Invited Lectures by:

Professor Earl E. Carstens

Dept. of Neurobiology, Physiology, & Behavior, University of California, USA

and:

Professor Martin Metz

Dept. of Dermatology and Allergy, Charité – Universitätsmedizin Berlin, Germany

Time: **15 December 2017 at 09:00-10.30**

Place: **Aalborg University, Fredrik Bajers Vej 7, room no. D2-106**

Abstract by Professor Earl E. Carstens

Rodent Models of Itch and Alloknesis: Implications for Theories of Itch-Pain Discrimination

Itch mediators act via specific molecular receptors expressed in cutaneous nerve endings to excite pruriceptors. These engage spinal circuits utilizing glutamate and neuropeptides, notably gastrin releasing peptide (GRP) and substance P (SP), to elicit scratching and itch sensation. In humans, most itch mediators elicit concomitant nociceptive sensations. This was verified in a rodent model. Itch mediators mainly elicited hindlimb scratches (indicative of itch) accompanied by some forelimb wiping (indicative of pain). These findings argue against strict itch- and pain-specific pathways, and instead support theories in which itch and pain coexist. The selectivity theory proposes that pain inhibits concomitant itch. The spatial contrast theory posits that localized activation of nociceptors elicits itch and pain while more widespread stimulation elicits only pain. The intensity theory holds that itch and pain are signaled by different rates of firing in a common neuronal pathway. Available evidence supports each theory. A common complaint of chronic itch patients is that light touch elicits itch, a phenomenon called alloknesis. In rodents, lightly touching the skin normally does not elicit scratching, but does so following the induction of acute or chronic itch. Spinal interneurons expressing neuropeptide Y (NPY) normally suppress alloknesis, and their inactivation results in alloknesis. In a chronic itch model, alloknesis was attenuated by disruption of SP but not GRP signaling in the spinal cord. This implies that low threshold mechanoreceptors are normally prevented by NPY from activating the spinal itch signaling pathway, but under chronic itch the NPY inhibition is reduced, allowing light touch to activate a potentially sensitized itch pathway.

Abstract by Professor Martin Metz

Impact of Chronic Pruritus in Dermatological Diseases

Many dermatological disorders are associated with pruritus. While in some diseases pruritus is a hallmark symptom, i.e. urticaria, atopic dermatitis or lichen planus, the prevalence and intensity of pruritus in other diseases and the impact of pruritus on the general well-being and quality of life is less well known. In recent years, there have been various reports on the prevalence and burden of itch in some selected skin diseases; a detailed characterization of the presence, intensity, localization, quality and impact of pruritus in different skin diseases was, as of yet, missing. We have therefore contacted more than 1,300 unselected, consecutive in- and outpatients with active dermatological disorders. Overall, we collected data from 880 patients with 19 different dermatological diagnoses including information on the on the presence, intensity, quality and localization of pruritus. Furthermore, we have analyzed the impact of pruritus on the quality of life, the effects on sleep and activity and work productivity and the impact of pruritus on suicidal ideation. In the presentation, I will summarize the main findings from our investigations, which indicate that chronic pruritus is not only common in many skin conditions, but also clearly associated with an impairment of quality of life, sleep, and work productivity, and that pruritus is linked to depression and suicidal ideations. Body maps of pruritus additionally show distinct bodily distribution patterns of pruritus, which, together with information on quality of pruritus and scratching behavior indicate disease specific pruritus characteristics in different dermatoses.

All interested are welcome!

Yours sincerely,

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