FIBROMYALGIA IN INFLAMMATORY AND ENDOCRINE DISORDERS DAVID S. HALLEGUA pdf

1: Dr. David Hallegua, MD â€“ Beverly Hills, CA | Rheumatology

Fibromyalgia in Children (David D. Sherry) â€¢ Fibromyalgia in Inflammatory and Endocrine Disorders (David S. Hallegua) â€¢ Chronic Fatigue Syndrome (Atul Deodhar) â€¢ The Functional Bowel Disorder Spectrum (Lucinda A.

Advanced Search Stress is now recognized as a significant contributory factor in the cause and progression of many diseases 1 â€” 3. The sympathetic nervous system SNS and the hypothalamo-pituitary-adrenocortical HPA axis, the principal pathways that respond to stress, exert tonic inhibitory control over the immune system through multiple coordinated networks involving glucocorticoids, catecholamines, neuropeptides, and cytokines 4 â€” 6. Stress can result in a resetting of immune responses with consequent impaired ability of the organism to mount an effective defense after onset of infection or chronic inflammatory disease. The exacerbatory effects of stress on many inflammatory diseases are well documented, although the evidence is rather more anecdotal than scientific. Paradoxically, protective effects of stress on inflammation have been reported 7 , 8. A major challenge in the field of psychoneuroimmunology lies in defining the highly complex interactions among the SNS, HPA axis, and immune system to elucidate the selective effects of stress on disease processes. One very important aspect of this research is establishing the relationship between the effects of early life stress and predisposition to disease in adulthood. The group of Neumann and colleagues 9 , 10 in Regensburg, Germany, has previously shown that in experimentally induced colitis, a rodent model of inflammatory bowel disease, mice subjected to chronic over-crowding and social defeat stress had increased severity of inflammation. This team has now developed a sophisticated paradigm of repeated maternal separation MS stress in neonatal mice, followed by chronic subordinate colony CSC stress in adulthood The paper, published in this issue of Endocrinology, contains two key observations: This has the potential to create a systemic proinflammatory milieu that may underlie the increased severity of disease. Negative experiences in childhood can disrupt behavioral and physiological adaptive responses to subsequent acute stressors later in life and may increase the risk of developing inflammatory disorders The paper by Veenema et al. The authors conclude that adverse early life experiences may make individuals more vulnerable to chronic stress later in life and consequently increase the risk of developing chronic inflammatory disease. But not all stressors elicit a negative response. Clearly it is important that children can mount a healthy, robust response to the many stressors encountered during their development, and it would be most undesirable and evolutionarily highly unlikely if all of these responses to acute stressors were to predispose toward serious illnesses in adulthood. One of the major questions arising from this field of research is, therefore, what defines a negative early childhood experience, compared with a healthy response to an environmental challenge, and once that distinction has been made, is there any way to intervene to mitigate or prevent long-term pathological consequences of a negative stress experience? Long-term alterations in neuroendocrine and cytokine responses have previously been reported subsequent to an acute stressor 13 , These changes may be associated with sensitization or desensitization of HPA axis activity, depending on whether the experimental paradigm is composed of homotypic or heterotypic stressors Induction of an acute stress response either neonatally 16 or in adulthood 8 , 17 can protect against the onset of inflammatory disease in mature rats, but protection is dependent on the timing of the stressor relative to induction of inflammation and whether plasma cytokine and corticosterone changes after the stressor exhibit a pro- or antiinflammatory profile Thus, the type and severity, and also the timing of the stressor relative to the onset of disease, may be crucial in determining pathophysiological consequences. A negative stressor may be of the type and severity that precipitates an alteration in circulating glucocorticoids and cytokines with a bias toward a proinflammatory milieu. Therefore, it would be of great interest to measure HPA axis activity and cytokine profiles in MS mice throughout the d period of CSC to determine whether there is an alteration in the balance of corticosterone and cytokine secretion. If there is, at which point does it begin to favor a proinflammatory environment, i. Learning more about the nature of an early life stressor, in particular the neuroendocrine-immune fingerprint of compounds

secreted in response to specific stressors, may go some way toward predicting the long-term consequences of a stressful episode on inflammation. In a separate but related context, Straub and Besedovsky 19 proposed the hypothesis that a relatively minor and transient infection may perturb the major systems that maintain homeostasis such that immune, nervous, and endocrine coordinates are reset at a level that can sensitize susceptible subjects to future immune challenge and predispose to chronic inflammatory diseases. Given that infections can simulate stress by activating the HPA axis and SNS as well as the immune system 20 , 21 , this hypothesis may be extended to fit stressful events in early life, with similar consequences for disease onset and progression. Identification of children subjected to severe stressful episodes, in conjunction with other information such as hereditary risk factors for conditions such as arthritis or asthma, would enable them to be monitored later in life for early markers of inflammation, offering the scenario of therapeutic intervention at the earliest opportunity. Although not measured by Veenema et al. In another study, rats subjected to MS exhibited altered responses to social defeat stress in adulthood along with alterations in central levels of serotonin Anxiety and depression have been associated with major dysfunctions in serotonergic and catecholaminergic pathways within the central nervous system. Evidence has emerged that manipulation of these neurotransmitters within the brain can have profound effects on the development of inflammation in a rat model of arthritis 23 and in patients with rheumatoid arthritis Therefore, it would be of some interest to measure changes in neurotransmitters within the brains of MS-CSC mice to determine any correlation between neurotransmitter levels and subsequent severity of colitis. Studies of this nature on coordinated neuroendocrine and neurotransmitter responses to early life stress may lead to a better understanding of how central neurotransmitters can influence peripheral inflammatory processes, a field of research that barely existed 10 yr ago. One further important issue highlighted by Veenema et al. Elevated corticotrophin-releasing factor mRNA in the paraventricular nucleus of the hypothalamus in MS-CSC mice is consistent with reports of increased corticotrophin-releasing factor production in posttraumatic stress disorder Research into these syndromes in humans has been limited by the lack of a reliable animal model. The MS-CSC mouse model of hypocortisolemia may serve as an appropriate model to study the complex neuroendocrinology underlying these hypocortisolemic disorders and may permit testing of therapeutic interventions such as glucocorticoid replacement or receptor antagonists to normalize cortisol secretion. Finally, one major difficulty in relating early life stress to risk of disease in humans is that it is very difficult to identify and quantify the nature and intensity of the stressor in childhood other than in extreme cases and draw any meaningful etiological relationships with probability of illness in adulthood. Most data relating adult illness to early childhood events derive from retrospective studies, and predictive data from longitudinal programs following children into adulthood are rare. The usefulness of animal studies in this area is that they can provide us with well-defined models of early life stress, which can be extrapolated to guide our design of testable hypotheses and protocols for prospective stress studies in humans.

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2: Table of contents for Fibromyalgia and other central pain syndromes

Fibromyalgia and Other Central Pain Syndromes This volume is the first comprehensive text devoted to fibromyalgia and other centrally mediated chronic pain syndromes. Leading experts examine the latest research findings on these syndromes and present evidence-based reviews of current controversies.

Menopausal status and biological variables DHEA-S levels did not differ significantly according to menopausal status. Regarding cytokine and chemokine differences, some statistically significant differences were noted according to menopausal status. Significant and positive correlations were found between IL-8 and all of the psychological measures for postmenopausal women only: Discussion The current study aimed to characterize basal cytokine profiles in pre- and postmenopausal women with FM through examination of serum concentrations of cytokines, chemokines, and inflammation-regulating hormones. Further, we sought to provide additional context to these findings through the examination of other factors that are likely to be contributory to inflammatory processes, namely menopausal status and psychological distress. Our findings revealed interesting differences in inflammatory processes when the menopausal status and psychological status of participants were taken into account. Premenopausal and postmenopausal participants reported similar levels of depressive symptoms and pain anxiety. Although premenopausal participants did report significantly higher levels of pain catastrophizing, the average scores on the PCS for these participants were similar to previous samples of individuals with FM. Our results suggested that IL-8 was correlated with depressive symptoms, pain catastrophizing, and pain-related anxiety for postmenopausal women but not for premenopausal women. These findings suggest a possible contributing role for IL-8 in psychological functioning in chronic pain, which is contrary to some previous findings. It is particularly important to acknowledge the relative scarcity of attention paid to the role of menopause and other age-related factors in FM, despite evidence that the prevalence of FM increases as individuals progress further into old age, 30 and that there appear to be changes in brain structure and function that have age-dependent implications for individuals with FM. While these findings stand at odds with the broader literature, largely comprising studies describing healthy samples, 10 , 12 they support previous findings for FM. A study by Bazzichi et al 18 reported no differences in IL-6 and IL-8 plasma levels between those with FM and a diagnosed psychiatric disorder like depression and anxiety and those with FM and no psychiatric diagnoses. The reason for this divergence is unclear, though these findings may suggest a unique immune characteristic of FM. Our results do not allow for inferences regarding the specificity of this effect in FM versus a general effect of the experience of chronic pain, but the relationship between IL-6 and depression in other chronic pain disorders warrants further study in the future. This null finding may have been attributable to a small sample size, however, which will be discussed later. Additional research of this relationship is therefore needed in larger samples. It is notable that DHEA-S levels did not correlate with pain intensity or cytokine or chemokine levels in our study, which were somewhat unexpected findings. However, it is conceivable that DHEA-S plays a more dynamic role in the regulation of inflammatory responses, as to an acute stressor, but does not have a detectable relationship in the basal state with these immune factors. It is also possible that the regulatory effects of DHEA-S are too complex to be adequately characterized using correlations. Previous evidence suggests that the effects of DHEA-S may be best described in their relation to cortisol 61 and testosterone levels, 62 both of which have implications for immune competence and markers of inflammation like IL-6 and IL-8, and may be affected by other underlying medical factors like hypothyroidism 63 or low estrogen. The current study was intended as a preliminary attempt to identify salient contributing factors to inflammatory processes in FM, thereby highlighting the need to account for these factors in future studies. Limitations A primary limitation of the current study is the size of our sample. As a result, the current study should be considered a pilot study that was exploratory in nature and was conceived as an attempt to detect novel associations between the study variables that could be more stringently examined in future studies. The limited

size of our sample means that our findings require replication in larger samples to ensure that they are reliable. Our results are similarly limited due to the unbalanced size of the groups; conclusions regarding our findings should be made with caution and with the understanding that there was a comparatively larger number of postmenopausal women, which may explain some degree of the difference in significant relationships between variables in the pre- and postmenopausal groups. Further, our presented findings do not provide adjusted significance values that account for the multiple comparisons posed by our analyses. We deemed this approach necessary, given the small size of our sample and our intention to identify novel effects, but the lack of adjustment to the presented probability values is nevertheless a significant limitation. Consequently, our findings should be interpreted with this caution in mind. Additionally, some recent evidence suggests that measuring reactivity, rather than basal levels, of IL-6 may constitute a more reliable measurement approach in individuals with FM. This approach relies on diagnosis from previous medical providers, which may be variable with regard to adherence to a strict set of diagnostic criteria for FM. Though we have no reason to suspect inaccurate FM diagnoses in our study sample, our results should be interpreted with this caution in mind. Similarly, the current study did not specifically target individuals with clinical mood disorders. It is possible that specific examination of individuals with diagnosed mood disorders such as major depressive disorder may demonstrate stronger relationships between distress and cytokine production if these relationships are examined according to sex- and age-specific factors, as in the current study. Due to differences in the processing of sampling assays compared with previous studies, we were also unable to provide comparisons with cytokine levels in healthy pre-and postmenopausal women, which could serve to provide additional context to our findings and would provide a clearer illustration of the processes of FM. Although FM is predominantly found in women, comparison of our findings with similarly aged men, both with and without FM, would also be beneficial to delineate the unique aspects of sex, aging, and inflammatory processes in FM. As a result, we urge attention to these issues in future studies. Despite these limitations, we propose that our findings regarding differential implications of psychological and menopausal status for IL-6, IL-8, and IL activity should be interpreted as preliminary findings that warrant further examination with additional sampling in a larger study in the future. Conclusion The current study sought to characterize differences in basal proinflammatory cytokine profiles in women with FM. While data are preliminary, the current study is among the first to present serum cytokine levels in women with FM, and the first to do so with specific attention paid to age-related and psychological factors. Our findings regarding the role of menopause are particularly important, as they highlight the potential importance of aging factors in understanding the development of FM in women. However, replication of our results in larger samples of women with FM is necessary. Our findings highlight an avenue for future research that may provide additional utility in characterization of the complex etiology and clinical presentation of FM. The authors report no other conflicts of interest in this work. Fibromyalgia prevalence, somatic symptom reporting, and the dimensionality of polysymptomatic distress: Arthritis Care Res Hoboken ;65 5: The American College of Rheumatology preliminary diagnostic criteria for fibromyalgia and measurement of symptom severity. Arthritis Care Res Hoboken ;62 5: Fibromyalgia criteria and severity scales for clinical and epidemiological studies: What is fibromyalgia, how is it diagnosed and what does it really mean? Arthritis Care Res Hoboken ;66 7: The contribution of pain, reported sleep quality, and depressive symptoms to fatigue in fibromyalgia. Genetic influences on the dynamics of pain and affect in fibromyalgia. The genetics of fibromyalgia syndrome. Cytokines play an aetiopathogenetic role in fibromyalgia: Rheumatology Oxford ;40 7: The interaction between autoimmune diseases and fibromyalgia: Expert Rev Clin Immunol. A meta-analysis of cytokines in major depression. Neuroendocrine immunology of fibromyalgia. Ann N Y Acad Sci. Cytokines and depression in cases with fibromyalgia. Inflammation and its discontents: Systematic review with meta-analysis: Circulating cytokine levels compared to pain in patients with fibromyalgia: Cytokine patterns in fibromyalgia and their correlation with clinical manifestations. The endotoxin-induced increase of cytokines is followed by an increase of cortisol relative to dehydroepiandrosterone DHEA in healthy male subjects.

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Dysregulation of IL production with aging: One year outcome of preadolescents with fibromyalgia. Psychiatric disorders in patients with fibromyalgia:

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3: ECU Libraries Catalog

Fibromyalgia in inflammatory and endocrine disorders / David S. Hallegua --Chronic fatigue syndrome / Atul Deodhar --The functional bowel disorder spectrum / Lucinda A. Harris and Lin Chang --Genitourinary associations with fibromyalgia / Daniel J. Wallace and Swamy Venuturupalli --Chronic low back pain / Thorsten Giesecke and Michael E.

Classification[edit] Fibromyalgia is classed as a disorder of pain processing due to abnormalities in how pain signals are processed in the central nervous system. Although mental disorders and some physical disorders commonly are co-morbid with fibromyalgia â€" especially anxiety, depression, irritable bowel syndrome , and chronic fatigue syndrome â€" the ICD states that these should be diagnosed separately. A review divides individuals with fibromyalgia into four groups as well as "mixed types": Fibromyalgia is often associated with anxiety and depressive symptoms. Although fibromyalgia is classified based on the presence of chronic widespread pain, pain may also be localized in areas such as the shoulders , neck , low back , hips , or other areas. Many sufferers also experience varying degrees of myofascial pain and have high rates of comorbid temporomandibular joint dysfunction. However, several hypotheses have been developed including "central sensitization". In these vulnerable individuals, psychological stress or illness can cause abnormalities in inflammatory and stress pathways which regulate mood and pain. Eventually, a sensitization and kindling effect occur in certain neurons leading to the establishment of fibromyalgia and sometimes a mood disorder. In simple terms, it can be described as the volume of the neurons being set too high and this hyper-excitability of pain processing pathways and under-activity of inhibitory pain pathways in the brain results in the affected individual experiencing pain. Some neurochemical abnormalities that occur in fibromyalgia also regulate mood, sleep, and energy, thus explaining why mood, sleep, and fatigue problems are commonly co-morbid with fibromyalgia. As the hippocampus plays crucial roles in maintenance of cognitive functions, sleep regulation, and pain perception, it was suggested that metabolic dysfunction of the hippocampus may be implicated in the appearance of these symptoms. Accordingly, a study that employed functional magnetic resonance imaging to evaluate brain responses to experimental pain among people with fibromyalgia found that depressive symptoms were associated with the magnitude of clinically induced pain response specifically in areas of the brain that participate in affective pain processing, but not in areas involved in sensory processing which indicates that the amplification of the sensory dimension of pain in fibromyalgia occurs independently of mood or emotional processes. There is also some data that suggests altered dopaminergic and noradrenergic signaling in fibromyalgia. One study found fibromyalgia patients exhibited higher plasma cortisol , more extreme peaks and troughs, and higher rates of dexamethasone non suppression. However, other studies have only found correlations between a higher Cortisol awakening response and pain, and not any other abnormalities in cortisol. Heart rate variabilities observed were different in males and females. Restorative sleep was correlated with improvement in pain related symptoms. Altered connectivity and decreased grey matter of the default mode network , [56] the insula , and executive attention network have been found in fibromyalgia. Increased levels of glutamate and glutamine have been observed in the amygdala, parts of the prefrontal cortex , the posterior cingulate cortex , and the insula, correlating with pain levels in FM. Decreased GABA has been observed in the anterior insular in fibromyalgia. However, neuroimaging studies, in particular neurochemical imaging studies, are limited by methodology and interpretation. One study found increased levels of pro-inflammatory cytokines in fibromyalgia, which may increase sensitivity to pain, and contribute to mood problems. There is no single test that can fully diagnose fibromyalgia and there is debate over what should be considered essential diagnostic criteria and whether an objective diagnosis is possible. In most cases, people with fibromyalgia symptoms may also have laboratory test results that appear normal and many of their symptoms may mimic those of other rheumatic conditions such as arthritis or osteoporosis. The most widely accepted set of classification criteria for research purposes was elaborated in by the Multicenter Criteria Committee of the American College of Rheumatology. These criteria, which are

known informally as "the ACR ", define fibromyalgia according to the presence of the following criteria: A history of widespread pain lasting more than three months â€” affecting all four quadrants of the body, i. Tender points â€” there are 18 designated possible tender points although a person with the disorder may feel pain in other areas as well. Diagnosis is no longer based on the number of tender points. The number of tender points that may be active at any one time may vary with time and circumstance. The WPI counts up to 19 general body areas [a] in which the person has experienced pain in the preceding two weeks. The revised criteria for diagnosis are:

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4: Is There a Link Between IBS, Fibromyalgia, and Celiac Disease? - MPR

This volume is the first comprehensive text devoted to fibromyalgia and other centrally mediated chronic pain syndromes. Leading experts examine the latest research findings on these syndromes and present evidence-based reviews of current controversies.

Preface Fibromyalgia is a syndrome characterized by pain, fatigue, depression, sleep disturbances, anxiety and many other problematic symptoms. According to statistics, it afflicts approximately percent of the human population and is more common in women than men. The Medical community seems to see the syndrome in a somewhat narrow way. They usually recognize that the syndrome often begins after a stressful moment in life e. Because of the common idea that fibromyalgia has no clear physiological causes, no targeted treatments exist, and the patients are often treated by various drugs such as antidepressants and anticonvulsants. The examples used in this article are thyroid hormones, CoQ10, and near-infrared. Fibromyalgia and thyroid hormones After becoming interested about the importance of thyroid function for health, I noticed that Dr. Lowe had made important research showing that fibromyalgia and clinical hypothyroidism have dozens of biochemical and physiological similarities. Lowe and Yellin have written an excellent review article on this issue. Similar extreme decreases in metabolic rate have also been noted in hypothyroidism and PCOS. Cordero has made important discoveries about the biochemical state of fibromyalgia patients. Also, the intracellular levels of ubiquinone coenzyme Q10 or CoQ10 were dramatically lower than in the healthy subjects. The interesting fact is that supplementation of CoQ10 reversed the above-mentioned biochemical abnormalities completely. The depression score has also been noted to decrease significantly from 22 to 6, while in the placebo group, the score increased to These same biomarkers are also importantly related to aging, so it could be said that fibromyalgia syndrome is functionally somewhat analogous to getting old. Instead, they are often higher than in healthy subjects. But despite these increases in plasma levels, it seems that the CoQ10 levels inside the cells are generally decreased in fibromyalgia patients, and the CoQ10 supplementation seems to generally benefit the patients. This results in chronic fatiguing illness that may be alleviated by natural mitochondrial enchancers such as CoQ Low levels of CoQ10 in cells has been noted in fibromyalgia, chronic fatigue syndrome, and periodontitis. There indeed is some evidence showing alleviation of migraine, periodontitis, and depression with a CoQ10 supplement. Some of these factors have also been shown to deplete CoQ This effect seems to be beneficial for many diseases. So far it has been demonstrated that near-infrared might be one of the most potent medical treatments for some chronic diseases. In two Brazilian studies, low level laser treatment LLLT with near-infrared was associated with a huge improvement in hypothyroid patients. Sunlight, incandescent lamps, halogen lamps, and heat lamps also provide near-infrared, and theoretically they should have similar biological effects. In many animal trials, LED lamps have been as useful as lasers. There are also some trials showing large biological effects with halogen-based Bioptron lamps. Some of the patients also received CoQ10, which probably accounts for some of the benefits, but I think that the improved intestinal function and decreased inflammation and oxidation must have been important factors as well. Antibiotics have also been shown to alleviate some of the fibromyalgia symptoms. None of these patients had celiac disease, but many of them had other symptoms of digestive system heartburn, constipation, irritable bowel. Disturbed energy-metabolism has also been noted in chronic fatigue syndrome CFS , but some of the markers seem to be worse in fibromyalgia. Patients with fibromyalgia and CFS seem to have some disturbances with AMPK phosphorylation in various cell types connective tissue, muscle cells, white blood cells. AMPK is quite important for cellular energy metabolism, so these problems might be importantly related to the metabolic problems of these diseases. In animal experiments, endotoxin LPS has been shown to cause these problems related to mitochondrial biogenesis. Mark Starr, the author of Hypothyroidism Type 2: Thus, it seems likely that besides fibromyalgia, there are many other painful conditions that involve disturbed energy-metabolism. Free fatty acids and energy metabolism: Young

fibromyalgia patients in Japan seem to have extremely high levels of free fatty acids NEFA , and it would be interesting to see if this applies to most of the adult patients as well. Because NEFAs seem to be able to inhibit some aspects of metabolism Randle cycle, thyroid hormone receptors, insulin signalling , they might be one factor contributing to the syndrome. Insulin resistance seems to be a potential factor causing disturbances of metabolism in cells. Even the descendants of diabetic patients have decreased muscle ATP production, even if there are no signs of inflammation. Diabetic patients have low hepatic ATP production. Amino acids glycine and histidine seem to have various beneficial health effects, at least in animal studies. They can improve NEFA oxidation and insulin resistance. Their consumption seems to decrease NEFA levels, inflammation, lipid peroxidation and other harms related to junk food and endotoxemia. The combination of glycine and cysteine has led to remarkable improvements in the metabolism of elderly human subjects. In animal studies, it has many protective effects on mitochondria, and in human studies, it has shown to be beneficial for patients suffering from chemotherapy, diabetes and heart failure. In adolescent Japanese fibromyalgia patients ubiquinol reduced form of CoQ10 lowered their cholesterol levels significantly. Also, thyroid hormones and near-infrared have been shown to decrease cholesterol levels. Fibromyalgia and CFS patients have higher levels of oxidative stress when compared to healthy controls. Oxidative stress is a common marker of eg. Women with fibromyalgia have over two-fold leptin levels compared to the healthy controls. Chronic inflammation can increase leptin levels. A recent study investigated the associations between the expression of some genes and fatigue diseases. In fibromyalgia patients, decreased blood flow has been noted in some parts of brain, compared to healthy subjects thalamus, nucleus caudatus, cerebral cortex. In CFS patients, decreased blood perfusion has been noted in their brainstem compared to healthy subjects. In one study, metabolism was also lower in brainstem and right mediofrontal cortex when compared to healthy subjects. Some proof of neuroinflammation has also been noted in limbic system, brainstem and thalamus. According to one study, women suffering from fibromyalgia have some evidence of neuropathy in their eyes. The researchers discussed that mycotoxins might be able to cause mitochondrial dysfunction, leading to disease. Therefore, it might be irrelevant. Even though it has many beneficial effects, which might be related to the powerful inhibition of NLRP3 inflammasome, it also seems to have some harmful effects on energy metabolism. Low-dose naltrexone is another popular drug for fibromyalgia and CFS. I have heard some good anecdotes about it, but have no more information. Treating endotoxemia and inflammation with an anti-inflammatory diet and supplements glycine, taurine might also be very important, because inflammation might be an important factor causing the energy-metabolism disturbances in these patients.

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5: Fibromyalgia - Wikipedia

Patients with fibromyalgia (FM) frequently have gastrointestinal symptoms and signs. This article critically reviews the available literature and concludes the following: evidence that inflammatory bowel disease is associated with FM is contradictory, but should be looked for in patients taking.

The workshop, organized by Dr. Stanley Pillemer and Dr. In his introductory remarks at the workshop, Dr. Investigators from diverse groups have been brought together to share perspectives, research advances, and research problems so that collective wisdom can be focused on particular areas of research. People with this syndrome may also experience sleep disturbances, morning stiffness, irritable bowel syndrome, anxiety, and other symptoms. Fibromyalgia affects 3 to 6 million Americans, and occurs primarily in women of childbearing age. However, children, the elderly, and men can also be affected. The Chronic Pain segment of the workshop was chaired by Dr. Researchers discussed their studies in this area outlining the current state-of-the-art and the relationship of the findings of pain studies to fibromyalgia. Casey, Professor of Neurology at the University of Michigan in Ann Arbor, said that fibromyalgia may be related to abnormalities of central pain processing, or processing of nociceptive nerve endings or pathways concerned with pain information in the central nervous system CNS. There are specialized nociceptors receptors for pain in skin and in muscle that are activated by mechanical stimuli. Diseases or injuries to the CNS can produce pain and can profoundly alter the perception of pain in animals and in humans. Casey mentioned that a PET positron emission tomography scan can measure regional blood flow changes in individuals experiencing pain. Ronald Dubner, Chairman of the Department of Oral, Cranial, Facial, and Biological Sciences, University of Maryland at Baltimore, said that persistent pain lasting for hours or days may lead to changes in the central nervous system and the peripheral nervous system, which consists of all the nerves that carry signals between the CNS and the rest of the body. He added that if the balance is altered between these two systems, this could lead to a further increase and spread of pain. Chemical changes may take place at the site of tissue damage and after nerve injury. There is increased activity of the nerves in the tissues of the body that causes changes plasticity in CNS functioning. It is this hyperactivity that may play a role in the amplification and the increased duration of pain. Bradley said that chronic pain is pain that persists despite medical or surgical interventions. Even after a year period, the majority of patients continue to have some pain and some symptoms. Bradley found that compared to healthy people, fibromyalgia patients have decreased cerebral brain blood flow and higher levels of substance P in their system. Substance P is a chemical involved in the transmission of pain signals via the nervous system. Jon Russell, Professor of Medicine and Clinical Immunology at the University of Texas Health Science Center at San Antonio, said that substance P levels are substantially elevated in spinal fluid of people with fibromyalgia compared to normal people. He found that in fibromyalgia patients, the pain threshold was much lower, stiffness was greater, physical function was more impaired, and anxiety and depression were greater. Wendy Sternberg, Professor in the Department of Psychology at Haverford College in Pennsylvania, found that female rodents, like their human counterparts, are more sensitive to certain painful stimuli than are males. She said that this sensitivity may be related to the estrogen cycle in females, since researchers have found that when estrogen levels are at their peak pain sensitivity is highest. Researchers have conducted treatment outcome studies looking at whether exercise training or combined biofeedback and exercise training or just biofeedback alone could increase self-efficacy in individuals with fibromyalgia. Keefe said that a new study, supported by NIAMS, will look at cognitive behavior in patients with fibromyalgia. He said that these include the psychological predisposition of patients, the methods of measuring pain thresholds, the lack of experimental controls, and the problems in measuring the entire experience. The Neuroendocrine segment was chaired by Dr. Neuroendocrinology refers to the study of the interactions between the nervous system and the endocrine hormonal system. Scientists discussed their studies pertaining to the stress system and its relationship to altered sleep, mood, pain perception, and

biological rhythms. The parallels between what is known about fibromyalgia and the implications for future research were mentioned. Every time you disturb this equilibrium, you disturb homeostasis the natural state of balance and cause stress. To give a little background, Dr. Chrousos said that the stress system consists of two components, a central nervous system component and a peripheral component. When it is activated, it helps us and then goes back to its normal state. Chrousos said that researchers at NIH have studied seasonal depression and found that people who suffer from this disorder have an underactive stress response. He added that patients with chronic fatigue syndrome and fibromyalgia also may belong in this group. Eve Van Cauter, Research Associate Professor in the Department of Medicine, Section of Endocrinology, at the University of Chicago in Illinois, demonstrated that sleep disturbances can go hand in hand with neuroendocrine disturbances. She emphasized that various hormones are secreted in rhythmic patterns and that single measurements of individual hormones have little value, because the body has different hormonal states of functioning at different times of the day. Van Cauter explained that the timing, duration, and quality of sleep are controlled by two major components of bodily functioning involved in sleep called the homeostatic and circadian components. The homeostatic component reflects the time that has elapsed since sleeping. The longer the time that has elapsed the greater is the tendency to sleep. On the other hand, the circadian component, which is related to the time of day, appears to be important in waking. With increasing age, the balance between the homeostatic and circadian components appears to shift. This may explain why a forty year old person who has had an unduly late night may be unable to sleep all day to make up the deficit, whereas a teenager may be able to do so. Van Cauter speculated that fibromyalgia also may be associated with an alteration in the balance between the homeostatic and circadian components of sleep. Subscribe Sleep is important in controlling the release of growth hormone. Waking suppresses the secretion of growth hormone but stimulates the release of cortisol. Fragmentation of sleep would tend to cause low levels of growth hormone and higher levels of cortisol. Van Cauter suggested that fibromyalgia patients may demonstrate hormonal profiles similar to those seen in people with fragmented sleep, but research is needed to test this. She described preliminary sleep studies showing that a drug called gamma hydroxybutyrate may stimulate slow wave deep, non-dreaming sleep. She found that the amount of growth hormone secreted at the beginning of the sleep period increased threefold, and there was a progressive increase in the amount of slow wave sleep, but only during the first 2 hours of sleep. Paul Plotsky, Professor in the Department of Psychiatry and Behavioral Sciences at Emory University in Atlanta, Georgia, said that for several months, studies were conducted in his laboratory on rat pups focusing on the effects of not handling newborn rat pups and separating them from their mothers. When compared to normal rats, deprived rats showed high degrees of fear and anxiety. Plotsky said that researchers can program an animal to have an overactive or underactive stress response to pain stimulation. He added that these changes seem to last the lifetime of the animal. She said that ovulation and menstrual cycles are initiated by the hypothalamic pituitary gonadal axis. The hypothalamus, a part of the brain, produces hormones that stimulate the pituitary gland, which lies under the hypothalamus, to release other hormones that affect the female gonads ovaries. She added that most women have a sufficient number of hormone pulses that cause them to ovulate when normal levels of progesterone are present. Goldstein, a Senior Investigator in the Clinical Neuroscience Branch of the National Institute of Mental Health, NIH, said that stress is neither a stimulus nor a response, but a condition where there is a discrepancy between the state that the organism is programmed to maintain and the state that is sensed. He discussed how the sympathetic nervous system a part of the nervous system that controls many of the involuntary activities of the glands, organs, and other parts of the body responds differently to particular stressors. Goldstein said that the closer you look at the sympathetic nervous system, the more complex it appears and the more difficult it is to precisely define it. Crofford said that a number of clinicians have observed that fibromyalgia can be precipitated by acute stress or psychological stress that might activate either the HPA axis or the sympathetic nervous system. Crofford discussed fibromyalgia studies at the National Institutes of Health where she collaborated with Drs. They found that patients with fibromyalgia had lower levels of cortisol a steroid stress

hormone , in their urine than did healthy people. She noted that cortisol is very important for all metabolic systems physical and chemical processes in the body. Fibromyalgia sufferers also showed increases in ACTH a hormone secreted by the pituitary gland secretion compared with healthy normal subjects when they were given corticotropin-releasing hormone a hormone that triggers ACTH secretion. The Sleep Disorders segment was chaired by Dr. Researchers discussed their studies in the following areas: He began by saying that a rapid increase in the knowledge of basic mechanisms during the past 5 to 10 years has revolutionized our understanding of sleep. McCarley said that we start out awake and then gradually progress to the deeper stages of sleep, which include slow wave, delta, restorative, and thenREM rapid eye movement. REM sleep is the phase that is associated with dreaming, and is promoted by cholinergic neurons nerve cells that produce a substance called acetylcholine. These neurons turn on before and during the onset of REM sleep. He said that as you go to sleep, your cholinergic neurons are quiet. Then they become active, and at a certain threshold REM sleep occurs. The cycle repeats itself all through the night. He outlined recent research that demonstrated that nerve cells in the hypothalamus may control sleeping and waking states. McCarley said that ongoing studies of the structure of the brain show exciting possibilities that these nerve cells may also coordinate other systems including those controlling stress responses and the endocrine glands. She said that there is much evidence that implicates interleukin-1 IL-1 in sleep regulation. IL-1 is a molecule that is produced by certain cells of the immune system and occurs naturally in the brain. Studies have shown that IL-1 induces excess sleep in rabbits, cats, rats, and monkeys. IL-1 antibodies, which block the action of IL-1, inhibit normal sleep and restorative sleep after sleep deprivation. Everson said that in sleep-deprived people psychiatric changes are marked and these include mood and dispositional changes, hallucinations, unusual body sensations, tremors, losses of equilibrium, slurred speech, and changes in brain waves. In addition, there are physiological changes such as a decrease in body temperature and in blood levels of cortisol. She said that sleep-deprivation is a risk factor in disease. Everson cited studies comparing sleep-deprived rats to rats that slept normally. She said that researchers demonstrated that sleep-deprived rats had a decline in blood levels of thyroid hormones that is consistent with central hypothyroidism in humans and experimental animals. She also found that the animals had depressed immune systems and bacteria appeared in their blood, indicating an increased susceptibility to infections. Evelyn Satinoff, Chair of the Psychology Department at the University of Delaware in Newark, discussed the importance of thermal or temperature factors on sleep. In young female rats, she demonstrated that sleep and body temperature are interdependent. However, when old rats that were no longer ovulating were compared to young female rats that had their ovaries removed, the animals without ovaries did not show a deterioration in their sleep or body temperature rhythms. The rhythms were actually more precise. Similar studies in males also excluded a role for sex hormones. Further investigation showed that the instability of temperature and sleep rhythms with aging occur independently of one another. Moldofsky said that fibromyalgia may be a model for sleep dysregulation. Moldofsky cited studies on normal healthy people who participated in a sleep study. At the completion of the talks, the chairmen summarized the results of the workshop.

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