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Thermogenic and psychogenic sweating in humans: Identifying eccrine glandular recruitment patterns from glabrous and non-glabrous skin surfaces

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ABSTRACT

In this experiment, psychogenic (mental arithmetic), thermogenic (mean body temperature elevation of 0.6 °C) and combined thermo-psychogenic treatments were used to explore eccrine sweat-gland recruitment from glabrous (volar hand and forehead) and non-glabrous skin surfaces (chest). It was hypothesised that each treatment would activate the same glands, and that glandular activity would be intermittent. Nine individuals participated in a single trial with normothermic and mildly hyperthermic phases. When normothermic, a 10-min arithmetical challenge was administered, during which sudomotor activity was recorded. Following passive heating and thermal clamping, sweating responses were again evaluated (10 min). A second arithmetical challenge (10 min) was administered during clamped hyperthermia, with its sudorific impact recorded. The activity of individual sweat glands was recorded at 60-s intervals, using precisely positioned, and uniformly applied, starch-iodide papers. Those imprints were digitised and analysed. Peak activity typically occurred during the thermo-psychogenic treatment, revealing physiologically active densities of 128 (volar hand), 165 (forehead) and 77 glands.cm^{-2} (chest). Except for the hand (46%), glands uniquely activated by one treatment were consistently < 10% of the total glands identified. Glandular activations were most commonly of an intermittent nature, particularly during the thermogenic treatment. Accordingly, we accepted the hypothesis that psychogenic, thermogenic and thermo-psychogenic stimuli activate the same sweat glands in both the glabrous and non-glabrous regions. In addition, this investigation has provided detailed descriptions of the intermittent nature of sweat-gland activity, revealing that a consistent proportion of the physiologically active glands are recruited during these thermal and non-thermal stimuli.

1. Introduction

Human eccrine sweating is stimulated by thermoregulatory mechanisms (Taylor and Machado-Moreira, 2013) and an array of non-thermal (*e.g.*, psychogenic) influences (Mekjavic and Eiken, 2006; Kenny and Journey, 2010; Kondo et al., 2010). Both types of sudomotor response appear as whole-body phenomena (Machado-Moreira and Taylor, 2012a, 2012b; Taylor and Machado-Moreira, 2013), although the non-glabrous (hairy) skin is more thermally responsive, whilst the glabrous skin appears more sensitive to non-thermal stimuli (*e.g.*, the volar surfaces of the hands [palms] and feet [soles]). Those functional, but also regional anatomical differences have been recognised for over 300 years (Grew, 1684), with local sweat rates varying across body segments due to differences in glandular density and sweat-gland output (sensitivity: Kuno, 1956; Sato, 1977; Taylor and Machado-Moreira, 2013). Surprisingly, it remains unknown whether those diverse sudorific stimuli activate the same, or separate and unique, pools of sweat glands, and so the primary objective of this investigation was to explore those possibilities.

The central (hypothalamic) control of thermoregulation is now well established (Ott, 1884; Isenschmid and Schnitzler, 1914; Barbour, 1921; Nakamura, 2011), even though the precise locations of those neurones remain uncertain (Farrell et al., 2013, 2015). Similarly, the neurones responsible for activating psychogenic sweating require identification, and appear to reside within various central locations (Craig et al., 2000; Nagai et al., 2004; Farrell et al., 2013, 2015),

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including supra-medullary sites (Taylor et al., 2008; Kenny and Journey, 2010; Kondo et al., 2010). Nevertheless, common neural pathways descending from the brain stem, have now been identified for both forms of sweating (Farrell et al., 2013), making it possible that those efferent signals also share common downstream pathways. Moreover, the terminal sympathetic neurones that drive the ubiquitous distributions of thermogenic and psychogenic sweating are cholinergic (Machado-Moreira et al., 2012). Accordingly, it is not such a big leap to consider the possibility that those neurones might also activate the same sweat glands, although that thesis challenges the long-held dogma that neurones with different neurotransmitters recruit unique sets of sweat glands during both thermogenic and psychogenic stimulation (Darrow, 1937; Chalmers and Keele, 1952; Kuno, 1956; Ogawa, 1975; Iwase et al., 1997).

In a previous communication (Machado-Moreira and Taylor, 2017), it was reported that sweat glands from the volar hand, that were not active during a thermal stress, could be activated when a non-thermal (psychogenic or cognitive) stimulus was applied during mild hyperthermia. Unfortunately, in that experiment, we were unable to differentiate between glands that were uniquely activated by the thermal treatment, and those recruited only during the psychogenic stimulation, since we lacked the ability to track the activation and deactivation of individual sweat glands. Furthermore, glands from only one body segment (hand) were investigated. Therefore, for this experiment, a colourimetric method was refined to provide that capability (serial starch-iodide imprints), and the time-dependent nature of psychogenic, thermogenic and combined thermal and psychogenic (thermo-psychogenic) sweating was investigated from three spinal segments, which included both glabrous and non-glabrous skin surfaces.

Using that method, two theories of glandular recruitment were investigated. Firstly, it was hypothesised that the same eccrine sweat glands would be recruited, regardless of whether the stimulation was of a thermal or cognitive nature. Secondly, the time dependency of glandular recruitment, including the duration of glandular activation and the re-activation of those sweat glands, was explored, and it was postulated that glandular activity would be intermittent, and not a continuous phenomenon. The resulting information will extend our understanding of sweat-gland recruitment patterns, and provide greater precision with regard to describing the neural control of human eccrine sweating.

2. Methods

2.1. Participants

Nine individuals participated in this experiment (four females and five males; age 29.0 y [standard deviation (SD) 4.4], body mass 75.2 kg [SD 8.0]). All procedures were approved by a Human Research Ethics Committee (University of Wollongong) in accordance with the National Health and Medical Research Council (Australia). Subjects provided written, informed consent before commencing. They were also screened to exclude those taking medication, with a history of shingles, heat illness or sweating disorders, or those with an allergy to iodine.

2.2. Procedures

2.2.1. Procedural overview

Supine (resting) subjects participated in a single trial in temperate conditions, lasting approximately 2.0–2.5 h, with every trial including two distinct thermal phases (Fig. 1; normothermia and mild hyperthermia) and three experimental treatment stages. In the first phase, which followed confirmation of normothermia with absolutely minimal sweat-gland activity, participants were exposed to a single, non-thermal stimulus (psychogenic treatment), applied in the form of a cognitive challenge (mental arithmetic; 10 min). The objective of this first treatment was to stimulate, and then to identify, eccrine sweat glands from three body segments (forehead, chest and palmar [volar] hand) that were activated during that non-thermal stimulus. The second phase followed another confirmation of normothermia with minimal sweat secretion. Subjects were passively heated to induce a clamped, mean body temperature elevation of 0.5–1.0 °C. Following the attainment of a thermal steady state, thermally activated sweat glands from those same body segments were identified (10 min; treatment two [thermogenic sweating]). That was followed by the third treatment, in which an identical cognitive challenge (10 min) was overlaid onto a clamped, mildly hyperthermic state, thereby permitting the identification of sweat glands activated during the combined thermo-psychogenic treatment. Indeed, we have previously shown that, without that thermal priming of the sweat glands, a more complete expression of the sudomotor impact of that psychogenic stimulus would not be revealed (Machado-Moreira and Taylor, 2012a).

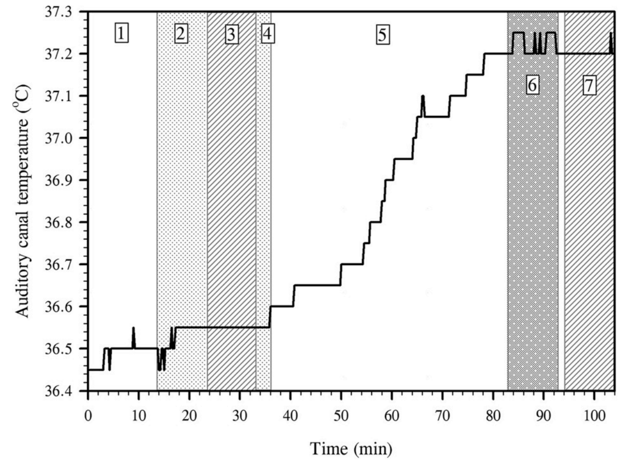

Fig. 1. An overview of the separate thermal phases of this experiment, overlaid onto the auditory canal temperatures of one participant (S5-M). Trials commenced with the stabilisation (10–15 min [step 1]) and confirmation of normothermia without sweat secretion (10 min [step 2]), after which subjects were exposed to the first non-thermal (psychogenic) treatment for 10 min (mental arithmetic [step 3]), followed by a normothermic reconfirmation (3 min [step 4]). Sweat-gland activity was investigated during that psychogenic treatment using serial, iodinated-paper imprints. The second phase commenced with passive heating (step 5). Once the desired elevation in auditory canal temperature was achieved (0.3–0.5 °C), and sweating clearly established, thermal clamping was initiated. When adequately clamped, sweat-gland activation was again investigated (10 min [step 6]). Finally, a second psychogenic treatment was applied when subjects were in that clamped, mildly hyperthermic state (thermo-psychogenic; 10 min [step 7]), with sweat-gland activity assessed for a third time.

2.2.2. Experimental standardisation

Subjects were instructed to avoid strenuous exercise within the 24 h preceding testing, alcohol on the previous evening and caffeinated drinks for 2 h prior to presentation. They were also asked to eat high-carbohydrate and low-fat meals prior to testing, and to arrive in a well-hydrated state. To that end, the consumption of 15 mL kg^{-1} of additional fluid before retiring, with a further 500 mL on the morning of testing, was prescribed. Hydration state was verified on presentation (urine specific gravity: Clinical Refractometer, Model 140, Shibuya Optical, Tokyo, Japan), with additional water (2.5 mL kg^{-1}) provided to individuals having a urine specific gravity > 1.029 (N = 3). To minimise non-thermal artefacts, subjects were blindfolded throughout testing, except during the two cognitive challenges.

2.2.3. Experimental routine

On arrival, participants provided a urine sample and routine physical characteristics were measured (air-conditioned laboratory: 20–21 °C), after which they dressed in a swimming costume and donned a water-perfusion garment (Cool Tubesuit, Med-Eng, Ottawa, Canada). Subjects then entered a thermally equilibrated, climate-controlled chamber (25–26 °C, 2.12 kPa water-vapour pressure [65% relative humidity]), adopted a supine position on a padded bed, with skin-site preparation (cleaning and shaving) and instrumentation commencing. Every trial followed the same time line (Fig. 1), commencing with a preparatory period (~30 min), followed by baseline stabilisation and verification periods. During that time, water from a bath (regulated at 34 °C) was circulated through the perfusion garment, and the feet were immersed in a second water bath (33 °C: both 38-L, Grant Instruments Ltd., Shepreth, U.K.). Those procedures ensured that participants, who were now blindfolded, were well-rested, euhydrated, thermally neutral (normothermic) and had no visible signs of eccrine sweating.

The first psychogenic treatment was applied immediately after the normothermic verification (mental arithmetic; 10 min), with deep-body temperature sustained within a 0.1 °C range. The blindfold was removed, and subjects were presented with a series of cards (after Machado-Moreira and Taylor, 2017), each containing a unique and moderately difficult arithmetic problem (equations: *e.g.*, 3792/5, 738/4). Cards were held above the face, with constant verbal encouragement provided to increase speed, accuracy and the number of problems completed. Cards were presented for 20 s only, and immediately replaced following either successful completion or reaching that time limit. There was no rest between cards. The aim was to maximise and standardise anxiety and psychological stress across individuals, with sudomotor data collection occurring throughout that treatment. Cards used in that challenge were set aside so that unique problems could be presented during the second cognitive treatment.

On completion of the first experimental treatment, the blindfold was replaced, followed by a re-verification of the normothermic state (3 min). Immediately thereafter, passive heating commenced (Fig. 1), with both water baths being heated: 42 °C for the feet and 48 °C for the perfusion suit. The aim was to produce a gradual elevation in heat storage. That was monitored from the auditory canal, with a temperature of 0.3–0.5 °C above the normothermic baseline being sufficient to achieve the target mean body temperature rise. On average, mean body temperature increased by 0.6 °C (SD 0.3). It is important to note that this was a rather mild thermal stimulus. Since heavy sweating would significantly and adversely affect the quality of the starch-iodide imprints, possibly compromising the precision of gland counting, it was deemed essential to avoid a greater thermal loading. Once the target elevation was achieved, and sweat-gland activity was visually apparent, that thermal state was clamped by manipulating both water-bath temperatures. That technique is well established within the current laboratory (Cotter et al., 1995; Burdon et al., 2017; Machado-Moreira et al., 2012), and was further developed for this investigation, with the stability of the auditory canal temperature again sustained within 0.1 °C. Data collection for thermogenic sweating followed (10 min). After a brief rest, the blindfold was removed and the second psychogenic treatment was applied using novel arithmetical problems (Fig. 1). After that thermo-psychogenic stage, testing was terminated and subjects recovered in the air-conditioned laboratory.

2.3. Physiological measurements

2.3.1. Sudomotor activity

Since this experiment required serial measurements of the activation, cessation and reactivation of individual eccrine sweat glands, then previous methods used to quantify discharged sweating during thermal and psychogenic treatments were inappropriate (capacitance hygrometry [Machado-Moreira and Taylor, 2012a, 2012b] and colourimetry [Machado-Moreira and Taylor, 2017]). Accordingly, a different approach was developed, although the technique chosen (colourimetry: starch-iodide paper) was not novel (Minor, 1927; Randall, 1946a, 1946b; Herrmann et al., 1952; Sato et al., 1988), but had extensive development prior to the advent of automated systems. A recent refinement of that technique (Gagnon et al., 2012) permitted the development of a method involving the serial collection of iodinated paper imprints, and that procedure was chosen for this investigation.

For the current experiment, 11 such imprints were recorded from each of three skin surfaces during the three data-collection periods: normothermic psychogenic, thermogenic and combined thermo-psychogenic treatments (Fig. 1). Those sweat-gland imprints were formed by the reaction of sweat with iodine impregnated into cotton paper, when that paper was held in contact with the skin. The process yielded short-duration impressions of the activated sweat glands, with skin imprints collected at 60-s intervals from each site, commencing at time zero. In addition, imprints were taken during the baseline period (at 6, 9 and 12 min; Fig. 1) to confirm that sweat-gland activity was either absent, or minimal.

Paper was prepared prior to each trial, using 100% cotton paper ($120\ g\ m^{-2}$; JD18C Southworth, Neenah Paper Inc., Neenah, U.S.A.) cut into rectangles (3 × 6 cm). Those rectangles were individually suspended within sealed glass jars containing ~0.3 g of solid iodine (48 h). Direct contact with the iodine was prevented. When saturated with iodine, the paper turned light brown, and the pieces were removed and labelled (coded) to permit subject, treatment type, sample site and time-point identification. They were then stored in air-tight containers. All paper handling, including the experimental and analysis periods, was performed without contact between the paper and skin, except during each measurement, which only involved contact with the skin of each subject.

Three skin regions were chosen for investigation: the forehead (glabrous or volar skin; first-second thoracic segments [T1-2]), due to its strong thermal response (Taylor and Machado-Moreira, 2013); the chest (fourth-fifth thoracic segments [T4-5]), a non-glabrous site with intermediate thermal responsiveness (Taylor and Machado-Moreira, 2013); and the volar surface of the hand (glabrous skin; third-sixth thoracic segments [T3-6]), because of its high reactivity to non-thermal stimuli (Machado-Moreira and Taylor, 2012a, 2012b). At each site, bespoke right-angle frames (perspex; $3\ cm^2$) were glued to the flattest local skin surface (Collodion U.S.P., Mavidon Medical 14 Products, FL, U.S.A.). For the forehead, the lower edge of the frame was centred between, and directly above, the eyebrows. The chest frame was positioned with its lower corner 2 cm medial to, and 2 cm above, the papilla of the left breast. The palmar frame was aligned with the base of the left ring finger and the fifth metacarpal. Those frames were positioned for ease of access by the experimenters. For each trial, a separate experimenter took responsibility for data collection at each skin site, whilst another administered the arithmetic problems.

For every 10-min treatment, a separate air-tight container of coded, iodinated paper (rectangles) was used for each skin site. Every surface imprint was collected using an identical procedure. Wearing barrier gloves, the experimenters positioned the paper rectangles above the corresponding frame, holding a padded perspex stamp (press) in the other hand. The timing was controlled by one experimenter, with imprints collected in an identical manner across sites. This first step involved the precise alignment of the iodinated paper with both the corner and one side of each frame. That alignment was essential, with rehearsal preceding testing, since successive imprints were to be used to identify gland locations and to track the activity of individual sweat glands. When told to start, the stamp was placed onto the paper, and light, but even, pressure was smoothly applied to initiate gentle and even skin contact. Time was counted aloud, with the duration for each stamp varying due to local differences in sweat-gland output: forehead 2 s, chest 3 s and hand 8 s. Those time limits prevented the merging of adjacent sweat spots that can occur when sweat droplets become too large. As each time elapsed, the stamp and iodinated paper were simultaneously, and vertically, removed to avoid smudging. The quality of each imprint was immediately examined, with a second (timed) imprint taken if needed. Those imprints were immediately placed into airtight containers. Between successive imprints, a second stamp, equipped with absorbent tissue, was used to remove excessive sweat (if required) at least 10 s before the next imprint was taken.

2.3.2. Body temperatures and heart rate

Deep-body temperature was estimated from an external auditory canal using an insulated, ear-moulded plug and thermistor (Edale instruments Ltd., Cambridge, U.K.). That method minimises artefactual influences associated with changes in facial skin temperature, which are further reduced in temperate conditions (Taylor et al., 2014). Thus, valid, reliable and responsive measures of deep-body temperature were obtained (Keatinge and Sloan, 1975; Todd et al., 2014), with that index known to track changes in the thermal energy content of the central blood volume (Taylor et al., 2014). In addition, eight skin temperatures were simultaneously measured (Type EU, Yellow Springs Instruments Co. Ltd., Yellow Springs, OH, U.S.A.): forehead, right scapula, right chest, right arm, left dorsal forearm, left dorsal hand, right anterior thigh and left posterior calf. Using area-weighted coefficients, those data were used to estimate mean skin temperature (Hardy and DuBois, 1938), and then, in combination with auditory canal temperature (80% weighting), to obtain mean body temperature (Vallerand et al., 1992). Temperatures were sampled at 15-s intervals (Grant Instruments Ltd., 1206 Series Squirrel, U.K.), with thermistors calibrated against a certified thermometer (Dobros total immersion, Dobbie Instruments, Sydney, Australia). Heart rate was measured throughout from ventricular depolarisation, with data recorded at 60-s intervals (Vantage NV, Polar Electro Sport Tester, Kempele, Finland).

2.4. Data analysis

Immediately following each trial, the sweat-gland imprints were digitised using a stereoscopic microscope (Nikon SMZ 800, Camera Head, Nikon Corp., Tokyo, Japan; Nikon DS Fi1 2/3-inch square pixel, 5.24 megapixel interline CCD). To standardise that process, a fourth (identical) right-angle frame was glued to the stage plate, permitting individual imprints to be inserted and digitised from identical positions. Using overlayed grids, the central square (1 cm^2) of each imprint was located and used for analysis, with nine sets of 11 digital images created for every participant; three skin sites with three treatments per site. Those images were imported into Adobe Photoshop (Adobe Photoshop CC), their backgrounds were made transparent and they were superimposed. Individual sweat glands appeared as separate dark marks, and this facilitated fine tuning the alignment of successively overlaid images.

Once aligned, the total number of activated sweat glands was identified for each site, and for each treatment (stimulation) within that site (after Machado-Moreira and Taylor, 2017). It is important to note that, as time progressed, more glands were activated, so the total number of active sweat glands almost invariably exceeded those counted within any one image. Therefore, the first part of these analyses was to identify all of the activated sweat glands, and their locations, within each of the nine sets of images for each participant. The highest counts were found during the combined thermo-psychogenic treatment. Those glands were assigned unique numbers (software markers) that were then attached to the gland marks within each set of images (Fig. 2), with the same number used when that gland was

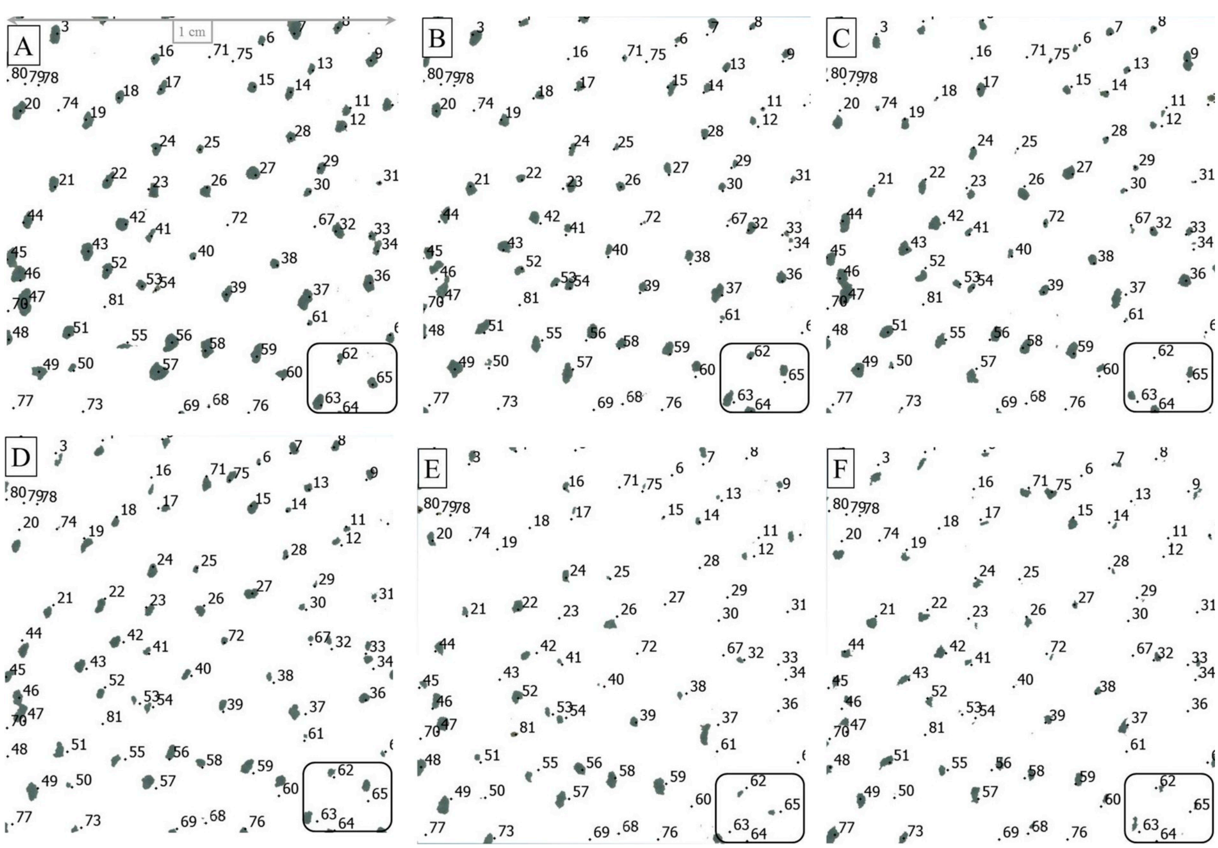

Fig. 2. Digitised eccrine sweat-gland imprints (1 cm^2) collected from a mildly hyperthermic participant at 60-s intervals during a combined (clamped) thermal and psychogenic treatment (10 min). Those images are from the chest of that individual (S6-F): average auditory canal temperature 37.1 °C (normothermia: 36.9 °C), skin temperature 36.4 °C (normothermia: 33.6 °C) and heart rate 86 b min^{-1} (normothermia: 65 b min^{-1}). During that treatment, 81 glands.cm^{-2} were active. Eleven imprints were obtained by gently pressing iodinated paper (3 s) onto a precisely defined skin target. Six time points are shown (A-F): 0, 2, 4, 6, 8 and 10 min. Glands were counted and labelled. Activated glands appear as irregular dark marks, and inactive glands are identified with small diamonds. Glands 62–65 are highlighted for illustrative purposes, with an average distance between glands 62 and 65 of 1.1 mm. Table 2 shows the sweat-gland activity patterns for just ten of those sweat glands during the three experimental treatments.

identified in either preceding or proceeding images.

Once the maximal density of site-specific sweat glands had been determined for each person, the following activity definitions were used to classify glandular activity within each treatment: (1) a dark mark (iodine reaction) appearing at a designated location - that gland was either initially activated, or it was reactivated following a period of inactivity (code 1); (2) a dark mark in the same location on a successive image(s) - that gland had remained continuously active during those time intervals (codes 2–11); and (3) no dark mark present in the designated location on a successive image(s) - the gland was no longer active (code 0). From those codes, it was possible to determine which glands were inactive (relative to the total number identified at each site), when individual glands were activated, as well as the frequency and duration of those activations, and whether or not those glands were continuously active or reactivated during the three treatments.

No attempt was made to differentiate between the sudomotor responses of the two genders, which may differ, depending on hormonal cycling, between each of the two experimental treatments. Instead, this was a single trial involving repeated measures, with each individual acting as her/his own control. Sweat-gland data were analysed using two-way Analysis of Variance with *post hoc* analyses performed when statistical differences were identified at the 5% level; Tukeys *HSD* statistic for between-treatment effects, and Dunnet's Multiple Comparisons for time effects. Data are reported as means with standard errors of those means (±), or standard deviations (SD) when describing data distributions.

3. Results

3.1. Verification of the normothermic and mildly hyperthermic states

Prior to testing, the average urine specific gravity of all participants was 1.016 (SD 0.01). Auditory canal and mean body temperatures did not differ significantly during the normothermic phase of this experiment ($P > 0.05$; Table 1), and no sudomotor activity was recorded on the iodine-impregnated paper at this stage. Mean skin temperatures were 0.1 °C higher during the psychogenic treatment ($P < 0.05$; Table 1), but that difference was physiologically insignificant. Therefore, during that phase, it was assumed that eccrine sweat-gland activation was evoked solely by the psychogenic treatment, with that stress also being reflected within the elevated heart rates ($P < 0.05$; Table 1). During passive heating, the average time to reach the target auditory canal temperature was 46.8 min (SD 15.7). That clamped, mildly hyperthermic state (thermogenic treatment) significantly elevated tissue temperatures and heart rate ($P < 0.05$; Table 1). When the psychogenic treatment was re-applied during clamped, but mild hyperthermia (thermo-psychogenic treatment), there was another chronotropic effect ($P < 0.05$), but none of the tissue temperatures was altered ($P > 0.05$; Table 1). Therefore, any additional sweat-gland activation during that thermo-psychogenic treatment can be attributed to overlaying the cognitive challenge onto the existing thermal feedback.

3.2. Sweat-gland recruitment

For each of the nine sets of digitised sweat-gland imprints, the activity patterns and codes defined above were applied, with Table 2 showing the codes for 10 eccrine sweat glands identified from the chest of the person shown in Fig. 2 (S6-F), but now across each of the three treatments. The maximal density of activated sweat glands for each site was determined for each individual, with maximal gland counts occurring during the thermo-psychogenic treatment, although some glands that were inactive during that treatment were activated during one of the other two treatments. Those maxima are presented in Table 3 for all participants, revealing site-specific glandular density maxima that ranged from 87 to 236 activated sweat glands.cm^{-2} at the forehead, 54–103 glands.cm^{-2} for the chest and 41–291 glands.cm^{-2} at the volar surface of the hand. Those inter-individual differences reflect variations in the sudomotor response to each treatment, with those range limits revealing low and high responders (respectively).

Within each site, those maximal glandular densities were not realised during every treatment. For instance, on average, the lowest, site-specific proportional densities occurred during the psychogenic treatment (all < 20% of the maximal glandular densities; Table 4), which reflects the eccrine responsiveness to that stimulation. However, since the intensities of the thermal and non-thermal treatments were not matched, then the lower psychogenic responsiveness may have been more related to the intensity of that stress *per se*, rather than to its non-thermal nature.

The highest proportional gland counts were observed when the cognitive challenge was combined with mild hyperthermia (thermo-psychogenic treatment; all > 85% of site-specific maxima; Table 4), refuting the suggestion that cognitive stress suppresses thermal sweating (Sugenoya et al., 1982). In those instances, it was assumed that the thermal sensitivity of those glands largely determined that outcome. That interpretation is supported by the proportional counts that were common to each of the treatment pairs (Table 4), with the greatest number of common glands being activated during both the thermogenic and thermo-psychogenic treatments. Thus, it is quite likely that the psychogenic and thermogenic treatments were not physiologically equivalent in their sudorific impact. Nevertheless, some 10–15% of the glands identified across all three treatments remained inactive during the most stressful (thermo-psychogenic) treatment. Instead, those glands were activated and identified in one of the other treatments. Furthermore, on average, < 15% of the glands identified during the thermo-psychogenic treatment were recruited during all three treatments (Table 4).

Since the primary objective was to determine whether or not the same eccrine sweat glands might be recruited during both the thermal and cognitive stimulations, then glands that were uniquely activated within each of the three treatments, but remained silent within the other two treatments, were determined (Table 4). With the exception of the volar hand during the combined thermo-psychogenic treatment, the number of glands that was uniquely activated by one of the three treatments was consistently < 10%. Given the probable error rates for such manual determinations, that outcome is interpreted to signify that

Table 1

Heart rate and auditory canal, mean skin and mean body temperatures during two thermal states (normothermia and clamped mild hyperthermia [thermogenic treatment]), and then during cognitive challenges (mental arithmetic) applied within each state (N = 9): normothermic psychogenic and thermo-psychogenic treatment. Data are means within each stage, with standard errors of the means in parenthesis. Symbols reveal statistically significant differences ($P < 0.05$): † = between the normothermic baseline and the psychogenic treatment; * = between the normothermic baseline and either the thermogenic or thermo-psychogenic treatments; ‡ = between the psychogenic and thermogenic treatments; and § = between the thermogenic and the thermo-psychogenic treatments.

Condition	Auditory canal temperature (°C)	Mean skin temperature (°C)	Mean body temperature (°C)	Heart rate (b.min^{-1})
Normothermic baseline	36.7 (0.1)	34.0 (0.2)	36.2 (0.1)	70 (3)
Normothermic psychogenic treatment	36.7 (0.1)	34.1 (0.2)†	36.2 (0.1)	77 (3)†
Thermogenic treatment	37.1 (0.1)*,‡	35.6 (0.3)*,‡	36.8 (0.1)*,‡	78 (3)*
Thermo-psychogenic treatment	37.1 (0.1)*	35.6 (0.3)*	36.8 (0.1)*	86 (3)*,§

Table 2
Individual activity patterns for physiologically active eccrine sweat glands of the chest of one subject (S6-F: overall glandular density 82 glands.cm^{-2}). Three experimental treatments (10 min) are shown for that site: a psychogenic treatment (normothermia: 52 glands.cm^{-2} activated in this treatment); a thermogenic treatment (clamped, mild hyperthermia: 78 glands.cm^{-2} activated); and a combined (clamped) thermal and psychogenic treatment (81 glands.cm^{-2} activated). Activity codes are defined within the text, with digitised images presented in Fig. 2 (chest: thermo-psychogenic treatment). The activity of the same ten glands (leftmost column, moving vertically) is illustrated for each treatment, with activity codes provided at 1-min intervals (moving left to right), starting at time zero. Glands were first identified (numbered), and their locations determined, during the thermo-psychogenic (rightmost) treatment, with glandular activity for those same glands tracked during each of the other treatments. Inactive glands are coded 'zero', initial activations have code 'one' and glands remaining active across successive 1-min periods are shown with incrementing numbers greater than one (moving left to right).

Minute	0	1	2	3	4	5	6	7	8	9	10	0	1	2	3	4	5	6	7	8	9	10	0	1	2	3	4	5	6	7	8	9	10
Gland	Psychogenic treatment											Thermogenic treatment											Thermo-psychogenic treatment										
10	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0	0	0	1	0	0	1	2	0	1	2	3	4	0	1	2	3
19	0	0	0	1	0	0	1	0	1	0	0	1	2	3	4	5	6	7	0	0	1	2	1	2	3	4	5	6	7	0	1	2	3
28	0	0	0	1	0	0	0	0	0	0	0	1	2	3	0	1	2	3	4	5	6	7	1	2	3	4	5	6	7	0	0	1	2
56	0	0	0	1	2	0	0	0	0	0	0	1	2	3	4	5	0	1	0	1	2	3	1	2	3	4	5	6	7	0	1	2	3
61	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	1	0	0	1	2	3	4	5	6	7	8	9	10	0
62	0	0	0	0	1	0	0	0	0	0	0	0	0	1	2	3	4	5	6	7	8	9	1	2	3	4	0	0	1	0	1	2	3
63	0	0	0	1	2	0	1	0	0	0	0	1	2	3	4	5	0	1	0	0	0	1	1	2	3	4	5	6	7	0	1	0	1
64	0	0	0	0	1	0	1	0	0	0	0	1	2	3	4	5	6	7	8	9	10	11	1	2	3	4	5	6	7	0	1	2	3
65	0	0	0	0	1	0	1	0	0	0	0	1	2	3	4	5	6	0	1	2	0	1	1	2	3	4	5	6	7	0	1	2	3
75	0	0	0	0	0	0	0	0	0	0	0	1	2	3	4	5	0	0	0	0	0	0	0	0	0	0	1	2	3	0	1	2	3

Table 3
Site-specific maximal glandular densities for eccrine sweat glands activated within three skin regions. Those values generally occurred during the combined thermo-psychogenic treatment, with additional glands being identified in each of the other two treatments (cognitive challenge and mild hyperthermia). Data are shown for each participant (gender codes indicated), with overall sample means and standard deviations (glands.cm^{-2}) derived for each site.

Subject	Forehead	Chest	Volar hand
S1-M	213	60	101
S2-M	236	80	75
S3-M	135	71	229
S4-M	234	75	44
S5-M	113	54	41
S6-F	194	82	146
S7-F	166	103	144
S8-F	103	71	80
S9-F	87	95	291
Mean	165	77	128

Table 4
An overview of sweat-gland activation for three skin regions (forehead, chest and volar hand) during psychogenic, clamped thermogenic and combined thermo-psychogenic treatments ($N = 9$). Maximal glandular densities are from Table 3 (means with standard deviations in parenthesis), as determined across the three treatments. Some glands were recruited in more than one treatment (common glands), others were uniquely activated within only one treatment. Proportional gland counts (%) are expressed with respect to site-specific maxima, derived on an individual basis and averaged (sample sizes remained constant throughout).

	Forehead	Chest	Hand
Maximal densities of activated glands (glands.cm^{-2})	165 (58)	77 (16)	128 (85)
Psychogenic treatment	14.9%	12.4%	19.9%
Thermogenic treatment	92.7%	93.7%	39.9%
Thermo-psychogenic treatment	92.5%	96.5%	86.4%
Glands unique to the psychogenic treatment	0.3%	0.0%	5.6%
Glands unique to the thermogenic treatment	6.9%	3.1%	7.4%
Glands unique to the thermo-psychogenic treatment	6.8%	6.1%	46.2%
Common glands: all three treatments	14.3%	12%	5.5%
Common glands: thermogenic + thermo-psychogenic	71.3%	78.3%	26.5%
Common glands: psychogenic + thermo-psychogenic	0.1%	0.1%	8.3%
Common glands: psychogenic + thermogenic	0.2%	0.3%	0.6%

these thermal and non-thermal stimuli both activated the same eccrine sweat glands. At the volar hand, ~45% of the identified sweat glands were uniquely activated during the thermo-psychogenic treatment (Table 4), and it appeared that such a combined stimulation was required for a more complete sudomotor expression of the resulting psychophysiological strain.

3.3. Time-dependent glandular recruitment

With regard to the time-dependent nature of sweat-gland activation (Table 5), four observations are highlighted. Firstly, regardless of the

Table 5
The time-dependent characteristics of eccrine glandular activations from the forehead, chest and volar hand during separate psychogenic, clamped thermogenic and thermo-psychogenic treatments ($N = 9$). Total gland counts, and their locations, determined across the three treatments (Table 3). Data are for whole glands (integers) and times to the nearest 30 s, presented as means with standards errors of the means (parenthesis) derived across treatment durations (10 min). *Definitions:* 'active' and 'inactive' states refer to any time interval during a treatment; 'continuously active' means active for the entire treatment (10 min); and 'reactivated' shows how many individual glands that were activated more than once within a treatment.

Forehead	Psychogenic	Thermogenic	Thermo-psychogenic
Glands that were active	30 (18)	154 (19)	152 (18)
Glands that were inactive	135 (19)	11 (4)	12 (3)
Glands continuously active	0	19 (9)	17 (6)
Duration of each activation (min)	1.5 (0.2)	5.5 (0.6)	6.5 (0.5)
Glands that were reactivated	10 (7)	113 (18)	89 (19)
Chest	Psychogenic	Thermogenic	Thermo-psychogenic
Glands that were active	10 (6)	72 (6)	74 (5)
Glands that were inactive	66 (7)	5 (2)	3 (1)
Glands continuously active	0	13 (4)	21 (7)
Duration of each activation (min)	1.5 (0.2)	6.5 (0.4)	7.5 (0.4)
Glands that were reactivated	3 (3)	46 (5)	33 (6)
Volar hand	Psychogenic	Thermogenic	Thermo-psychogenic
Glands that were active	25 (11)	54 (21)	111 (27)
Glands that were inactive	103 (27)	73 (16)	17 (7)
Glands continuously active	0	0	< 1
Duration of each activation (min)	1.0 (0.1)	1.5 (0.1)	3.0 (0.4)
Glands that were reactivated	5 (2)	23 (11)	57 (15)

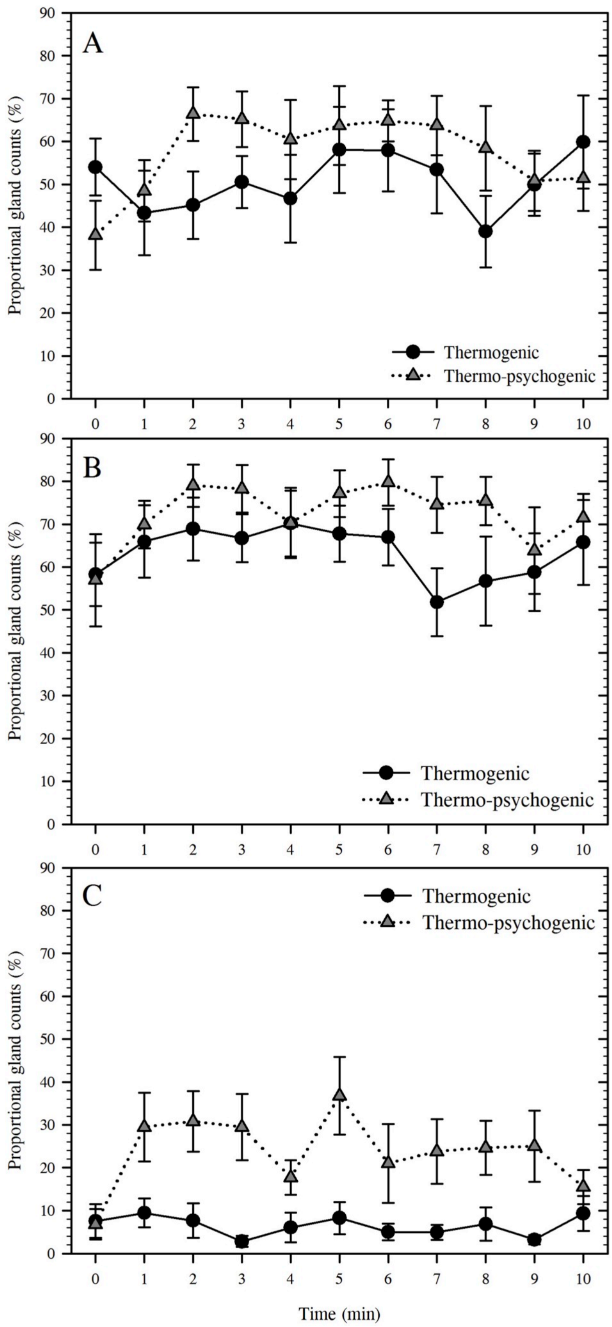

Fig. 3. Time-dependant sweat-gland recruitment patterns for the forehead (A), chest (B) and the volar hand (C) during the clamped thermogenic and thermo-psychogenic treatments ($N = 9$). Data are proportional gland counts (%), relative to the site-specific maximal number of activated glands identified for each individual (Table 3), and are presented as means and standard errors of those means.

site investigated, more glands remained inactive during the psychogenic stimulation than during either of the other two treatments. During the thermogenic and thermo-psychogenic treatments, that trend was reversed, with the least number of glands now remaining inactive, although reversal was incomplete at the volar hand. Secondly, no sweat glands were (continuously) active for the entire psychogenic treatment (Table 5). Regardless of the treatment, continuous sudomotor activity was rarely evident at the volar hand, but occurred more frequently at the forehead and chest. Thirdly, the duration of those glandular activations revealed both site- and treatment-specific tendencies (Table 5); universally shorter for the psychogenic treatment, as well as for the volar hand, regardless of the treatment. Fourthly, the psychogenic treatment was associated with the reactivation of fewer glands than the other two treatments (Table 5). Less than 35% of the site- and treatment-specific sweat glands were reactivated during the psychogenic treatment, with those proportions doubling during the thermogenic stimulation (Table 5).

To better illustrate the time-dependency of sweat-gland recruitment, and its intermittent nature, Fig. 3 displays proportional glandular activities for the thermogenic and thermo-psychogenic treatments for each skin site. If the first 2 min of each treatment are disregarded for the latter treatment, due to an assumed latency between the commencement of non-thermal stimulation and its physiological impact, then it is apparent that, as time progressed during those two treatments, a consistent proportion of sweat glands was active within each treatment. From Table 5, it is apparent that 40–85% of those glands either remained continuously active or were reactivated within those two treatments, as described by Randall (1946b). Therefore, since the proportional activations remained stable, then it can be assumed that some previously reactivated, and other silent, glands replaced those that ceased secretion during any sampling period. Significant differences (over time) were not evident between the forehead and chest for either of those treatments ($P > 0.05$). However, a significant, time-related difference was found between the thermogenic and thermo-psychogenic treatments when the forehead and chest were compared with the volar hand ($P < 0.05$).

4. Discussion

This experiment has, perhaps for the first time, revealed that efferent signals of either thermal or psychogenic origin will, for the most part, activate the same eccrine sweat glands, and not a unique subset of glands. That recruitment characteristic was evident at the forehead and chest regardless of the treatment applied, and at the volar hand during the psychogenic and thermogenic treatments. However, ~45% of the glands activated at the hand appeared to be uniquely related to the combined thermo-psychogenic treatment. Secondly, the intermittent (on-off) nature of sweat-gland activation was clearly demonstrated. For thermogenic stimuli, that oscillating activation pattern is well known, but it seems that the nature of those oscillations (glandular reactivation) might differ between the psychogenic and thermogenic treatments. These largely descriptive outcomes are important, not just for their own sake, but because they help us to better understand the neural mechanisms that modulate human eccrine sweat glands during thermal and non-thermal stress exposures.

It is important to first note that only the combined thermo-psychogenic treatment was likely to approach maximal activation of the full complement of eccrine sweat glands within any skin region. However, not even that treatment was aimed at achieving maximal glandular activation, which can approach 90–95% of the glands present within any skin region (Thompson, 1954; Willis et al., 1973), although that requires more extreme thermal loads (Randall, 1946a). Moreover, the intensities of the cognitive and thermal stimuli were not matched, increasing the inter-individual variability of the resulting sudorific impact due to variations in responsiveness to those treatments. Matching those intensities in the future would be helpful, and may perhaps clarify some aspects of this experiment. It must also be remembered that colourimetric indices only identify discharged sweat. Thus, the absence of sweat at the skin surface does not mean that the glands were not stimulated. Instead, some activated glands may not have produced primary (precursor) sweat at a rate sufficient to exceed ductal reabsorption, thereby preventing its discharge onto the skin. Nonetheless, the physiologically active glandular densities identified in the current study (165 [forehead], 128 [volar hand] and 77 glands.cm^{-2} [chest]) are, with the exception of the volar hand, comparable to those found within the literature (Taylor and Machado-Moreira, 2013; 186 [SD 81], 518 [SD 187] and 94 [SD 30] glands.cm^{-2}

[respectively]).

In addition to confirming that both thermal and non-thermal stimuli can elicit sweat from the glabrous and non-glabrous skin surfaces (Machado-Moreira and Taylor, 2012a, 2012b), the unique contribution from this research has been the demonstration that, at the forehead and chest, < 10% of the identified sweat glands were uniquely activated by any one of the three treatments. This was most reliably demonstrated when the thermogenic and thermo-psychogenic treatments were compared (Table 4), since the psychogenic stimulus was not as effective at producing discharged sweat within all participants. When considered in combination with evidence for common descending, sudomotor paths from the brain stem (Farrell et al., 2013), along with the demonstration that thermal and non-thermal stimuli both result in the cholinergic activation of the eccrine sweat glands (Machado-Moreira et al., 2012), the lack of evidence for the recruitment of a unique subset of glands supports the hypothesis that those glands are linked to their central controllers by common efferent pathways (Machado-Moreira and Taylor, 2012b). Thus, whilst those signals may arise from different central controllers, they may travel downstream along shared neural networks. That hypothesis requires neurological verification.

An apparent anomaly concerning that interpretation is the observation that ~45% of the glands activated at the volar hand (a glabrous site) were only turned on during the thermo-psychogenic treatment. It is possible the psychogenic and thermogenic treatments were either insufficiently effective, or that sweat-gland priming was required for their sudorific impact to be expressed (Machado-Moreira and Taylor, 2012a). Accordingly, one must not overlook the possibility that those apparently uniquely recruited glands were actually stimulated during the other two treatments, but their activation went undetected, due to local differences in precursor sweat production and reabsorption rates. Since, even during combined exercise and mild hyperthermia, the sweat-gland outputs from the volar surfaces of the hands and feet (1.9 and 0.6 μg.gland^{-1}.min^{-1}, respectively) represent < 25% of the flows observed from the forehead and chest (9.0 and 9.6 μg.gland^{-1}.min^{-1}, respectively; Taylor and Machado-Moreira, 2013), then that explanation is plausible.

Whilst the authors support those interpretations, it is also possible that sudomotor control of the volar hands (and feet) differs from that of the rest of the body. After all, appropriately hydrated palmar skin increases tactile sensitivity, contact friction and grip (Edelberg, 1961; Adelman et al., 1975), and since humans evolved from arboreal species that relied upon hand grip for survival (Folk and Semken, 1991; Best and Kamilar, 2018), then an evolutionary case may exist for variations in the central control of the volar surfaces. Regardless of the underlying mechanism, it is now clear that investigators need to include both glabrous and non-glabrous sites when evaluating sudomotor responses to thermogenic and psychogenic stimuli.

We now turn to the time-dependent nature of sweat-gland activation (Table 5). The intermittent activity of sweat glands was first described by Takahara (1936), and further elaborated by Kuno (1938), Randall (1946b), Wyndham (1973) and Nishiyama et al. (2001). Sweat secretion typically follows an oscillating pattern, and most pertinent to our discussion is the synchronisation of that rhythm within, but also across body segments, including glabrous (volar hand and foot) and non-glabrous sites (Nakayama and Takagi, 1959; van Beaumont et al., 1966; Hagbarth et al., 1972; Ogawa et al., 1977; Taylor and Machado-Moreira, 2013). This provides strong evidence for neural (sympathetic) linkages between those glands and a common central controller, leading us to reject the possibility that the volar skin is uniquely controlled. However, the oscillations above relate only to thermogenic sweating.

During the psychogenic treatment, the average durations of glandular activation across all sites were similar to those observed when the sudorific impact of all treatments was assessed at the volar hand. Those durations ranged between 1 and 3 min (Table 5). Much longer activations were observed at the forehead and chest during the thermogenic and thermo-psychogenic treatments (5.5–7.5 min; Table 5). Those observations require verification. Nevertheless, the between-treatment differences were also apparent when the patterns of glandular reactivation were evaluated (Table 5); an observation that appeared consistent with variations in activation duration. Indeed, it was invariably observed that the number of reactivated glands during the psychogenic treatment approached an order of magnitude lower than recorded for the other two treatments (Table 5), although that may reflect variations in stimulus intensity rather than differences in neural control.

In a previous investigation, we described the progressive activation of sweat glands during an identical psychogenic stimulation (Machado-Moreira and Taylor, 2017), and while the sweat rate remained stable, the number of activated sweat glands seemed to increase, similar to that seen during other non-thermal stimuli (Amano et al., 2011). Unfortunately, the colourimetric technique used in our previous investigation was insensitive to glandular deactivation, leading to a misinterpretation of those observations. In the current experiment, patterns of activation and deactivation were more realistically tracked (Table 5 and Fig. 3), and it is now apparent that, during the thermogenic and thermo-psychogenic treatments, the proportion of active sweat glands remained surprisingly consistent across successive sampling periods. Furthermore, those active glands were not necessarily the same glands, as previously inactive glands were frequently recruited, as described by others (Nishiyama et al., 2001). To our knowledge, this is the first time that such an observation has been described, and if considered with the evidence that the sweat rate of the volar hand remains stable beyond the second minute of an almost identical experimental treatment (Fig. 4A from Machado-Moreira and Taylor, 2017), then it follows that both the local sweat rate and the number of activated glands remain stable during those stimulations, but with individual glands alternating between active and inactive states.

5. Conclusion

The current observations support the hypothesis that psychogenic, thermogenic and thermo-psychogenic stimuli all activate the same eccrine sweat glands, across both glabrous and non-glabrous skin regions. In addition to that novel outcome, this experiment has yielded detailed evidence concerning the intermittent activity of those glands, revealing that the number of activated glands during thermogenic and thermo-psychogenic stimuli typically represent a consistent proportion of the physiologically active glands identified within each body segment.

Conflicts of interest

There are no conflicts of interest to declare.

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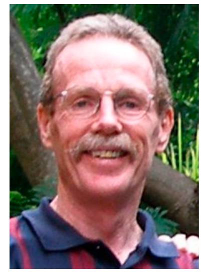

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