



Review

Clinical and experimental aspects of cutaneous neurogenic inflammation

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Abstract:

The aim of this paper is to present the state of knowledge on cutaneous neurogenic inflammation.

Peripheral effector functions served by afferent sensory neurons underlie the so-called neurogenic inflammation. The mechanism of cutaneous neurogenic inflammation is connected with the release of neuropeptides from the sensory endings. They also exert a number of functions within the immune system. The activity of neuropeptides in the inflammation of the skin can be observed in the form of erythema, edema, hyperthermia and pruritus. Beside these peptides and their receptors, inflammatory skin response, is regulated by tryptase and proteinase-activated receptor 2 (PAR-2). Capsaicin decreases effects of inflammation-induced sensory neuropeptides, which was used in the treatment of diseases caused by inflammation. The activity of transient receptor potential vanilloid receptor 1 (TRP-V1) is associated with the neurogenic inflammation. In inflammatory processes, the neuro-immuno-cutaneous system undergoes activation, which is responsible for triggering and maintaining the inflammatory conditions, both in the healthy skin as well as in the pathological conditions, like psoriasis. Skin exposure to UV radiation influences the neuro-immuno-cutaneous system and causes the release of neuropeptides, thereby eliciting inflammatory response in photodermatitis.

In conclusion, understanding the mechanisms and the factors controlling neurotransmitters and their receptors will lead to the identification of novel therapeutic targets for the treatment of cutaneous diseases e.g. pruritus, psoriasis, alopecia areata.

Key words:

cutaneous neurogenic inflammation, skin, neuropeptides, receptors, capsaicin
