56th Annual Scientific Meeting

THE SCIENCE OF SELF-REGULATION AND RESILIENCE THROUGH BIOFEEDBACK

May 13-16, 2026 | Baltimore

applied psychophysiology & biofeedback

aapb

an international society for mind-body research, health care, and education

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Saturday, May 16

4:15 pm -5:45 pm

BOS26: **Autonomic Dysfunction and Sensitized Chronic Pain**

Presented By: Peter Behel, MA, BCB; JP Ginsberg, PhD; Meghan Varner, BS, DPT

Session Type: 90-minute Symposium/Break Out Session

CE Credits: 1.5

"Sensitized Chronic Pain (SCP) and Autonomic Nervous System (ANS)-Hypothalamic/Pituitary/Adrenal (HPA) Axis Stress Responding"

Plan of the Presentation

i. An Early Model of Sensitized Chronic Pain
ii. Nociplastic Pain
iii. Functional Neurological Disorder (FND) and Sensitized Chronic Pain (SCP)
iv. Hypopituitarism Effects of SCP on HPA
v. Interventions that Shift ANS Towards Restoration of Normal HPA Function
    a. Autonomic Rehabilitation
        ?Photobiomodulation?
        Self-regulation (HRVB, 'Coherent Breathing', etc.)

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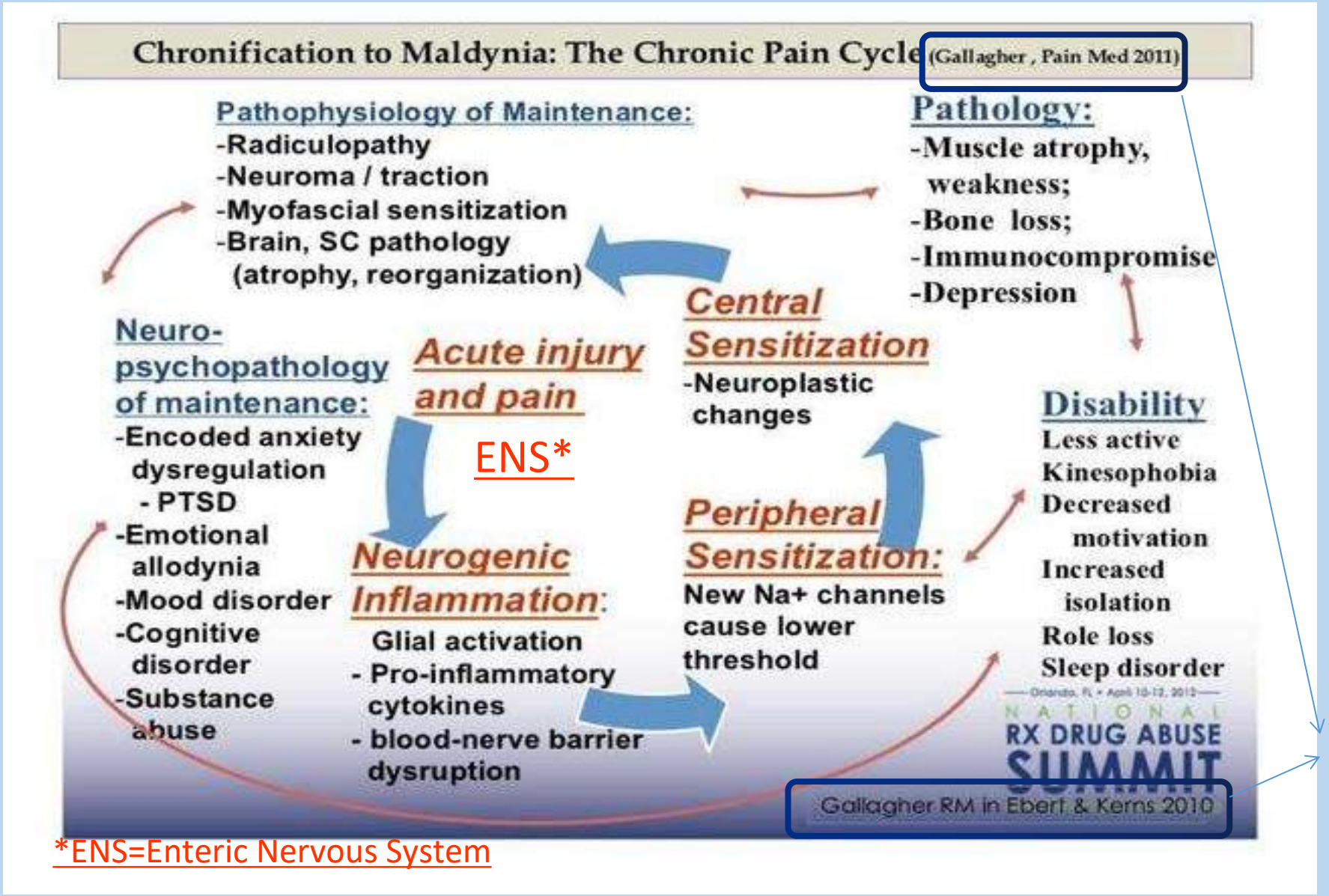
AN EARLY MODEL OF SENSITIZED CHRONIC PAIN



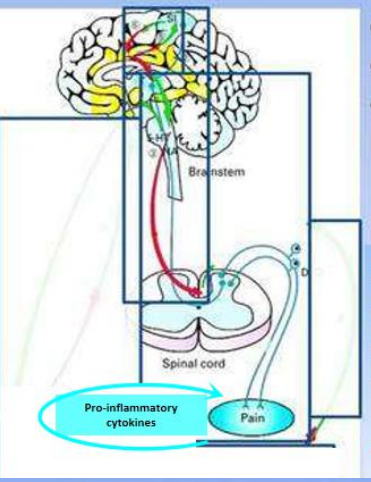
*ENS=Enteric Nervous System

(Note: This model was produced 15+ years ago, before the definition of nociplastic pain was introduced by IASP)

Chronic pain may be due to lasting tissue damage or psychiatric factors.

ACUTE NOCICEPTIVE PAIN NEGATIVE FEEDBACK (SHUT-OFF)

Blackburn-Munro et al (2001). p. 1013.

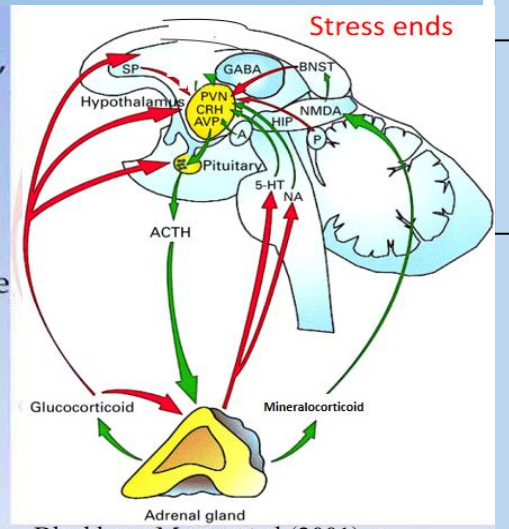


- Inflammation / nerve injury stimulate nociceptive information to dorsal horn
- Ascends to brainstem, gated in thalamus
- Cognitive appraisal in SI cortex
- Acute pain increases arousal via sympathetic and GC routes (excitatory reciprocal link between somatosensory and limbic cortices)

- →**Stress response**
- →Descending pain modulation
- → Pain ends
- → HPA stress response negative feedback signals to cortex
- →**Pain and stress response end**

NORMAL HPA STRESS RESPONSE NEGATIVE FEEDBACK (SHUT-OFF)

- Glucocorticoid → negative feedback via GR of HC, PVN, P, and AC
  - ↓CRH, AVP release
  - ↓ACTH release
  - ↓GC
  - ↓IL-1β
- Mineralocorticoid → negative feedback via GR in HC
  - ↑Glu →GABA↑
- Brainstem 5-HT/NE release
- Amy
- P ineal?
- neurokinin SP?



Blackburn-Munro et al (2001). p. 1010.

Chronic Pain includes a physiological stress response

STRESS OF CHRONIC PAIN CAUSES CENTRAL SENSITIZATION AND DEPRESSION

- 'HPA drive' →
- **Pain does not end →**
- **Stress does not end →**
- **'HPA overdrive' →**
- **Loss of GC inhibition of pro-inflammatory cytokines**
- **Proliferation of peripheral inflammation**
- **Heightened pain**
- **Disinhibition of descending cortical pain modulation ('nociceptive braking')**
- **Depletion of catecholaimes – noradrenaline from locus coeruleus and dopamine from hypothalamus**
- **Depressed behavior and mood**
- **"THE IMMUNE RUNAWAY TRAIN"**

"Dysfunction of the hypothalamo-pituitary–adrenal axis has been implicated in a variety of chronic pain conditions and might also be associated with increased risk of developing mood disorders."

Blackburn-Munro, G., & Blackburn-Munro, R. E. (2001). Chronic pain, chronic stress and depression: coincidence or consequence?. Journal of neuroendocrinology, 13(12), 1009-1023.

Blackburn-Munro, G. (2004). Hypothalamo-pituitary-adrenal axis dysfunction as a contributory factor to chronic pain and depression. Current pain and headache reports, 8(2), 116-124.

Nociceptive pain

• physiological response to a pain generating stimulus,

Neuropathic pain

• pathological abnormalities in pain receptor structure, neural transmission, and/or processing of a pain generating stimulus

Nociplastic pain

• introduced by the International Association for the Study of Pain (IASP) in 2017

• Definition: “Pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain.”

https://www.iasp-pain.org/Education/Content.aspx?ItemNumber=1698].

Characteristics of nociplastic pain:

- Pain originated in times of stress
- Pain without an injury
- Inconsistent symptoms
- Large number of symptoms
- Symptoms spread/move
- Triggered by stress
- Symmetrical symptoms
- Delayed onset
- Childhood adversity
- Lack of a physical diagnosis

IASP
INTERNATIONAL ASSOCIATION FOR THE STUDY OF PAIN

Fitzcharles, M. A., Cohen, S. P., Clauw, D. J., Littlejohn, G., Usui, C., & Häuser, W. (2021). Nociplastic pain: towards an understanding of prevalent pain conditions. *The Lancet*, *397*(10289), 2098-2110.

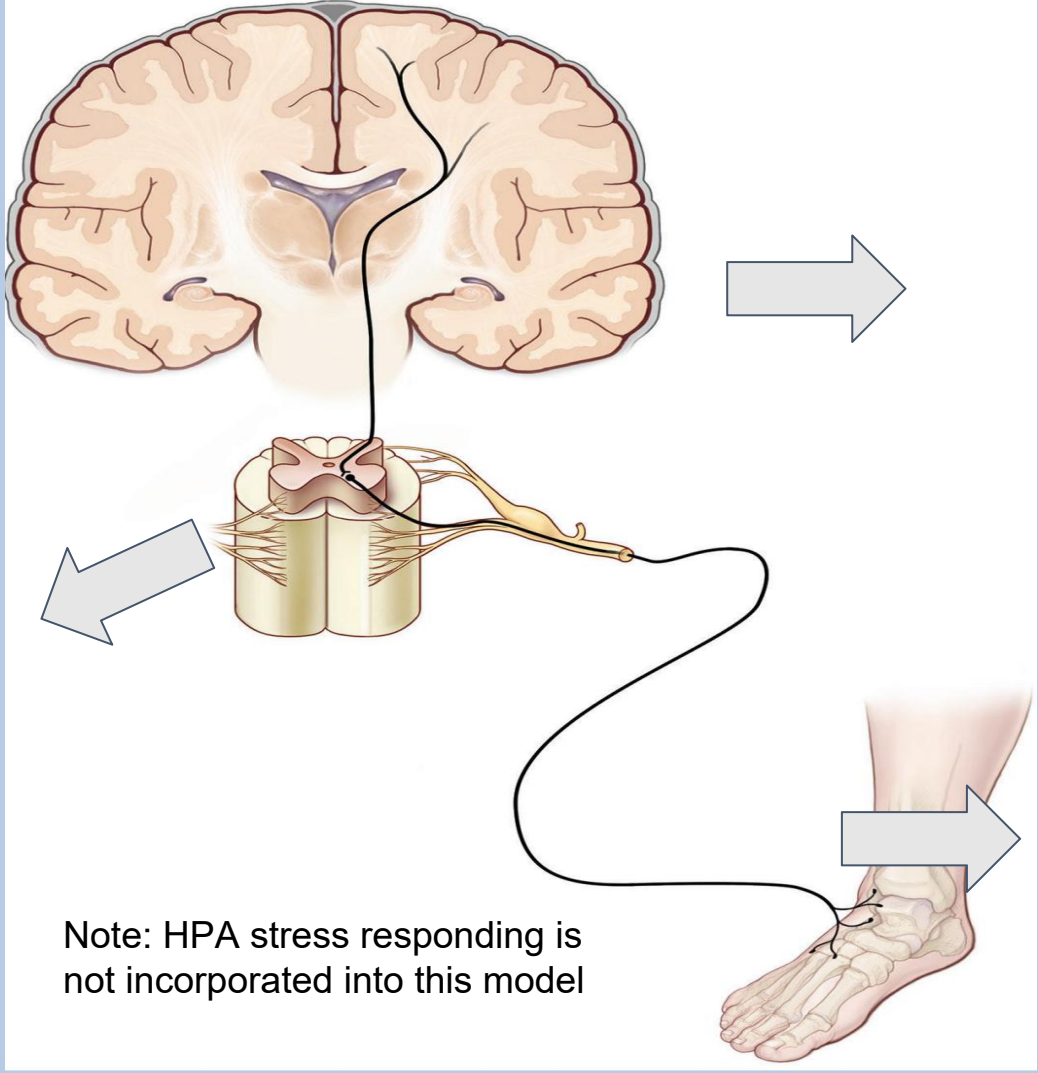
Features of nociplastic pain

Combined peripheral and central pain sensitization

- Hyper-responsiveness to painful and non-painful sensory stimuli
- Associated features
- Fatigue
- Sleep disturbance
- Cognitive disturbances
- Hypersensitivity to environmental stimuli
- Anxiety and depressed mood

Spinal mechanisms

- Regional clustering and convergence of signals from different pain loci
- Spinal cord reorganisation
- Amplified spinal reflex transmission
- Diminished spinal inhibition
- Wind-up and temporal summation
- Glial cell activation



Supraspinal mechanisms

- Hyper-responsiveness to pain stimuli
- Hyperactivity and connectivity in and between brain re gi ons involved in pain
- Increased activity of brain regions involved in pain inhibition (ie, descending inhibitory pathways)
- Elevated cerebrospinal fluid substance P and glutamate concentrations, decreased GABAergic transmission
- Changes in the size and shape of grey and white matter regions involved in pain processing
- Glial cell activation

Peripheral features

- Minor local muscle pathology (eg, changes in pH, muscle fibre composition, and latent and active trigger points)
- Peripheral sensitisation (eg, expansion of receptive fields, elevated cytokine and
- chemokine concentrations)
- Hyperalgesia, dysesthesia, and allodynia
- Localised or diffuse tenderness, or both

Fitzcharles, M. A., Cohen, S. P., Clauw, D. J., Littlejohn, G., Usui, C., & Häuser, W. (2021). Nociplastic pain: towards an understanding of prevalent pain conditions. *The Lancet*, *397*(10289), 2098- 2110.

Sensitized Chronic Pain

- Sensitized chronic pain (central sensitized pain) is a pain condition characterized by persistent pain that *arises* in the central nervous system (CNS)
- Chronic pain is long standing pain that persists beyond the usual recovery period or occurs along with a chronic health condition
- Whether nociceptive, neuropathic, and/or nociplastic in origin, sensitization can occur when the pain becomes chronic.
- Causes: Chronic nociceptive nociceptive and/or nociplastic pain; infections and some medications
- Symptoms: Hypersensitivity to touch, temperature, or movement; allodynia (pain in response to normally non-painful stimuli, such as light touch); hyperpathia (exaggerated pain response to painful stimuli)
- Diagnosis: Physical examination, medical history; neurological tests; Imaging studies (e.g., MRI, CT scan)
- Treatment: Medications (e.g., anticonvulsants, antidepressants, opioids); physical therapy, occupational therapy; interventional pain management, psychological interventions (e.g., psychotherapy, psychophysiological therapy)
- Associated with a wide range of pain, medical, and mental health diagnoses
- Not all chronic pain has an identifiable cause
    - Pain can persist even after a known underlying cause is treated and resolved

https://www.ninds.nih.gov/health-information/disorders/central-pain-syndrome

Central sensitization, chronic pain, and other symptoms: Better understanding, better management Mary M. Volcheck, Stephanie M. Graham, Kevin C. Fleming, Arya B. Mohabbat, Connie A. Luedtke Cleveland Clinic Journal of Medicine Apr 2023, 90 (4) 245-254; DOI: 10.3949/ccjm.90a.22019

Dydyk AM, Chiebuka E, Stretanski MF, et al. Central Pain Syndrome. [Updated 2025 May 3]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK553027/

Nijs J, George S, Clauw D et al. (2021). Central sensitisation in chronic pain conditions: latest discoveries and their potential for precision medicine. *The Lancet Rheumatology,* 3, e383-e392

Sensitized Chronic Nociplastic Pain and FND

https://neurosymptoms.org/en/

“These are genuine symptoms which relate to a problem in nervous system functioning rather than disease.”

Jon Stone, PhD
Professor
School of Neurological and Cardiovascular Sciences
Ted M Burns Prize for Humanism in Neurology
https://orcid.org/0000-0001-9829-8092
View Scopus Profile

FND Guide
neurosymptoms.org

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Home / Symptoms / Common Associated Symptoms / Pain

Pain

What is Persistent/Chronic Pain?

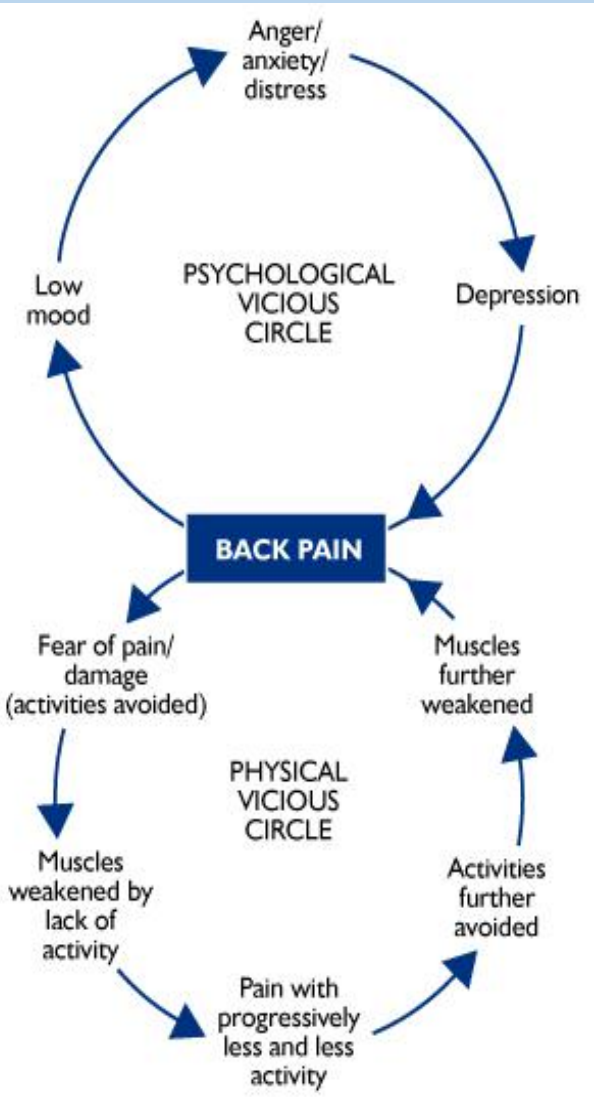
Persistent or Chronic Pain is a very common symptom in people with FND. It is important to recognise that if you do have chronic pain as part of your illness, the two things are probably related.

We have learnt a lot about the mechanisms of chronic pain in the last 10-20 years. Chronic (long-lasting) pain is different to the type of acute (sudden) pain that you get if you cut your finger. It has different mechanisms in the body.

Persistent pain is commonly due to an increase in the ‘volume knob’ in the pain pathways in the nervous system – both the brain and spinal cord. When pain comes from the brain like this is also called chronic primary pain, and also nociplastic plain (pronounced NO-SEE-PLASTIC pain). Common types of nociplastic pain include Fibromyalgia, most people with chronic back and neck pain, and many others.

Persistent/Chronic Pain – also called Nociplastic Pain is a medical condition in its own right

People with chronic pain suffering chronic pain can’t just ‘turn the volume knob down’. The pain is ACTUALLY happening, and at a level in the nervous system out of immediate control.



Funded by Scottish Government
The Scottish Government

https://neurosymptoms.org/en/symptoms/common-associated-symptoms/pain/

Chronic pain in the context of Functional Neurological Disorder (FND)

“Functional neurological disorder (FND) is characterised by neurological symptoms, such as seizures and abnormal movements. Despite its significance to patients, the clinical features of chronic pain in people with FND, and of FND in people with chronic pain, have not been comprehensively studied. Conclusions Pain symptoms and pain-related diagnoses are common in FND. Classification systems and treatments should routinely consider pain as a comorbidity in patients with FND.”

Steinruecke, M., Mason, I., Keen, M., M cWhirter, L., Carson, A. J., Stone, J., & Hoeritzauer, I. (2024). Pain and functional neurological disorder: a systematic review and meta-analysis. *Journal of Neurology, Neurosurgery & Psychiatry, 95(9), 874-885.*

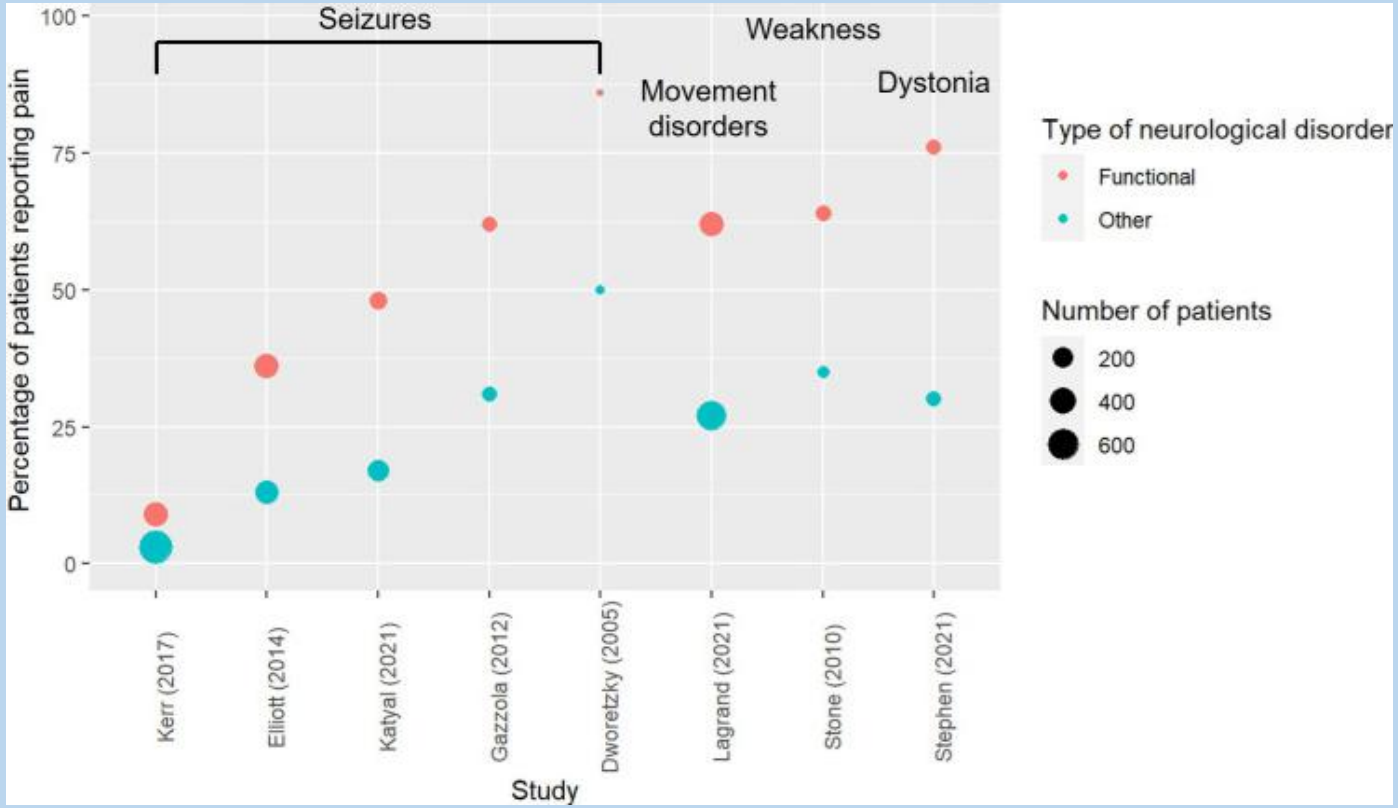
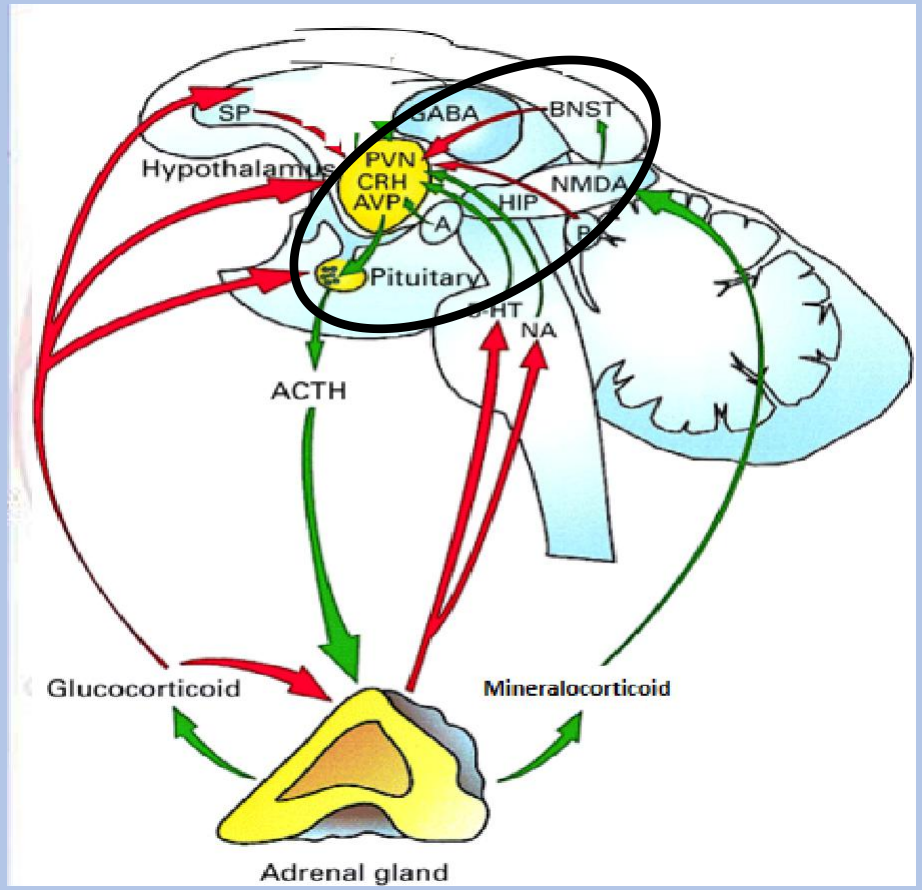


Figure 2 Proportion of patients with FND reporting chronic pain compared with patients with other neurological conditions. FND, functional neurological disorder.

Functional Secondary Hypopituitarism (HP)

- Temporary pituitary insufficiency or reduction in pituitary hormone production
- Caused by extreme or severe systemic factors, not physical tumor or structural damage. Adaptive shutdown of the pituitary due to chronic disease, inflammation, malnutrition, acute psychological stress, prolonged exercise, physical trauma, major surgery, and withdrawal from corticosteroid
- Associated with centralized pain disorders
- HPA axis is the primary stress response system producing cortisol and dampening immune response
- May present with hyper- or hypocortisolism
- Altered downstream signaling from the HPA axis
- Depletion - the hypothalamus, pituitary, adrenals, gonads, or thyroid cannot sustain the demands of severe pain
- Diminished end-organ hormones in serum due to uncontrolled pain include cortisol, pregnenolone, DHEA, and testosterone
- With or without involvement of the posterior pituitary
- Intrinsic inability of anterior pituitary to produce hormones or from an insufficient effect of hypothalamic trophic hormones on adenohypophyseal hormones.
- Diagnosed by measuring basal pituitary and specific target hormone levels



BNST = bed nucleus of stria terminalis (‘extended amygdala’)
PVN = paraventricular nucleus (releases CRH and AVP)
HIP = hippocampus
A = amygdala
CRH = corticotropin release hormone (goes from PVN to anterior pituitary via hypophysial portal system)
AVP = arginine vasopressin (goes from PVN to posterior pituitary via hypophysial portal system)

Hypopituitarism

- Normal BNST activity stimulates HPA axis response to stress
- Chronic stress promotes BNST activity via the amygdala (Glu) and brainstem arousal (5HT, NE) networks
- Over time, chronic BNST stimulation of HPA activity becomes feedback suppressed (GABA)
- Promotes negative affect via limbic connectivity with neocortical-striatal information
    - Loss of functional connectivity between the BNST and dorsolateral prefrontal and ventromedial orbitofrontal cortex
    - Loss of prefrontal top-down inhibitory influence that these regions exert on stress-responding neurocircuitry
    - Leads to persistent anxiety

Functional Secondary Hypopituitarism References

Eller-Smith, O. C., Nicol, A. L., & Christianson, J. A. (2018). Potential Mechanisms Underlying Centralized Pain and Emerging Therapeutic Interventions. Frontiers in cellular neuroscience, 12, 35. https://doi.org/10.3389/fncel.2018.00035

Iglesias P. (2024). An Update on Advances in Hypopituitarism: Etiology, Diagnosis, and Current Management. Journal of clinical medicine, 13(20), 6161. https://doi.org/10.3390/jcm13206161

Kim S. Y. (2015). Diagnosis and Treatment of Hypopituitarism. Endocrinology and metabolism (Seoul, Korea), 30(4), 443–455. https://doi.org/10.3803/EnM.2015.30.4.443

Tennant, F. The Physiologic Effects of Pain on the Endocrine System. Pain Ther 2, 75–86 (2013). https://doi.org/10.1007/s40122-013-0015-x

Tennant F, Hermann L. Normalization of serum cortisol concentration with opioid treatment of severe chronic pain. Pain Med. 2002;3:132–4.

Tennant F, Hermann L. Using biologic markers to identify legitimate chronic pain. Am Clin Lab. 2002;21(5):14–5, 18.

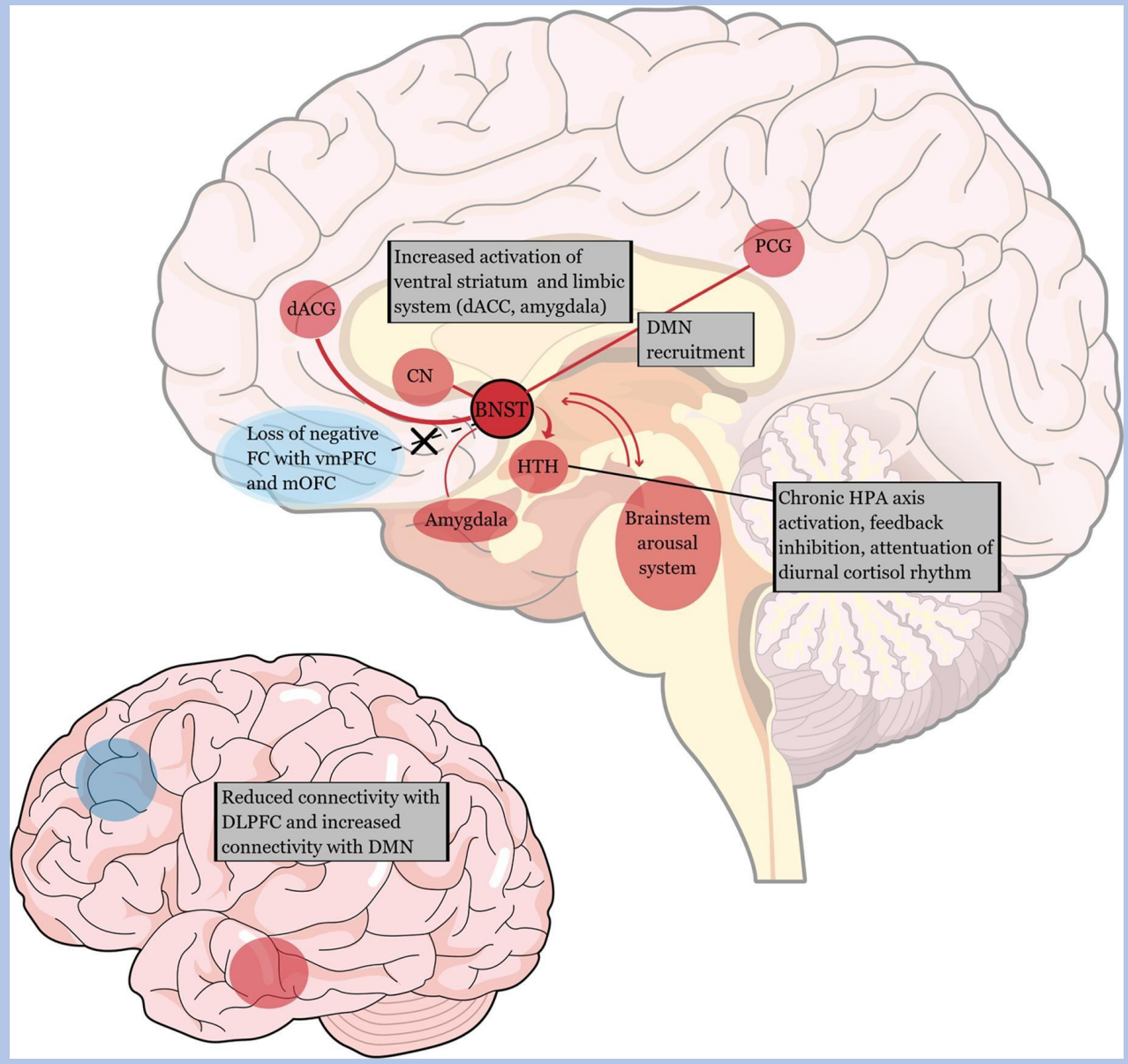


Figure: Awasthi, S., Pan, H., LeDoux, J. E., Cloitre, M., Altemus, M., McEwen, B., ... & Stern, E. (2020). The bed nucleus of the stria terminalis and functionally linked neurocircuitry modulate emotion processing and HPA axis dysfunction in posttraumatic stress disorder. NeuroImage: Clinical, 28, 102442. (Creative Commons)

Behavioral Interventions for Chronic Pain:
Shift of ANS Towards Restoration of Normal HPA Function

- Nutrition
- Stress Management
- Mindfulness/Meditation
- Yoga
- Photobiomodulation (PBM) and Impaired HPA mitochondrial function
- **Autonomic Rehabilitation, HRV Biofeedback, Autonomic Self-Regulation, Coherent Breathing …**

Sensitized Chronic Pain and Mitochondrial Abnormality

- Mitochondria play a key role in the physiologic stress response through dynamic interactions with stress-associated neuroendocrine, metabolic, and inflammatory pathways.
- Recent clinical evidence suggests that mitochondria are involved in the pathological processes underlying several psychiatric disorders.
- Animal studies and emerging clinical evidence indicate that exposure to chronic or severe stressors can alter mitochondrial DNA and mitochondrial function
- Chronic psychological stress triggers excessive mitochondrial fission in humans
- Disrupts mitochondrial regulation of stress reactivity systems: HPA, SAM and ANS

Picard, M., & McEwen, B. S. (2018). Psychological Stress and Mitochondria: A Conceptual Framework. Psychosomatic medicine, 80(2), 126–140. https://doi.org/10.1097/PSY.0000000000000544

Yu, T., Wang, L., Zhang, L., & Deuster, P. A. (2023). Mitochondrial Fission as a Therapeutic Target for Metabolic Diseases: Insights into Antioxidant Strategies. Antioxidants, 12(6), 1163. https://doi.org/10.3390/antiox12061163

Impaired HPA Mitochondrial Function and Photobiomodulation (PBM)

“PBM has analgesic potential and a safe profile for managing chronic pain, especially in cases difficult to control with conventional therapies. However, the variability of clinical parameters and limited follow-up still hinder more comprehensive recommendations.”

Ferreira, L. M. A., Oliveira, A. B. C., Mendes, J. J. B., Costa, G. V., Silva, I. R., Santos, G. N., Pereira, G. S., & Silva, M. L. (2026). Photobiomodulation in chronic pain: a systematic review of randomized clinical trials. Frontiers in integrative neuroscience, 20, 1717372. https://doi.org/10.3389/fnint.2026.1717372

“ …since dysrhythmic gamma oscillations are associated with neurodegenerative diseases and pain syndromes, including migraine with aura and fibromyalgia, we suggest that transcranial PBM should target diseases where patients are affected by impaired neural oscillations and aberrant brain wave patterns.”

Liebert, A., Capon, W., Pang, V., Vila, D., Bicknell, B., McLachlan, C., & Kiat, H. (2023). Photophysical Mechanisms of Photobiomodulation Therapy as Precision Medicine. Biomedicines, 11(2), 237. https://doi.org/10.3390/biomedicines11020237

“In the intranasal PBM method, the light source is located inside the nostril at the back of the nose and due to a thinner thickness of the ethmoid plate, it can directly irradiate subcortical (hypothalamus, thalamus, amygdala, hippocampus) and cortical (orbitofrontal cortex) structures of the limbic system in the brain … “

Salehpour, Farzad, Gholipour-Khalili, Sevda, Farajdokht, Fereshteh, Kamari, Farzin, Walski, Tomasz, Hamblin, Michael R., DiDuro, Joseph O. and Cassano, Paolo. "Therapeutic potential of intranasal photobiomodulation therapy for neurological and neuropsychiatric disorders: a narrative review" Reviews in the Neurosciences, vol. 31, no. 3, 2020, pp. 269-286. https://doi.org/10.1515/revneuro-2019-0063

Photobiomodulation (PBM) and Mitochondrial Cytochrome C Oxidase

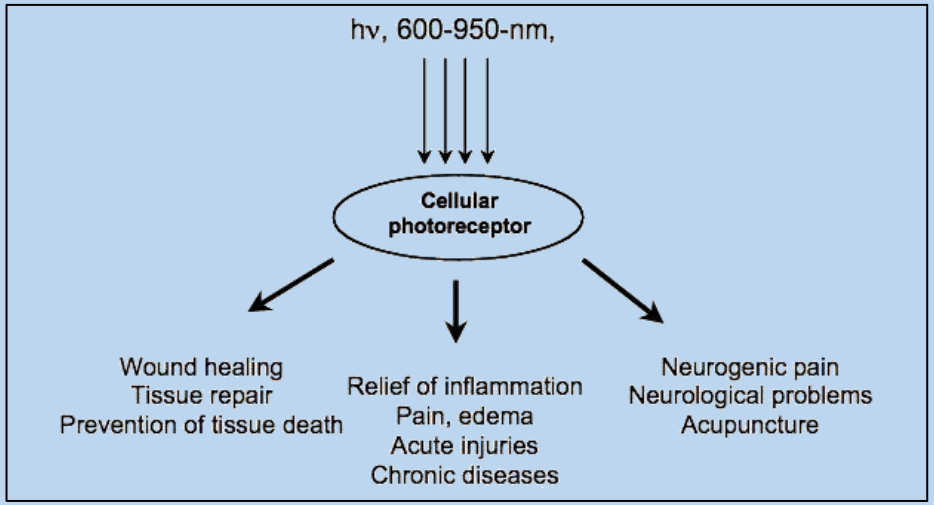
“ … emerging evidence indicates that chronic stress generates maladaptive alterations in mitochondria, which contribute to allostatic processes, ultimately promoting aging and disease.”

Daniels, T. E., Olsen, E. M., & Tyrka, A. R. (2020). Stress and Psychiatric Disorders: The Role of Mitochondria. Annual review of clinical psychology, 16, 165–186. https://doi.org/10.1146/annurev-clinpsy-082719-104030

“ … mitochondrial cytochrome c oxidase (Cco) is a photoreceptor that mediates the beneficial effects of low intensity infrared and near-infrared light on mammalian cells and tissues … ”

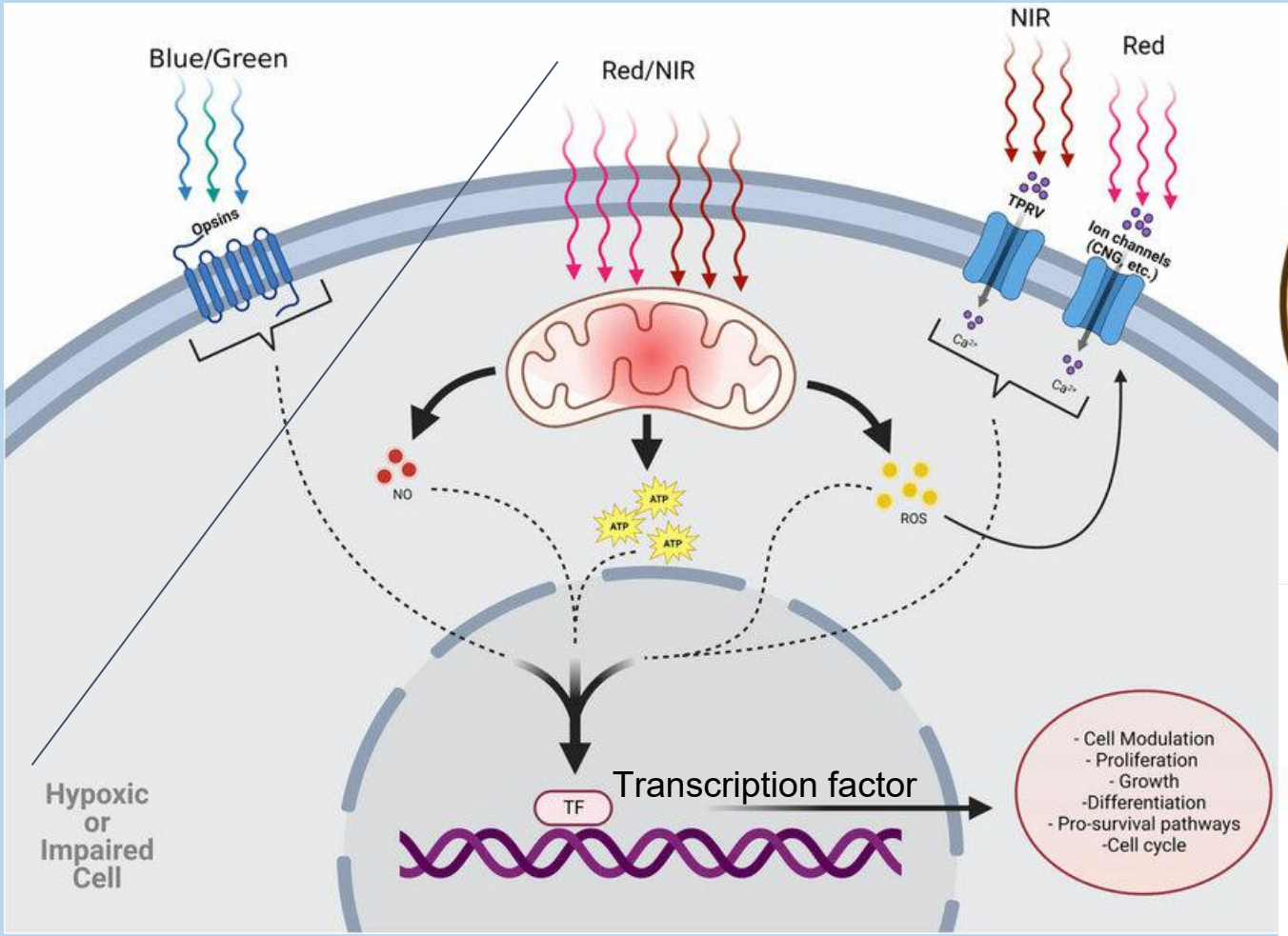
Ball, K. A., Castello, P. R., & Poyton, R. O. (2011). Low intensity light stimulates nitrite-dependent nitric oxide synthesis but not oxygen consumption by cytochrome c oxidase: Implications for phototherapy. Journal of Photochemistry and Photobiology B: Biology, 102(3), 182-191.

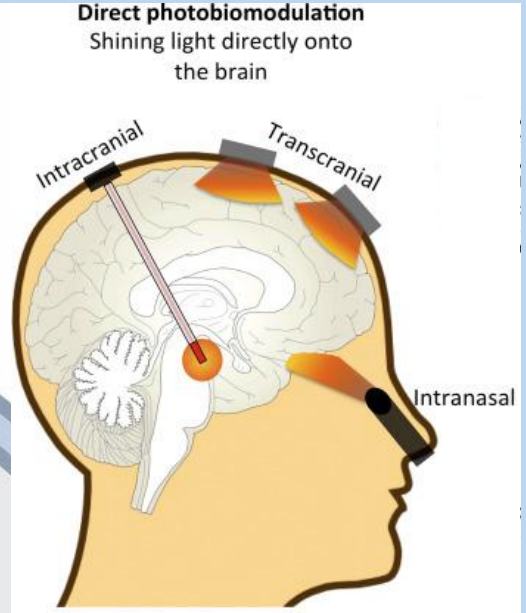
- PBM is also called low-level light therapy (LLT) or red/near-infrared light therapy (650-850 nm)
- Stimulates mitochondrial activity and increases cellular energy (ATP)
- Mitochondrial stimulation increases cytochrome c oxidase activity, accelerates cellular repair
- Reduces reactive oxidative species (ROS) and cellular stress
- Targets mitochondrial cytochrome c oxidase (CCO)
- Reduces mitochondrial fission
- Modulates mitochondria apoptosis
- Enhances cell survival by reducing apoptosis (anti-apoptotic)
- Produces an anti-inflammatory response
- Reduces acute and chronic inflammation
- Reduces stress, anxiety, and depression
- Decreases brain inflammation.
- Safe, non-invasive



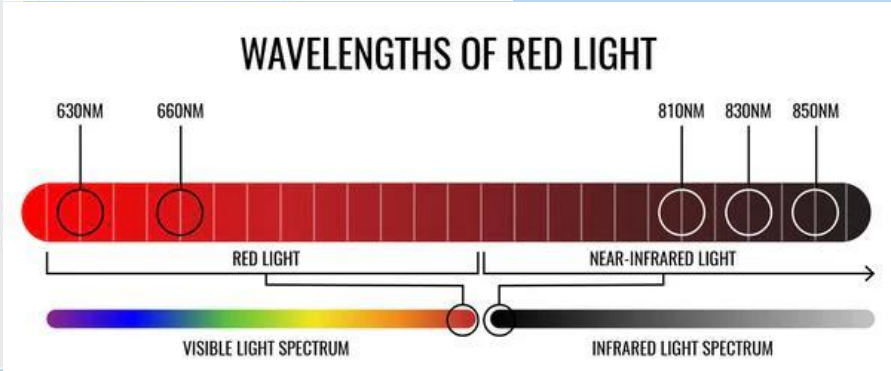
Hamblin M. R. (2018). Mechanisms and Mitochondrial Redox Signaling in Photobiomodulation. Photochemistry and photobiology, 94(2), 199–212. https://doi.org/10.1111/php.12864

Giménez, M. C., Luxwolda, M., Van Stipriaan, E. G., Bollen, P. P., Hoekman, R. L., Koopmans, M. A., Arany, P. R., Krames, M. R., Berends, A. C., Hut, R. A., & Gordijn, M. C. M. (2023). Effects of Near-Infrared Light on Well-Being and Health in Human Subjects with Mild Sleep-Related Complaints: A Double-Blind, Randomized, Placebo-Controlled Study. Biology, 12(1), 60. https://doi.org/10.3390/biology12010060





Gordon, L. C., & Johnstone, D. M. (2019). Remote photobiomodulation: an emerging strategy for neuroprotection. Neural Regeneration Research, 14(12), 2086-2087.



Chromophores involved in PBM include mitochondrial Cytochrome C, membrane receptors such as opsins and light-sensitive ion channels.
Note: The blue/green opsin photoreceptors are not involved in intranasal PBM (ipBM) stimulation of HPA activity,

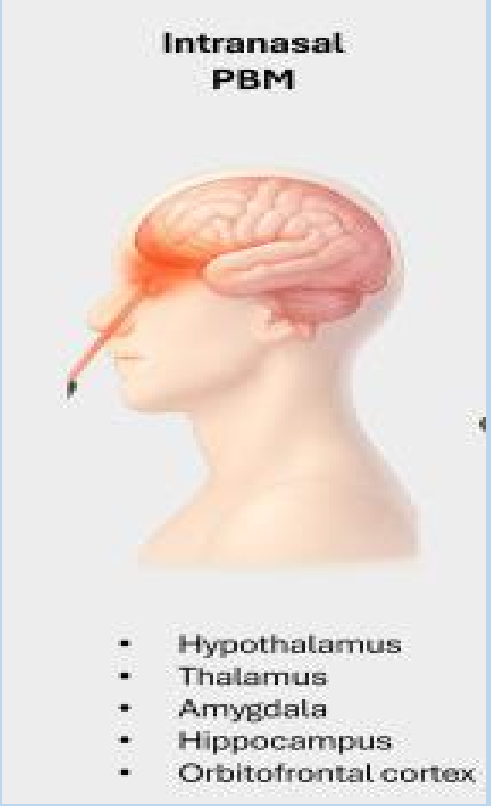
Barolet, Augustin & Martinez Villarreal, Amelia & Jfri, Abdulhadi & Litvinov, Ivan & Barolet, Daniel. (2023). Low-Intensity Visible and Near-Infrared Light-Induced Cell Signaling Pathways in the Skin: A Comprehensive Review. Photobiomodulation Photomedicine and Laser Surgery. 41. 147-166. 10.1089/photob.2022.0127. Figure on ResearchGate. Available from: https://www.researchgate.net/figure/Chromophores-involved-in-PBM-include-mitochondrial-CCO-membrane-receptors-such-as-opsins_fig3_370119740 [accessed 20 Apr 2026]

Intranasal Photobiomodulation (iPBM) for Sensitized Chronic Pain and Autonomic Rehabilitation: A New Pathway to Modulate ANS Regulation of HPA

- Delivered via the nostrils
- Option for managing chronic stress and improving mental health.
- NIR (750-850 nm) penetrates thin cribriform plate to effect the olfactory bulb, orbitofrontal cortex
- Red light therapy (650-750 nm) reduces cortisol
- Mitigates neuroinflammation
- Promotes neuronal survival
- Antidepressant effects
- Combined with transcranial light reduces anxiety, fear, and anger

Evidence and Application

- Animal models show reduced neuronal apoptosis caused by chronic restraint stress
- In human applications, used for
  - Managing stress
  - Dementia
  - PTSD
  - Improvements in sleep and cognitive function



De Ridder, D., Hamblin, M. R., & Vanneste, S. (2026). Transcranial Photobiomodulation for Neuromodulation of Brain Disorders: A Perspective. Neuromodulation: Technology at the Neural Interface.

Van Lankveld, H., Chen, JX, Zhong, XZ, Chen, JJ. (2026). Intranasal photobiomodulation: an energy efficient paradigm for cortical and subcortical stimulation bioRxiv 2026.03.03.709361; doi: https://doi.org/10.64898/2026.03.03.709361 (This article is a preprint and has not been certified by peer review)

Yoo SH. (2021). Intranasal Photobiomodulation Therapy for Brain Conditions: A Review. Medical Lasers 10:132-137. https://doi.org/10.25289/ML.2021.10.3.132

Additional PBM Reading

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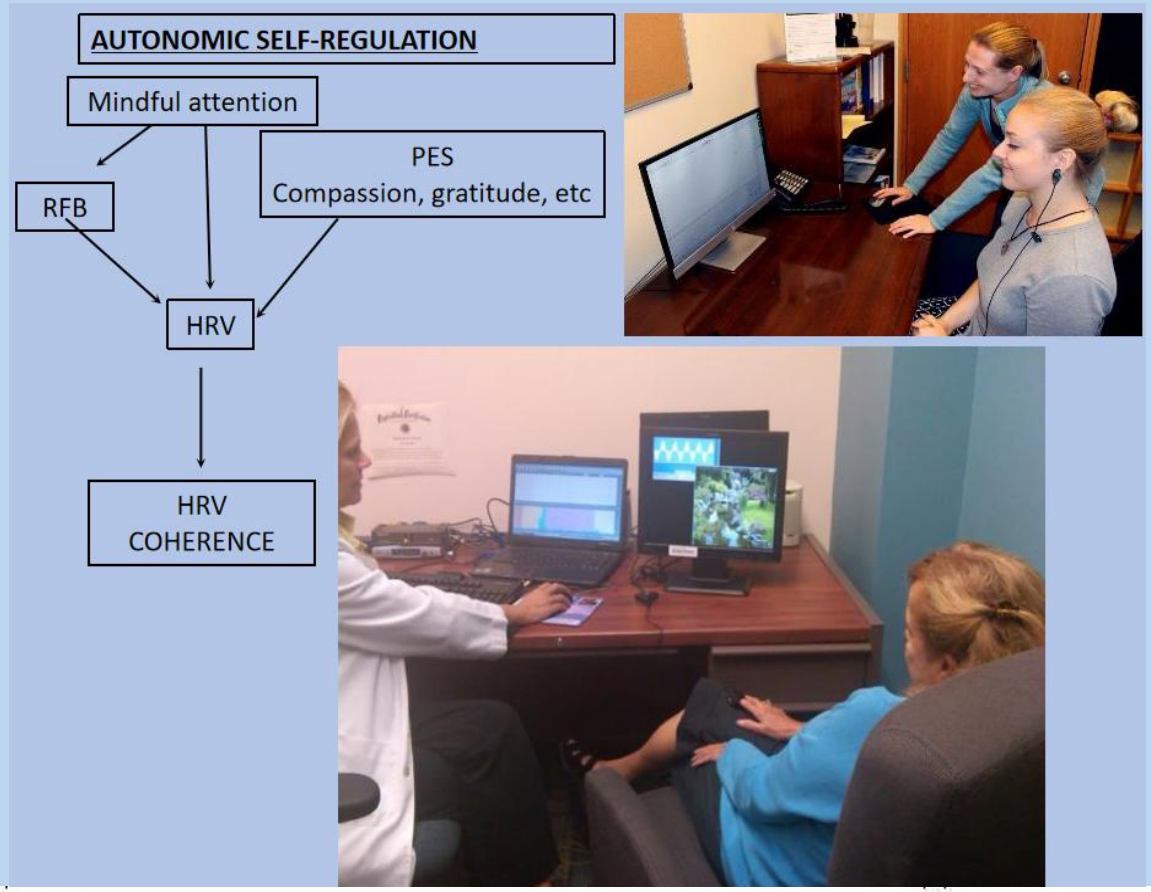
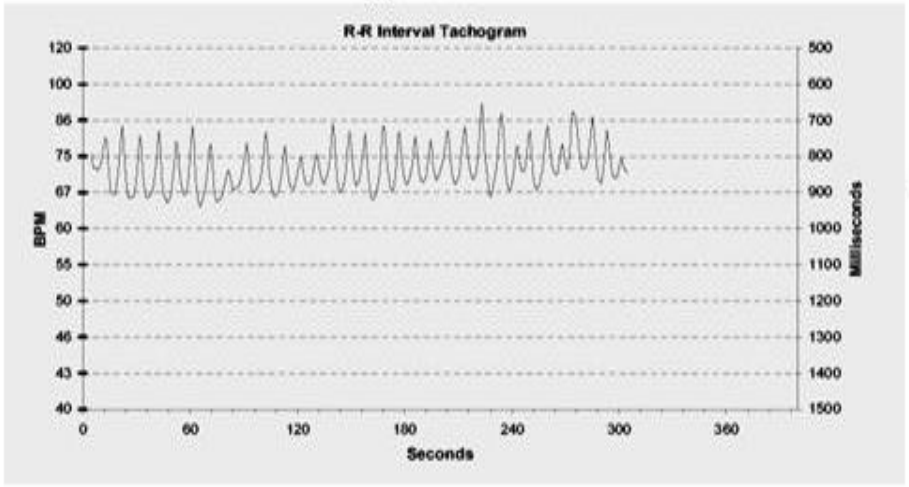
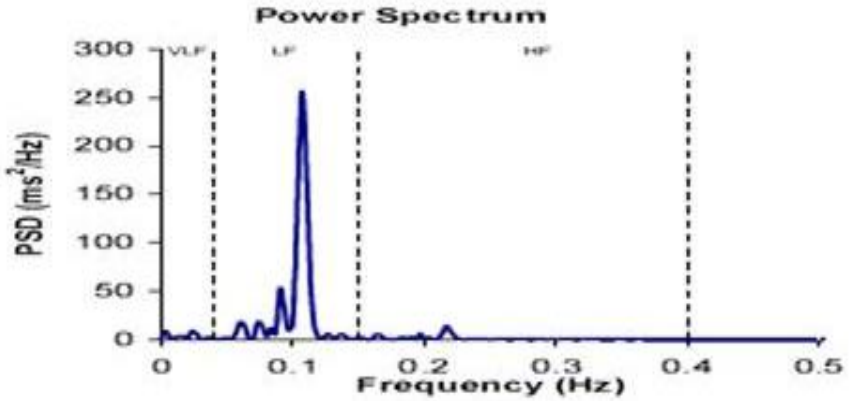
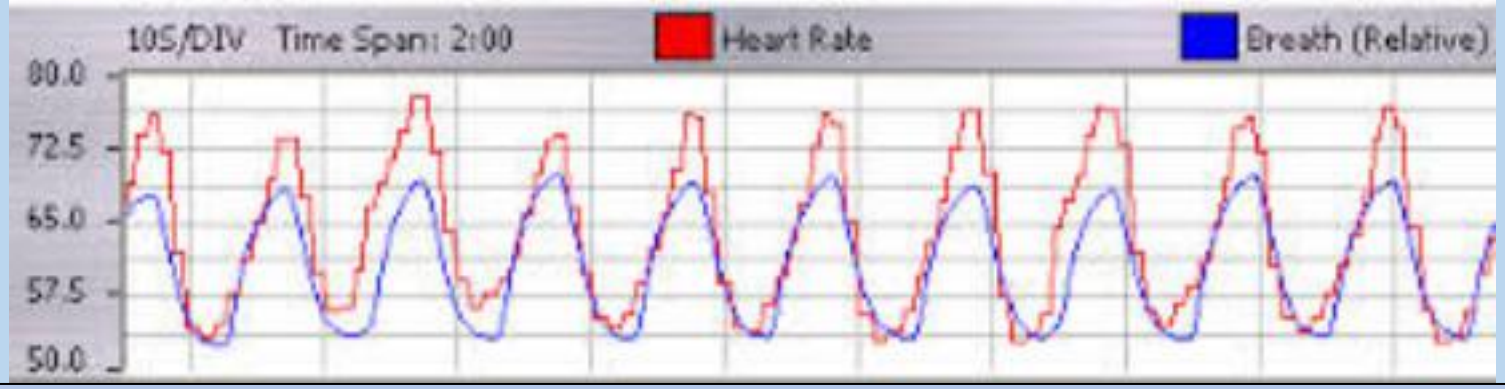
Autonomic Rehabilitation through Breath Control:
HRV Biofeedback, Autonomic Self-Regulation, Coherent Breathing, etc







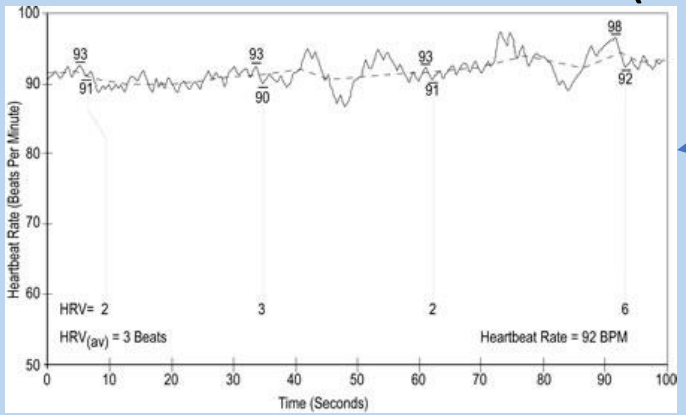


Resonance is the tendency of a system to oscillate with greater amplitude at some frequencies than at others. Relative maximum frequency of oscillation is the system's **resonance frequency**. At these resonance frequency, even small periodic driving forces can produce large amplitude oscillations

Pushing a person in a swing is an example of resonance. Pushing a swing in time with its resonant frequency will make the swing go higher and higher (maximum amplitude), while attempts to push it at a faster or slower tempo results in smaller arcs.

Coherence of Cardiac Rhythm: RSA and Effect of RFB on HRV

coherence.com (Richard Brown, MD and Stephen Elliott, Ph.D.)



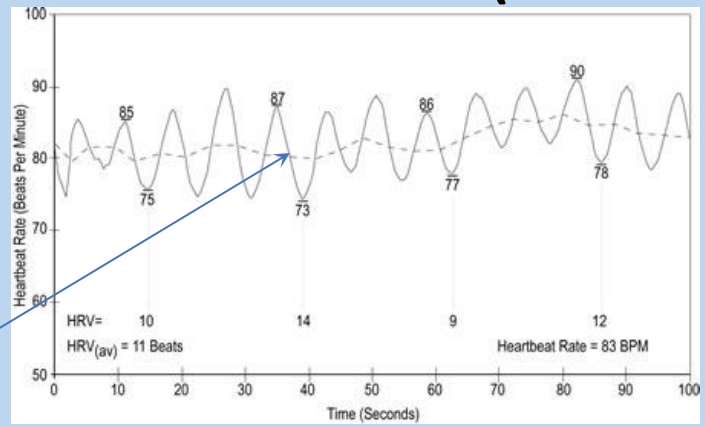
30 BrPM (0.5 Hz) , HRV(avg) = 2

7.5 BrPM (0.125 Hz), HRV(avg) = 11

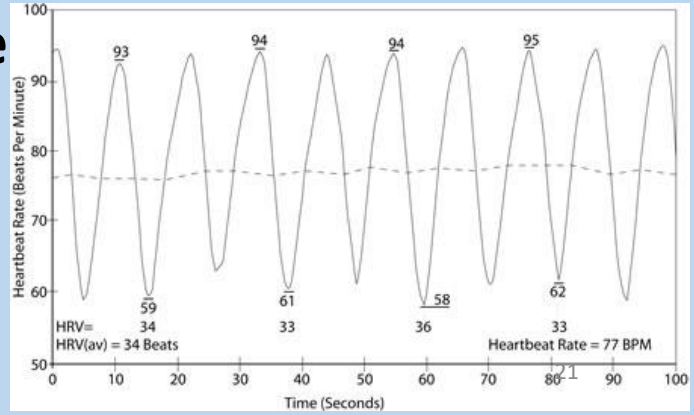
5.5 BrPM (0.092 Hz), HRV(avg) = 34

Baroreflex activates resonance ('Coherence')

The difference between the max and min HR for each cycle is shown along the center; averaging across consecutive cycles yields HRV(avg), one of the many measures of HRV.



'Resonance Frequency Breathing' RFB →



Properties of HRV Coherence

- Produced by resonant frequency breathing (RFB)
- RFB occurs ~0.1 Hz (=6 cycles/minute=10 sec/cycle=10 sec/ period)
- Produced by HRV Biofeedback
- Also produced by other stimuli at 0.1 Hz frequency (e.g. rhythmical muscle tension, chanting, picture presentation, etc.)
- Due to interactions between cycles of respiratory sinus arrhythmia (RSA) and baroreflex feedback control of vasomotor tone
- 0° phase between respiratory and sinusoidal HR cycles
- 90° phase between baroreflex and sinusoical HR cycles
- Associated with maximum RSA (max-min HR over respiratory cycle)
- Discrete sharp peak in power spectrum at resonant frequency
- Associated with improved adaptive behaviors
    - alertness, responsiveness
    - emotional self-regulation
    - cognitive function
- distinct from "relaxation"
- Healthy people do not have Coherence during non RFB periods
- Extended high coherence may be a sign of inflexibility of cardiac adjustment