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RESEARCH ARTICLE

Skin tattooing impairs sweating during passive whole body heating

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[1]*Integrative Physiology and Health Science, Alma College, Alma, Michigan;* [2]*Applied Physiology and Wellness, Southern Methodist University, Dallas, Texas;* [3]*Health Sciences, Boston University, Boston, Massachusetts;* [4]*Applied Clinical Research, University of Texas Southwestern Medical Center, Dallas, Texas;* [5]*Department of Physiology, University of Kentucky College of Medicine, Lexington, Kentucky; and* [6]*Department of Neurology and Neurotherapeutics, University of Texas Southwestern Medical Center, Dallas, Texas*

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Luetkemeier MJ, Allen DR, Huang M, Pizzey FK, Parupia IM, Wilson TE, Davis SL. Skin tattooing impairs sweating during passive whole body heating. *J Appl Physiol* 129: 1033–1038 2020. First published September 3, 2020; doi:10.1152/japplphysiol.00427.2019.—Tattooing of the skin involves repeated needle insertions to deposit ink into the dermal layer of the skin, potentially damaging eccrine sweat glands and the cutaneous vasculature. This study tested the hypothesis that reflex increases in sweat rate (SR) and cutaneous vasodilation are blunted in tattooed skin (TAT) compared with adjacent healthy skin (CON) during a passive whole body heat stress (WBH). Ten individuals (5 males and 5 females) with a sufficient area of tattooed skin participated in the study. Intestinal temperature (T_{int}), skin temperature (T_{skin}), skin blood flow (laser Doppler flux; LDF), and SR were continuously measured during normothermic baseline (34°C water perfusing a tube-lined suit) and WBH (increased T_{int} 1.0°C via 48°C water perfusing suit). SR throughout WBH was lower for TAT compared with CON ($P = 0.033$). Accumulated sweating responses during WBH (area under curve) were attenuated in TAT relative to CON (23.1 ± 12.9, 26.9 ± 14.5 mg/cm^2, $P = 0.043$). Sweating threshold, expressed as the onset of sweating in time or T_{int} from the initiation of WBH, was not different between TAT and CON. Tattooing impeded the ability to obtain LDF measurements. These data suggest that tattooing functionally damages secretion mechanisms, affecting the reflex capacity of the gland to produce sweat, but does not appear to affect neural signaling to initiate sweating. Decreased sweating could impact heat dissipation especially when tattooing covers a higher percentage of body surface area and could be considered a potential long-term clinical side effect of tattooing.

NEW & NOTEWORTHY This study is the first to assess the reflex control of sweating in tattooed skin. The novel findings are twofold. First, attenuated increases in sweat rate were observed in tattooed skin compared with adjacent healthy non-tattooed skin in response to a moderate increase (1.0°C) in internal temperature during a passive whole body heat stress. Second, reduced sweating in tattooed skin is likely related to functional damage to the secretory mechanisms of eccrine sweat glands, rendering it less responsive to cholinergic stimulation.

eccrine; skin blood flow; sweat rate; thermoregulation

INTRODUCTION

The popularity of decorative skin tattoos has increased in the past decade to become normative within popular Western culture. It is estimated that ~24% of Americans from late childhood to 60 yr of age have at least one tattoo, and the prevalence of tattoos among younger individuals, professional athletes, and military populations is even higher (16). Modern tattooing techniques involve repetitively puncturing the skin with a series of needles 50–3,000 times per minute to permanently deposit pigments or ink at a depth of 1–5 mm into the dermal layer of the skin. This process results in short-term complications (resolving in a few weeks) such as discomfort, erythema, bleeding, inflammation, and destruction of the cellular interface that connects the epidermal and dermal layers (20, 35). The incidence rate of long-term complications is ~7% and includes contact dermatitis and superficial/deep local infections (16, 18).

Another potential long-term complication could be damage to the eccrine sweat gland, leading to an impaired sweating response. An early case study reported decreased sweating in tattooed skin compared with control skin during a 15 min period of local heating (9). However, the study was limited as only results from one subject were reported and sweat rate (SR) was the only variable collected. Despite these limitations, this study provides the first evidence that sweat responses could be impaired in tattooed skin.

Luetkemeier et al. (23) identified lower sweat rates and higher sweat-sodium concentrations induced via a quantitative pilocarpine iontophoresis test (QPIT) in tattooed skin. Elevated sodium concentrations in sweat observed during QPIT are suggestive of impaired sodium reabsorption in the eccrine duct via endothelial sodium channels and the cystic fibrosis transmembrane regulator (31). In contrast, lower absolute sweat rates likely indicate a reduction in cholinergic cell signaling or other alterations in the secretory cells in the eccrine coil of the gland (34). Luetkemeier et al. (23) concluded that hypohidrosis and excess sodium losses caused by tattooing could predispose individuals to heat illnesses and hyponatremia during heavy physical activity in hot environmental conditions. Although these QPIT findings are informative, the technique has a number of limitations including *1*) delivery of only one submaximal cholinergic dose; *2*) underlying current-induced changes from the iontophoresis procedure; and *3*) findings representing only postganglionic responses, as it bypasses normal sudomotor pathways associated with a rise in core temperature.

Rogers et al. (32) recently examined sweat function in tattooed skin during exercise. Contrary to previous findings, no

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differences were observed in sweat rates collected via absorbent patches between tattooed skin and control skin of various body locations during 20 min of intermittent exercise (4 bouts of exercise for 5 min at rating perceived exertion of 15 separated by 1 min of rest in room temperature conditions). However, it is difficult to determine the magnitude of thermal stimulus of this exercise as neither internal nor skin temperature was reported. It is possible that this 20 min of exercise was not a sufficient thermal stimulus to allow potential differences in sweat rate to be differentiated between tattooed skin and healthy control skin.

In light of the limitations of the previous studies, nonmetabolic thermal-induced sweating can be assessed in a controlled manner via passive whole body heating (WBH) in which 45°C–50°C water is perfused through a tube-lined suit (44). Using this WBH experimental approach, sweating and skin blood flow have been systematically studied in the following conditions: aging (37), bedrest (26), hypohydration (28, 41), hypoxia (22), multiple sclerosis (1), nicotine/smoking (27), plasma osmolality (3, 14), and skin grafts (12, 13). It is currently unclear whether sweating and skin blood flow responses of tattooed skin are reflexively altered during WBH. This information is important to determine if the sweating dysfunction associated with skin tattooing has a peripheral neural component (nerve fiber density, sympathetic varicosities, or neurotransmitter release) or is confined to the gland. Therefore, the aim of this study was to test the hypothesis that reflex increases in sweat rate and cutaneous vasodilation were blunted in tattooed skin compared with adjacent healthy skin during a passive WBH stress.

METHODS

Subjects. Participants were required to have tattooed skin on their upper or lower arm with a minimum surface area of 5.6 cm^2 (TAT) and non-tattooed skin (CON) with the same minimum surface area adjacent to the tattooed skin. The minimum surface area for tattooed skin is twice the size of the ventilated capsules used for sweat measurement (see description in *Instrumention*) to ensure all skin under the capsule is completely tattooed. Participants with hypertension, cardiovascular, respiratory, metabolic, and/or neurological disorders were excluded from the study. Ten subjects (5 males, 5 females) were recruited for the study after receiving a complete explanation of the procedures and providing written informed consent before participation in the study. To avoid the progesterone-related shift in the thermoregulatory set point, all female subjects were tested within the first 5–10 days from menses, thus during the follicular phase of their menstrual cycle (7). All procedures were approved by the Institutional Review Boards at Southern Methodist University (Dallas, TX) and conformed to the Declaration of Helsinki. Subject characteristics are described in Table 1.

Based on observed differences in sweat rate in tattooed and control skin reported by Luetkemeier et al. (23), an effect size of 0.78 was calculated. Eleven subjects per group were derived from a power analysis ($\alpha = 0.05$, power = 0.80) to find an anticipated difference in SR between tattooed and control skin during WBH. Eleven subjects were recruited to participate with 10 subjects completing the entire WBH protocol.

Instrumentation. Internal temperature (intestinal temperature; T_{int}) was assessed using an ingestible telemetry thermometry pill (HQ Inc., Palmetto, FL) taken ~1–2 h before the collection of baseline measurements. Mean skin temperature (T_{skin}) was measured via the weighted average of thermocouples (Type T-epoxy coated tip, Omega) attached to six skin sites (chest, upper back, lower back, abdomen, thigh, and calf) (40). Thermocouples were interfaced to a thermocouple meter (TC-2000, Sable Systems International) calibrated using a temperature calibration unit (Model CL26, Omega). Local T_{skin} was monitored at both the TAT and CON sites using small teflon thermocouples (IT-18, Physitemp Instruments) interfaced to the thermocouple meter. Arterial blood pressure was obtained via auscultation of the brachial artery (SunTech Medical Instruments, Raleigh, NC). Heart rate was measured using an electrocardiogram (ECG; Solar 8000i, General Electric, NY) interfaced with a cardiotachometer (CWE, Ardmore, PA). Sweat rate was measured using capacitance hygrometry (Vaisala, Woburn, MA) by perfusing 100% nitrogen at a flow rate of 300 mL/min through a ventilated capsule (surface area = 2.83 cm^2) placed on TAT and CON skin of the exposed arm (5, 19). Laser Doppler flux (LDF; an index of skin blood flow) was measured at TAT and CON skin sites of the exposed dorsal forearm positioned at heart level via integrated laser Doppler probes (PF413, Perimed, Ardmore, PA) connected to a laser Doppler flowmeter (PF 5010, Perimed). Probes were fitted inside thermostatic probe holders (PF 450, Perimed) connected to a local heating device (PF 5020, Perimed) capable of controlling local skin temperature at that site. Local heating devices were only utilized to elicit maximal skin blood flow responses following WBH and not to clamp skin temperature during WBH. To modify T_{skin} and T_{int}, subjects were fitted in a tube-lined water perfusion suit (Allen-Vanguard Technologies Inc., Ottawa, ON, Canada) that covered their whole body with the exception of the head, feet, hands, and instrumented areas (TAT and adjacent CON skin). The water perfusion suit was connected to water baths and an external pump to induce a thermal stress by controlling both water temperature and flow.

Table 1. *Subject characteristics*

Subject	Sex	Age, yr	Height, cm	Mass, kg
1	M	39	180.3	88.0
2	F	26	173.2	65.0
3	F	29	173.4	77.3
4	M	47	180.5	90.1
5	F	19	165.1	61.8
6	M	35	188.8	96.5
7	M	23	171.5	56.6
9	M	19	186.5	80.8
10	F	24	165.5	93.9
11	F	21	170.1	71.0
Mean		28	175.5	78.1
SD		9	8.2	14.1

F, female; M, male.

Protocol. Following instrumentation, the participants laid supine on a patient bed for a 10-min normothermic baseline period with 34°C water perfusing the suit. Immediately following the baseline period, passive whole body heating (WBH) was performed by perfusing 48°C water through the suit until T_{int} increased 1.0°C. The following variables were continuously measured throughout the heating protocol: T_{int}, T_{sk}, heart rate, LDF, and SR. Upper arm cuff blood pressures were taken at 10 min intervals. As the suit was not in contact with the regions of skin where LDF and SR data were assessed, responses from these areas are presumed to be due entirely to thermoregulatory reflex-mediated neural modulation of efferent activity in response to changes in T_{int} induced by the suit. Upon achieving the desired increase in internal temperature (1.0°C), T_{int} was stabilized by lowering the temperature of the water perfusing the suit to 42°C. Once a stable elevated T_{int} was established, WBH measurements were recorded before the subjects were returned toward normothermic baseline T_{int} levels by decreasing the temperature of the water perfusing the suit to 16°C. During this time period, local heating was performed at the sites of LDF collection on the forearm by setting thermostatic probe holders to 44°C. Local heating was terminated after 30 min.

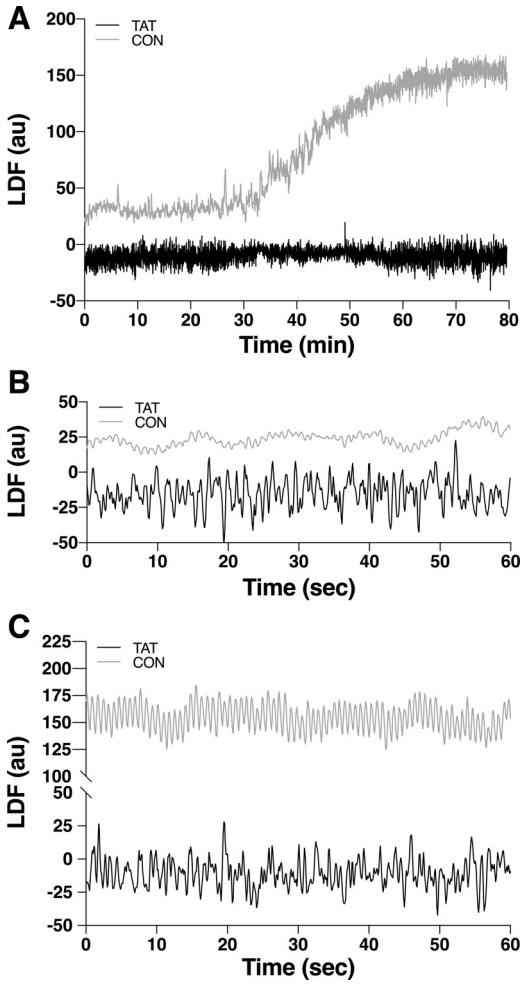

Fig. 1. A representative tracing of the LDF signal in one participant in tattooed skin (gray tracing) and control skin (black tracing) during the entire WBH period (*A*), during the first 60 s of WBH (*B*), and the final 60 s of WBH (*C*). LDF, an index of skin blood flow, was not calculated or analyzed due to the absence of an appropriate increase in LDF during WBH (*A*) and the lack of typical beat-by-beat and rhythmical alterations in the LDF signal (*B* and *C*) in tattooed skin. CON, healthy adjacent control skin; LDF, laser Doppler flux; TAT, tattooed skin; WBH, whole body heat stress.

All the participants with the exception of one completed the entire WBH protocol. One male subject experienced a run of premature ventricular contractions after reaching a 0.7°C rise in T_{int} at which time WBH was terminated. Data for this subject were excluded from all analyses.

Data analyses. All data were continuously acquired at a sampling rate of 200 Hz on a data acquisition system (Biopac, Santa Barbara, CA). Changes in sweat rates and T_{skin} relative to normothermic baseline conditions were determined for each 0.1°C increase in T_{int}. Sweating thresholds and slopes of the response from the onset of sweating as a function of both time and T_{int} in TAT and CON were determined using a segmental linear regression analysis based on a least-squares regression technique for each participant (8).

LDF measurements were not analyzed or reported due to several factors. First, an appropriate increase in the LDF signal during WBH was absent in tattooed skin (Fig. 1*A*). Second, typical beat-by-beat and rhythmical alterations at baseline (Fig. 1*B*) and peak heating (Fig. 1*C*) were not observed in the LDF signal of tattooed skin. These observations were consistent in the tattooed skin of all the participants. To the authors' knowledge, this has not been previously reported and are unsure if ink pigments within the tattoo are acting as chromophores or as reflectants to the 780 nm laser light. Pulse oximetry and similar related measurements have reported errors in measurements when placed above certain nail polish (6, 39), suggesting a potential rationale for the inability to collect laser Doppler flowmetry measurements in the skin.

Statistical analyses. All values are presented as means ± SD. A two-way within-factors (skin type and increases in T_{int}) repeated-measures ANOVA was utilized to compare SR and T_{skin} of skin (TAT vs. CON) as a function of T_{int} (0.1°C increments). Total sweat production (area under the curve) and sweating onset (time and temperature) values between TAT and CON were compared using paired *t* tests. For any significant main effects between skin type, an effect size [partial eta squared (η^2) or Cohen's *d*] was calculated to provide practical significance. All analyses were conducted using GraphPad Prism 8.3.1 (GraphPad Software Inc., La Jolla, CA). Statistical significance was accepted at $P < 0.05$.

RESULTS

Sweat rate. Sweat rates at normothermic baseline before WBH were not different between skin sites (TAT: 0.083 ± 0.025 mg·cm^{-2}·min^{-1}, CON: 0.080 ± 0.023 mg·cm^{-2}·min^{-1}, $P = 0.117$). The average change in sweat rates from normothermic baseline for TAT were significantly less than for CON during WBH, as indicated by a significant main effect of skin type ($P = 0.033$, $\eta^2 = 0.413$, Fig. 2). The total amount of sweat produced throughout WBH (area under the curve) was less for TAT compared with CON ($P = 0.043$, $d = -0.28$, Fig. 3). Total sweat production was less in TAT compared with CON in 7 of the 10 subjects following WBH (Fig. 3).

Sweat onset. No differences were observed in the time or temperature associated with the onset of sweating between TAT and CON. TAT and CON began sweating at 15.3 ± 5.6 min and 16.1 ± 6.1 min ($P = 0.087$), respectively, after the start of WBH, which corresponded to core temperatures of 36.96 ± 0.31°C and 36.94 ± 0.31°C ($P = 0.405$). Slopes of the sweating response from the onset of sweating were significantly lower in TAT compared with CON (TAT: 0.62 ± 0.29, CON: 0.74 ± 0.30, $P = 0.047$).

Skin temperature. Skin temperatures at normothermic baseline before WBH were not different between skin sites (TAT: 30.69 ± 0.90 mg·cm^{-2}·min^{-1}, CON: 30.81 ± 1.42 mg·cm^{-2}·

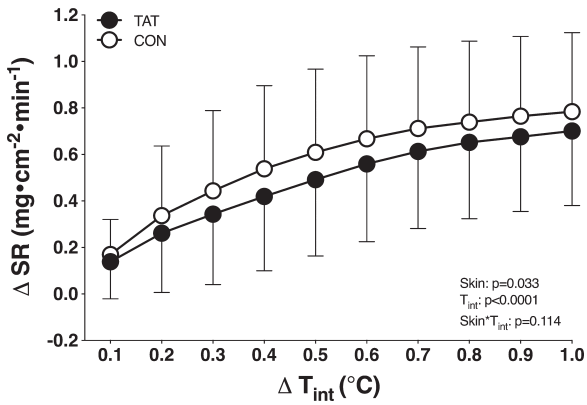

Fig. 2. Mean changes (±SD) in sweat rate (ΔSR; mg·cm^{-2}·min^{-1}) from baseline as a function of changes in intestinal temperature (ΔT_{int}, °C) during passive whole body heating in tattooed skin (TAT) and healthy adjacent control skin (CON) of 10 participants (5 males, 5 females). Differences were observed between TAT and CON (skin main effect; $P = 0.033$) using a 2-way within-factors repeated-measures ANOVA.

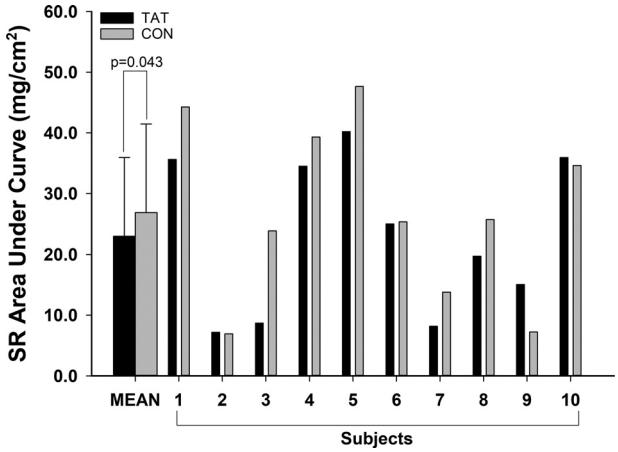

Fig. 3. Mean changes (thick bars; ± SD) and individual changes (thin bars) in total sweat accumulation (area under the curve; mg/cm^2) following a 1.0°C increase in intestinal temperature during passive whole body heating in tattooed skin (TAT) and healthy adjacent control skin (CON) of 10 participants (5 males, 5 females). Differences were observed between TAT and CON (P = 0.043) using a paired t test.

min^{-1}, P = 0.818). No differences were observed in the increase of local T_{skin} between TAT and CON when expressed as a change from normothermic baseline (Fig. 4).

DISCUSSION

The primary new finding in the current study is that peripheral nonglabrous skin of the arm containing tattoos has reduced sweat rates, and thus potential heat loss capacity, during WBH compared with adjacent skin without tattoos. Differences in eccrine sweat rate between TAT and CON were not associated with delays in sweating thresholds during WBH, but rather the total quantity of sweat production and sweat rate per increase in internal temperature. These data indicate that the collateral effects of the tattooing process negatively impact eccrine sweat gland function and could be considered a potential long-term complication of this cosmetic procedure.

This study extends previous cholinergic agonist-induced observations of lower sweat rates and higher sodium-sweat concentrations in tattooed skin compared with adjacent healthy control skin (23). Although these initial observations provided evidence of impaired sweating in tattooed skin, the functional effects on sudomotor and glandular secretions were still unclear due to a number of limitations with the QPIT methodology. In the present study, WBH was used to increase skin sympathetic nerve activity (11, 43), to induce cholinergic sweating, and other in vivo factors important in engaging and sustaining the sweating process (4, 10, 31, 34). During WBH, significantly less sweat was produced in TAT versus CON skin. The difference in the total amount of reflex sweat collected from tattooed skin was ~85% of that collected from control skin, whereas in the previous study (23), the sweat collected from tattooed skin was only 53% of the control skin. This could indicate that the decrement in sweating due to tattooing was diminished in thermoregulatory sweating compared with a single dose of acetylcholine agonist. It is plausible that the sweating deficiency is flow rate-dependent (greater differences are seen at certain flow rates) as pilocarpine iontophoresis dose is near maximal while WBH encompasses both low and high sweat rates associated with the gradual rise in core temperature. Alternately, QPIT acts directly and WBH indirectly to increase sudomotor activity to release acetylcholine to induce sweating. As such, the sweating deficiency may be confined to the postsynaptic cholinergic pathway (type-3 muscarinic receptor stimulation, G-protein transduction, phospholipase cyclase activation, inositol triphosphate and diacylglycerol production, Ca^{++} entry and release, and Cl^- channel activation) (25). Combined, these data indicate that tattooing results in a quantitative deficiency in the capacity of surface fluid secretions of eccrine sweat glands (~15% reduction) that was maintained throughout the whole body heat stress resulting in a 3.79 mg/cm^2 difference in total sweat. Although this is a small amount, it may be amplified and become more important in individuals that are heavily tattooed over larger surface areas of the body.

The onset of sweating was not different following the initiation of whole body heating for both TAT and CON skin. Likewise, no differences were observed in the core temperature associated with the onset of sweating. Taken together, this indicates that there is no delay in sweating in response to a threshold rise in core temperature. Classically, the sweating threshold has been interpreted as being associated with the neural control and the slope of the sweating responses as an effect on the gland itself (15). The slopes of the sweating responses associated with increasing T_{int} were significantly less for TAT versus CON. Taken together, it is likely that the neural innervation and initial sudomotor transduction of the neural signal to the sweat glands remains intact and functional in tattooed skin. We hypothesize that skin-sympathetic nerve activity would be similar between adjacent skin areas, but the local cholinergic nerves may have been damaged by the tattooing process. Although these variables were not directly assessed in the current study, it appears that there is adequate innervation and neural transmitter release to retain relative functionality.

No differences were observed in the skin temperature between TAT and CON during WBH (Fig. 4) likely indicating that attenuated sweating was not enough to cause an elevation in local T_{skin} in TAT above that of CON in the current study. Therefore, it was unlikely that differences in sweat rates

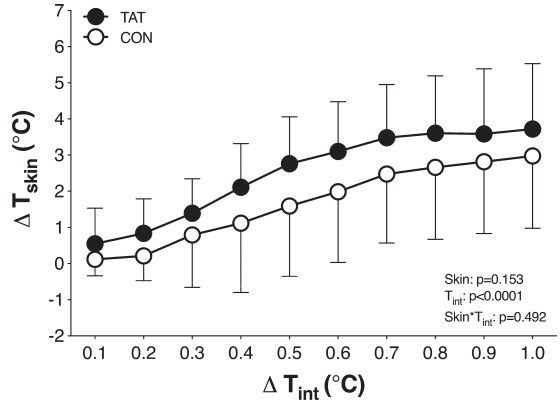

Fig. 4. Mean changes in local skin temperature (ΔT_{skin}, °C) from baseline (±SD) as a function of changes in intestinal temperature (ΔT_{int}, °C) during passive whole body heating in tattooed skin (TAT) and healthy adjacent control skin (CON) of 10 participants (5 males, 5 females).

between TAT and CON were related to a local cooling suppression effect (29, 38).

The precise mechanisms responsible for attenuated sweat responses are unknown. Likely, candidates include physical damage to the gland during the tattooing process and sustained/repeated immunological responses to the injection and presence of ink and dyes (16). The tattooing process can involve repeated (up to 3,000 times per minute) puncture trauma within a 1–5 mm depth range, and thus may have the potential to directly damage eccrine sweat gland secretory coils and reabsorptive ducts. The skin is classified as thick or thin (relating to the epidermis) due to presence of hair follicles (glabrous or nonglabrous). A tattoo can be applied to both skin types. In thin nonglabrous skin of the upper arm (a common tattoo site), the dermal depth is ~1.0–1.4 mm (21) with a hypodermal depth of ~12 mm (24, 30) depending on precise location, sex, and genetics. Eccrine secretory coils are located in the reticular layer of the dermis and superficial hypodermis, while the ducts transverse the dermis and epidermis to the skin surface. Thus, these structures appear to be within the needle puncture depth of the ink delivery procedure. Although tattoo trauma is unique, burns and skin grafts may offer some interpretive insight to the current data. Davis et al. (13) have identified long-term reductions in cutaneous vasodilation and sweating capability in grafted skin 4 to 8 yr postsurgery. These deficits in eccrine sweat function in grafted skin are observed with both WBH (12) and direct postsynaptic cholinergic stimulation (12). This seems to indicate that eccrine sweat glands have limitations in their regenerative capacity when damaged or sliced.

In addition to physical trauma, there are also reports of hypersensitivity reactions to pigments (especially certain red inks and dyes) and reports of granulomatous reactions and sarcoidosis (2, 17, 33, 42). The ink pigments are solid particles (e.g., metal salts and organic compounds) suspended in diluent (e.g., alcohol or water). Combining different pigments makes different shades and colors. Although most of the pigments are fairly inert, some may be immunoreactive and in certain environments such as UV exposure can heighten or repeat reactions (35, 36). Tattooing is considered a cosmetic procedure and thus is less regulated in the United States than medical procedures, and not all inks fully detail their contents. It is currently unclear if and how hypersensitivities and granulomatous reactions affect eccrine sweat gland function.

Limitations and future considerations. There are several limitations of the current study. First, tattooed skin in the current study included the following differences that were difficult to control: procedures (i.e., tattoo artists, application guns, needles, etc.), types of inks, ink colors, designs, and age from initial tattooing. Therefore, it is unclear how these uncontrolled factors impacted the observed impairments in sweating. Second, skin blood flow could not be determined in tattooed skin by laser Doppler flowmetry likely due to the pigments in the tattoo ink. Other measurements, including laser scatter, that allow the laser frequency to be altered allowing penetration into inked areas of the tattoo may be useful in determining skin blood flow. Lastly, the WBH procedure experimentally clamps skin temperature under the water perfusion suit, creating an uncompensable heat stress. Whole body heating was used as the experimental paradigm to increase sudomotor drive, as this can be controlled and is independent of changes in metabolism and arterial blood pressure associated with exercise. Thus, caution should be employed when extending or comparing the current findings to compensable and exercise heat stresses. Now that these decrements in eccrine sweat function have been identified in tattooed skin, further research is warranted to more precisely characterize the impact of this finding on sweat gland thermoregulatory function in a compensable environment and ultimately assess whether attenuated sweating responses in tattooed skin covering large skin surface areas translate to increased risk of heat-related injury.

Conclusion. Tattooed skin demonstrates an attenuated sweat rate in response to passive whole body heating compared with adjacent healthy control skin. These data may indicate functional damage to the secretory coil of eccrine sweat glands rendering it less responsive to cholinergic stimulation. As tattooed skin still sweats in response to WBH and sweating onset times are similar between tattooed and control skin, neural signaling mechanisms remain intact. However, it is unknown if neural structures are spared during the trauma induced by tattooing, or if there is some plasticity and return of function after the tattooing process. Combined with previous studies (23), which also suggested potential functional damage of the eccrine sweat gland duct, there appears to be long-term consequences of the tattooing process that have not previously been considered. Decreased sweating in tattooed skin could impact heat dissipation, especially when tattooing covers a higher percentage of body surface area and could be considered a potential clinical side effect of tattooing.

ACKNOWLEDGMENTS

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DISCLOSURES

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

AUTHOR CONTRIBUTIONS

M.J.L., D.R.A., M.H., T.E.W., and S.L.D. conceived and designed research; M.J.L., D.R.A., M.H., F.K.P., I.M.P., and S.L.D. performed experiments; M.J.L., D.R.A., F.K.P., I.M.P., and S.L.D. analyzed data; M.J.L., D.R.A., M.H., T.E.W., and S.L.D. interpreted results of experiments; M.J.L. and S.L.D. prepared figures; M.J.L., T.E.W., and S.L.D. drafted manuscript; M.J.L., D.R.A., M.H., F.K.P., I.M.P., T.E.W., and S.L.D. edited and revised manuscript; M.J.L., D.R.A., M.H., F.K.P., I.M.P., T.E.W., and S.L.D. approved final version of manuscript.

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