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Temperature receptors in cutaneous nerve endings are not thermostat molecules that induce thermoregulatory behaviors against thermal load

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Dear Editor-in-Chief,

The conclusion suggested by Shigeo Kobayashi's paper "Temperature receptors in cutaneous nerve endings are thermostat molecules that induce thermoregulatory behaviors against thermal load"[1] is not at all justified.

The discussion about comparators, comparing neuronal set-points with neuronal signals representing actual temperatures, and in case of a difference between such 2 signals, actuating effector mechanisms, has occupied the thermal community for decades. The first very old thermoregulatory models were based on such "thermostats," thought as neuronal circuits, located in the central thermoregulatory areas. Shigeo Kobayashi transfers such "thermostats" from the central circuit level to the peripheral molecular level of single thermoreceptor channels. No doubt: thermoTRPchannels exhibit, as many other identified membrane channels, an on/off or switching behavior, i.e. according to defined thresholds they are either open or closed for certain substances. However, this property does not at all qualify them for being "thermostats inducing thermoregulatory behaviors."

Two reasons prevent such a conclusion:

1. The discussed switching behavior, and by this the postulated "thermostat," are present on the channel level, whereas the efferent signals actuating thermoregulatory behaviors are on the neuronal circuit (or system) level. According to basic neurophysiological facts,[2] between these 2 processes, peripheral channel activities and central effector commands, there are a lot of integrative and circuit processes, during which an on/off-behavior will vanish and may re-appear, repetitively. The (discrete) on/off-signals of many single channels will add up to continuous membrane current signals, which may generate again discrete signals, in form of action potential trains, containing continuous information by modulation of their firing rates. Such signals on the nerve fiber level converge via synaptic transmission with many other signals and are submitted to extensive circuit processing. There is no reason to assuming that a single channel "thermostat" information is still present and relevant. Moreover, switching behavior may also occur on a more central level and disappear by integration processes, as e.g. shown for the thermoafferent system.[3] In summary, a phenomenon observed on the basic molecular level is not a relevant information parameter on a neuronal circuit or system level. This holds, by the way, for both technical and biological systems.
2. Shigeo Kobayashi is convinced that the thermoregulatory system needs one or more "thermostats." Thus, he was looking, as others before, for substrates possibly constituting such devices, and he found something. However, the first models postulating central set-points, comparators or "thermostats" have been recognized since a lot of time as non-biological, inadequate and obsolete.[4] Also recent reviews[5–7] have clearly demonstrated that substrates as comparators and thermostats are totally superfluous in a thermoregulatory control-loop, containing both various feedback loops and (for thermoregulatory behaviors) feedforward branches. Both autonomous and behavioral mechanisms are inherent parts of such a complex multi-effector system.

A re-introduction of the set-point/comparator (thermostat) concept, but this time not even on a central system level, but on a peripheral molecular channel level, would be a tragic error, suitable to re-start on old confusing set-point discussion, which finally had come to a converging end.

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