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Conference report

Conference Report of the 4th Symposium of the German-Endocrine–Brain–Immune Network together with the German Zoological Society, Bayreuth, Germany, October 3–6, 2005

The 4th Symposium of the German Endocrine Brain Immune Network (GEBIN) was held from October 3rd until 6th at the campus of the University of Bayreuth, Bavaria, Germany. As a special of this meeting, the symposium was held together with the 98th Congress of the German Zoological Society (GZS) in order to intensify the network idea of the GEBIN.

1. The plenary sessions together with the German Zoological Society

In two outstanding plenary talks, Robert Dantzer, Bordeaux, France, and Shamgar Ben-Eliahu, Tel Aviv, Israel, summarized their extensive research. Robert Dantzer bridged the important gap between sickness behavior on one side and depression on the other. He presented convincing arguments that systemic and CNS-borne cytokines play an extremely important role for both entities. Shamgar Ben-Eliahu demonstrated important links between the stress system and tumor metastasis. His research lead to the evolution of novel therapeutic strategies to inhibit surgery—associated stress which might decrease surgery—associated metastatic spreading.

2. Peripheral neuroendocrine immune connections

In the more specific part of the GEBIN meeting, Jörg Reichrath, Dermatology, Homburg, demonstrated immunomodulatory effects of 1,25-dihydroxyvitamin D, derivatives thereof, and their anti-proliferative and differentiating effects. In addition, these hormones can generate tolerance via $CD4^+CD25^+$ T-cells. Furthermore, he showed that immune cells have the potential to locally produce biologically activate vitamin D, which feeds-back on immune cells by the vitamin D receptor.

In the session relating to peripheral neuroimmune interactions, Fiebich et al., Psychiatry, Freiburg, provided new mechanistic insights into the anti-inflammatory activities of 5-HT3-receptor antagonists such as tropisetron, which inhibits T-cell activation by targeting the calcineurin pathway. Härle et al., Rheumatology, Regensburg, demonstrated novel effects of the peripheral sympathetic nervous system on $CD4^+CD25^+$ T-cells in the collagen-induced arthritis model. He showed that the sympathetic nervous system stimulates a proinflammatory phenotype of these cells.

In the session on neuroendocrinology and immune functions, Kraus et al., Pharmacology and Toxicology, Magdeburg, demonstrated a novel interaction of the opioid and cannabinoid system in Jurkat T-cells. He demonstrated how cannabinoids signal via STAT5 to upregulate IL-4 secretion. He showed, by using transcription factor decoy oligonucleotides, that IL-4 transactivates the μ-opioid receptor gene via STAT6. This cooperative effect of both systems may contribute to the analgesic effect of cannabinoids. Beat Lutz, Max Planck Institute for Psychiatry, Munich, demonstrated how the cannabinoid system is important to normalize and maintain the body's homeostasis. He showed that endogenous cannabinoids trigger obesity and that antagonists to endogenous cannabinoid receptors induce weight loss.

Capellino et al., Rheumatology, Regensburg, demonstrated that different estrogen metabolites have opposing effects on an immune response. She showed that 16α-, 4-, and 2-hydroxyesterone confer anti-proliferative effects and that 2-hydroxyestradiol exerts mitogenic effects in activated human monocytic cells at low concentrations of 10^{-9}–10^{-11} mol/l. A local dysregulation of estrogen metabolism in chronic synovitis in rheumatoid arthritis patients might be involved in the pathogenesis of this disease. Orsal et al., Psychosomatic Clinic, Berlin, reported on the influence of progesterone derivatives on neurogenic inflammation. Dydrogesterone induced a decrease in NK-cells and an increase of Th2 cytokines by decreasing Th1 cytokines. These studies gave a good example for the cross-talk of the nervous, endocrine, and immune system during inflammation and pain.

Several reports were devoted to allergic skin diseases. Daniltchenko et al., Psychosomatic Clinic, Berlin, demonstrated an impact of stress on the worsening of dermatitis via alteration of neurogenic inflammation and the hypothalamic–pituitary-axis (HPA)-activation. Rohleder et al., Biopsychology, Dresden, demonstrated an alteration of

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the cortisol response and glucocorticoid sensitivity in atopic men and women during stressful conditions.

Böhm et al., Dermatology, Münster, reported how the α-MSH system links the endocrine and the immune system. He showed data, that human basophils express functional receptors for the neuropeptide α-MSH, which inhibits expression of IL-4. Moreover, in a murine model of allergic airway inflammation, systemic treatment with α-MSH resulted in a significant reduction of allergen-specific IgE production, eosinophil influx, and IL-4 production.

3. Stress, behavior, and immune function

Niemi and Pacheco-Lopez et al., Psychology and Behavioral Immunobiology, Zürich, demonstrated how the immune system can be conditioned. In an immune conditioning model with cyclosporin A, they demonstrated how the human immune system can "learn immunosuppression" by a conditioned stimulus. They also indicated that the insular cortex is the most important brain area for these effects. Knackstedt et al., Pediatrics, Berlin, demonstrated how maternal stress may increase the susceptibility towards a Th2-driven immune response thus rendering mice to a higher risk for atopic diseases like asthma in offsprings. Reber et al., Zoology, Regensburg, provided evidence that chronic social stress in mice decreases the severity of acute colitis, which was followed by impaired wound healing and higher mortality. Engler et al., Psychology and Behavioral Immunobiology, Zürich, showed how social stress can impair the anti-inflammatory capacity of glucocorticoids in termination of inflammatory processes. He also found a redistribution of $CD11b^{+}$ cells from the bone-marrow to the spleen during social stress. Dawils et al., Animal Physiology, Bayreuth, demonstrated a negative impact of acute stressors on the susceptibility to NK-sensitive tumor metastasis and how the susceptibility may be attenuated by different social housing conditions.

4. Neuroimmune–endocrine network in psychiatric disorders and neuroimmunology of the CNS

Metselaar et al., Pharmaceutical Sciences, Utrecht, illustrated in animal models how effective glucocorticoids in a liposomal formulation can achieve high tissue concentrations thus greatly reducing local immunopathology in arthritis. This therapeutic intervention may be useful during flare-ups of arthritis and encephalomyelitis. Dimitrov et al., Neuroendocrinology, Lübeck, demonstrated how sleep can modulate the immune response by increasing IL-12 production form pre-myeloid dendritic cell precursors. Schwarz et al., Psychiatry, Munich, reported an over-stimulation of the tryptophan degrading enzyme (IDO) mediated by proinflammatory cytokines in patients with depressive disorders. Müller and Riedel et al., Psychiatry, Munich, demonstrated that inhibition of COX-2 confers an anti-inflammatory effect via the inhibition of prostaglandin E2 and pro-inflammatory cytokines. They pointed out that COX-2 inhibition has therapeutic effects in early stages of schizophrenia and in depression. Musil et al., Psychiatry, Munich, demonstrated that macrophage inhibitory factor (MIF) is significantly elevated in depressed patients. MIF is secreted by the pituitary gland together with ACTH. The significance of up-regulated MIF levels is not yet clear but might be a factor for increased circulating cytokines in these diseases. Müller, Psychiatry, Munich, summarized the growing evidence that central inflammatory mechanisms play an important role in the long-term neurodegenerative changes in depression by inducing neuronal apoptosis and exciting neurons.

Thanks to the excellent organization of Volker Stefanski and Dietrich von Holst not only the scientific program but also social aspects of this meeting were admirable (see also www.gebin.org).

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