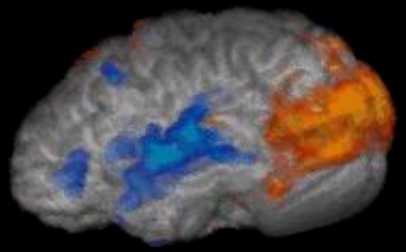
Stres, meditace & resilience



Petr Bob, Centrum pro neuropsychiatrický výzkum traumatického stresu, 1. lékařská fakulta UK

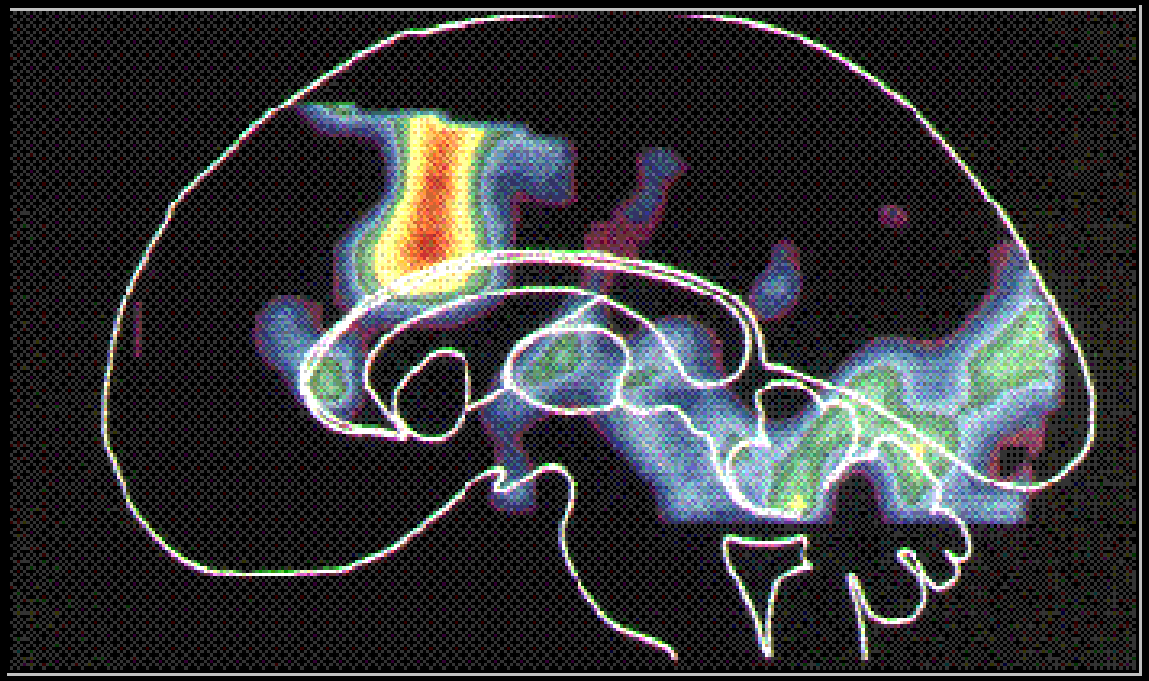
Stres je charakterizován těžce rozhodnutelnou situací, která je náročná z hlediska řešení a působí jako negativní emoce, strachu, úzkosti a ohrožení.

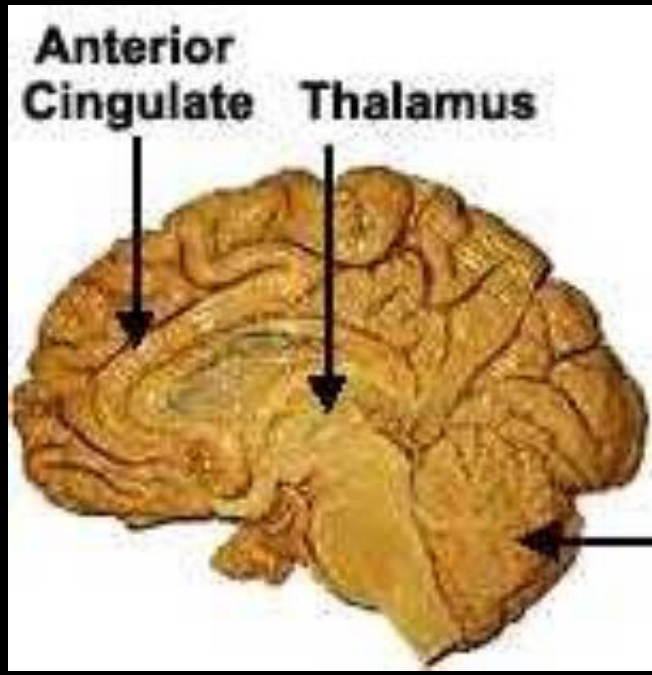
V případě že tento výběr řešení není možný dochází ke kognitivnímu konfliktu. Jako experimentální model stresové situace bývá používán Stroopův test.

červená

zelená modrá

K detekci kognitivního konfliktu („Nevím co mám dělat“) dochází v souvislosti s aktivaci anteriorního cingulárního kortexu, která má za následek aktivaci centrální autonomní sítě a stresovou reakci.





Pojem disociace a intrapsychického konfliktu rozpracoval také Sigmund Freud a tento pojem stál u zrodu psychoanalýzy a dalších směrů v psychoterapii.

Neřešitelnost konfliktu může vést k DISOCIACI, která představuje událost ve vědomí nejčastěji v reakci na traumatický stres, která nezapadá do stávajících kognitivních schémat v důsledku přítomnosti kognitivního konfliktu.

PIERRE JANET
(1859-1947)

Podle stávajících nálezů působení traumatického stresu a disociace narušuje nejen normální funkce vědomí člověka ale zároveň s tím také normální funkce CNS zejména pak funkce limbického systému a může vést k limbické iritabilitě jejímž projevem je hyperexcitabilita neuronů limbického systému.

J Neuroimmune Pharm (2006) 1: 421–427
DOI 10.1007/s11481-006-9036-0

INVITED REVIEW

Stress-Induced Immune Dysregulation: Implications for Wound Healing, Infectious Disease and Cancer

Jonathan P. Godbout · Ronald Glaser

Received: 20 April 2006 / Accepted: 13 July 2006 / Published online: 10 August 2006

Abstract The communication between the central nervous system and the immune system occurs via a complex network of bidirectional signals linking the nervous, endocrine and immune systems. The field of psychoneuroimmunology (PNI) has provided new insights to help understand the pathophysiological processes that are linked to the immune system. Work in this field has established that psychological stress disrupts the functional interaction between the nervous and immune systems. Stress-induced immune dysregulation has been shown to be significant enough to result in health consequences, including reducing the immune response to vaccines, slowing wound healing, reactivating latent herpesviruses, such as Epstein–Barr virus (EBV), and enhancing the risk for more severe infectious disease. Chronic stress/depression can increase the peripheral production of proinflammatory cytokines, such as interleukin (IL)-6. High serum levels of IL-6 have been linked to risks for several conditions, such as cardiovascular disease, type 2 diabetes, mental health complications, and some cancers. This overview will discuss the evidence that psychological stress promotes immune dysfunction that negatively impacts human health.

Key words stress · immunity · cytokines · behavior

Abbreviations

HPA	hypothalamic–pituitary–adrenal
SNS	sympathetic nervous system
GC	glucocorticoids
EBV	Epstein–Barr virus
NPC	nasopharyngeal carcinoma
BL	Burkitt's lymphoma

Introduction

Psychological stress can be defined as a perceived stress that influences an individual's ability to cope with life events. It is clear that psychological stress impacts immune

Immunol Res (2014) 58:193–210
DOI 10.1007/s12026-014-8517-0

IMMUNOLOGY AT STANFORD UNIVERSITY

Effects of stress on immune function: the good, the bad, and the beautiful

Firdaus S. Dhabhar

Firdaus S. Dhabhar

Published online: 6 May 2014

Abstract Although the concept of stress has earned a bad reputation, it is important to recognize that the adaptive purpose of a physiological stress response is to promote survival during fight or flight. While long-term stress is generally harmful, short-term stress can be protective as it prepares the organism to deal with challenges. This review discusses the immune effects of biological stress responses that can be induced by psychological, physiological, or physical (including exercise) stressors. We have proposed that short-term stress is one of the nature's fundamental but under-appreciated survival mechanisms that could be clinically harnessed to enhance immunoprotection. Short-term (i.e., lasting for minutes to hours) stress experienced during immune activation enhances innate/primary and adaptive/secondary immune responses. Mechanisms of immune-enhancement include changes in dendritic cell, neutrophil, macrophage, and lymphocyte trafficking, maturation, and function as well as local and systemic production of cytokines. In contrast, long-term stress suppresses or dysregulates innate and adaptive immune responses by altering the Type 1–Type 2 cytokine balance, inducing low-grade chronic inflammation, and suppressing numbers, trafficking, and function of immunoprotective cells. Chronic stress may also increase susceptibility to some types of cancer by suppressing Type 1 cytokines and protective T cells and increasing regulatory/suppressor T cell function. Here, we classify immune responses as being protective, pathological, or regulatory, and discuss "good" versus "bad" effects of stress on health. Thus, short-term stress can enhance the acquisition and/or expression of immunoprotective (wound healing, vaccination, anti-infectious agent, anti-tumor) or immuno-pathological (pro-inflammatory, autoimmune) responses. In contrast, chronic stress can suppress protective immune responses and/or exacerbate pathological immune responses. Studies such as the ones discussed here could provide mechanistic targets and conceptual frameworks for pharmacological and/or biobehavioral interventions designed to enhance the effects of "good" stress, minimize the effects of "bad" stress, and maximally promote health and healing.

Review TRENDS in Immunology Vol.26 No.12 December 2005

The role of stress-response systems for the pathogenesis and progression of MS

Stefan M. Gold[1], David C. Mohr[2], Inge Huitinga[3], Peter Flachenecker[4], Esther M. Sternberg[5] and Christoph Heesen[6]

Disease progression in multiple sclerosis (MS) – an inflammatory demyelinating and neurodegenerative disease with a presumed T-cell driven autoimmune origin – has long been hypothesized to be associated with stress. However, this notion has only recently been supported by prospective clinical studies. Several clinical and molecular studies in MS and its animal models have recently shown disruptions in the communication between the immune system and the two major stress response systems, the hypothalamo–pituitary–adrenal (HPA) axis and the autonomic nervous system. Insensitivity to glucocorticoid and β-adrenergic modulation might be involved in overshooting inflammation in MS, whereas hyperactivity of the HPA axis has been linked to neurodegeneration and increased disability. Here, we integrate findings from molecular, cellular, experimental, clinical and epidemiological research to describe the involvement of stress response systems in MS pathogenesis and progression.

Jiným příkladem jsou poznatky o vlivu stresu na roztroušenou sklerózu

Outline

Figures (1)

ELSEVIER

Handbook of Clinical Neurology

Volume 181, 2021, Pages 119-126

Chapter 8 - The stress-axis in multiple sclerosis: Clinical, cellular, and molecular aspects

Jeroen Melief [1], Inge Huitinga [2, 3], Stefan M. Gold [4, 5]

Show more

https://doi.org/10.1016/B978-0-12-820683-6.00008-7 Get rights and content

Part of volume:

The Human Hypothalamus: Neuroendocrine Disorders

Edited by Dick F. Swaab, Ruud M. Buijs, Paul J. Lucassen, Ahmad Salehi, Felix Kreier

Stres prostřednictvím limbického systému významně ovlivňuje endokrinní systém, tzv. hypothalamo-hypofyzo-adrenální osa – kortisol se vylučuje z nadledvinek a další změny.

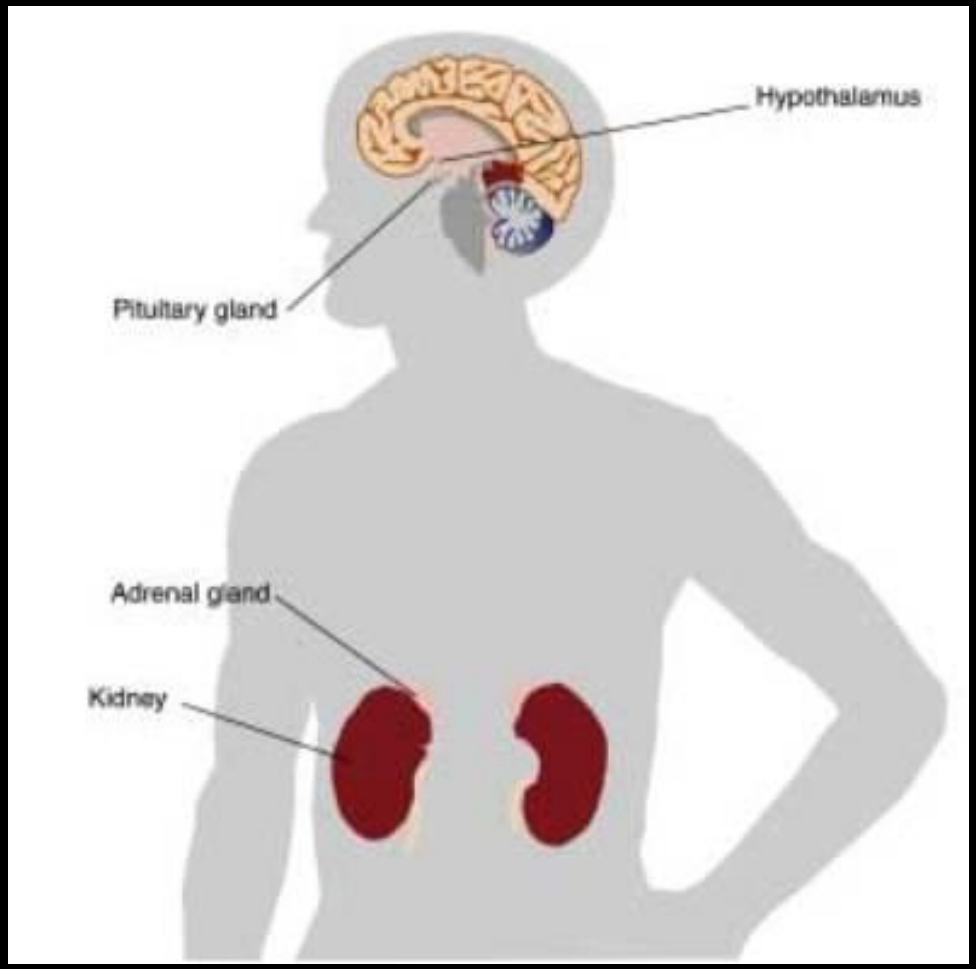
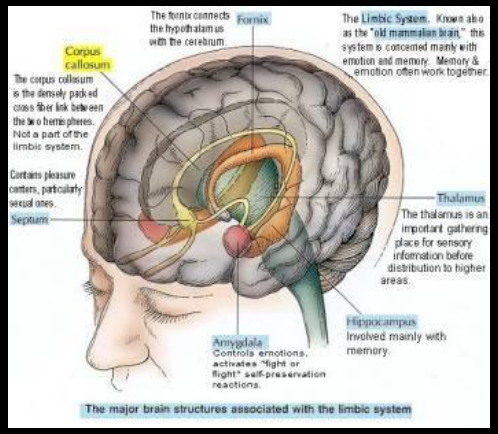
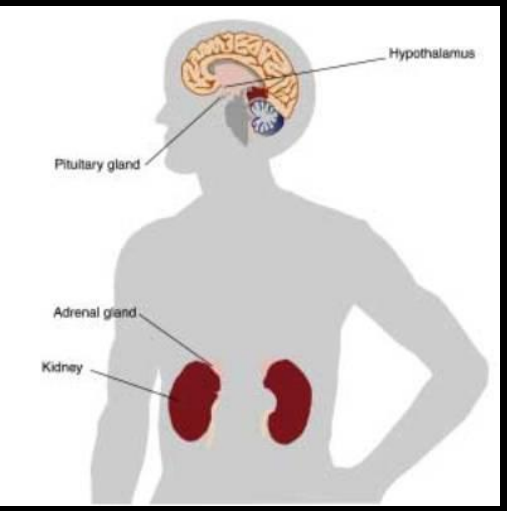


Fig. http://www.montana.edu/wwwai/imsd/alcohol/Vanessa/vwendocrine.htm

Stres prostřednictvím hippocampu, hypothalamu, hypofýzy (pituitary) a dřeně nadledvinek (adrenal gland) zvyšuje hladiny kortizolu. Paradoxně PTSD má za následek snížení kortizolu.





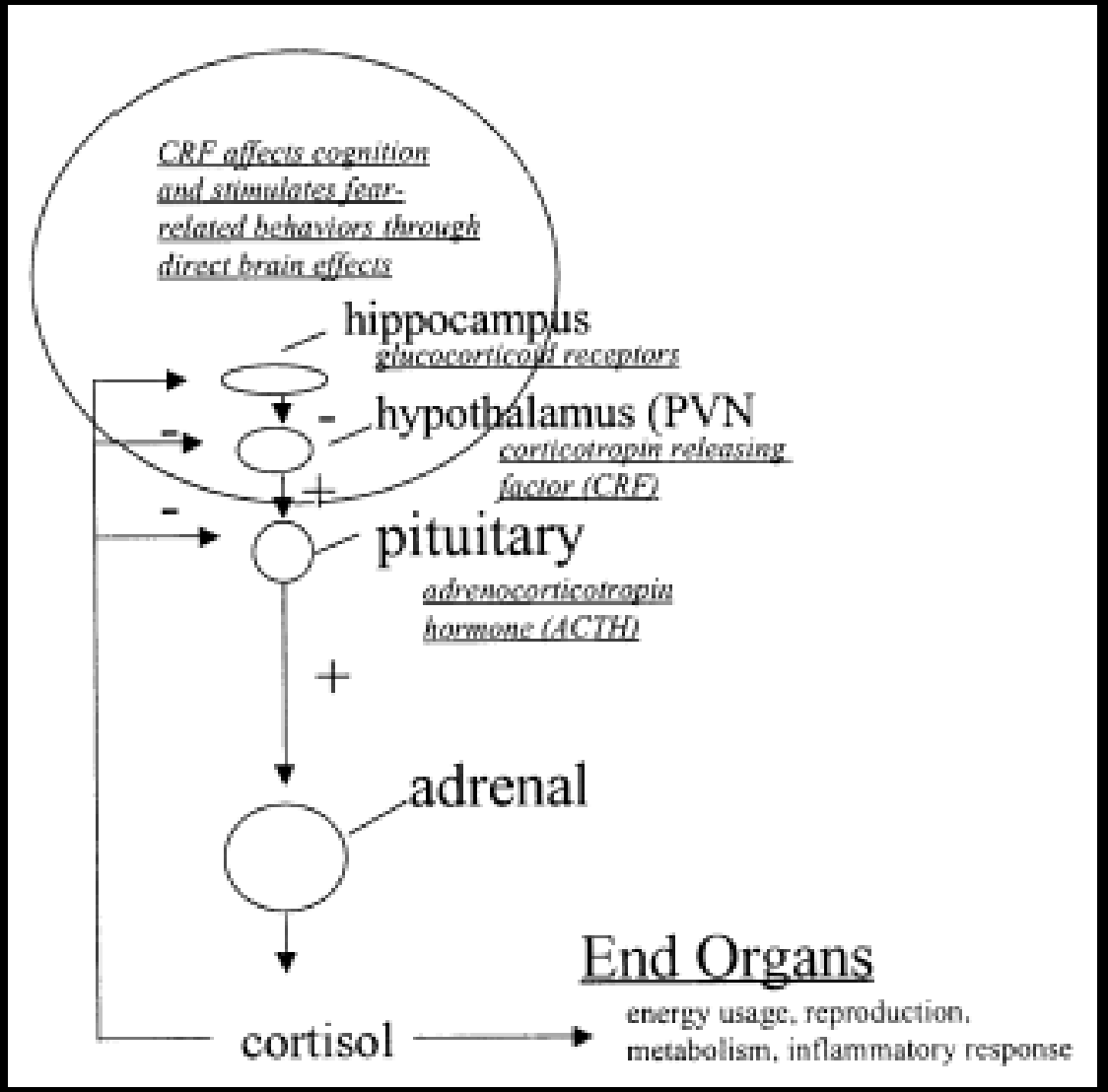
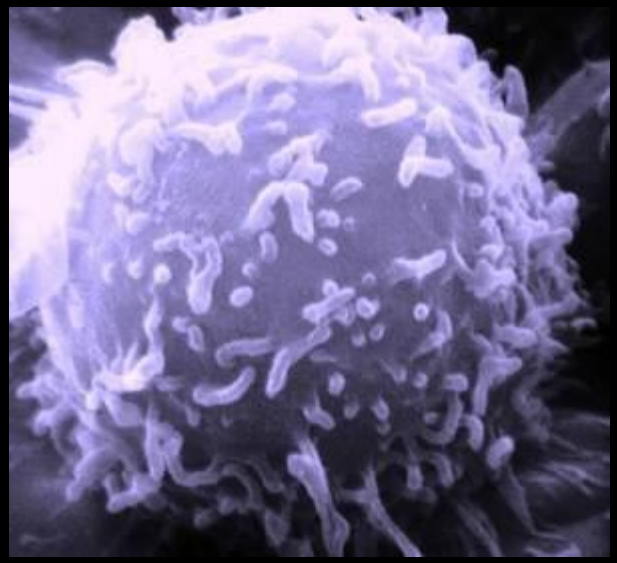
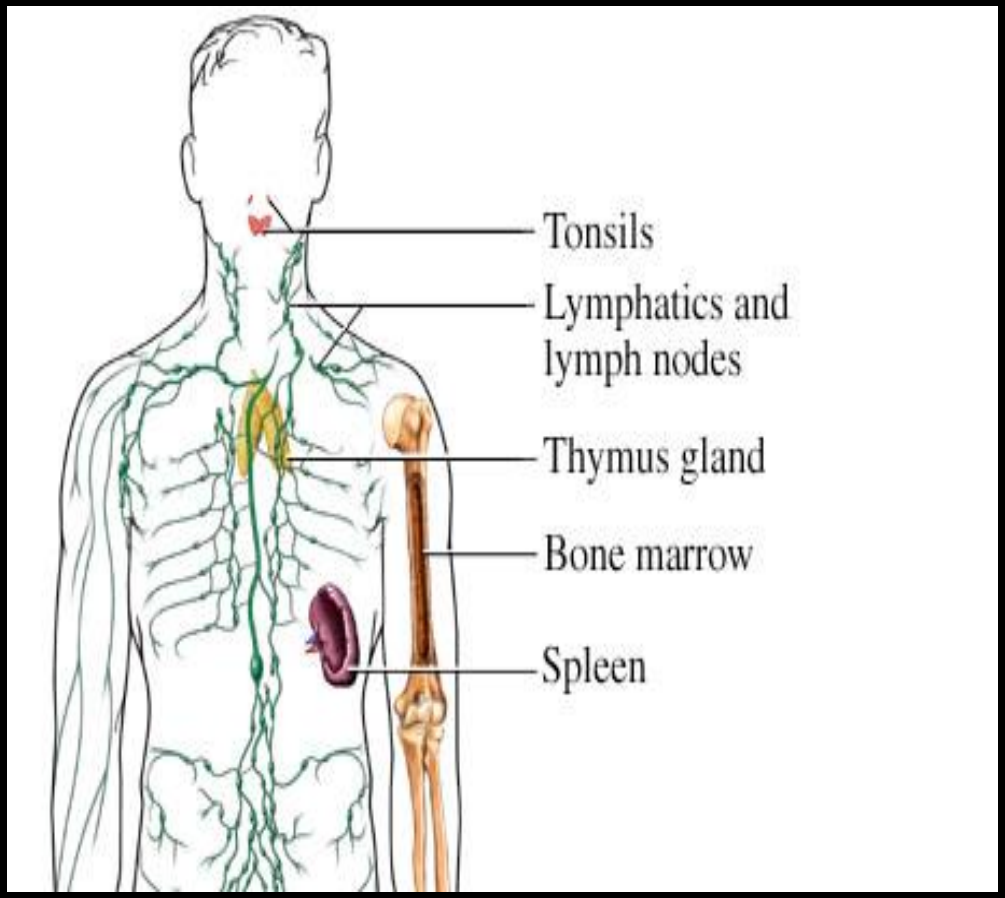
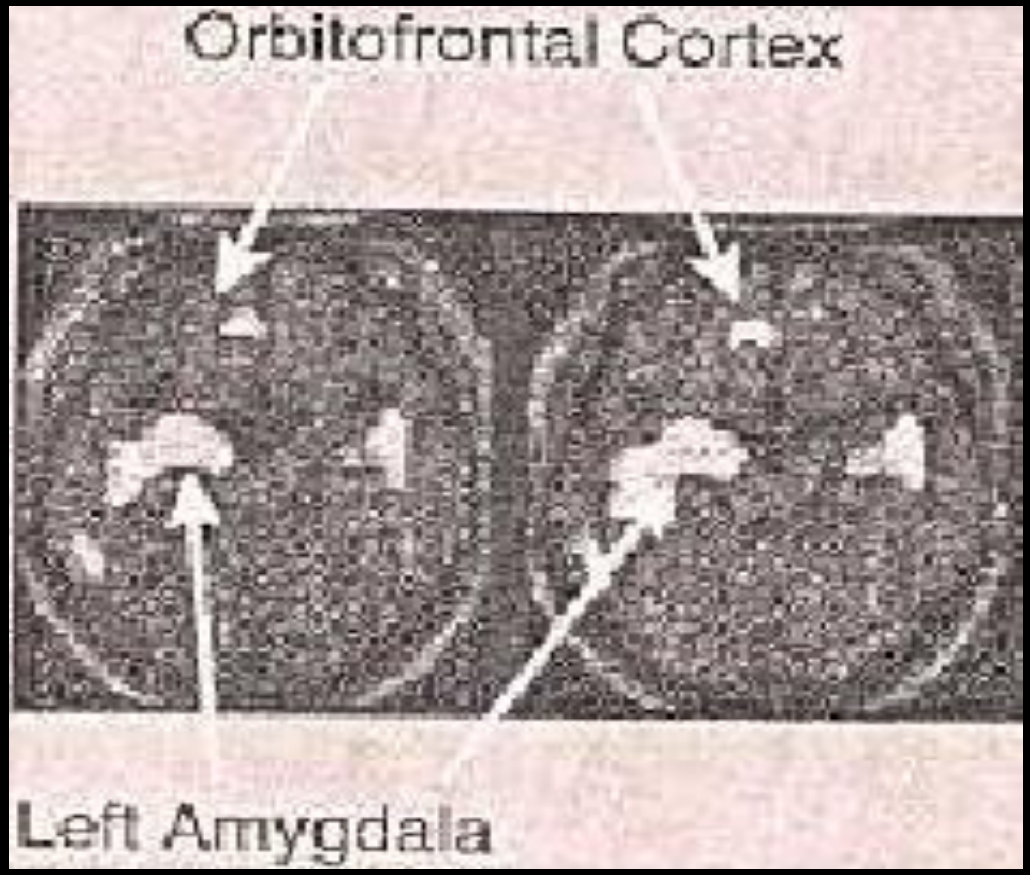


Fig. Bremner, 1999; Juruena et al., 2020

Stress prostřednictvím limbického systému ovlivňuje imunitní funkce. Obecně je známo, že stres snižuje imunitu například prostřednictvím kortisol nebo může vést k zánětům. Stres může také ovlivňovat imunitní buňky a signální molekuly například cytokiny (proteiny, peptidy, or glycoproteiny jako například interleukin-6 [IL-6])

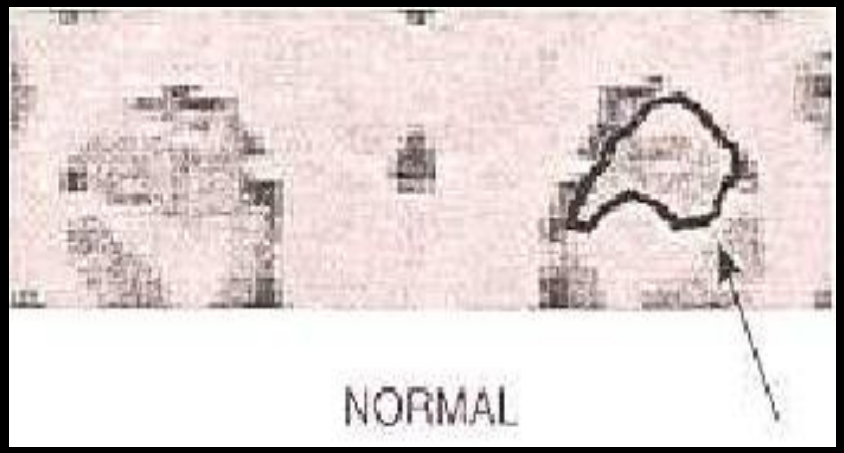


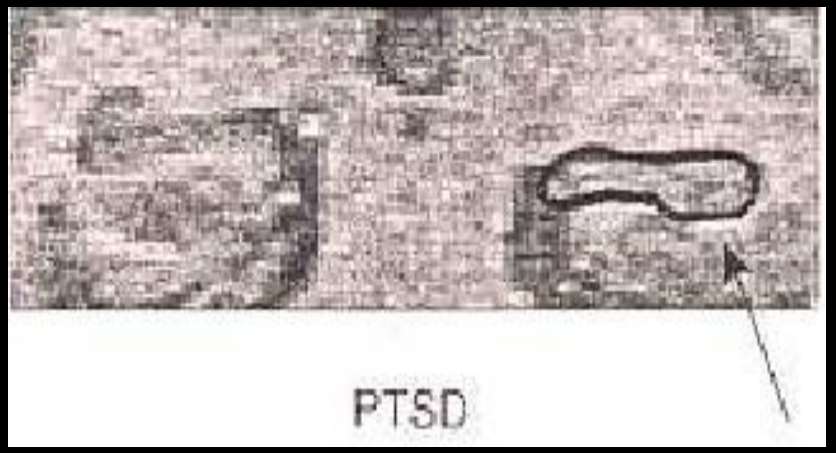




Po podnětu, který vyvolává strach můžeme pozorovat výraznější MRI aktivaci v levé amygdale u pacientů s postraumatickou stresovou poruchou (vpravo) ve srovnáni s kontrolní skupinou (Bremner, 2003; Teicher, 2006; Sharp, 2017; Juruena et al., 2020; Leistner & Menke, 2020).

Působení traumatického stresu na mozek je doloženo také dalšími měřícími metodami jako jsou např. zobrazovací metody.

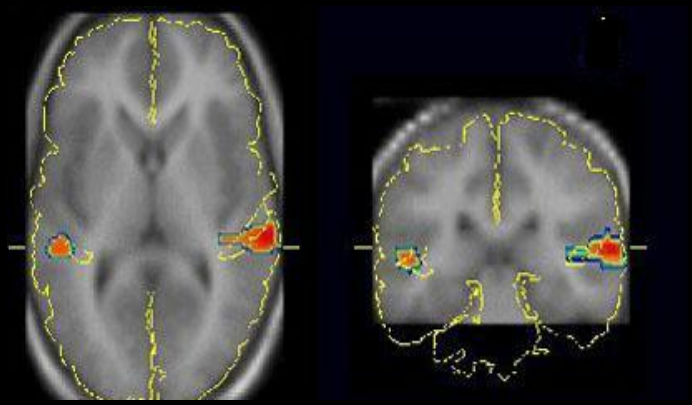




MRI hippocampu u zdravých jedinců ve srovnání s jedinci s PTSD vykazuje zřetelné zmenšení objemu (Bremner, 2003; Pitman, 2012; Kunimatsu, 2020).

Vlivem traumatického stresu bylo také k doloženo zmenšení objemu corpus callosum a porušení interakcí mezi hemisférami, které může vykazovat vztah k alexithymii a disociaci.

(Teicher, 2003, 2006; Milani et al., 2017; Ju et al., 2020.



Mezi vlivy traumatického stresu byl také zjištěn závažný vliv verbálního a emočního týrání dětí na neurobiologické funkce a strukturální uspořádání mozku.

(Teicher et al., 20!6).

Doložená evidence ukazuje že stres vztahující se k verbálnímu týrání může snižovat objem bílé hmoty v některých oblastech temporálních laloku.

BIOL PSYCHIATRY 2009;65:227–234

Preliminary Evidence for White Matter Tract Abnormalities in Young Adults Exposed to Parental Verbal Abuse

Jeewook Choi, Bumseok Jeong, Michael L. Rohan, Ann M. Polcari, and Martin H. Teicher

Background: Psychiatric sequelae of exposure to parental verbal abuse (PVA) appear to be comparable with that of nonfamilial sexual abuse and witnessing domestic violence. Diffusion tensor imaging (DTI) was used to ascertain whether PVA was associated with abnormalities in white matter (WM) tract integrity.

Methods: 1271 healthy young adults were screened for exposure to childhood adversity. Diffusion tensor imaging was collected on 16 unmedicated subjects with history of high-level exposure to PVA but no other form of maltreatment (4 male/12 female subjects, mean age 21.9 ± 2.4 years) and 16 healthy control subjects (5 male/11 female subjects, 21.0 ± 1.6 years). Group differences in fractional anisotropy (FA), covaried by parental education and income, were assessed using tract-based spatial statistics (TBSS).

Results: Three WM tract regions had significantly reduced FA: 1) arcuate fasciculus in left superior temporal gyrus, 2) cingulum bundle by the posterior tail of the left hippocampus, and 3) the left body of the fornix. Fractional anisotropy in these areas was strongly associated with average PVA scores ($r_s = -.701, -.801, -.524$, respectively) and levels of maternal verbal abuse. Across groups, FA in region 1 correlated with verbal IQ and verbal comprehension index. Fractional anisotropy in region 2 was inversely associated with ratings of depression, dissociation, and limbic irritability. Fractional anisotropy in region 3 was inversely correlated with ratings of somatization and anxiety.

Conclusions: Exposure to PVA may be associated with alteration in the integrity of neural pathways with implications for language development and psychopathology.

THE JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY

ACAMH THE ASSOCIATION FOR CHILD AND ADOLESCENT MENTAL HEALTH

Journal of Child Psychology and Psychiatry 57:3 (2016), pp 241–266 doi:10.1111/jcpp.12507

Annual Research Review: Enduring neurobiological effects of childhood abuse and neglect

Martin H. Teicher[1,2] **and Jacqueline A. Samson**[1,2]

[1]Department of Psychiatry, Harvard Medical School, Boston, MA; [2]Developmental Biopsychiatry Research Program, McLean Hospital, Belmont, MA, USA

Background: Childhood maltreatment is the most important preventable cause of psychopathology accounting for about 45% of the population attributable risk for childhood onset psychiatric disorders. A key breakthrough has been the discovery that maltreatment alters trajectories of brain development. **Methods:** This review aims to synthesize neuroimaging findings in children who experienced caregiver neglect as well as from studies in children, adolescents and adults who experienced physical, sexual and emotional abuse. In doing so, we provide preliminary answers to questions regarding the importance of type and timing of exposure, gender differences, reversibility and the relationship between brain changes and psychopathology. We also discuss whether these changes represent adaptive modifications or stress-induced damage. **Results:** Parental verbal abuse, witnessing domestic violence and sexual abuse appear to specifically target brain regions (auditory, visual and somatosensory cortex) and pathways that process and convey the aversive experience. Maltreatment is associated with reliable morphological alterations in anterior cingulate, dorsal lateral prefrontal and orbitofrontal cortex, corpus callosum and adult hippocampus, and with enhanced amygdala response to emotional faces and diminished striatal response to anticipated rewards. Evidence is emerging that these regions and interconnecting pathways have sensitive exposure periods when they are most vulnerable. **Conclusions:** Early deprivation and later abuse may have opposite effects on amygdala volume. Structural and functional abnormalities initially attributed to psychiatric illness may be a more direct consequence of abuse. Childhood maltreatment exerts a prepotent influence on brain development and has been an unrecognized confound in almost all psychiatric neuroimaging studies. These brain changes may be best understood as adaptive responses to facilitate survival and reproduction in the face of adversity. Their relationship to psychopathology is complex as they are discernible in both susceptible and resilient individuals with maltreatment histories. Mechanisms fostering resilience will need to be a primary focus of future studies. **Keywords:** Child abuse; neglect; neuroimaging; resilience; stress.

Curr Psychiatry Rep (2017) 19: 69
DOI 10.1007/s11920-017-0825-3

Trauma, PTSD, and the Developing Brain

Ryan J. Herringa[1]

Published online: 19 August 2017

Abstract

Purpose of Review PTSD in youth is common and debilitating. In contrast to adult PTSD, relatively little is known about the neurobiology of pediatric PTSD, nor how neurodevelopment may be altered. This review summarizes recent neuroimaging studies in pediatric PTSD and discusses implications for future study.

Recent Findings Pediatric PTSD is characterized by abnormal structure and function in neural circuitry supporting threat processing and emotion regulation. Furthermore, cross-sectional studies suggest that youth with PTSD have abnormal frontolimbic development compared to typically developing youth. Examples include declining hippocampal volume, increasing amygdala reactivity, and declining amygdala-prefrontal coupling with age.

Summary Pediatric PTSD is characterized by both overt and developmental abnormalities in frontolimbic circuitry. Notably, abnormal frontolimbic development may contribute to increasing threat reactivity and weaker emotion regulation as youth age. Longitudinal studies of pediatric PTSD are needed to characterize individual outcomes and determine whether current treatments are capable of restoring healthy neurodevelopment.

Introduction

Approximately two-thirds of youth are exposed to trauma during childhood, and many develop PTSD as a result [1]. By age 18, roughly 8% of traumatized youth have met criteria for a diagnosis of PTSD, with numbers rising up to 40% in cases of sexual abuse and assault [1]. In addition to the psychological suffering imposed, PTSD is associated with lower academic achievement, and increasing incidence of depression, suicide attempts, and substance abuse into adulthood [2]. Childhood trauma and PTSD also pose a tremendous societal cost in terms of health care utilization and financial outlay. For example, the sequelae of childhood maltreatment, including PTSD, are estimated to cost the USA over $500 billion annually [3]. These sobering statistics highlight the need to elucidate neurodevelopmental disruptions in youth with PTSD, with the aim of mitigating its effects throughout the lifespan.

Neuroimaging studies in adult PTSD suggest structural and functional abnormalities in frontolimbic circuitry supporting threat processing and emotion regulation. Briefly, structural brain meta-analyses in adult PTSD show decreased gray matter volume in the dorsal anterior cingulate cortex (dACC) and

Review › Int Rev Neurobiol. 2020;152:1-22. doi: 10.1016/bs.irn.2020.01.004.
Epub 2020 Feb 19.

Stress, the brain, and trauma spectrum disorders

J Douglas Bremner [1], Matthew T Wittbrodt [2]

Affiliations + expand

PMID: 32450992 PMCID: PMC8214870 DOI: 10.1016/bs.irn.2020.01.004

Free PMC article

Abstract

This chapter reviews the relationship between stress and brain function in patients with neuropsychiatric disorders, with an emphasis on disorders that have most clearly been linked to traumatic stress exposure. These disorders, which have been described as trauma spectrum disorders, include posttraumatic stress disorder (PTSD), a subgroup of major depression, borderline personality disorder (BPD) and dissociative disorders; they share in common a neurobiological footprint, including smaller hippocampal volume, and are distinguished from other disorders that may share symptom similarities, like some of the anxiety disorders, but are not as clearly linked to stress. The relationship between environmental events such as stressors, especially in early childhood, and their effects on brain and neurobiology is important to understand in approaching these disorders as well as the development of therapeutic interventions. Addressing patients with stress-related disorders from multiple developmental (age at onset of trauma) as well as levels of analysis (cognitive, cultural, neurobiological) approaches will provide the most complete picture and result in the most successful treatment outcomes.

Keywords: Borderline personality disorder; Child abuse; Child development; Child development disorders; Depressive disorder; Dissociative disorders; Neurobiology

Journal of Psychiatric Research 128 (2020) 5–15

Contents lists available at ScienceDirect

Journal of Psychiatric Research

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

Structural and functional brain alterations in psychiatric patients with dissociative experiences: A systematic review of magnetic resonance imaging studies

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[c] Department of Clinical Psychology, University of Groningen, the Netherlands

ARTICLE INFO

Keywords:
Dissociation
Neuroimaging
MRI
Depersonalization
Derealization

ABSTRACT

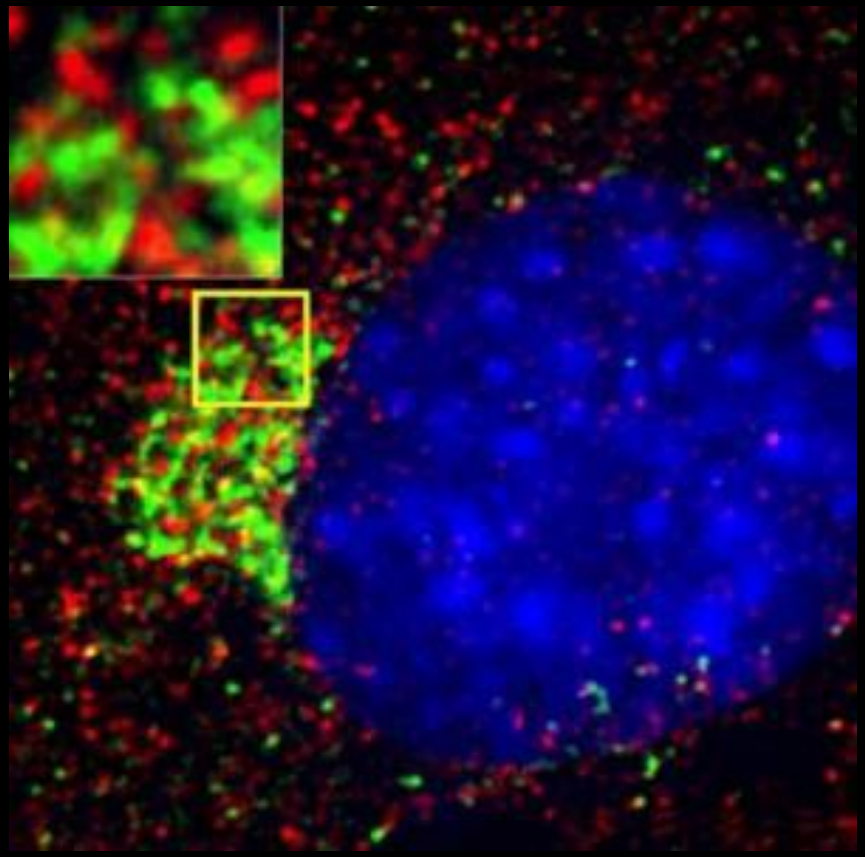
Introduction: There is currently no general agreement on how to best conceptualize dissociative symptoms and whether they share similar neural underpinnings across dissociative disorders. Neuroimaging data could help elucidate these questions.

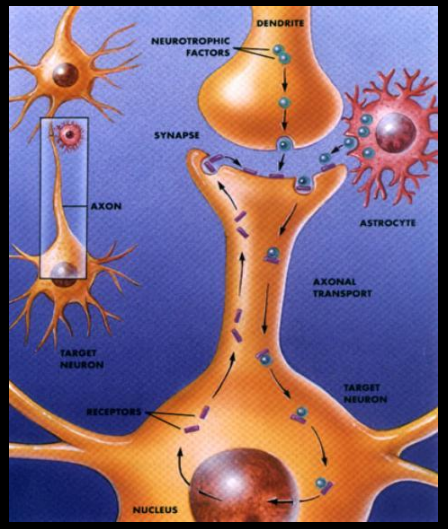
Objectives: The objective of this review is to summarize empirical evidence for neural aberrations observed in patients suffering from dissociative symptoms.

Methods: A systematic literature review was conducted including patient cohorts diagnosed with primary dissociative disorders, post-traumatic stress disorder (PTSD), or borderline personality disorder.

Results: Results from MRI studies reporting structural (gray matter and white matter) and functional (during resting-state and task-related activation) brain aberrations were extracted and integrated. In total, 33 articles were included of which 10 pertained to voxel-based morphology, 2 to diffusion tensor imaging, 10 to resting-state fMRI, and 11 to task-related fMRI. Overall findings indicated aberrations spread across diverse brain regions, especially in the temporal and frontal cortices. Patients with dissociative identity disorder and with dissociative PTSD showed more overlap in brain activation than each group showed with depersonalization/derealization disorder.

Conclusion: In conjunction, the results indicate that dissociative processing cannot be localized to a few distinctive brain regions but rather corresponds to differential neural signatures depending on the symptom constellation.





Vlivem stresu dochází ke snížení exprese neurotrofních faktorů např. BDNF, které má za následek změny v synaptické plasticitě, poruchy konsolidace paměti a může být příčínou neurodegenerativního procesu.

(Bondar & Merkulova, 2016; Phillips, 2017; Notaras & Buuse, 2020).

Na straně druhé integrované stavy vědomí, například v souvislosti s psychoterapií nebo meditací pravděpodobně mohou integrovat mozkové funkce.
(Baars, 2002; Stuckey et al., 2005)

-Znamená to že integrace mysli ovlivňuje mozek?

Neuroreport. 2005 November 28; 16(17): 1893–1897.

Meditation experience is associated with increased cortical thickness

Sara W. Lazar[a], Catherine E. Kerr[b], Rachel H. Wasserman[a,b], Jeremy R. Gray[c], Douglas N. Greve[d], Michael T. Treadway[a], Metta McGarvey[e], Brian T. Quinn[d], Jeffery A. Dusek[f,g], Herbert Benson[f,g], Scott L. Rauch[a], Christopher I. Moore[h,i], and Bruce Fischl[d,j]

Abstract

Previous research indicates that long-term meditation practice is associated with altered resting electroencephalogram patterns, suggestive of long lasting changes in brain activity. We hypothesized that meditation practice might also be associated with changes in the brain's physical structure. Magnetic resonance imaging was used to assess cortical thickness in 20 participants with extensive Insight meditation experience, which involves focused attention to internal experiences. Brain regions associated with attention, interoception and sensory processing were thicker in meditation participants than matched controls, including the prefrontal cortex and right anterior insula. Between-group differences in prefrontal cortical thickness were most pronounced in older participants, suggesting that meditation might offset age-related cortical thinning. Finally, the thickness of two regions correlated with meditation experience. These data provide the first structural evidence for experience-dependent cortical plasticity associated with meditation practice.

NeuroImage 61 (2012) 181–187

Contents lists available at SciVerse ScienceDirect

NeuroImage

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

Bridging the hemispheres in meditation: Thicker callosal regions and enhanced fractional anisotropy (FA) in long-term practitioners

Eileen Luders [a], Owen R. Phillips [a], Kristi Clark [a], Florian Kurth [b], Arthur W. Toga [a,*], Katherine L. Narr [a]

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ARTICLE INFO

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Corpus callosum
DTI
Mindfulness
MRI
Plasticity

ABSTRACT

Recent findings suggest a close link between long-term meditation practices and the structure of the corpus callosum. Prior analyses, however, have focused on estimating mean fractional anisotropy (FA) within two large pre-defined callosal tracts only. Additional effects might exist in other, non-explored callosal regions and/or with respect to callosal attributes not captured by estimates of FA. To further explore callosal features in the framework of meditation, we analyzed 30 meditators and 30 controls, carefully matched for sex, age, and handedness. We applied a multimodal imaging approach using diffusion tensor imaging (DTI) in combination with structural magnetic resonance imaging (MRI). Callosal measures of tract-specific FA were complemented with other global (segment-specific) estimates as well as extremely local (point-wise) measures of callosal micro- and macro-structure. Callosal measures were larger in long-term meditators compared to controls, particularly in anterior callosal sections. However, differences achieved significance only when increasing the regional sensitivity of the measurement (i.e., using point-wise measures versus segment-specific measures) and were more prominent for microscopic than macroscopic characteristics (i.e., callosal FA versus callosal thickness). Thicker callosal regions and enhanced FA in meditators might indicate greater connectivity, possibly reflecting increased hemispheric integration during cerebral processes involving (pre)frontal regions. Such a brain organization might be linked to achieving characteristic mental states and skills as associated with meditation, though this hypothesis requires behavioral confirmation. Moreover, longitudinal studies are required to address whether the observed callosal effects are induced by meditation or constitute an innate prerequisite for the start or successful continuation of meditation.

Psychiatry Research: Neuroimaging 191 (2011) 36–43

Contents lists available at ScienceDirect

Psychiatry Research: Neuroimaging

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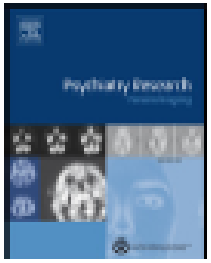

Mindfulness practice leads to increases in regional brain gray matter density

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ABSTRACT

Therapeutic interventions that incorporate training in mindfulness meditation have become increasingly popular, but to date little is known about neural mechanisms associated with these interventions. Mindfulness-Based Stress Reduction (MBSR), one of the most widely used mindfulness training programs, has been reported to produce positive effects on psychological well-being and to ameliorate symptoms of a number of disorders. Here, we report a controlled longitudinal study to investigate pre–post changes in brain gray matter concentration attributable to participation in an MBSR program. Anatomical magnetic resonance (MR) images from 16 healthy, meditation-naïve participants were obtained before and after they underwent the 8-week program. Changes in gray matter concentration were investigated using voxel-based morphometry, and compared with a waiting list control group of 17 individuals. Analyses in *a priori* regions of interest confirmed increases in gray matter concentration within the left hippocampus. Whole brain analyses identified increases in the posterior cingulate cortex, the temporo-parietal junction, and the cerebellum in the MBSR group compared with the controls. The results suggest that participation in MBSR is associated with changes in gray matter concentration in brain regions involved in learning and memory processes, emotion regulation, self-referential processing, and perspective taking.

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Short-term meditation induces white matter changes in the anterior cingulate

Yi-Yuan Tang[a,b,1], Qilin Lu[a], Xiujuan Geng[c], Elliot A. Stein[c], Yihong Yang[c], and Michael I. Posner[b,1]

The anterior cingulate cortex (ACC) is part of a network implicated in the development of self-regulation and whose connectivity changes dramatically in development. In previous studies we showed that 3 h of mental training, based on traditional Chinese medicine (integrative body–mind training, IBMT), increases ACC activity and improves self-regulation. However, it is not known whether changes in white matter connectivity can result from small amounts of mental training. We here report that 11 h of IBMT increases fractional anisotropy (FA), an index indicating the integrity and efficiency of white matter in the corona radiata, an important white-matter tract connecting the ACC to other structures. Thus IBMT could provide a means for improving self-regulation and perhaps reducing or preventing various mental disorders.

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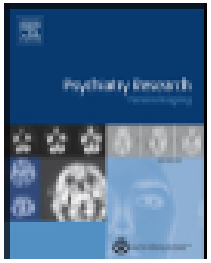

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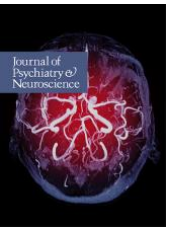
Mindfulness-based treatments for posttraumatic stress disorder: a review of the treatment literature and neurobiological evidence



Jenna E. Boyd, MSc; Ruth A. Lanius, MD, PhD; Margaret C. McKinnon, PhD, CPsych

Mindfulness-based treatments for posttraumatic stress disorder (PTSD) have emerged as promising adjunctive or alternative intervention approaches. A scoping review of the literature on PTSD treatment studies, including approaches such as mindfulness-based stress reduction, mindfulness-based cognitive therapy and metta mindfulness, reveals low attrition with medium to large effect sizes. We review the convergence between neurobiological models of PTSD and neuroimaging findings in the mindfulness literature, where mindfulness interventions may target emotional under- and overmodulation, both of which are critical features of PTSD symptomatology. Recent emerging work indicates that mindfulness-based treatments may also be effective in restoring connectivity between large-scale brain networks among individuals with PTSD, including connectivity between the default mode network and the central executive and salience networks. Future directions, including further identification of the neurobiological mechanisms of mindfulness interventions in patients with PTSD and direct comparison of these interventions to first-line treatments for PTSD are discussed.

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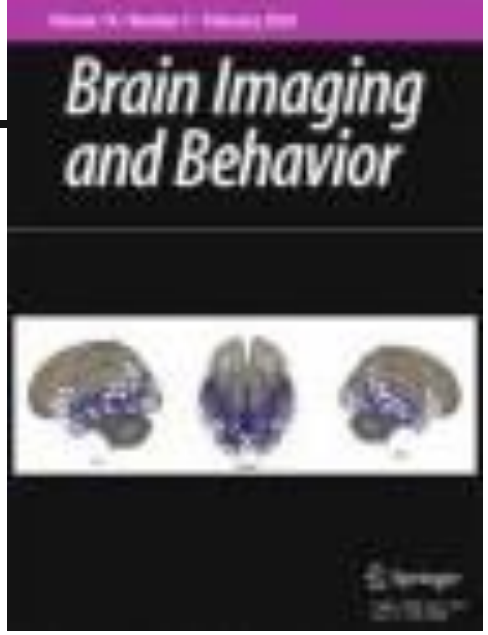

REVIEW ARTICLE

Mindfulness related changes in grey matter: a systematic review and meta-analysis

Cyril R. Pernet[1] · Nikolai Belov[2] · Arnaud Delorme[3,4] · Alison Zammit[1]

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Abstract

Knowing target regions undergoing strfuncti changes caused by behavioural interventions is paramount in evaluating the effectiveness of such practices. Here, using a systematic review approach, we identified 25 peer-reviewed magnetic resonance imaging (MRI) studies demonstrating grey matter changes related to mindfulness meditation. An activation likelihood estimation (ALE) analysis (n = 16) revealed the right anterior ventral insula as the only significant region with consistent effect across studies, whilst an additional functional connectivity analysis indicates that both left and right insulae, and the anterior cingulate gyrus with adjacent paracingulate gyri should also be considered in future studies. Statistical meta-analyses suggest medium to strong effect sizes from Cohen's d ~ 0.8 in the right insula to ~ 1 using maxima across the whole brain. The systematic review revealed design issues with selection, information, attrition and confirmation biases, in addition to weak statistical power. In conclusion, our analyses show that mindfulness meditation practice does induce grey matter changes but also that improvements in methodology are needed to establish mindfulness as a therapeutic intervention.

Děkuji Vám za pozornost.

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