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Unravelling some of the complexities concerning the neural control of human eccrine sweating

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Abstract
The widely accepted, though not unequivocal, opinion concerning thermal and psychological (psychogenic) seating is tha the former is cholinergically mediated (Dale & Feldberg, 1934), while the latter is of noradrenergic origin (Robertshaw, 1977). Moreover, psychological sweating is thought to be elicited by a different neural centre (Ogawa, 1975), possibly through separate pathways (Chalmers & Keele, 1952) that exclusively innervate the glabrous (non-hairy) skin of the hands and feet (Darow, 1937, Kuno, 1956, Ogawa, 1975). Evidence for the cholinergic modulation of thermal sweating is incontrovertible. However, evidence supporting the theoretical control of psychological sweating is less than convincing. Following observations of Chalmers & Keele (1952) and Kuno (1956) first Ogawa (1975), and then Ogawa et al. (1977) hypothesised the existence of separate non-thermal pathways, and this was further developed by lwase et al (1997). Their hypothetical model, in addition to assigning thermal seating to the non-glabrous (hairy) sites and psychological sweating to glabrous skin surfaces, proposed the existence of inhibitory affects of thermal loading upon psychological sweating, and of psychological stress upon thermal sweating. Two recently completed projects were designed to test the underlying assumptioins of this model, and this research is summarised below.

Keywords
human, control, eccrine, neural, unravelling, concerning, sweating, complexities

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UNRAVELLING SOME OF THE COMPLEXITIES CONCERNING THE NEURAL CONTROL OF HUMAN ECCRINE SWEATING

Christiano A. Machado-Moreira, Peter L. McLennan, Stephen Lillioja, Joanne N. Caldwell, Wilko van Dijk and Nigel A.S. Taylor

INTRODUCTION

The widely accepted, though not unequivocal, opinion concerning thermal and psychological (psychogenic) sweating is that the former is cholinergically mediated (Dale & Feldberg, 1934), while the latter is of noradrenergic origin (Robertshaw, 1977). Moreover, psychological sweating is thought to be elicited by a different neural centre (Ogawa, 1975), possibly through separate pathways (Chalmers & Keele, 1952) that exclusively innervate the glabrous (non-hairy) skin of the hands and feet (Darrow, 1937; Kuno, 1956; Ogawa, 1975). Evidence for the cholinergic modulation of thermal sweating is incontrovertible. However, evidence supporting the theoretical control of psychological sweating is less than convincing.

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METHODS

The first project involved six separate experiments conducted across eight body segments, in which 38 glabrous and non-glabrous skin sites were studied in 30 passively heated individuals. For this work, core temperature was first elevated by ~0.5°C and clamped, using a combination of heated air (36°C, 60% relative humidity), and heated water passed through a water-perfusion garment (40-46°C; Figure 1). This led to the establishment of low-intensity, steady-state thermal sweating, the purpose of which was to provide the best possible opportunity for sweat glands to respond to psychological stimulation: mental arithmetic (1 min) and a painful stimulus (15 s of strong palmar pressure). Sweating responses were evaluated from discharged sweat, measured using ventilated sweat capsules (1.40 and 3.16 cm^2) glued to each site, to prevent leakage and pressure-induced artefacts.

In the second project, nine subjects participated in a four-phase experiment, in which palmar pain, mental arithmetic and static, handgrip exercise were used to evoke sweating in the first three treatments, performed in series. The first state was thermoneutral rest (air: 27-28°C). These stimuli were reapplied following passive, whole-body heating (water-perfusion garment (48°C) and foot bath (42°C); Figure 1) that induced a core temperature elevation of 0.5°C above its thermoneutral level. This thermal state was then clamped, and a systemic atropine sulphate infusion (0.04

mg.kg^{-1}) was administered intravenously. Water bath and perfusion garment temperatures were modified to sustain the clamp, with core temperature changing by <0.1°C. This clamping was essential to prevent the gradual rise in core temperature that would otherwise accompany a systemic cholinergic blockade within hot conditions. Finally, in the fourth phase of these trials, heating was increased in an attempt to re-establish thermal sweating. Discharged sweating responses were measured from the forehead, right forearm (dorsal surface), right hand (dorsal and palmar surfaces), and the right calf (upper medial surface) using ventilated sweat capsules (3.16 cm^2), with primary sweating evaluated from changes in palmar skin conductance.

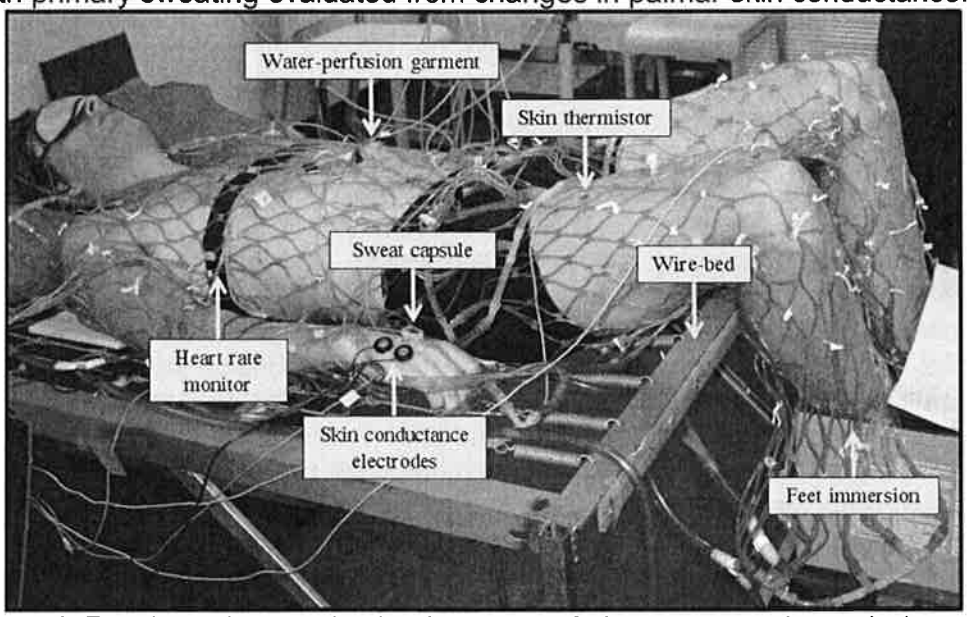

Figure 1. Experimental set-up showing the water-perfusion garment and water bath used to heat and clamp body temperature.

RESULTS

In the first series of experiments, both psychological stimulation and acute pain resulted in significant elevations in sweat secretion from more than 70% of the sites investigated (psychological: 28 of 38 sites (P<0.05); pain: 23 of 32 sites (P<0.05)). These changes were evident within both the glabrous and non-glabrous skin surfaces, and occurred without changes in mean body temperature (P>0.05). Indeed, there were no consistent sudomotor response differences between the glabrous and non-glabrous skin surfaces, and at no site was thermal sweating inhibited during a non-thermal stimulation.

During the blockade trials, the non-thermal treatments elicited significant sweating only from the palm when subjects were thermoneutral (P<0.05), and this secretion was evident from both the sweat capsule and skin conductance measures. However, following passive heating, these stimuli induced secretion from every site (P<0.05), as observed within the first experimental series. Conversely, with the establishment of the cholinergic blockade, none of these non-thermal challenges could evoke either primary or discharged sweating (P>0.05). But when the thermal clamp was removed, and core and skin temperatures were deliberately driven upwards, thermal sweating was restored (P<0.05), even though each subject was still under the influence of

atropine. Since atropine is a competitive antagonist for acetylcholine, it can be assumed that this blockade breakthrough occurred due to a thermally mediated increase in acetylcholine release. Therefore, residual sweating in the presence of such a blockade cannot simply be assumed to represent evidence for the participation of other neurotransmitters in the control of this sweating.

CONCLUSION

Observations from the first series of experiments appear to refute the existence of the independent modulation of either thermal or psychological sweating from separate skin surfaces. Indeed, both forms of secretion appear to be ubiquitous. Furthermore, these experiments failed to show any evidence for a reciprocal inhibition of sweating, as first postulated by Kuno (1956).

The second experiment, through the use of a thermal clamp during a cholinergic blockade, has established the dependence of both thermally and non-thermally mediated sweating upon acetylcholine, from both the glabrous and non-glabrous skin surfaces. Indeed, the complete absence of sweating during the blockade makes it highly unlikely that any other neurotransmitter was significantly participating in these sudorific responses prior to the blockade. Moreover, whilst these observations unequivocally confirm the cholinergic determination of thermal sweating (Chalmers & Keele, 1952; Foster & Weiner, 1970), they also now extend this control to include these non-thermal sudomotor drives, for which noradrenergic efferents had previously been postulated (Robertshaw, 1977; Nakazato *et al.*, 2004).

REFERENCES

Chalmers, T.M., & Keele, C.A. (1952). The nervous and chemical control of sweating. *Brit. J. Dermatol.*, **64:** 43-54.

Dale, H.H., & Feldberg, W. (1934). The chemical transmission of secretory impulses to the sweat glands of the cat. *J. Physiol.*, **82:** 121-128.

Darrow, C.W. (1937). Neural mechanisms controlling the palmar galvanic skin reflex and palmar sweating. *Arch. Neurol. & Psychiatry.*, **37:** 641-663.

Foster K.G., & Weiner, J.S. (1970). Effects of cholinergic and adrenergic blocking agents on the eccrine sweat glands. *J. Physiol.*, **210:** 883-895.

Iwase, S., Ikeda, T., Kitazawa, H., Hakusui, S., Sugenoya, J., & Mano, T. (1997). Altered response in cutaneous sympathetic outflow to mental and thermal stimuli in primary palmoplantar hyperhidrosis. *J. Auton. Nerv. Sys.*, **64:** 65-73.

Kuno, Y. (1956). *Human perspiration*. C.C. Thomas, Springfield, Illinois.

Nakazato, Y., Tamura, N., Ohkuma, A., Yoshimaru, K., & Shimazu, K. (2004). Idiopathic pure sudomotor failure: Anhidrosis due to deficits in cholinergic transmission. *Neurol.*, **63:** 1476-1480.

Ogawa, T. (1975). Thermal influence on palmar sweating and mental influence on generalized sweating in man. *Jap. J. Physiol.*, **25:** 525-536.

Ogawa, T., Asayama, M., & Ito, M. (1977). Comparison of sudomotor neural activities between palmar and non-palmar sweating. *Proceedings of the XVIII International Congress of Neurovegetative Research*. Nov 4-6, 1977. Tokyo, Japan. Pp. 236-238.

Robertshaw, D. (1977). Neuroendocrine control of sweat glands. *J. Invest. Dermatol.*, **69:** 121-129.

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