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Editorial

Neonatal Capsaicin Treatment, a Potent Method for TRPV1-Related Physiological Studies

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Editorial

The Transient Receptor Potential Vanilloid 1 (TRPV1) channel is a member of a larger family of transient receptor potential ionic channels [1]. It is activated by noxious heat, as well as by protons, capsaicin and some endogenous factors [2]. For this reason, TRPV1 has been widely used in physiological researches, such as touch, pain, and taste, including body temperature, via the mouse model for the TRPV1 knockout in order to investigate in detail the mechanisms of TRPV1 [3-5].

Capsaicin is the main component of hot chili peppers and it is responsible for their spicy flavor and pungent taste. It acts as a neurotoxin and specifically destroys a subset of small-diameter primary afferents [6]. As described above, the TRPV1 receptors are activated by vanilloids like capsaicin, and the binding of at least two capsaicin molecules is required for complete activation of this channel [7]. Studies on various sensory mechanisms mediated by TRPV1 have been conducted via repeated treatment of capsaicin in adult rodents [8]. However, related researches on TRPV1 is still limited to temperature sensing or nociceptive threshold [9,10].

Previous report indicated that a subcutaneous injection of capsaicin into the newborn rat pups leads to spontaneous TRPV1 desensitization in the L5 spinal dorsal root ganglia [11]. That is, neonatal capsaicin treatment to the rodent is easily induced TRPV1 desensitization without hassle of repeated capsaicin treatment, and it would be possible to observe a variety of physiological changes relating to TRPV1 desensitization. However, there are few reports on the physiological effect of TRPV1 desensitization via neonatal capsaicin treatment [11-13]. Therefore, this editor would like to list the not well known physiological changes due to desensitization of TRPV1.

Firstly, it would be a good investigation tool for the research of noxious heat stimuli. It was well known that TRPV1 responds to noxious heat (> 43°C), therefore, neonatal capsaicin-treated rats showed a deficiency of noxious heat responses due to a systemic loss of capsaicin-sensitive TRPV1. As compared to the control, withdrawal latency to radiant infrared heat and hot water (43°C) was decreased to about 50% in the neonatal capsaicin-treated rats [11].

Secondly, neonatal capsaicin-treated rats showed hyperthermia similar with the TRPV1 antagonists-treated rats [9]. The body temperature of the rats in the group with the neonatal capsaicin treatment was maintained one degree higher than nai:ve rats [11]. The difference between TRPV1 antagonist and neonatal capsaicin treatment is that hyperthermia was induced chronically in neonatal capsaicin treated-rats. Therefore, neonatal capsaicin treatment would be a potential value as a model for the study of hyperthermia mechanism through TRPV1 desensitization [11,13].

Thirdly, recent report indicated that heat shock factor (Hsf) 1 genes were abnormally expressed in the neonatal capsaicin-treated rats [11]. Hsf1 not only is a key transcription factor in heat response but also controls heat shock proteins. Hsf1 expression also coincides with daily body temperature oscillations [14]. According to the report, expression of Hsf1 in the liver was increased in a pattern similar to that in the hypothalamus during the daytime. Nai:ve rats having a nocturnal behavior showed a decreased expression of Hsf1 during the daytime [11]. That is, circadian temperature rhythm would be affected by abnormally high temperature in the capsaicin-treated rats. The report would be able to provide important information of the clock gene networks.

Lastly, brown adipose tissue (BAT) is active in human newborns, in which it is responsible for maintaining body temperature. In the present report, neonatal capsaicin-treated rats were associated with long-lasting hyperthermia [13]. Therefore, BAT activity may be affected by an abnormal increase in core body temperature. According to the report, it was confirmed that the size of BAT abnormally increased and the expression levels of leptin were significantly decreased in the neonatal capsaicin-treated rats, compared to the normal rats [13]. A deficiency in leptin has been associated with an increased frequency of infection [15]. Actually, up to 2,000 colonies of Staphylococcus aureus and 1,200 colonies of Streptococcus agalactiae were identified in a dermal region of the neonatal capsaicin-treated rats [13]. Neonatal capsaicin treatment may be useful for investigating the association between hyperthermia and infectious disease.

Conclusion

Little-knownTRPV1-related physiological changes by neonatal capsaicin treatment have been described in this editorial. Neonatal capsaicin treatment affected to desensitize in systemic TRPV1 including dorsal root ganglia. As the result, abnormal noxious heat sensation, core body temperature, and clock gene expression were induced, and bacterial infection was also induced by function disruption of BAT. From this information, the unbeknown physiological changes that appear in association with the TRPV1 desensitization and various mechanisms would be able to investigate using neonatal capsaicin treatment.

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