chirosacademy

BASIC SCIENCE LEVEL 2

6. Communicating Chiropractic's impact on the Brain

Outline

- All the functions of the prefrontal cortex
- Prefrontal cortex function during alert (non-stressful) conditions
- The impact of stress on the prefrontal cortex
- The Hand model of the prefrontal cortex, limbic brain and stress
- Stress alone can cause subluxations
- Traumatic experiences and stress
- How stress shuts down the prefrontal cortex and boosts the limbic brain
- Symptoms of chronic stress
- The strong link between stress, inflammation and most chronic diseases of today

Mental Health

Immune system

Inflammation

ANS regulation

Endocrine

Gastrointestinal

Pain & stiffness

Sleep

High blood pressure

High heart rate

Attention and focus

Chiropractic Adjustments change processing in the Prefrontal Cortex!!!

Coronary artery disease
Diabetes
Cancer
Obesity
Alzheimer's disease
Endocrine disorders
ADHD
Autism
Autoimmune
Fibromyalgia
CFS
Chronic pain
Major Depression
PTSD
Anxiety
Bipolar disorder
Schizophrenia
High blood pressure
Addiction

What can be done to help a person struggling with after-effects from a traumatic event?

- Main Aim is to restore balance between the rational and emotional brain!

- **Top down** (targeting the MPFC and your memory to be more rationally present in your body and emotions)
  - Psychotherapy for desensitization
  - CBT to stop the reactivity
  - Re-framing past trauma (you can change your own memories)
  - Mindfulness mediation (becoming present in your MPFC)
  - Yoga, Tai Chi (learning to inhabit your body)
  - **(Chiropractic by affecting PFC processing)**

- **Bottom up** (targeting brainstem and effects of overactive amygdala through touch, breathing, movement and biochemistry to activate the parasympathetic NS to promote healing, sleep and digestion)
  - Breathing exercises
  - Massage
  - Antidepressants/Antianxiety/Antipsychotic medication
  - Nutrition/supplements
  - Yoga, Tai Chi (conscious movement)
  - Neurofeedback
  - **Chiropractic (touch, effect on bigger muscles)**

Van der Kolk, B. A. (2015). The body keeps the score: Brain, mind, and body in the healing of trauma: Penguin Books.

JCC Journal of Contemporary Chiropractic

CHRONIC LOW BACK PAIN, PTSD, AND DEPRESSION: A CASE FOR USING THERAPEUTIC NEUROSCIENCE EDUCATION AND MANUAL THERAPY TO SUPPORT ENGAGEMENT IN PSYCHOLOGICAL SERVICE

Nathan Hinkeldey, DC[1], James Leonard, DC[2], Trevor McArthur, DC[2]

ABSTRACT

Objectives: To demonstrate the benefit that a strong patient-provider relationship can have in treating a patient with chronic low back pain and mental health conditions using manual therapy and therapeutic neuroscience education.

Clinical Presentation: A 32-year-old male veteran had dull, central low back pain. Clinical testing revealed the back pain was predominantly mechanical; however, an affective component to pain was also identified. His symptoms responded favorably to an end-range loading exam and had a directional preference for lumbar extension. He was also involved in outpatient mental health treatment.

Intervention and Outcome: The classification using the McKenzie Method of Diagnosis and Therapy (MDT) was found to be a lumbar posterior derangement. Treatment included spinal manipulative therapy, repetitive end-range loading, and therapeutic exercise for 6 visits over 5 months. Mechanically, the patient experienced complete functional improvement, 3 months of little to no pain, and a decrease on the PROMIS Pain Interference Scale 6B of 8 points (14 to 6). Initially, he believed his suicidal ideations were a result of his pain presentation; however, reduction of his pain did not alleviate the ideation. He voluntarily reported to the emergency department and received appropriate mental health care.

Conclusion: There is a strong relationship between depression, PTSD, and low back pain. Therefore, healthcare providers treating low back pain need should be aware of mental health diagnoses that impact pain presentations when making treatment recommendations. *(J Contemporary Chiropr 2018;1:26-29)*

Key Indexing Terms: Chiropractic; Depression; Mental Health; Post-Traumatic Stress Disorder

INTRODUCTION

Post-traumatic stress disorder (PTSD) impacts both civilian and military personnel at different rates. Per the National Center for PTSD, 7-8% of people in the United States will have PTSD at some point in their lifetime with incidence being higher 10% in women than 4% in men; however, further evaluation of military personnel revealed incidence of PTSD in Veterans serving in Operations Iraqi Freedom and Enduring Freedom of 11-20%, in the Gulf War of 12%, and the Vietnam War of 30%. (1) Incidence of PTSD is further elevated in the chronic pain population with authors citing incidence of 51% in the chronic low back pain population and 50% in the post motor vehicle accident population. A case series from a Veteran Healthcare Administration chiropractic clinic reported 68% of the population treated for low back pain presented with comorbid PTSD. (2) PTSD is a common comorbid condition found in patients with spine pain that direct access providers may see.

The latter is especially relevant to primary care providers (PCP) and chiropractors because, of those patients with spine pain, 53% prefer to report to their PCP and 28% to their chiropractor for treatment. (3) In greater than 85% of the population with low back pain, a structural source cannot be identified; healthcare providers continue to seek a specific structural abnormality or specific disease instead of identifying psychological factors. This results in greater expense and burden. (4) Psychosocial factors are much stronger predictors of low back pain presence and poor treatment outcomes. (5,6) Chronic pain, PTSD, and

Information from a trusted person can be very powerful!

Hinkeldey, N., Leonard, J., & McArthur, T. (2018). Chronic low back pain, PTSD, and Depression; A case for using therapeutic neuroscience education and manual therapy to support engagement in psychological service. Journal of Contemporary Chiropractic, 1(1), 26-29.

What you tell your patient matters!

JOURNAL OF MANUAL & MANIPULATIVE THERAPY, 2017
VOL. 25, NO. 5, 227–234
https://doi.org/10.1080/10669817.2016.1231860

Taylor & Francis
Taylor & Francis Group

The effect of manual therapy and neuroplasticity education on chronic low back pain: a randomized clinical trial

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ABSTRACT

Objective: To determine if a neuroplasticity educational explanation for a manual therapy technique will produce a different outcome compared to a traditional mechanical explanation.
Methods: Sixty-two patients with chronic low back pain (CLBP) were recruited for the study. Following consent, demographic data were obtained as well as pain ratings for low back pain (LBP) and leg pain (Numeric Pain Rating Scale), disability (Oswestry Disability Index), fear-avoidance (Fear-Avoidance-Beliefs Questionnaire), forward flexion (fingertips-to-floor), and straight leg raise (SLR) (inclinometer). Patients were then randomly allocated to receive one of two explanations (neuroplasticity or mechanical), a manual therapy technique to their lumbar spine, followed by post-intervention measurements of LBP, leg pain, forward flexion, and SLR.
Results: Sixty-two patients (female 35 [56.5%]), with a mean age of 60.1 years and mean duration of 9.26 years of CLBP participated in the study. There were no statistically significant interactions for LBP ($p = .325$), leg pain ($p = .172$), and trunk flexion ($p = .818$) between the groups, but SLR showed a significant difference in favor of the neuroplasticity explanation ($p = .041$). Additionally, the neuroplasticity group were 7.2 times (95% confidence interval = 1.8–28.6) more likely to improve beyond the MDC on the SLR than participants in the mechanical group.
Discussion: The results of this study show that a neuroplasticity explanation, compared to a traditional biomechanical explanation, resulted in a measureable difference in SLR in patients with CLBP when receiving manual therapy. Future studies need to explore if the increase in SLR correlated to changes in cortical maps of the low back.

KEYWORDS
Pain; brain; plasticity; education; manual therapy; straight leg raise; remapping

Discussion: The results of this study show that a **neuroplasticity explanation**, compared to **a traditional biomechanical explanation**, resulted in a measureable difference in SLR in patients with CLBP when receiving manual therapy. Future studies need to explore if the increase in SLR correlated to changes in cortical maps of the low back.

Louw, A., Farrell, K., Landers, M., Barclay, M., Goodman, E., Gillund, J., . . . Timmerman, L. (2017). The effect of manual therapy and neuroplasticity education on chronic low back pain: a randomized clinical trial. Journal of Manual & Manipulative Therapy, 25(5), 227-234.

The Effect of Neuroscience Education on Pain, Disability, Anxiety, and Stress in Chronic Musculoskeletal Pain

Adriaan Louw, PT, MAppSc, Ina Diener, PT, PhD, David S. Butler, PT, EdD, Emilio J. Puentedura, PT, DPT

ABSTRACT. Louw A, Diener I, Butler DS, Puentedura EJ. The effect of neuroscience education on pain, disability, anxiety, and stress in chronic musculoskeletal pain. Arch Phys Med Rehabil 2011;92:2041-56.

Objective: To evaluate the evidence for the effectiveness of neuroscience education (NE) for pain, disability, anxiety, and stress in chronic musculoskeletal (MSK) pain.

Data Sources: Systematic searches were conducted on Biomed Central, BMJ.com, CINAHL, the Cochrane Library, NLM Central Gateway, OVID, ProQuest (Digital Dissertations), PsycInfo, PubMed/Medline, ScienceDirect, and Web of Science. Secondary searching (PEARLing) was undertaken, whereby reference lists of the selected articles were reviewed for additional references not identified in the primary search.

Study Selection: All experimental studies including randomized controlled trials (RCTs), nonrandomized clinical trials, and case series evaluating the effect of NE on pain, disability, anxiety, and stress for chronic MSK pain were considered for inclusion. Additional limitations: studies published in English, published within the last 10 years, and patients older than 18 years. No limitations were set on specific outcome measures of pain, disability, anxiety, and stress.

Data Extraction: Data were extracted using the participants, interventions, comparison, and outcomes (PICO) approach.

Data Synthesis: Methodological quality was assessed by 2 reviewers using the Critical Review Form–Quantitative Studies. This review includes 8 studies comprising 6 high-quality RCTs, 1 pseudo-RCT, and 1 comparative study involving 401 subjects. Most articles were of good quality, with no studies rated as poor or fair. Heterogeneity across the studies with respect to participants, interventions evaluated, and outcome measures used prevented meta-analyses. Narrative synthesis of results, based on effect size, established compelling evidence that NE may be effective in reducing pain ratings, increasing function, addressing catastrophization, and improving movement in chronic MSK pain.

Conclusions: For chronic MSK pain disorders, there is compelling evidence that an educational strategy addressing neurophysiology and neurobiology of pain can have a positive effect on pain, disability, catastrophization, and physical performance.

Key Words: Education; Musculoskeletal System; Neurophysiology; Neurosciences; Pain; Rehabilitation.

PAIN IS A POWERFUL motivating force that guides treatment-seeking behaviors in patients.[1-3] Patient education has long been explored in the management of pain, anxiety, and stress associated with low back pain (LBP).[4-7] In the orthopedic domain, there are a number of studies on the effect of patient education on pain, with outcomes ranging from "excellent"[8] to "poor."[9,10] The study by Udermann et al[8] demonstrated that introduction of an individualized educational booklet on back biomechanics can result in decreased pain and frequency of LBP episodes in patients with chronic LBP (CLBP). In contrast to those findings, 2 systematic reviews[9,10] on the effect of individualized and/or group education for LBP and mechanical neck pain showed little efficacy for such education.

Most education programs for orthopedic patient populations have used anatomic and biomechanical models for addressing pain,[4,11-14] which not only have shown limited efficacy,[4,11,12,15,16] but may even have increased patient fears, anxiety, and stress, thus negatively impacting their outcomes.[11,17-19] Several educational strategies are advocated for patients with LBP, including biomechanical/back school type of education, evidence-based guideline education (ie, *The Back Book*[20]), cognitive behavioral therapy, and recently, neuroscience education (NE).

NE can be best described as an educational session or sessions describing the neurobiology and neurophysiology of pain, and pain processing by the nervous system. Instead of a

List of Abbreviations

BPPT	brachial plexus provocation test
CFS	chronic fatigue syndrome
CLBP	chronic low back pain
CONSORT	Consolidated Standards of Reporting Trials
LBP	low back pain
MSK	musculoskeletal
NE	neuroscience education

Neuroscience education about pain helps on its own!!

Conclusions: For chronic MSK pain disorders, there is compelling evidence that an educational strategy addressing neurophysiology and neurobiology of pain can have a positive effect on pain, disability, catastrophization, and physical performance.

Louw, A., Diener, I., Butler, D. S., & Puentedura, E. J. (2011). The effect of neuroscience education on pain, disability, anxiety, and stress in chronic musculoskeletal pain. *Archives of Physical Medicine and Rehabilitation, 92*(12), 2041-2056.

Results from the 2012 National Health Interview Survey

Spine

SPINE Volume 42, Number 23, pp 1810–1816

HEALTH SERVICES RESEARCH

The Prevalence, Patterns, and Predictors of Chiropractic Use Among US Adults

Results From the 2012 National Health Interview Survey

Jon Adams, PhD,* Wenbo Peng, PhD,* Holger Cramer, PhD,*,† Tobias Sundberg, PhD,*,‡
Craig Moore, Masters of Clinical Trials Research,* Lyndon Amorin-Woods, MPH,§
David Sibbritt, PhD,* and Romy Lauche, PhD*

Benefits reported under chiropractic care:

- 67% helped them to improve overall health and made them feel better.
- 42% reported sleeping better
- 40% reported reduced stress or helped them to relax.
- 39% reported easier for them to cope with their health problems
- 33% reported a sense of control over their health
- 27% reported felt better emotionally
- 13% reported improved their relationships with others

Adams J, Peng W, Cramer H, et al. The Prevalence, Patterns, and Predictors of Chiropractic Use Among US Adults: Results From the 2012 National Health Interview Survey. *Spine (Phila Pa 1976).* Dec 1 2017;42(23):1810-1816.

Abstracts and references for you!

Opinion TRENDS in Cognitive Sciences Vol.11 No.9

The amygdala and ventromedial prefrontal cortex in morality and psychopathy

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Recent work has implicated the amygdala and ventromedial prefrontal cortex in morality and, when dysfunctional, psychopathy. This model proposes that the amygdala, through stimulus-reinforcement learning,

particular, moral transgressions are judged to be less rule-contingent than are conventional transgressions; individuals are less likely to state that moral, rather than conventional, transgressions are permissible in the

Blair, R. J. R. (2007). The amygdala and ventromedial prefrontal cortex in morality and psychopathy. Trends Cogn Sci, 11(9), 387-392. doi:https://doi.org/10.1016/j.tics.2007.07.003

Impairment of social and moral behavior related to early damage in human prefrontal cortex

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The long-term consequences of early prefrontal cortex lesions occurring before 16 months were investigated in two adults. As is the case when such damage occurs in adulthood, the two early-onset patients had severely impaired social behavior despite normal basic cognitive abilities, and showed insensitivity to future consequences of decisions, defective autonomic responses to punishment contingencies and failure to respond to behavioral interventions. Unlike adult-onset patients, however, the two patients had defective social and moral reasoning, suggesting that the acquisition of complex social conventions and moral rules had been impaired. Thus early-onset prefrontal damage resulted in a syndrome resembling psychopathy.

Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. Nature Neuroscience, 2, 1032. doi:10.1038/14833

Review Article

Brain and Neuroscience Advances

Brain and Neuroscience Advances
Volume 2: 1–15

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journals.sagepub.com/home/bna
SAGE

The dynamics of disordered dialogue: Prefrontal, hippocampal and thalamic miscommunication underlying working memory deficits in schizophrenia

David A Kupferschmidt[1] and Joshua A Gordon[1,2]

Abstract

The prefrontal cortex is central to the orchestrated brain network communication that gives rise to working memory and other cognitive functions. Accordingly, working memory deficits in schizophrenia are increasingly thought to derive from prefrontal cortex dysfunction coupled with broader network disconnectivity. How the prefrontal cortex dynamically communicates with its distal network partners to support working memory and how this communication is disrupted in individuals with schizophrenia remain unclear. Here we review recent evidence that prefrontal cortex communication with the hippocampus and thalamus is essential for normal spatial working memory, and that miscommunication between these structures underlies spatial working memory deficits in schizophrenia. We focus on studies using normal rodents and rodent models designed to probe schizophrenia-related pathology to assess the dynamics of neural interaction between these brain regions. We also highlight recent preclinical work parsing roles for long-range prefrontal cortex connections with the hippocampus and thalamus in normal and disordered spatial working memory. Finally, we discuss how emerging rodent endophenotypes of hippocampal- and thalamo-prefrontal cortex dynamics in spatial working memory could translate into richer understanding of the neural bases of cognitive function and dysfunction in humans.

Kupferschmidt, D. A., & Gordon, J. A. (2018). The dynamics of disordered dialogue: Prefrontal, hippocampal and thalamic miscommunication underlying working memory deficits in schizophrenia. Brain and Neuroscience Advances, 2, 2398212818771821.

Best Practice & Research Clinical Gastroenterology
Vol. 18, No. 4, pp. 663–680, 2004
doi:10.1016/j.bpg.2004.04.010
available online at http://www.sciencedirect.com

4

Central nervous system involvement in functional gastrointestinal disorders

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Although functional gastrointestinal disorders (FGID) are common, their pathophysiology remains incompletely understood. It is generally accepted that dysfunction of the bidirectional pathways between the gastrointestinal tract and the central nervous system (the 'brain–gut axis') at any level can cause FGID symptoms. In this review article, we focus on the role of the central nervous system in the brain–gut axis.

First, we describe the functional anatomy of the brain–gut axis. Second, we focus on the results from brain-imaging studies both in healthy volunteers and in FGID patients. These new investigational techniques made identification of brain regions critically involved in processing of visceral afferent information possible. Differences in central nervous system response to visceral stimuli between controls and FGID patients will be highlighted. Third, we will address the issue of high comorbidity with psychiatric disorders. Some hypotheses about common pathophysiological substrates will be discussed.

Van Oudenhove, L., Demyttenaere, K., Tack, J., & Aziz, Q. (2004). Central nervous system involvement in functional gastrointestinal disorders. Best Practice & Research Clinical Gastroenterology, 18(4), 663-680.

J Neural Transm (2009) 116:941–952
DOI 10.1007/s00702-009-0243-8

BASIC NEUROSCIENCES, GENETICS AND IMMUNOLOGY - REVIEW ARTICLE

Neurotransmitters and prefrontal cortex–limbic system interactions: implications for plasticity and psychiatric disorders

Alberto Del Arco · Francisco Mora

Received: 20 November 2008 / Accepted: 11 May 2009 / Published online: 28 May 2009

Abstract The prefrontal cortex (PFC) efferent projections to limbic areas facilitate a top-down control on the execution of goal-directed behaviours. The PFC sends glutamatergic outputs to limbic areas such as the hippocampus and amygdala which in turn modulate the activity of the nucleus accumbens (NAc). Dopamine and acetylcholine neurons in the brainstem and basal forebrain/septal areas, which send outputs to NAc, hippocampus and amygdala, are also regulated by PFC glutamatergic projections, and seem to be of special relevance in modulating motor, emotional and mnemonic functions. Both the physiological and pathological changes in the PFC influence the activity of these limbic areas and the associated with psychiatric disorders or due to environmental-dependent plasticity, can change PFC–limbic system interactions.

Keywords Dopamine · Glutamate · Acetylcholine · NMDA · Corticosterone · Prefrontal cortex · Hippocampus · Amygdala · Nucleus accumbens · Schizophrenia · Environmental enrichment, social isolation

Prefrontal cortex involvement in motor, emotional and mnemonic functions

Neuropsychopharmacology (2017), 1–9

www.neuropsychopharmacology.org

Disrupted Glutamatergic Transmission in Prefrontal Cortex Contributes to Behavioral Abnormality in an Animal Model of ADHD

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[1]*Department of Physiology and Biophysics, School of Medicine and Biomedical Sciences, State University of New York at Buffalo, Buffalo, NY, USA*

Spontaneously hypertensive rats (SHR) are the most widely used animal model for the study of attention deficit hyperactivity disorder (ADHD). Here we sought to reveal the neuronal circuits and molecular basis of ADHD and its potential treatment using SHR. Combined electrophysiological, biochemical, pharmacological, chemicogenetic, and behavioral approaches were utilized. We found that AMPAR-mediated synaptic transmission in pyramidal neurons of prefrontal cortex (PFC) was diminished in SHR, which was correlated with the decreased surface expression of AMPAR subunits. Administration of methylphenidate (a psychostimulant drug used to treat ADHD), which blocks dopamine transporters and norepinephrine transporters, ameliorated the behavioral deficits of adolescent SHR and restored AMPAR-mediated synaptic function. Activation of PFC pyramidal neurons with a CaMKII-driven Gq-coupled designer receptor exclusively activated by designer drug also led to the elevation of AMPAR function and the normalization of ADHD-like behaviors in SHR. These results suggest that the disrupted function of AMPARs in PFC may underlie the behavioral deficits in adolescent SHR and enhancing PFC activity could be a treatment strategy for ADHD.
Neuropsychopharmacology advance online publication, 22 March 2017; doi:10.1038/npp.2017.30

Cheng, J., Liu, A., Shi, M. Y., & Yan, Z. (2017). Disrupted glutamatergic transmission in prefrontal cortex contributes to behavioral abnormality in an animal model of ADHD

Molecular Psychiatry (2000) 5, 142–149

www.nature.com/mp

ORIGINAL RESEARCH ARTICLE

Disease-specific alterations in frontal cortex brain proteins in schizophrenia, bipolar disorder, and major depressive disorder

NL Johnston-Wilson[1], CD Sims[2], J-P Hofmann[2], L Anderson[2], AD Shore[3], EF Torrey[4], RH Yolken[1] and the Stanley Neuropathology Consortium

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Severe psychiatric disorders such as schizophrenia, bipolar disorder and major depressive disorder are brain diseases of unknown origin. No biological marker has been documented at the pathological, cellular, or molecular level, suggesting that a number of complex but subtle changes underlie these illnesses. We have used proteomic technology to survey post-mortem tissue to identify changes linked to the various diseases. Proteomics uses two-dimensional gel electrophoresis and mass spectrometric sequencing of proteins to allow the comparison of subsets of expressed proteins among a large number of samples. This form of analysis was combined with a multivariate statistical model to study changes in protein levels

Johnston-Wilson, N. L., Sims, C. D., Hofmann, J. P., Anderson, L., Shore, A. D., Torrey, E. F., & Yolken, R. H. (2000). Disease-specific alterations in frontal cortex brain proteins in schizophrenia, bipolar disorder, and major depressive disorder. Molecular Psychiatry, 5, 142. doi:10.1038/sj.mp.4000696

Abnormal protein and mRNA expression of inflammatory cytokines in the prefrontal cortex of depressed individuals who died by suicide

Ghanshyam N. Pandey, PhD; Hooriyah S. Rizavi, MS; Hui Zhang, PhD; Runa Bhaumik, PhD; Xinguo Ren, MD

Background: Depression and stress are major risk factors for suicidal behaviour, and some studies show abnormalities of proinflammatory cytokines in the serum and cerebrospinal fluid (CSF) of depressed and suicidal patients. However, it is not clear if similar abnormalities of cytokines are present in the brain of suicidal and depressed patients. **Methods:** We therefore determined the mRNA (using real-time polymerase chain reaction) and protein (using enzyme-linked immunosorbent assay and Western Blot) expression levels of interleukin (IL)-1β, IL-6, tumour necrosis factor (TNF)-α, lymphotoxin A, lymphotoxin B, IL-8, IL-10 and IL-13 in the prefrontal cortex (PFC) obtained from 24 depressed individuals who died by suicide and 24 nonpsychiatric controls. **Results:** We observed that the mRNA and protein levels of IL-1β, IL-6, TNF-α, and lymphotoxin A were significantly increased, and levels of anti-inflammatory cytokine IL-10, and of IL-1 receptor antagonist (IL-1RA) were significantly decreased in the PFC of depressed individuals who died by suicide compared with controls. There were no significant differences in the protein and mRNA levels of IL-8 and IL-13 in the PFC. **Limitations:** The main limitation of this study is that some of the suicide group had been taking antidepressant medication at the time of death. **Conclusion:** Our results suggest that alterations of cytokines may be associated with the pathophysiology of depressed suicide and there may be an imbalance between pro- and anti-inflammatory cytokines in people who die by suicide. The causes of these increases in the brain of people who die by suicide, therefore, need to be investigated further.

Pandey, G. N., Rizavi, H. S., Zhang, H., Bhaumik, R., & Ren, X. (2018). Abnormal protein and mRNA expression of inflammatory cytokines in the prefrontal cortex of depressed individuals who died by suicide. Journal of psychiatry & neuroscience: JPN, 43(6), 376.

Mol Neurobiol (2015) 51:1353–1367
DOI 10.1007/s12035-014-8814-6

Glutamate Release Machinery Is Altered in the Frontal Cortex of Rats with Experimental Autoimmune Encephalomyelitis

Natalí L. Chanaday · A. Alejandro Vilcaes · Ana L. de Paul · Alicia I. Torres · Alicia L. Degano · German A. Roth

Received: 22 April 2014 / Accepted: 11 July 2014 / Published online: 19 July 2014

Abstract Experimental autoimmune encephalomyelitis (EAE) is an animal model that mimics many of the clinical and pathological features of the human disease multiple sclerosis (MS). Both are inflammatory demyelinating and neurodegenerative pathologies of the central nervous system associated with motor, sensory, and cognitive deficits. In MS, gray matter atrophy is related to the emergence of cognitive deficits and contributes to clinical progression. In particular, prefrontal cortex injury and dysfunction have been correlated to the development of fatigue, one of the most common and disabling symptoms in MS. However, the molecular bases of these changes remain unknown. Taking advantage of EAE similitude, we phosphorylation and dispersion. These changes were associated with reduced synaptic vesicle mobility, with no alterations in synaptosomal morphology as evidenced by electron microscopy. The present are the first pieces of evidence unraveling the molecular mechanisms of frontal cortex neuronal dysfunction in EAE and, possibly, MS.

Keywords Experimental autoimmune encephalomyelitis · Multiple sclerosis · Synapsin · Glutamate release · Synaptosomes · Release machinery

Chanaday, N. L., Vilcaes, A. A., de Paul, A. L., Torres, A. I., Degano, A. L., & Roth, G. A. (2015). Glutamate Release Machinery Is Altered in the Frontal Cortex of Rats with Experimental Autoimmune Encephalomyelitis. Molecular Neurobiology, 51(3), 1353-1367. doi:10.1007/s12035-014-8814-6

Behavioral/Systems/Cognitive

Global Connectivity of Prefrontal Cortex Predicts Cognitive Control and Intelligence

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[1]Psychology Department, Washington University, St. Louis, Missouri 63130, [2]Institute of Cognitive Science, University of Colorado Boulder, UCB 345, Boulder, Colorado 80309, [3]Department of Psychology, University of Ljubljana, Ljubljana, Slovenia, and [4]Department of Psychiatry, Yale University School of Medicine, New Haven, Connecticut 06519

Control of thought and behavior is fundamental to human intelligence. Evidence suggests a frontoparietal brain network implements such cognitive control across diverse contexts. We identify a mechanism—global connectivity—by which components of this network might coordinate control of other networks. A lateral prefrontal cortex (LPFC) region's activity was found to predict performance in a high control demand working memory task and also to exhibit high global connectivity. Critically, global connectivity in this LPFC region, involving connections both within and outside the frontoparietal network, showed a highly selective relationship with individual differences in fluid intelligence. These findings suggest LPFC is a global hub with a brainwide influence that facilitates the ability to implement control processes central to human intelligence.

Cole, M. W., Yarkoni, T., Repovš, G., Anticevic, A., & Braver, T. S. (2012). Global connectivity of prefrontal cortex predicts cognitive control and intelligence. *Journal of Neuroscience, 32*(26), 8988-8999.

Behavioral/Cognitive

Emotion Regulation and Trait Anxiety Are Predicted by the Microstructure of Fibers between Amygdala and Prefrontal Cortex

Annuschka Salima Eden,[1] Jan Schreiber,[4] Alfred Anwander,[4] Katharina Keuper,[1,3] Inga Laeger,[2] Peter Zwanzger,[2] Pienie Zwitserlood,[5] Harald Kugel,[6] and Christian Dobel[1,3]

[1]Institute for Biomagnetism and Biosignalanalysis and [2]Department of Psychiatry, University Hospital, 48149 Münster, Germany, [3]Otto Creutzfeldt Center for Cognitive and Behavioural Neuroscience, 48149 Münster, Germany, [4]Max Planck Institute for Human Cognitive and Brain Sciences, 04103 Leipzig, Germany, [5]Westfälische Wilhelms-Universität, 48149 Münster, Germany, and [6]Center of Radiology, Faculty of Medicine, 48149 Münster, Germany

Diffusion tensor imaging revealed that trait anxiety predicts the microstructural properties of a prespecified fiber tract between the amygdala and the perigenual anterior cingulate cortex. Besides this particular pathway, it is likely that other pathways are also affected. We investigated white matter differences in persons featuring an anxious or a nonanxious personality, taking into account all potential pathway connections between amygdala and anxiety-related regions of the prefrontal cortex (PFC). Diffusion-weighted images, measures of trait anxiety and of reappraisal use (an effective emotion-regulation style), were collected in 48 females. With probabilistic tractography, pathways between the amygdala and the dorsolateral PFC, dorsomedial PFC, ventromedial PFC, and orbitofrontal cortex (OFC) were delineated. The resulting network showed a direct ventral connection between amygdala and PFC and a second limbic connection following the fornix and the anterior limb of the internal capsule. Reappraisal use predicted the microstructure of pathways to all calculated PFC regions in the left hemisphere, indicating stronger pathways for persons with high reappraisal use. Trait anxiety predicted the microstructure in pathways to the ventromedial PFC and OFC, indexing weaker connections in trait-anxious persons. These effects appeared in the right hemisphere, supporting lateralization and top-down inhibition theories of emotion processing. Whereas a specific microstructure is associated with an anxious personality, a different structure subserves emotion regulation. Both are part of a broad fiber tract network between amygdala and PFC.

Key words: amygdala; DTI; PFC; reappraisal use; top-down inhibition; trait anxiety

Eden, A. S., Schreiber, J., Anwander, A., Keuper, K., Laeger, I., Zwanzger, P., . . . Dobel, C. (2015). Emotion regulation and trait anxiety are predicted by the microstructure of fibers between amygdala and prefrontal cortex. *Journal of Neuroscience, 35*(15), 6020-6027.

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Prefrontal Cortex GABAergic Deficits and Circuit Dysfunction in the Pathophysiology and Treatment of Chronic Stress and Depression

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Abstract

Psychiatric diseases, notably major depression, are associated with imbalance of excitatory and inhibitory neurotransmission within the prefrontal cortex (PFC) and related limbic brain circuitry. In many cases these illnesses are precipitated or exacerbated by chronic stress, which also alters excitatory and inhibitory neurotransmitter systems. Notably, exposure to repeated uncontrollable stress causes persistent changes in the synaptic integrity and function of the principal glutamatergic excitatory neurons in the PFC, characterized by neuronal atrophy and loss of synaptic connections. This can lead to dysfunction of the PFC circuitry that is necessary for execution of adaptive behavioral responses. In addition, an emerging literature shows that chronic stress also causes extensive alteration of GABAergic inhibitory circuits in the PFC, leading to the hypothesis that inhibitory neurotransmitter deficits contribute to changes in PFC neuronal excitability and cognitive impairments. Here we review evidence in rodents and human, which point to the mechanisms underlying stress-induced alterations of GABA transmission in the PFC, and its relevance to circuit dysfunction in mood and stress related disorders. These findings suggest that alterations of GABA interneurons and inhibitory neurotransmission play a causal role in the development of stress-related neurobiological illness, and could identify a new line of GABA related therapeutic targets.

Ghosal, S., Hare, B. D., & Duman, R. S. (2017). Prefrontal cortex GABAergic deficits and circuit dysfunction in the pathophysiology and treatment of chronic stress and depression. *Current opinion in behavioral sciences, 14*, 1-8.

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Original Article

ORIGINAL ARTICLE

The Number of Parvalbumin-Expressing Interneurons Is Decreased in the Prefrontal Cortex in Autism

Ezzat Hashemi[1,3], Jeanelle Ariza[1,3], Haille Rogers[1,3], Stephen C. Noctor[2,4] and Verónica Martínez-Cerdeño[1,3,4]

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Abstract

The cognitive phenotype of autism has been correlated with an altered balance of excitation to inhibition in the cerebral cortex, which could result from a change in the number, function, or morphology of GABA-expressing interneurons. The number of GABAergic interneuron subtypes has not been quantified in the autistic cerebral cortex. We classified interneurons into 3 subpopulations based on expression of the calcium-binding proteins parvalbumin, calbindin, or calretinin. We quantified the number of each interneuron subtype in postmortem neocortical tissue from 11 autistic cases and 10 control cases. Prefrontal Brodmann Areas (BA) BA46, BA47, and BA9 in autism and age-matched controls were analyzed by blinded researchers. We show that the number of parvalbumin+ interneurons in these 3 cortical areas—BA46, BA47, and BA9—is significantly reduced in autism compared with controls. The number of calbindin+ and calretinin+ interneurons did not differ in the cortical areas examined. Parvalbumin+ interneurons are fast-spiking cells that synchronize the activity of pyramidal cells through perisomatic and axo-axonic inhibition. The reduced number of parvalbumin+ interneurons could disrupt the balance of excitation/inhibition and alter gamma wave oscillations in the cerebral cortex of autistic subjects. These data will allow development of novel treatments specifically targeting parvalbumin interneurons.

Hashemi, E., Ariza, J., Rogers, H., Noctor, S. C., & Martínez-Cerdeño, V. (2017). The Number of Parvalbumin-Expressing Interneurons Is Decreased in the Prefrontal Cortex in Autism. Cerebral Cortex, 27(3), 1931-1943. doi:10.1093/cercor/bhw021

Original Investigation

Role of the Medial Prefrontal Cortex in Impaired Decision Making in Juvenile Attention-Deficit/Hyperactivity Disorder

Tobias U. Hauser, PhD; Reto Iannaccone, MS; Juliane Ball, PhD; Christoph Mathys, PhD; Daniel Brandeis, PhD; Susanne Walitza, MD; Silvia Brem, PhD

IMPORTANCE Attention-deficit/hyperactivity disorder (ADHD) has been associated with deficient decision making and learning. Models of ADHD have suggested that these deficits could be caused by impaired reward prediction errors (RPEs). Reward prediction errors are signals that indicate violations of expectations and are known to be encoded by the dopaminergic system. However, the precise learning and decision-making deficits and their neurobiological correlates in ADHD are not well known.

OBJECTIVE To determine the impaired decision-making and learning mechanisms in juvenile ADHD using advanced computational models, as well as the related neural RPE processes using multimodal neuroimaging.

DESIGN, SETTING, AND PARTICIPANTS Twenty adolescents with ADHD and 20 healthy adolescents serving as controls (aged 12-16 years) were examined using a probabilistic reversal learning task while simultaneous functional magnetic resonance imaging and electroencephalogram were recorded.

MAIN OUTCOMES AND MEASURES Learning and decision making were investigated by contrasting a hierarchical Bayesian model with an advanced reinforcement learning model and by comparing the model parameters. The neural correlates of RPEs were studied in functional magnetic resonance imaging and electroencephalogram.

RESULTS Adolescents with ADHD showed more simplistic learning as reflected by the reinforcement learning model (exceedance probability, P_x = .92) and had increased exploratory behavior compared with healthy controls (mean [SD] decision steepness parameter β: ADHD, 4.83 [2.97]; controls, 6.04 [2.53]; P = .02). The functional magnetic resonance imaging analysis revealed impaired RPE processing in the medial prefrontal cortex during cue as well as during outcome presentation (P < .05, family-wise error correction). The outcome-related impairment in the medial prefrontal cortex could be attributed to deficient processing at 200 to 400 milliseconds after feedback presentation as reflected by reduced feedback-related negativity (ADHD, 0.61 [3.90] μV; controls, −1.68 [2.52] μV; P = .04).

CONCLUSIONS AND RELEVANCE The combination of computational modeling of behavior and multimodal neuroimaging revealed that impaired decision making and learning mechanisms in adolescents with ADHD are driven by impaired RPE processing in the medial prefrontal cortex. This novel, combined approach furthers the understanding of the pathomechanisms in ADHD and may advance treatment strategies.

Supplemental content at jamapsychiatry.com

Hauser, T. U., Iannaccone, R., Ball, J., Mathys, C., Brandeis, D., Walitza, S., & Brem, S. (2014). Role of the medial prefrontal cortex in impaired decision making in juvenile attention-deficit/hyperactivity disorder. *JAMA psychiatry, 71*(10), 1165-1173.

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Prefrontal atrophy, disrupted NREM slow waves, and impaired hippocampal-dependent memory in aging

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Abstract

Aging has independently been associated with regional brain atrophy, reduced non-rapid eye movement (NREM) slow-wave activity (SWA), and impaired long-term retention of episodic memories. However, that the interaction of these factors represents a neuropatholgical pathway associated with cognitive decline in later life remains unknown. Here, we show that age-related medial prefrontal cortex (mPFC) grey-matter atrophy is associated with reduced NREM SWA activity in older adults, the extent to which statistically mediates the impairment of overnight sleep-dependent memory retention. Moreover, this memory impairment was further associated with persistent hippocampal activation and reduced task-related hippocampal-prefrontal cortex connectivity, potentially representing impoverished hippocampal-neocortical memory transformation. Together, these data support a model in which age-related mPFC atrophy diminishes SWA, the functional consequence of which is impaired long-term memory. Such findings suggest that sleep disruption in the elderly, mediated by structural brain changes, represent a novel contributing factor to age-related cognitive decline in later life.

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Mander, B. A., Rao, V., Lu, B., Saletin, J. M., Lindquist, J. R., Ancoli-Israel, S., . . . Walker, M. P. (2013). Prefrontal atrophy, disrupted NREM slow waves and impaired hippocampal-dependent memory in aging. Nature Neuroscience, 16(3), 357.

frontiers in
HUMAN NEUROSCIENCE

MINI REVIEW ARTICLE
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Neurobiological foundations of multisensory integration in people with autism spectrum disorders: the role of the medial prefrontal cortex

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This review aims to relate the sensory processing problems in people with autism spectrum disorders (ASD), especially multisensory integration (MSI), to the role of the medial prefrontal cortex (mPFC) by exploring neuroanatomical findings; brain connectivity and Default Network (DN); global or locally directed attention; and temporal multisensory binding. The mPFC is part of the brain's DN, which is deactivated when attention is focused on a particular task and activated on rest when spontaneous cognition emerges. In those with ASD, it is hypoactive and the higher the social impairment the greater the atypical activity. With an immature DN, cross-modal integration is impaired, resulting in a collection of disconnected fragments instead of a coherent global perception. The deficit in MSI may lie in the temporal synchronization of neural networks. The time interval in which the stimulation of one sensory channel could influence another would be higher, preventing integration in the typical shorter time range. Thus, the underconnectivity between distant brain areas would be involved in top-down information processes (relying on global integration of data from different sources) and would enhance low level perception processes such as over focused attention to sensory details.

Keywords: autism spectrum disorders (ASD), multisensory integration, medial prefrontal cortex (mPFC), default network, temporal multisensory binding

Martínez-Sanchis, S. (2014). Neurobiological foundations of multisensory integration in people with autism spectrum disorders: the role of the medial prefrontal cortex. *Frontiers in Human Neuroscience, 8*, 970

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WILEY NMR IN BIOMEDICINE

RESEARCH ARTICLE

Medial prefrontal cortex deficits correlate with unrefreshing sleep in patients with chronic fatigue syndrome

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Unrefreshing sleep is a hallmark of chronic fatigue syndrome/myalgic encephalomyelitis (CFS). This study examined brain structure variations associated with sleep quality in patients with CFS. 38 patients with CFS (34.8 ± 10.1 years old) and 14 normal controls (NCs) (34.7 ± 8.4 years old) were recruited. All subjects completed the Hospital Anxiety and Depression Scale, Pittsburgh Sleep Quality Index (PSQI), and Chalder Fatigue Scale (CFQ) questionnaires. Brain MRI measures included global and regional grey and white matter volumes, magnetization transfer T_1 weighted (MT-T1w) intensities, and T_1 weighted (T1w) and T_2 weighted spin echo signal intensities. We performed voxel based group comparisons of these regional brain MRI measures and regressions of these measures with the PSQI and CFQ scales adjusted for age, anxiety and depression, and the appropriate global measure. In CFS patients, negative correlations were observed in the medial prefrontal cortex (mPFC) between PSQI and MT-T1w intensities (family-wise error corrected cluster, $P_{FWE} < 0.05$) and between PSQI and T1w intensities ($P_{FWE} < 0.05$). In the same mPFC location, both MT and T1w intensities were lower in CFS patients compared with NCs (uncorrected voxel $P < 0.001$). This study is the first to report that brain structural differences are associated with unrefreshing sleep in CFS. This result refutes the suggestion that unrefreshing sleep is a misperception in CFS patients and further investigation of this symptom is warranted.

KEYWORDS

inferior fronto-occipital fasciculus, medial prefrontal cortex, MRI, sleep quality, voxel based morphometry

Shan, Z. Y., Kwiatek, R., Burnet, R., Del Fante, P., Staines, D. R., Marshall-Gradisnik, S. M., & Barnden, L. R. (2017). Medial prefrontal cortex deficits correlate with unrefreshing sleep in patients with chronic fatigue syndrome. *NMR in Biomedicine, 30*(10), e3757.

frontiers in Psychiatry

REVIEW
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Autoimmune Aspects of Neurodegenerative and Psychiatric Diseases: A Template for Innovative Therapy

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Neurodegenerative and psychiatric diseases (NPDs) are today's most important group of diseases, surpassing both atherosclerotic cardiovascular disease and cancer in morbidity incidence. Although NPDs have a dramatic impact on our society because of their high incidence, mortality, and severe debilitating character, remarkably few effective interventions have become available. The current treatments, if available, comprise the lifelong intake of general immunosuppressants to delay disease progression or neurotransmitter antagonists/agonists to dampen undesired behaviors. The long-term usage of such medication, however, coincides with often severe adverse side effects. There is, therefore, an urgent need for safe and effective treatments for these diseases. Here, we discuss that many NPDs coincide with subtle chronic or flaring brain inflammation sometimes escalating with infiltrations of lymphocytes in the inflamed brain parts causing mild to severe or even lethal brain damage. Thus, NPDs show all features of autoimmune diseases. In this review, we postulate that NPDs resemble autoimmune-driven inflammatory diseases in many aspects and may belong to the same disease spectrum. Just like in autoimmune diseases, NPD symptoms basically are manifestations of a chronic self-sustaining inflammatory process with detrimental consequences for the patient.

de Haan, P., Klein, H. C., & 't Hart, B. A. (2017). Autoimmune Aspects of Neurodegenerative and Psychiatric Diseases: A Template for Innovative Therapy. Frontiers in Psychiatry, 8(46). doi:10.3389/fpsyt.2017.00046

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Illness Progression, Recent Stress and Morphometry of Hippocampal Subfields and Medial Prefrontal Cortex in Major Depression

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Abstract

Background—Longitudinal studies of illness progression in Major Depressive Disorder (MDD) indicate that the onset of subsequent depressive episodes becomes increasingly decoupled from external stressors. A possible mechanism underlying this phenomenon is that multiple episodes induce long-lasting neurobiological changes that confer increased risk for recurrence. Prior morphometric studies have frequently reported volumetric reductions in MDD—especially in medial prefrontal cortex (mPFC) and the hippocampus— but few studies have investigated whether these changes are exacerbated by prior episodes.

Treadway, M. T., Waskom, M. L., Dillon, D. G., Holmes, A. J., Park, M. T. M., Chakravarty, M. M., . . . Fava, M. (2015). Illness progression, recent stress, and morphometry of hippocampal subfields and medial prefrontal cortex in major depression. *Biological Psychiatry, 77*(3), 285-294.

8th International IEEE EMBS Conference on Neural Engineering
Shanghai, China, May 25 - 28 , 2017

Study of intrinsic functional connectivity from medial prefrontal cortex in female with major depressive disorders

X. Zhang, Y. Tang, S. Tong, *Senior Member, IEEE* and Y. Li*, *Member, IEEE*

***Abstract*—Major depressive disorder (MDD) is associated with excessive self-focus and a tendency to engage in self-reflection. Women are more inclined to form negative thoughts when facing problems such as social rejection. The neuroimaging studies addressing MDD females are still rare. The medial prefrontal cortex, a central node of anterior default mode network (DMN), plays an important role in the pathophysiology of MDD. In this study, we explored the inter-regional correlation feature of mPFC with whole brain through functional connectivity (FC) analysis of resting state functional magnetic resonance imaging (rs-fMRI) data. Increased FC with left anterior cingulate cortex (ACC_L) was found in female MDD patients compared with healthy controls. Furthermore, the FC strength of ACC_L was positively correlated with clinical scores, which implied the important role of mPFC-ACC pathway in MDD females. Moreover, decreased FC with right middle temporal gyrus (mTG_R) and left middle temporal gyrus (mTG_L) was found in MDD females, which may result from the low self-evaluation in MDD. This work provides additional insights in understanding the pathophysiological mechanisms of MDD women.**

Functional communication between brain regions plays a critical role in complex cognitive process (e.g self-reflection). Resting-state functional connectivity (FC) reflects temporal correlation between blood oxygen level-independent (BOLD) signals from different brain regions [3]. Findings in MDD using resting state functional magnetic resonance imaging (rs-fMRI) suggest abnormal FC within the default mode network (DMN) as well as the salience network and central executive network [4]. The medial prefrontal cortex (mPFC), as a central node of DMN, is closely linked to low self-reflection when facing negative thoughts in female MDD patients [4]–[7].

In this study, we used seed-based resting state functional connectivity analysis to examine the intrinsic inter-regional correlation feature of neuronal activities in female MDD patients. We hypothesized that MDD females would have altered FC in brain regions involved with cognition/emotion regulation.

Zhang, X., Tang, Y., Tong, S., & Li, Y. (2017, 25-28 May 2017). *Study of intrinsic functional connectivity from medial prefrontal cortex in female with major depressive disorders.* Paper presented at the 2017 8th International IEEE/EMBS Conference on Neural Engineering (NER)

So what does this all mean to us chiros?

- What can we claim?
- What can we not claim?
- Why? Why not?
- How do you explain this to patients?
- How are subluxations linked to symptoms?
- How can you measure ‘stress’ and our effect in practice?

What can we claim?

Someone has noticed that ‘x’ has improved under your care

- That’s really interesting! We don’t actually have any direct evidence showing chiropractic improves ‘x’ but
- ‘We have scientific evidence that chiropractic care impacts the the prefrontal cortex’
- This therefore could explain why you feel ‘x’ has improved under chiropractic care, because the Prefrontal Cortex is known to be implicated or plays a major role in ‘x’
- This is something I have seen before

What can we claim?

Someone calls asking if chiropractic care can fix/cure/help with ‘x’

- We don’t actually have any direct clinical evidence showing chiropractic improves ‘x’ but
- ‘We have scientific evidence that chiropractic care impacts the prefrontal cortex’
- And the prefrontal cortex is known to play a major role in ‘x’
- So its possible chiropractic care may help with ‘x’
- But I cannot promise to fix/cure ‘x’ because there is not enough research done yet about chiropractic and ‘x’
- Although we have had many cases here in this clinic that have been helped with ‘x’

How solid is the evidence we change PFC processing?

- 4 publications showing N30 SEP peak changes with Heidi adjusting
- Validated by Drewes & Lelic (with Heidi there adjusting only)
- Drewes and Lelic did all analysis showing the N30 SEP changes were PFC processing changes
- BUT:
  - Can other chiros also produce changes in N30 SEP amplitudes?
  - Does it matter which segment is thrust upon (VS vs non-VS)? – Collecting data right now at CCR in NZ!!!!
  - Does technique matter?
  - Does skill level matter?

- No evidence yet this change in PFC processing is a good thing (I assume it is because Im biased pro chiropractic, and in light of the positive feedback from patients and subjects in the studies)

- No evidence for 'improved' PFC function
  - But I assume that is why other studies are showing improved HRV, and why chiro patients claim they feel better, have better health, personal interactions improve, cope better, improved JPS in elbow and ankle, improved SMI, improved MMI, etc, etc

- Is this solid evidence? NO…… but its sure as shit better evidence than that squashed nerve theory!

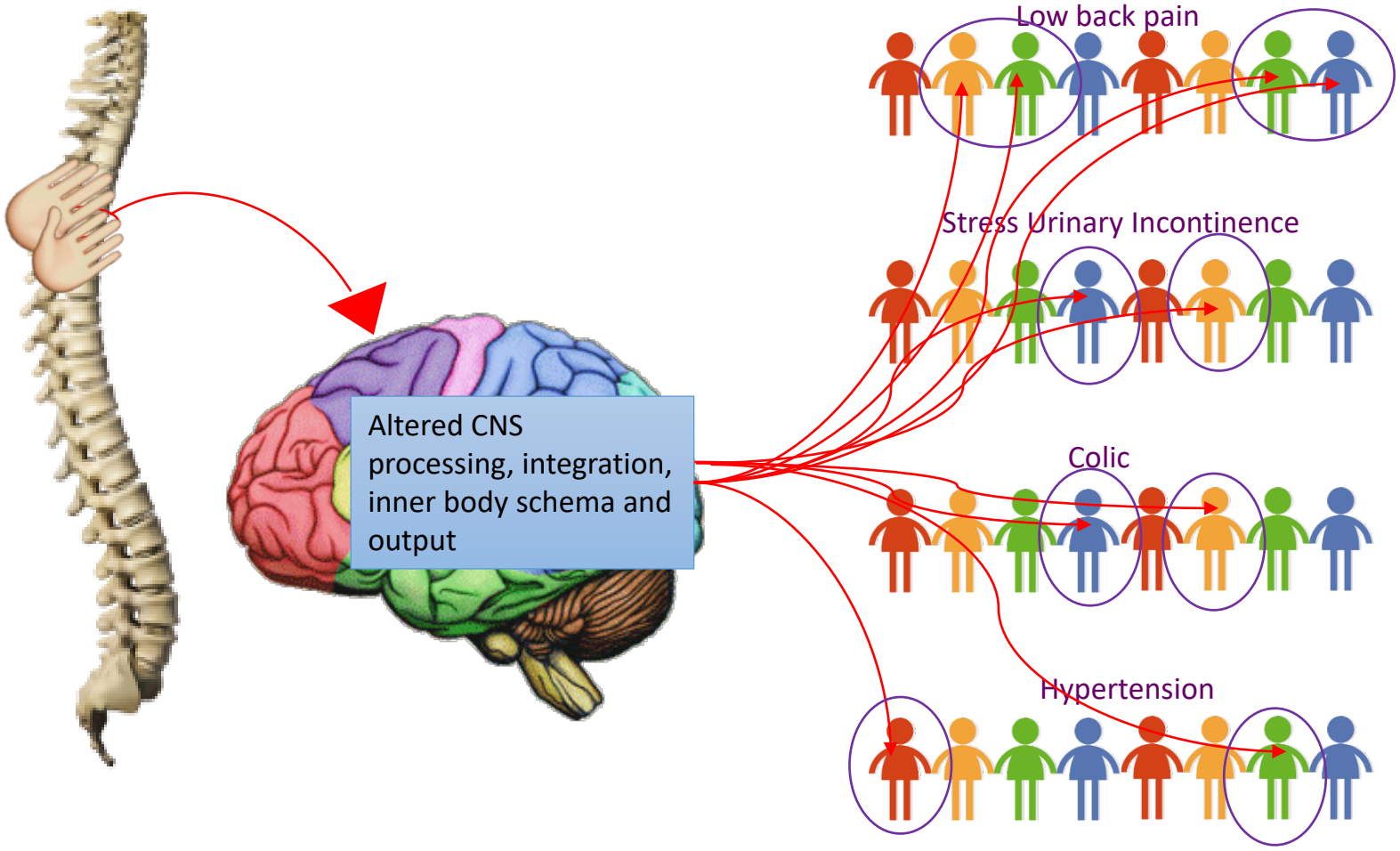
So what can you say?

- There is a growing body of evidence that suggests….
- There is emerging evidence that suggests….
- There is novel evidence that suggests….

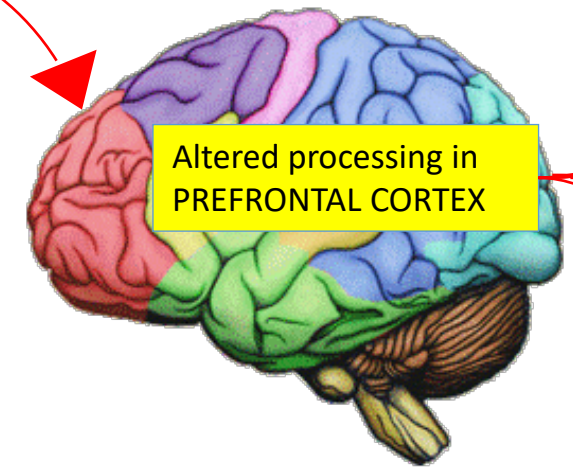
- We appear to improve the communication between the spine and the brain and this appears to affect the accuracy with which the brain can perceive what is going on inside and outside of the body

- Every animation we make has been VERY CAREFULLY worded to be accurate according to the limited evidence that we do have

RESPONDERS vs NON-RESPONDERS



RESPONDERS vs NON-RESPONDERS



Coronary artery disease

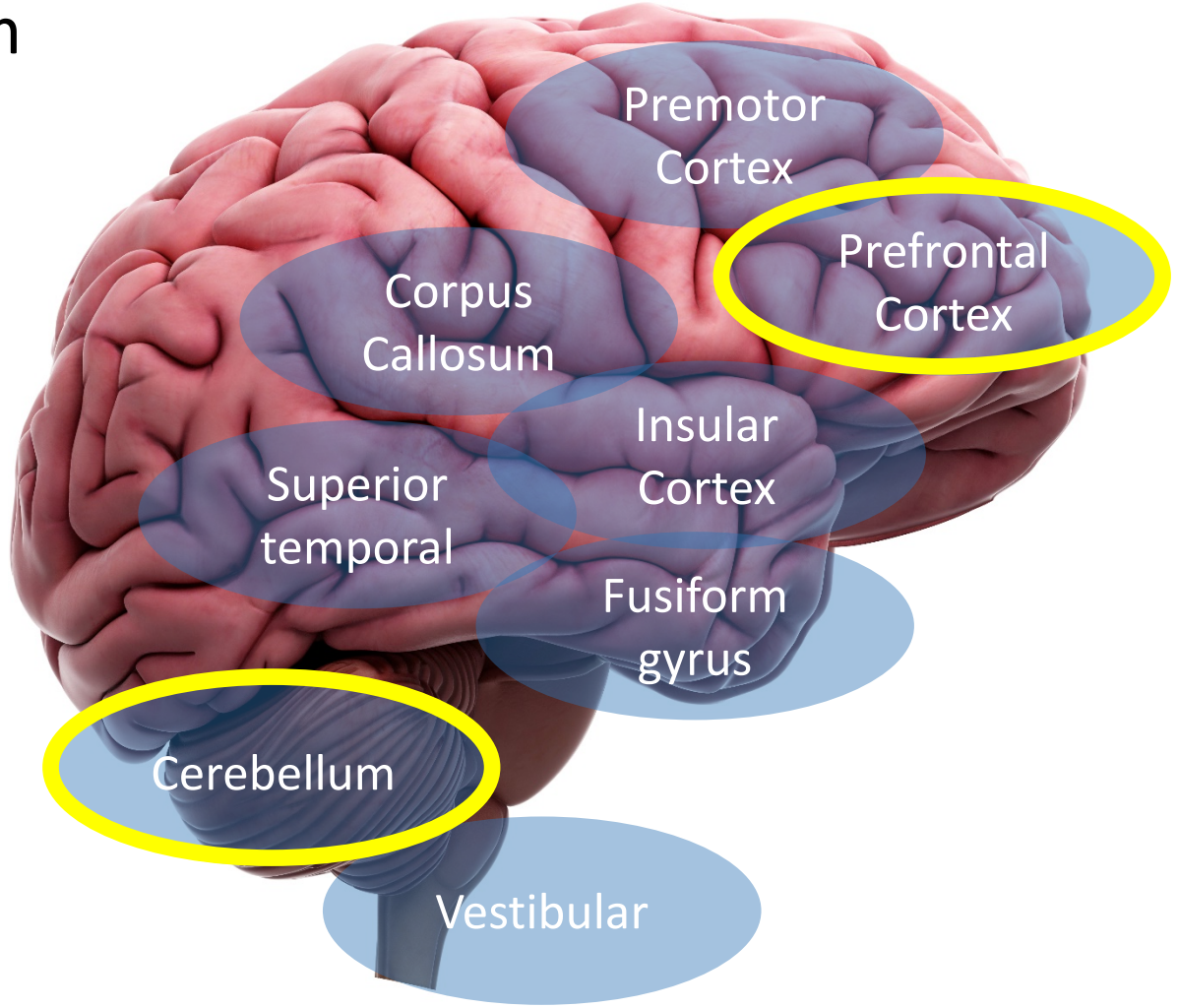
Autoimmune Disorders (e.g. MS)

Anxiety and/or Depression

ADHD and/or Autism

Example Autism

- Many brain regions known to be affected in kids with Autism



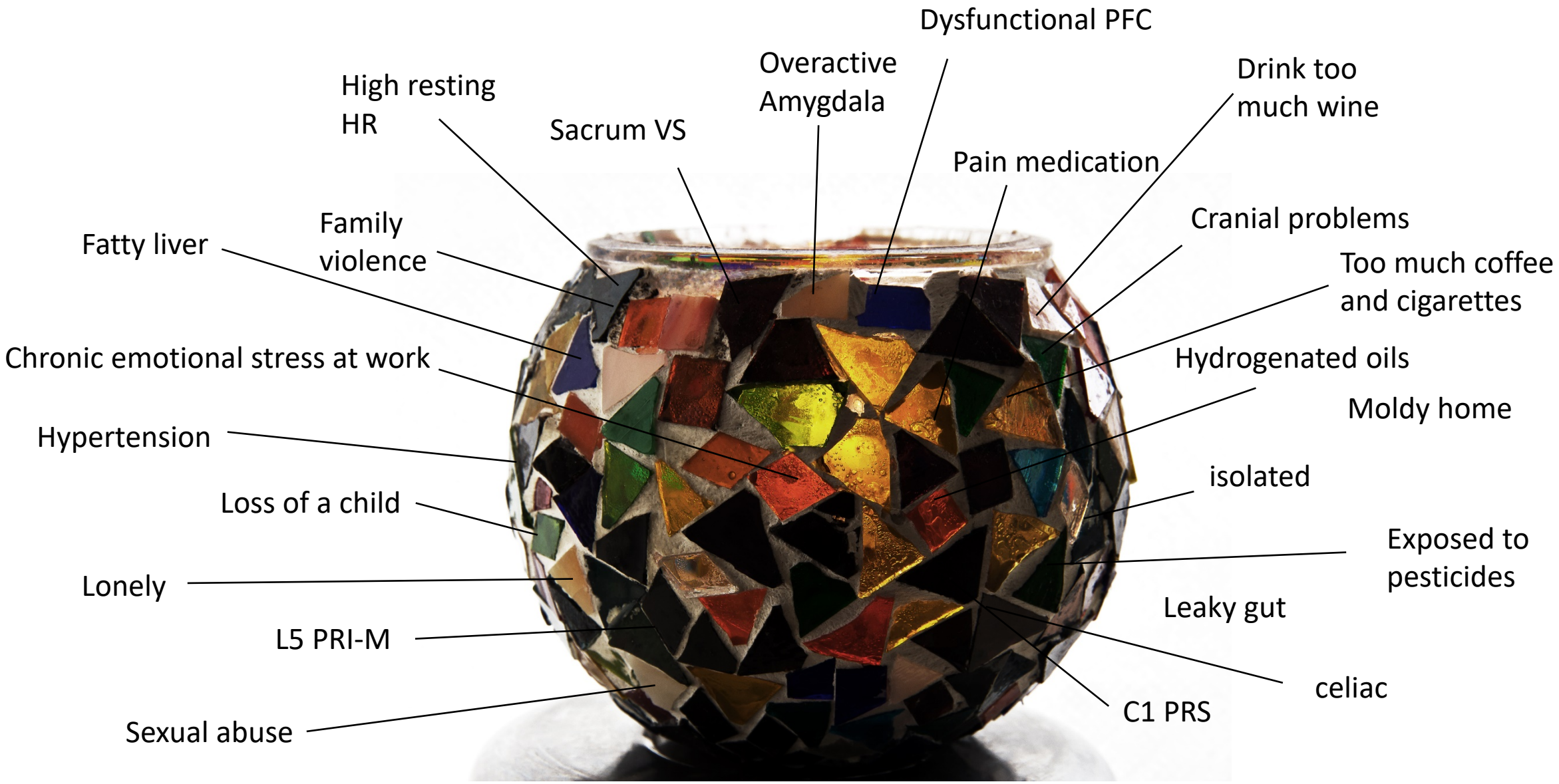
- Only PFC and Cerebellum known to be affected by adjustments

Your health status is like a Mosaic Vase

Shoenfeld Y, Isenberg DA. The mosaic of autoimmunity. Immunol Today. 1989/04/01/ 1989;10(4):123-126; Sharif K, Watad A, Coplan L, et al. The role of stress in the mosaic of autoimmunity: An overlooked association. Autoimmunity Reviews. 2018;17(10):967-983; Perricone C, Agmon-Levin N, Shoenfeld Y. Novel pebbles in the mosaic of autoimmunity. BMC Medicine. 2013/04/04 2013;11(1):101.

Your symptoms: chronic low back pain, headaches, very anxious, cry a lot, feel down, low motivation to do anything, tired all the time, diarrhea, bloating and constipation

Shoenfeld Y, Isenberg DA. The mosaic of autoimmunity. Immunol Today. 1989/04/01/ 1989;10(4):123-126; Sharif K, Watad A, Coplan L, et al. The role of stress in the mosaic of autoimmunity: An overlooked association. Autoimmunity Reviews. 2018;17(10):967-983; Perricone C, Agmon-Levin N, Shoenfeld Y. Novel pebbles in the mosaic of autoimmunity. BMC Medicine. 2013/04/04 2013;11(1):101.



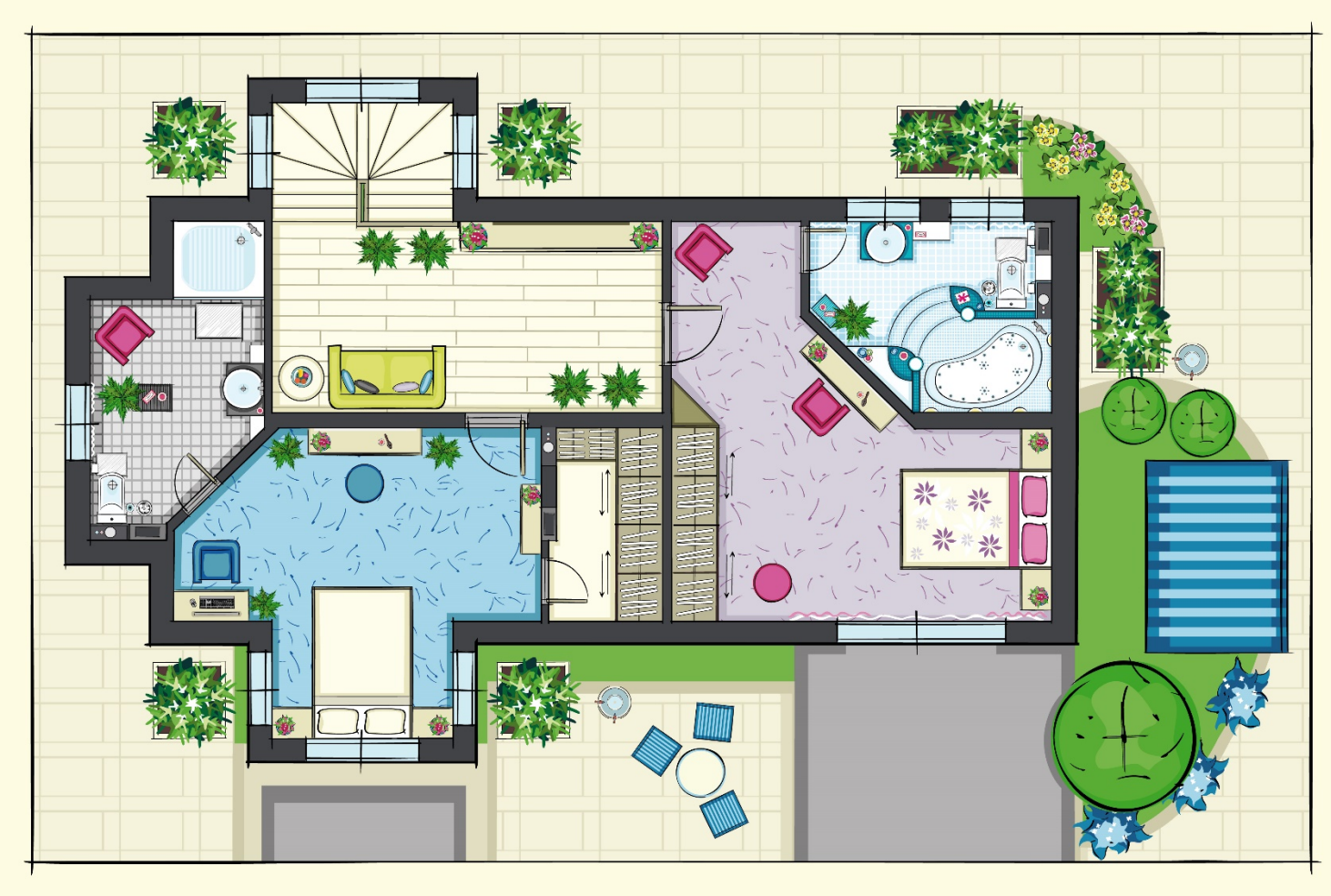
Shoenfeld Y, Isenberg DA. The mosaic of autoimmunity. Immunol Today. 1989/04/01/ 1989;10(4):123-126; Sharif K, Watad A, Coplan L, et al. The role of stress in the mosaic of autoimmunity: An overlooked association. Autoimmunity Reviews. 2018;17(10):967-983; Perricone C, Agmon-Levin N, Shoenfeld Y. Novel pebbles in the mosaic of autoimmunity. BMC Medicine. 2013/04/04 2013;11(1):101.

What can we NOT claim

- chiropractic can fix or cure ‘x’

Why can we NOT claim we can fix/cure any of these?

- Because there is no CLINICAL evidence!



How can you measure ‘stress’ effects on the body in practice?

- Stress questionnaires
- EEG
- HRV
- Resting HR

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ORIGINAL ARTICLE

Heart Rate Variability, Prefrontal Neural Function, and Cognitive Performance: The Neurovisceral Integration Perspective on Self-regulation, Adaptation, and Health

Julian F. Thayer, Ph.D. · Anita L. Hansen, Ph.D. · Evelyn Saus-Rose, Cand. Psychol. · Bjorn Helge Johnsen, Ph.D.

Published online: 8 May 2009

Abstract
Background In the present paper, we describe a model of neurovisceral integration in which a set of neural structures involved in cognitive, affective, and autonomic regulation are related to heart rate variability (HRV) and cognitive performance.
Methods We detail the pathways involved in the neural regulation of the cardiovascular system and provide pharmacological and neuroimaging data in support of the neural structures linking the central nervous system to HRV in humans. We review a number of studies from our group showing that individual differences in HRV are related to performance on tasks associated with executive function and prefrontal cortical activity. These studies include comparisons of executive- and nonexecutive-function tasks in healthy participants, in both threatening and nonthreatening conditions. In addition, we show that manipulating resting HRV levels is associated with changes in performance on executive-function tasks. We also examine the relationship between HRV and cognitive performance in ecologically valid situations using a police shooting simulation and a naval navigation simulation. Finally, we review our studies in anxiety patients, as well as studies examining psychopathy.
Conclusion These findings in total suggest an important relationship among cognitive performance, HRV, and prefrontal neural function that has important implications for both physical and mental health. Future studies are needed to determine exactly which executive functions are associated with individual differences in HRV in a wider range of situations and populations.

Keywords Executive function · Prefrontal · Heart rate variability · Cognition · Health

Introduction

Any comprehensive model of health must account for the complex mix of cognitive, affective, behavioral, and physiological factors that contribute to individual differences in health and disease. For example, individual differences in blood pressure, pulse pressure, and pulse wave velocity have been related to cognitive function across the lifespan [1,

Conclusion These findings in total suggest an important relationship among cognitive performance, HRV, and prefrontal neural function that has important implications for both physical and mental health. Future studies are needed to determine exactly which executive functions are associated with individual differences in HRV in a wider range of situations and populations.

Thayer, J. F., Åhs, F., Fredrikson, M., Sollers III, J. J., & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: implications for heart rate variability as a marker of stress and health. Neuroscience and Biobehavioral Reviews, 36(2), 747-756.

Heart rate variability great measure to monitor better PFC, & Amygdala function and emotional regulation

In addition to being linked to vmPFC and amygdala modulation, emotion regulation is linked to HRV (Appelhans and Luecken, 2006; Thayer and Brosschot, 2005). Individuals with greater emotion regulation ability have been shown to have greater levels of resting HRV (Appelhans and Luecken, 2006; Thayer and Lane, 2009). In addition, during successful performance on emotion regulation tasks HRV appears to be increased (Butler et al., 2006; Ingjaldsson et al., 2003; Smith et al., 2011).

Thayer JF, Åhs F, Fredrikson M, Sollers III JJ, Wager TD. A meta-analysis of heart rate variability and neuroimaging studies: implications for heart rate variability as a marker of stress and health. Neurosci Biobehav Rev. 2012;36(2):747-756. p.750

frontiers in Psychology

ORIGINAL RESEARCH
published: 10 March 2015
doi: 10.3389/fpsyg.2015.00261

Resting heart rate variability predicts self-reported difficulties in emotion regulation: a focus on different facets of emotion regulation

DeWayne P. Williams[1], Claudia Cash[1,2], Cameron Rankin[1], Anthony Bernardi[1], Julian Koenig[1] and Julian F. Thayer[1]*

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The *Model of Neurovisceral Integration* suggests that vagally mediated heart rate variability (vmHRV) represents a psychophysiological index of inhibitory control and thus, is associated with emotion regulation capacity. Over the past decade, growing empirical evidence supports this notion, showing that those with higher resting vmHRV can regulate negative emotions more adequately. However, to our knowledge, no study has previously examined how resting vmHRV may relate to everyday perceived difficulties in emotion regulation. The present study attempts to examine such relationship in 183 undergraduate students (98 female, 60 minority, mean Age = 19.34). Resting vmHRV was collected during a 5-min resting baseline period, and everyday difficulties in emotion regulation were assessed using the Difficulties in Emotion Regulation Scale (DERS). Controlling for potential covariates (including both trait anxiety and rumination), results revealed a negative relationship between resting vmHRV and DERS such that lower resting vmHRV was associated with greater difficulties in emotional regulation, especially a lack of emotional clarity and impulse control, as indicated by the respective subscales of the DERS. These findings provide further evidence for the *Neurovisceral Integration Model*, suggesting that emotion regulation and autonomic regulation share neural networks within the brain. Moreover, the present study extends prior research by highlighting two distinct facets of emotion regulation (impulse control and emotional clarity) that should be of particular interest when investigating the link between emotion regulation, resting vmHRV, and related health outcomes including morbidity and mortality.

Williams, D. P., Cash, C., Rankin, C., Bernardi, A., Koenig, J., & Thayer, J. F. (2015). Resting heart rate variability predicts self-reported difficulties in emotion regulation: a focus on different facets of emotion regulation. Frontiers in Psychology, 6, 261.

PLOS ONE

RESEARCH ARTICLE

Investigating the Associations of Self-Rated Health: Heart Rate Variability Is More Strongly Associated than Inflammatory and Other Frequently Used Biomarkers in a Cross Sectional Occupational Sample

Marc N. Jarczok[1], Marcus E. Kleber[1], Julian Koenig[2]*, Adrian Loerbroks[3], Raphael M. Herr[4,1], Kristina Hoffmann[1], Joachim E. Fischer[1], Yael Benyamini[5], Julian F. Thayer[2,1]

1 Mannheim Institute of Public Health, Social and Preventive Medicine, Mannheim Medical Faculty, Heidelberg University, Mannheim, Germany, **2** The Ohio State University, Department of Psychology, Columbus, Ohio, United States of America, **3** Institute of Occupational and Social Medicine, Medical Faculty, Heinrich-Heine-University Düsseldorf, Düsseldorf, Germany, **4** Department of Clinical Psychology, University of Amsterdam, Amsterdam, The Netherlands, **5** Bob Shapell School of Social Work, Tel Aviv University, Tel Aviv, Israel

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Jarczok, M. N., Kleber, M. E., Koenig, J., Loerbroks, A., Herr, R. M., Hoffmann, K., . . . Thayer, J. F. (2015). Investigating the associations of self-rated health: heart rate variability is more strongly associated than inflammatory and other frequently used biomarkers in a cross sectional occupational sample. PLoS ONE [Electronic Resource], 10(2), e0117196.

Journal of Neuroimmunology 260 (2013) 28–36

Contents lists available at SciVerse ScienceDirect

Journal of Neuroimmunology

journal homepage: www.elsevier.com/locate/jneuroim

Vagal nerve activity as a moderator of brain–immune relationships

Hideki Ohira[a,*] Masahiro Matsunaga[b] Takahiro Osumi[c] Seisaku Fukuyama[d,e] Jun Shinoda[d]

ABSTRACT

We investigated whether vagal tone, as assessed by heart rate variability (HRV), moderates the neural correlates of immune and physiological responses to acute stress. Participants with low and high baseline HRV underwent a reversal learning task as an acute stressor. Natural killer cells, norepinephrine, and adrenocorticotropic hormone in peripheral blood changed with acute stress in the high HRV group only. Activity in the prefrontal cortex and striatum correlated with the immune and physiological indices in the high HRV group. High vagal tone may reflect more flexible top-down brain regulation of immune and physiological activity.

Heart rate variability

Ohira, H., Matsunaga, M., Osumi, T., Fukuyama, S., Shinoda, J., Yamada, J., & Gidron, Y. (2013). Vagal nerve activity as a moderator of brain–immune relationships. *Journal of Neuroimmunology, 260*(1-2), 28-36.

associated with HF-HRV. Importantly, the regions that have been associated with various immune functions as well as those identified as being associated with HRV are part of a medial prefrontal-brainstem network that Lane and Wager (2009) have suggested as being critically involved in the regulation of the autonomic nervous system, the endocrine system and the immune system as well as the related functions of pain, emotion, and behavioral regulation.

Kul'chyns'kyi, A. B., Kyjenko, V. M., Zukow, W., & Popovych, I. L. (2017). Causal neuro-immune relationships at patients with chronic pyelonephritis and cholecystitis. correlations between parameters EEG, HRV and white blood cell count. Open Medicine, 12(1), 201-213.

Younes et al. Chiropractic & Manual Therapies (2017) 25:33
DOI 10.1186/s12998-017-0167-6

Chiropractic & Manual Therapies

RESEARCH Open Access

Effect of spinal manipulative treatment on cardiovascular autonomic control in patients with acute low back pain

Mohamed Younes[1,2], Karine Nowakowski[3], Benoit Didier-Laurent[3], Michel Gombert[3] and François Cottin[1,2*]

Abstract

Background: This study aimed to quantify the effect of spinal manipulative treatment (SMT) from an analysis of baroreflex, systolic blood pressure and heart rate variability (HRV) on patients with acute back pain. It was hypothesized that SMT would increase the parasympathetic cardiovascular autonomic control.

Methods: Twenty-two patients with acute back pain were randomly divided into two groups: one receiving sham treatment (Sham) and the other receiving SMT. Recordings were completed during the first day and the seventh day, immediately before and after treatment on both days. ECG and systolic blood pressure were continuously recorded to compute cardiovascular variability and baroreflex sensitivity components. The perceived level of pain was measured with the numeric pain scale (NPS) 48 h before, just before and just after each treatment. The NPS ranged from 0 to 100% (peak of pain before treatment). ECG and systolic blood pressure recordings were analyzed in time frequency domain using the Smoothed pseudo Wigner-Ville distribution.

Results: Root mean square of the successive differences, high frequency power of the heart rate variability, and high frequency baroreflex sensitivity differences between post and pre tests were higher in the SMT group than in the Sham group ($p < 0.01$), whereas no differences were observed with the other heart rate variability components. Also, no differences were observed with the systolic blood pressure components. Although the estimated pain scale values decreased over time, no difference was observed between the SMT and Sham groups.

Conclusions: This seems to be the first study to assess the effect of SMT on both heart rate variability and baroreflex sensitivity in patients with acute back pain. SMT can be seen to provoke an increase in parasympathetic control known to relate to a person's healthy state. Thus, cardiovascular variability analysis may be a useful tool for clinicians to quantify and objectify the beneficial effects of spinal manipulation treatment.

Keywords: Baroreflex, Blood pressure, Heart rate, Autonomic nervous system, Back pain, Spinal manipulation, Randomized study, Pain scale

Younes, M., Nowakowski, K., Didier-Laurent, B., Gombert, M., & Cottin, F. (2017). Effect of spinal manipulative treatment on cardiovascular autonomic control in patients with acute low back pain. Chiropractic & Manual Therapies, 25(1), 33.

HRV Summary

- HRV correlates with your self-reported health level
- HRV correlates with your ability to regulate your emotions
- HRV correlates with regulation of immune and physiological activities
- HRV is a measure of 'vagal tone' (the vagus nerve function)

- The vagus nerve is the parasympathetic NS (the healing component)

- **HRV correlates with PFC activity**, meaning it correlates with your PNS function (your healing functions of the body), regulation of emotions, relates to self-reported measures of health, relates to regulation of immune and physiological function

- **Chiropractors affect the PFC!!**
- **Chiropractic affects HRV (parasympathetic response)!!**

Thank You

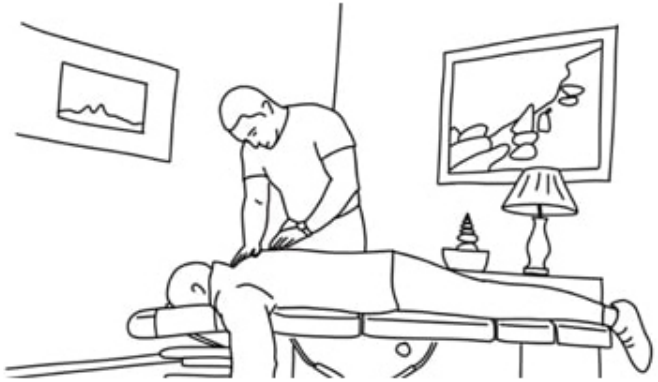
DR. HEIDI HAAVIK

ENLIGHTENING THE WORLD ABOUT THE SCIENCE OF CHIROPRACTIC

HAAVIK RESEARCH

Introduction to Chiropractic Care

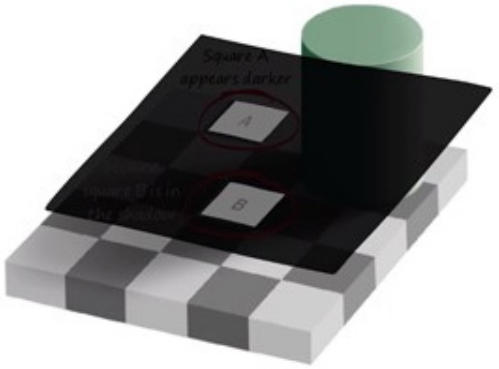
The introduction to chiropractic video series is the perfect way to gain an understanding of why chiropractic care may help you and your family.



The Beginners Guide to Chiropractic

In this first introductory video we explore what chiropractic is all about, and how it works, then we briefly explore the evidence informed effects of chiropractic care.

View video ›



How the Brain Perceives the World

Did you know that your brain and central nervous system are constantly changing? It's quite amazing – from one day to the next your brain is not the same.

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The Beginners Guide to Chiropractic

The Beginners Guide to Chiropractic

The word chiropractic derives from the Greek words "cheir", meaning hand, and "practikos" meaning skilled in or concerned with. The origin of the word chiropractic can be traced back to D.D. Palmer who coined it in 1895 when he founded chiropractic.

> Chiropractic care is really about total health and wellbeing

What does a Chiropractor do?

A chiropractor is a healthcare professional who specializes in the health and function of the spine and nervous system. Because of this focus on the spine, many people think chiropractors can only help with problems such as back pain, neck pain and headaches. They can often help with these issues but there is much more to chiropractic than just pain.

This is the first video in our animated series "Introduction to Chiropractic". In this video, we outline what a chiropractor does, then we briefly explore the effects of care. It is a perfect one to watch for anyone that is curious about chiropractic care, and how it can help their family.

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The Beginners Guide to Chiropractic

In this first introductory video we explore what chiropractic is all about, and how it works, then we briefly explore the evidence informed effects of chiropractic care.

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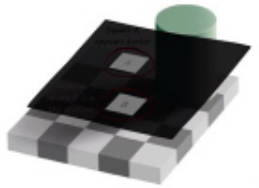


How the Brain Perceives the World

Did you know that your brain and central nervous system are constantly changing? It's quite amazing – from one day to the next your brain is not the same.

View video ›

Break the Pain Cycle

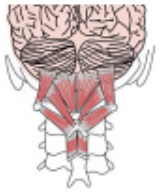
Did you know that pain is created in your brain to let you know that something is not ok within your body? Feeling pain is good because it is actually helpful and informative.

View video ›

Chiropractic Care and Migraines

Did you know that 1 in 6 people in the world experience migraines regularly? The World Health Organisation consider them to be the most debilitating of all neurological disorders.

View video ›



Chiropractic Affects your Brain

Your brain receives information about your body from the environment and your organs. Did you know that the muscles in your body are also sensory organs?

View video ›

What is that Pop?

If you have been adjusted before by a chiropractor you may have noticed a strange popping sound. Don't worry – it is just the formation of gas within a joint.

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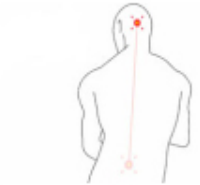

Lower Back Pain

Scientists have worked out that at any one time, over 500,000,000 people around the world are suffering from low back pain and it is now the leading cause of disability worldwide.

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Growing Pains

We've all heard of growing pains right? But did you know that what we call growing pains aren't associated with growing? So they're not actually growing pains at all.

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Pain and the Immune System

Research studies have shown that the way you feel pain all depends on what's going on for you – and most importantly – what you think and feel about the situation.

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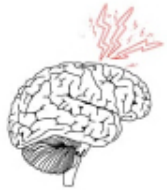

Chiropractic and Headaches

Headaches are a sign that something is not right. Your brain will create for you the sensation of pain if it thinks there is something wrong or if there is a potential problem.

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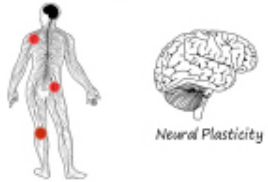

Pain is Created in Your Brain

Did you know that the scientists now know that the feeling of pain is something your brain decides that you should experience – if it believes that there is a problem?

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Chronic Pain

Chronic pain is the second-most common reason people see a doctor and miss work. More than one-third of people with chronic pain become disabled by their pain to some degree.

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Chiropractic Research

Research summary articles to read, download and print (members only) all backed by the latest scientific research studies.

Chronic Pain

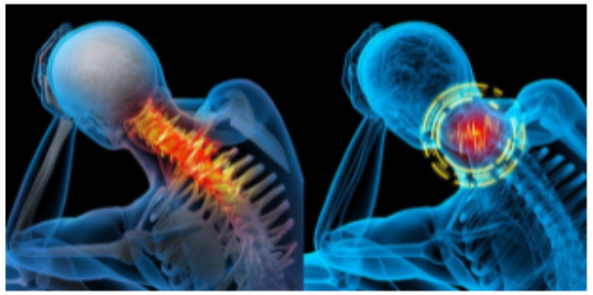
Chronic pain that has persisted for more than 3 months is no longer protective, nor informative. So, what is chronic pain and what can you do about it?

Read more »

Pain is in the Brain

Sometimes pain persists long after tissue damage has actually healed. When pain persists for more than three months we call this chronic pain.

Read more »



Neck Pain

Up to half the world's population suffers from neck pain at some stage. For some, one big problem is that it just keeps coming back, or becomes chronic.

Read more »

UNDERSTANDING PAIN

Dr. Kelly Holt
BSc, BSc(Chiro), PGDipHSc, PhD

Dr. Heidi Haavik
BSc(Physiol), BSc(Chiro) PhD

Experiencing pain is normal. Everyone experiences pain now and then.[1] Pain is supposed to be protective to make you stop doing things that may be dangerous.[2] But chronic pain that has persisted for more than 3 m hs is no longer protective, nor tive.[3] So, what is chronic pain and out it if you suffer from it?

PAIN IS CREATED IN THE BRAIN

Dr. Kelly Holt
BSc, BSc(Chiro), PGDipHSc, PhD

Dr. Heidi Haavik
BSc(Physiol), BSc(Chiro) PhD

Did you know that scientists now know the feeling of pain is something your brain decides you should experience if it believes there is some tissue damage in your body?[1] In fact, your brain can decide that you should feel pain even if it only thinks there is a potential threat of tissue damage!!![2-5]

It may seem strange, but it's totally up to your brain to decide whether you should feel pain or not. Your brain may decide you should experience pain even if you have no actual tissue damage yet,[6] or your brain may not create the feeling of pain for you when tissue damage has actually occurred![7 8]

heals the problem.[1] This pain is helpful and informative.[1] If we listen to our body these pain experiences can be a good thing.

But for some people, pain can persist even after the initial injury that caused it has healed.[9 11 12] And for some people, the pain

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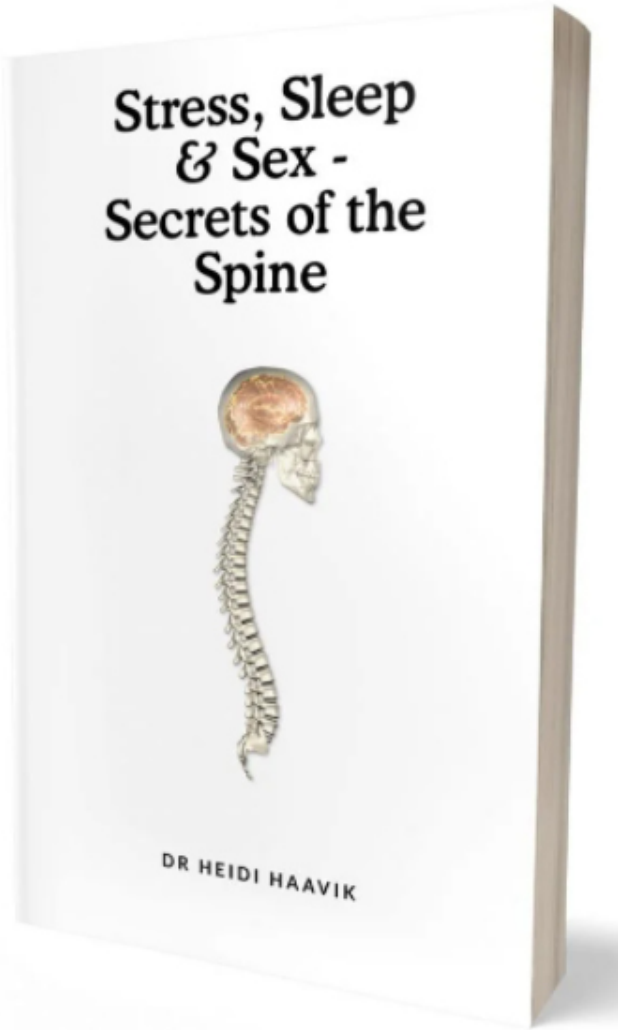
NECK PAIN AND FALLS RISK

Dr. Kelly Holt
BSc, BSc(Chiro), PGDipHSc, PhD

Dr. Heidi Haavik
BSc(Physiol), BSc(Chiro) PhD

Neck pain is very common throughout the world.[1] Up to half of all people around the world suffer from neck pain at some stage each year.[2-5] For some people, one big problem with neck pain is that it just keeps coming back, or becomes chronic, and may even increase their risk of suffering from a fall.[2 4 6 7]

Scientists know that your brain uses sensory information from your muscles and joints around your spine to help control your balance and posture and to make sure you're moving properly.[1,2] When your brain takes sensory information and uses it to help guide movements and control muscles we call this sensorimotor function.[8] One particular study looked at whether neck pain has an impact on proper sensorimotor function in older people.[7] In this study, the researchers ran a whole lot of tests of sensorimotor function, like how well the study participants controlled the movement of their eyes and how good their balance was, and they took into account their age and other conditions that they suffered from.[7]



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