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Immune Mechanisms of Pain and Analgesia is the first volume to discuss a new concept of immune-neural interplays leading to pain or analgesia. It argues the classical view that pain and its control are restricted to the nervous system, offering a comprehensive overview of the emerging area of immune mechanisms in pain and its control.

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Glial cells surrounding the damaged nerves release cytokines and proinflammatory mediators that activate resident immune cells and recruit circulatory immune cells. Toll-like receptors on the glial cells play a crucial role in the pathogenesis of chronic pain. Animal models indicate an immune mechanism of neuropathic pain.

Neuroimmune mechanisms of pain: Basic science and ...
Overview: This translational research program addresses pathophysiological processes related to neuropathic pain and the potential confluence of chronic pain, autoimmunity, infectious diseases, and their intersections in human patients. Chronic neuropathic pain can affect any part of the body and can occur due to a variety of insults, infections, autoimmune or metabolic disorders (e.g., diabetic peripheral neuropathy).

Mechanisms of Pain and Immune Processes - Andrew Mannes
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Abstract. The role of central glial cells in the mechanisms underlying pain has been intensively studied in the last two decades. Most studies on glia and pain focused on the potential detrimental role of glial cells following noxious stimulus/insults manifested as an "activation" or a "reactive" state (increase in glial marker expression and production of proinflammatory/nociceptive molecules).

Nonneuronal central mechanisms of pain: glia and immune ...
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Immune Mechanisms of Pain and Analgesia is the first volume to discuss a new concept of immune-neural interplays leading to pain or analgesia. It argues the classical view that pain and its control are restricted to the nervous system, offering a comprehensive overview of the emerging area of immune mechanisms in pain and its control. It challenges the traditional view that pain sensation or suppression is attributed exclusively to the nervous system and presents a critical analysis of this new concept. The book is written by an internationally recognized group of researchers and discusses complex and controversial issues such as cytokines and their pain-exacerbating but also analgesic effects, the production of opioids by immune cells, peripheral analgesic and anti-inflammatory actions of opioids, immunomodulatory effects of opiates, and immunosuppressive effects of pain.

This volume represents edited material that was presented at a conference on brainstem modulation of spinal nociception held in Beaune, France during July, 1987. Pain Modulation, Volume 77 in the series Progress in Brain Research reviews, analyses and suggests new research strategies on several relevant topics including: the endogenous opioid peptides; sites of action of opiates; the role of biogenic animes and non-opioid peptides in analgesia; dorsal horn circuitry; behavioural factors in the activation of pain modulating networks and clinical studies of nociceptive modulation.

"International classification of diseases-11 (ICD-11) classifies chronic primary pain conditions like temporomandibular pain disorders and fibromyalgia syndrome under Chapter 21 ("symptoms, signs or clinical findings, not elsewhere classified"). such conditions lack well-defined etiology and diagnosis. Our understanding of chronic primary pain conditions is incomplete, and these conditions are not just limited to anomalies in sensory perception of pain but involve multiple systems. The immune system is one of those contributors to the pathophysiology of chronic primary pain. This thesis explores the role of the immune system in pain modulation. Specifically, we will focus on two chronic primary pain conditions: temporomandibular disorder (TMD) and fibromyalgia syndrome (FMS). Chapter I would summarize the literature on the general involvement of the immune system in enhancing and resolving pain, and how immune cells and neurons interact due to neuro-immune molecular overlap. Chapter II will describe my first research project representing an important example of reverse translational research in the area of pain genetics and immunology. Here, we found an association between an inflammatory mediator, epiregulin (EREG), and chronic TMD through statistical genetics approaches. TMD, a major cause of nondental pain in the orofacial region, is characterized by craniofacial pain involving the joint, masticatory muscles, or muscle innervations of the head and neck. We found that loss of function EREG genetic variants are associated with chronic TMD and chronic pain intensity. Next, we found that the same genetic variants are analgesic during the acute stages of pain development. We then validated these associations in the large independent cohort. Finally, we were able to confirm this dichotomous role of EREG in pain through animal pain models. Chapter III deciphers the role of the immune response in another chronic musculoskeletal pain condition-fibromyalgia syndrome (FMS). FMS, a common rheumatic disease, is characterized by chronic widespread pain, fatigue, and, sleep and cognitive difficulties. Pathogenesis of this syndrome remains elusive leading to a lack of objective diagnosis and specific treatment. Although the immune system's involvement in FMS is irrefutable, the specifics are yet to be deciphered. Furthermore, numerous studies have described the presence of small fiber neuropathy in FMS patients, but the mechanism of development of this neuropathy is unknown. In chapter III, we investigate peripheral blood mononuclear cells (PBMCs) through flow cytometry and differential gene expression in a case-control manner. We found that the FMS cases have fewer circulating natural killer (NK) cells. Furthermore, these cells were activated and exhausted in FMS patients. Co-culturing these cells with HLA-/- cell line (an activation stimulus for NK cells) showed that the NK cells from FMS patients are hyperactive compare to controls. Lastly, skin biopsies from an independent cohort showed increased expression of ULBP (NK activation ligand) and recruitment of NK cells on the peripheral nerves of the patients. In summary, this thesis advances our current understanding of the immune system's involvement in chronic primary pain conditions. Firstly, it demonstrates the dichotomies role of EREG in pain development being protective against acute pain but contributing to chronic pain. Secondly, we found the contribution of NK cells to FMS through its association with peripheral nerves in FMS. Both of these findings are novel steppingstones on our understanding of the pathophysiology of chronic pain and have therapeutics implementations in the treatment of chronic primary pain conditions"--

The Janeway's Immunobiology CD-ROM, Immunobiology Interactive, is included with each book, and can be purchased separately. It contains animations and videos with voiceover narration, as well as the figures from the text for presentation purposes.

Biomarkers have potential utility in the treatment of pain as diagnostics and for quantification of drug efficacy and safety. A qualified biomarker will capture overlapping disease mechanisms and will be responsive to treatment. The necessity for these strict requirements renders it difficult to discover new biomarkers, particularly one that is reliable, practical and non-invasive, and simple for routine utilisation. This thesis demonstrates that two approaches may be useful to overcome these challenges: bottom-up and top-down biomarker discovery and development. Current animal models of neuropathic pain are inadequate to develop biomarkers as they only cover 'no pain' and 'high pain': not the heterogeneity that exists between these extremes. Therefore, a novel rat model of graded neuropathic pain was developed by advancing the existing chronic constriction injury model. Sciatic nerve and subcutaneous chromic gut sutures were varied, resulting in 'dose-dependent' behavioural allodynia. Allodynia was correlated with microglial activation marker expression in the ipsilateral lumbar dorsal horn of the spinal cord, suggesting that changes in behaviour are associated with disease mechanisms. A literature review of the pathophysiological mechanisms of pain, filtered by the criterion for accessible biomarkers, revealed that the peripheral immune system was the ideal target for the bottom-up approach. As such, the graded model was then used to explore peripheral immune mechanisms in order to begin the process of construct validation of potential neuropathic pain biomarkers. It was demonstrated that peripheral immune cells significantly contribute to chronic constriction injury-induced allodynia, as adoptive transfer of splenocytes or peripheral blood mononuclear cells from high pain donors to low pain recipients potentiates allodynia. Intrathecal transfer of high pain immune cells to low pain recipients potentiated allodynia, confirming that infiltrating immune cells are not passive bystanders, but actively contribute to nociceptive hypersensitivity in the lumbar spinal cord. The graded transcriptome of dorsal horn of the ipsilateral lumbar spinal cord was compared with that in the blood, identifying chemokines and transcription factors as potential blood-borne biomarkers of neuropathic pain. The top-down approach explored the utility of saccadic eye movements as an objective, functional biomarker of sedation, an adverse effect associated with opioid treatment of pain. This study compared the interaction between sleep deprivation and opioids on opioid-naïve with opioid-tolerant participants. The naive-participant study evaluated the effects of sleep deprivation alone, morphine alone and the combination; the tolerant-participant study compared day-to-day effects of alternate-daily-dosed buprenorphine and the combination of buprenorphine on the dosing day with sleep deprivation. Psychomotor impairment was measured using saccadic eye movements, other oculomotor measures and an alertness visual analogue scale (VAS). Saccadic eye movements demonstrated an additive interaction between acute opioids and sleep deprivation, however the nature of the interaction between chronic buprenorphine and sleep deprivation remained unclear. This study revealed greater saccadic eye movement, but not VAS impairment in tolerant versus naive participants, suggesting that chronically dosed patients may not become tolerant to the sedative effects of opioids. These findings open up a number of new opportunities for pain biomarker development within the peripheral immune system, identify potential pain biomarker candidates, as well as further validating saccadic eye movement analysis as a biomarker of sedation. This thesis highlights that bottom-up and top-down approaches are appropriate methods for biomarker discovery and development.

Several new developments in the field of neuroimmunology with focus on the brain-to-immune system communication have been the incentive for this PIR volume. It covers topics such as brain-immune interactions, the impact of stress on the immune response, pain and immunosuppression, the modulation of inflammation and pain by the sympathetic nervous system, consequences of nerve injury for the immune system, neuronal mechanisms of immune cell recruitment, and the modulation of the immune response by corticotropin-releasing hormone or adenosine. The authors are a unique group of scientists who are all interested in brain-to-immune interactions; however, each from a different perspective. The volume will serve both neurobiologists and immunologists to understand the influence of the central nervous system on peripheral inflammation. Many aspects of this book will also be stimulating for researchers in the pain field.

Within the past few years, it has become recognized that the immune system communicates to the brain. Substances released from activated immune cells (cytokines) stimulate peripheral nerves, thereby signaling the brain and spinal cord that infection/inflammation has occurred. Additionally, peripheral infection/inflammation leads to de novo synthesis and release of cytokines within the brain and spinal cord. Thus, cytokines effect neural activation both peripherally and centrally. Through this communication pathway, cytokines such as interleukin-1, interleukin-6 and tumor necrosis factor markedly alter brain function, physiology and behavior. One important but underrecognized aspect of this communication is the dramatic impact that immune activation has on pain modulation. The purpose of this book is to examine, for the first time, immune-to-brain communication from the viewpoint of its effect on pain processing. It is aimed both at the basic scientist and health care providers, in order to clarify the major role that substances released by immune cells play in pain modulation. This book contains chapters contributed by all of the major laboratories focused on understanding how cytokines modulate pain. These chapters provide a unique vantage point from which to examine this question, as the summarized work ranges from evolutionary approaches across diverse species, to the basics of the immune response, to the effect of cytokines on peripheral and central nervous system sites, to therapeutic potential in humans.

This volume addresses neuronal pain mechanisms at the peripheral, spinal and supraspinal level which are thought to significantly contribute to pain and which may be the basis for the development of new treatment principles. Chapters on nociceptive mechanisms in the peripheral nociceptive system address the concept of hyperalgesic priming, the role of voltage-gated sodium channels in different inflammatory and neuropathic pain states, the hyperalgesic effects of NGF in different tissues and in inflammatory and neuropathic pain states, and the contribution of proteinase activated receptors (PAR) to the development of pain in several chronic pain conditions. Chapters on nociceptive mechanisms in the spinal cord address the particular role of NO and of glial cell activation in the generation and maintenance of inflammatory and neuropathic pain and it discusses the potential role of local inhibitory interneurons, of the endogenous endocannabinoid system and the importance of non-neuronal immune mechanisms in opioid signaling in the control of pain. Furthermore, it is presented how spinal mechanisms contribute to the expression of peripheral inflammation.

The Oxford Handbook of the Neurobiology of Pain represents a state of the art overview of the rapidly developing field of pain research. As populations age, the number of people in pain is growing dramatically, with half the population living with pain. The opioid crisis has highlighted this problem. The present volume is thus very timely, providing expert overviews of many complex topics in pain research that are likely to be of interest not just to pain researchers, but also to pain clinicians who are seeking new therapeutic opportunities to develop analgesics. Many of the topics covered are of interest to neuroscientists, as pain is one of the most amenable sensations for mechanistic dissection. The present volume covers all aspects of the topic, from a history of pain through invertebrate model systems to the human genetics of pain and functional imaging. Chapters include the role of ion channels, the opioid system, the immune and sympathetic systems, as well as the mechanisms that transform acute to chronic pain. Migraine and the interplay between sleep and pain are also discussed. New technology in the form of transgenic animals, chemogenetics, optogenetics, and proteomic analyses are providing significant advances in our research and are covered as well. Demystifying pain through an understanding of its fundamental biology, as outlined in this volume, is the most direct route to ameliorating this vast human problem.

To determine if stress exerts its effect on pain via GC-GR activity, mice were treated with a GR antagonist prior to stress. Indeed, GR blockade attenuated stress effects on pain-like behavior after nerve injury. Importantly, besides perceived stressors, there are multiple stimuli that induce GC release, including bodily injury, illness, and exercise. Hence, in broad contexts, exposure to GCs may impact pain. To determine if exposure to elevated GCs is sufficient to produce pain enhancement, mice were treated systemically with corticosterone (CORT) prior to nerve injury (in lieu of stress). Like the stressor, this procedure also increased allodynia in mice. Together, these data indicate that stress-induced potentiation of allodynia is at least partially mediated by GR.