set at $P < 0.05$.

and mean skin temperatures, as well as heart rate and percent heart rate reserve increased over the exposure period (main effect of time: all $P < 0.001$). However, no between-group differences (main effect of group: all $P > 0.05$) or time*group interaction effects (all $P > 0.05$) were observed for core and mean skin temperatures as well as percent heart rate reserve. In contrast, a main effect of group ($P = 0.03$) was detected for absolute heart rate such that it was greater for T2D relative to control at the end of the exposure ($P = 0.02$). Lastly, cumulative body heat storage (T2D: 380 (93) kJ vs. Control: 358 (172) kJ, $P = 0.67$; Figure 1) and the change in mean body temperature (T2D: 1.2 (0.3)°C vs. Control: 1.3 (0.7)°C, $P = 0.69$) over the exposure was also similar between groups.

## Discussion

In contrast to our hypothesis, we showed that unacclimatized, but physically active, older adults with and without relatively well-controlled T2D displayed similar capacities to dissipate heat during a brief, 3-h extreme passive heat exposure. Consequently, they demonstrated comparable elevations in body heat storage, core temperature, as well as cardiovascular strain.

To prevent continued increases in core temperature during heat exposure, the rate of heat loss must balance the rate of heat gained. As depicted in Figure 1, total heat gain exceeded evaporative heat loss at the start of exposure in both groups, which is the typical response observed at the start of a heat exposure due to a lag in the activation of heat loss responses of cutaneous vasodilation and sweating [21]. Consequently, both groups showed rapid increases in body heat storage in the early period of heat exposure, which were followed by reduced, yet sustained rates of body heat storage, and therefore core temperature in the final 30-min. This was due to progressive, albeit similar, decreases in dry heat gain, which were paralleled by concomitant increases in evaporative heat loss that were also similar between groups. However, the increases in evaporative heat loss were insufficient to offset the greater heat gains in the final 30-min. Consequently, heat balance was not achieved. In this context, we previously showed that, during passive exposure to identical ambient conditions (44°C and 30% relative humidity), unacclimatized, but physically active, healthy older adults displayed an attenuated increase in evaporative heat loss during the first hour of exposure resulting in a greater amount of heat stored relative to young (18–30 years), physically active, adults. This attenuation persisted for the duration of the exposure despite a greater rate of dry heat gain (~6 W/m$^2$) [2], leading to a 1.8-fold greater body heat storage relative to their younger counterparts. In the current study, we anticipated that body heat storage would be even worse in older adults with T2D given that age-related

**Table 2.** Heat exchange and core and mean skin temperatures as well as heart rate and percent heart rate reserve at the start and end of a 3-h extreme passive heat exposure in older adults with and without type 2 diabetes.

| | T2D | CON | ANOVA *P*-Values | | |
|---|---|---|---|---|---|
| Variable | Mean (SD) | Mean (SD) | Group | Time | Interaction |
| Metabolic heat production ($W/m^2$) | | | | | |
| Start exposure | 51 (5) | 54 (7) | 0.31 | 0.008 | 0.41 |
| End exposure | 54 (5) | 56 (8) | | | |
| Dry heat gain ($W/m^2$)[a] | | | | | |
| Start exposure | 59 (12) | 59 (13) | 0.88 | <0.001 | 0.75 |
| End exposure | 41 (9) | 40 (11) | | | |
| Total heat gain ($W/m^2$)[b] | | | | | |
| Start exposure | 110 (12) | 113 (15) | 0.75 | <0.001 | 0.49 |
| End exposure | 95 (10) | 96 (14) | | | |
| Evaporative heat loss ($W/m^2$) | | | | | |
| Start exposure | 69 (16) | 69 (11) | 0.89 | <0.001 | 0.96 |
| End exposure | 90 (9) | 90 (13) | | | |
| Body heat storage ($W/m^2$)[c] | | | | | |
| Start exposure | 39 (11) | 44 (16) | 0.29 | <0.001 | 0.52 |
| End exposure | 4 (4) | 6 (7) | | | |
| Core temperature (°C) | | | | | |
| Start exposure | 37.4 (0.4) | 37.3 (0.3) | 0.63 | <0.001 | 0.37 |
| End exposure | 37.8 (0.3) | 37.8 (0.3) | | | |
| Mean skin temperature (°C) | | | | | |
| Start exposure | 35.9 (0.4) | 35.9 (0.4) | 0.40 | <0.001 | 0.07 |
| End exposure | 36.5 (0.4) | 36.3 (0.4) | | | |
| Heart rate (beats/min) | | | | | |
| Start exposure | 84 (14) | 77 (12) | 0.03 | <0.001 | 0.11 |
| End exposure | 102 (17) | 90 (15) | | | |
| Heart rate reserve (%) | | | | | |
| Start exposure | 9 (8) | 10 (6) | 0.33 | <0.001 | 0.08 |
| End exposure | 31 (12) | 25 (12) | | | |

Abbreviations: T2D, participants with type 2 diabetes; CON, participants without type 2 diabetes. Heat exchange data are averages of the first (start) and last (end) 30 min of the 3-h extreme heat exposure (44°C, 30% relative humidity). [a]Total heat gain represents the combination of metabolic heat production and dry heat gain. [b]Rate of body heat storage was calculated as the difference between total heat gain and evaporative heat loss. All variables were analyzed using a two-way ANOVA with the factors of group (Control and T2D) and time (Start, End). Significance was set at $P < 0.05$.

impairments in cutaneous vasodilation and sweating are exacerbated in individuals with T2D [8,29], which could affect whole-body heat exchange. However, we showed no significant between-group differences in whole-body evaporative heat loss and dry heat gain. Consequently, older adults with and without well-controlled T2D demonstrated a similar elevated body heat storage and therefore core temperature (Figure 1 and Table 2).

The lack of T2D-related impairment in whole-body heat loss and therefore similar thermal and cardiovascular strain may in part be attributed to the fact that our participants were engaging in regular physical activity, were relatively well-controlled, and were free of T2D-related complications including neuropathies. Altogether, this may have mitigated any reductions in thermoeffector function that would have compromised the body's physiological capacity to dissipate heat [6,30,31]. Furthermore, it is important to note that participants in this study remained physically inactive (seated) for the duration of the exposure. In situations requiring moderate-to-high increases in metabolic rate (≥200 $W/m^2$), which augment the rate that heat must be dissipated (e.g. activities of daily living such as housekeeping and maintenance, work, others), habitually-physically active older adults with relatively-well controlled T2D exhibit marked reductions in whole-body evaporative heat loss that exacerbate body heat storage relative to their healthy counterparts [12,13]. Therefore, the lack of T2D-related impairment in whole-body heat loss in the current study may be attributed to the requirement for heat loss (i.e. total heat gain) being insufficient to observe such impairments.

Although body heat storage and core temperature were similar in older adults with and without T2D, these outcomes may have important health implications. Current World Health Organization

(WHO) recommendations indicate that a 1°C rise in body temperature is inadvisable and may pose a health concern [32]. In this study, however, we observed a modest increase in core temperature of 0.4–0.5°C across both groups after 3 h; an increase that does not represent a clinical sign of heat stress. However, the cumulative change in body heat storage in older adults both with and without T2D was equivalent to an increase in mean body temperature of ~1.2°C, exceeding WHO limits after 3 h. These observations further reinforce the notion that rectal temperature often underestimates heat stored within the body, and thus, whole-body thermal strain [28].

An important consideration of the current study is that both the older adults with and without T2D continued to store heat at the end of the exposure (Figure 1 and Table 2), indicating that body temperature would continue to rise with a more extended heat exposure. If left unchecked, a gradual accumulation of heat in the body could lead to dangerous elevations in body temperature paralleled by potentially greater elevations in cardiovascular strain. In comparison, the rate of heat storage at the end of the exposure in the young adults in our previous study approached zero [2], indicating that a more prolonged exposure (>3 h) would be associated with negligible further increases in body temperature and therefore possibly cardiovascular strain. Therefore, extended extreme heat exposures without preventative measures (e.g. use of air conditioners, fans, proper hydration, etc.) may present a greater health concern to older adults with and without T2D compared to their younger counterparts. Future studies are required to assess the impact of day-long heat exposures in whole-body heat exchange.

While our study provides valuable new insights into the effects of well-controlled T2D on the body's physiological capacity to dissipate heat during extreme passive heat exposure, some limitations must be considered including the specific environmental conditions and exposure duration, a relatively small sample size, a fairly short duration of diabetes, as well as our inability to comment on the effects of specific medications on whole-body heat dissipation and therefore body temperature regulation during extreme passive heat exposure.

In summary, we showed that unacclimatized, physically active, older adults with relatively-well controlled T2D displayed a similar physiological capacity to dissipate heat compared to their healthy counterparts during a brief, 3-h extreme passive heat exposure.

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## Disclosure statement

No conflict of interest, financial or otherwise, are declared by the author(s).

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