The possible trigger of the thermoregulation in human skin at hyperthermic irritation: A phase transition at 41 °C.

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Local hyperthermic irritation of the skin leads to increased perfusion, which performs the thermoregulation. From heat tolerance experiments it is derived, that this thermoregulation is more than heat transport. Thermoregulation at hyperthermic exposition of the skin is mainly the removal of the liberated histohormones. These histohormones are histamine, kinins and prostaglandines, which are affecting the smooth muscles, thus producing the enlarged perfusion of the blood vessels, and which (mainly histamin) are stimulating the endothelial pinocytosis, thus producing the edema or the blister in the case of thermal injury (1).

The heat induced liberation process of these histohormons at weak hyperthermic exposition (T <45°C) is unknown. On the one hand a neural feedback is detectable, because a cut and degeneration of the sensory fibres influences the flare of the irritated field. On the other hand a local reaction is observed, which is independent on reflectory neural output (1).

For exact determination of the regulating activity of the skin a heat flux calorimeter according to (2) was used. The heat flux into the skin was measured at defined temperatures between skin temperature (30-35 °C) and 49 °C at the forearm and thorax. The results are identical with respect to the regulatory phenomenon: The heat flux detected regulatory change of the blood flow is not a steady function of the temperature. Up to 37 °C the local blood flow is unchanged. Between 37 and 45 °C the blood flow increases to a maximum value, which is not affected by a further increase of the temperature.

The temperature dependent change of the blood flow clearly indicates, that a phase transition between 37 and 45 °C with the transition temperature T_t = 41 °C is linked to the heat regulation of the skin. The hypothesis is discussed, that a transmitter is released on the phase transition of a lipid structure. This may be a membrane of a vesicular component of the mast cells or the terminal neural formations.

A phase transition at 41 °C is known from dispersed dipalmitoyllecithin, which is the main component of the lung alveolar surfactant and which generates domains in biological systems, as indicated by the phase transition at 41 °C in the plasma membranes of E.coli (3).

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2) Sick, H., zur Mühlen, J. and Wienert, V. (1980) Thermochimica Acta 40, 19-27.
3) Träuble, E. Eibl, H. and Sawada, H. (1974) Naturwissenschaften 64, 344 - 354.