Chronic Pain and Neuroplasticity (NP): The basics.
-NP is the new science that will win cases for you by explaining why the patient's multiple complaints are real and claim-related.
-This is the second in a multi-part series that will explain the science of neuroplasticity and how it relates to various aspects of claims, and how it puts big, scientific holes in the denial of many claims.

-NP refers to changes in nerve pathways and synapses (connections between nerve endings) which are due to injury or changes in behavior, environment and neural processes. NP explains how the brain changes throughout life.

-Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage. This common definition acknowledges that pain is a conscious experience involving interpretation of (painful) sensory input that is influenced by emotional, pathological, and cognitive factors, as well as previous pain experiences. Pain may be nociceptive, neuropathic, or mixed. Nociceptive pain can be described as normal pain. Neuropathic pain is pain that arises from a lesion, disease, or dysfunction in the nervous system and is maintained by a dysfunction in the nerve pathways or by abnormal signaling to the CNS (central nervous system). Chronic pain is the result of neuroplasticity gone bad!

-To understand pain, we must first understand normal pain pathways. Input or pain signals travel from neuron (cells of the nervous system) to neuron, and as the signal goes across the neuron to neuron connections (synapses), it is affected by a variety of neurotransmitters and neuromodulators. In normal pain signals, the pain is not modulated, inhibited, or amplified by these neurochemicals either on the incoming (presynaptic) or the receiving (postsynaptic) side of the synapses. Neural damage to either the CNS or PNS (peripheral nervous system) provokes maladaptive responses that amplify the pain signals, sensitization! The amplification of the signal eventually crescendos in a vast multiplication of the original signal. The remarkable thing about sensitization is that there need not be pathophysiologic damage to the neurons themselves! Multiple processes are involved, including CNS/PNS, neuron cell death, disinhibition of pain signals, altered gene expression, and abnormal cellular connectivity between neurons. Neuroimmune interactions also underlie

many of these mechanisms. Imaging studies have shown that sensitization conditions are actually associated with functional, structural and chemical changes in the nerves and brain! This multidimensional process warrants consideration as a chronic disease not only affecting sensory and emotional processing but also producing an altered brain state seen on imaging.

-The understanding of sensitization of pain from a peripheral site is a major scientific discovery of recent times that enhances clinical understanding of chronic pain. One of the mysteries of pain patients is what appears to be the new occurrence in the CNS of other painful conditions such as fibromyalgia, interstitial cystitis, TMJ, and irritable bowel syndrome. Glial cell (types of brain cells which are far more prevalent than neurons in the central nervous system) become activated and produce Neuroinflammation, which is clearly an underlying mechanism for the centralization of peripheral pain. The basic idea of central sensitization is that the features of the CNS change in ways that amplify and distort pain so that it no longer solely reflects the activity of the peripheral nerves. A hallmark of this is new areas of pain.

-The exact transition from acute to chronic pain is incompletely understood and clearly involves central sensitization. Our current definitions of chronic pain are: pain that lasts longer than expected, or pain greater than three months, or pain that persists after healing has occurred. Sensitization is often involved, which means the pain is magnified. Unlike acute pain which can usually be precise identified, patients with chronic pain often describe it as a more diffuse pain, and difficult to localize- describing it as vague and moving around. Over time, the pain becomes dissociated from its original cause. Since a family history of central sensitization is common, this implies genetic influences which may respond to environmental influences.

-Per the American Medical Association Guides to the Evaluation of Permanent Impairment, 5th Edition, it notes that peripheral and central sensitization do occur, and that the patient receives the pain is real.

-Per the American Medical Association Guides to the Evaluation of Permanent Impairment, 6th Edition, it notes that there is accumulating evidence that persistent pain should be considered a disease entity in its own right. Indeed, permanent changes in the responsiveness of both the peripheral and central nervous system can persist even after all tissue

healing has ensued; thus, persistent pain can become a self-perpetuating condition. The individual is often left with ongoing pain without identifiable signs of the original inciting disease process that initiated the pain. This results in a multitude of consequences that can lead to significant impairment for the individual affected, including physical impairment, mood dysfunction, and social disruption. Concerning sensitization, it may be localized or generalized, with most or all of the body affected. This is due to changes in the PNS or CNS, with resultant hyperalgesia (an exaggerated pain response from a usually painful stimulation) and/or allodynia (pain due to a non-noxious stimulus that does not normally provoke pain).

-From the DOWC (Div. Of Workers Comp-Colorado) , online edition of the Medical Treatment Guidelines:
CENTRAL SENSITIZATION: The experience of pain evoked by the excitation of neurons or nerve fibers that normally relay non-painful sensations to the spinal cord. This results when incoming pain signals act on a sensitized CNS. Experimental data suggest that pathways normally carrying pain signals themselves become overstimulated and/or fail to respond to inhibitory influences causing increased pain**.** An example is 'wind-up' which occurs when cells in the spinal cord increase their rate of discharge in response to repeated stimulation by pain receptors.

-American Academy of Pain Medicine (AAPM) 28th Annual Meeting: February 24, 2012. Patients with chronic low back pain (CLBP) have altered brain connectivity on functional magnetic resonance imaging compared with healthy controls, and experience temporary changes in this pattern when their pain is exacerbated, a new study reported by lead author Marco Loggia, PhD, from Harvard Medical School. "In this study we show, for the first time that clinical pain severity in CLBP patients appears to be encoded in brain connectivity patterns". Catherine Bushnell, PhD, president of the Canadian Pain Society and professor of anesthesia and neurology at McGill University, Montreal, Quebec, said, "This exciting study adds to the growing literature indicating that chronic pain alters the brain. Showing changes in connectivity between brain regions important for mood and cognitive function could help explain why pain patients frequently develop anxiety disorders and have problems with memory and decision making.

-Three excellent articles for basic science of pain and neuroplasticity:

1. Maldynia: Pathophysiology and Management of Neuropathic and Maladaptive Pain-a report of the AMA Council on Science and Public Health. Journal of pain medicine 2010. By multiple MDs in anesthesiology, psychiatry, and occupational services.
2. Demystifying pain pathways. Journal of Practical Pain Management. March 2013. Robert Roth, PhD, pharmacologist, Temple University; Joseph Purrglozzi, MD, Johns Hopkins University, and Robert Taylor, PhD,NEMA research.
3. Central sensitization: common etiology and somatoform disorders. Journal of Practical Pain Management. March 2013. Albert Roberts, M.D., University of Texas, Carol Lourdy, PhD, University of Texas and Robert Gatget, PhD, University of Texas.

-I hope this wasn't too technical or boring for you. This was part 2 in my series. The remained of this series will deal with the important new science of NP, what bad NP does to people, how to treat (and not mistreat) patients with NP, and how to use this science to win your case!