Can Nutrition be Part of the Solution to Concussions and TBI's?

Dr. Garrett Wdowin

Today's Plan

- Terminology
- Concussion Pathology
- Important Roll of Nutrition and Nutraceuticals
- Answer the Following Question Confidently

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Can Nutrition be Part of the Solution to Concussions and TBI's?

3

Yes

4

Absolutely, Nutrition is Part of the Solution to Concussions and TBI's

5

Garrett Wdowin NMD, FAARM, ABAAHP

- Ivy League Football Championship Team, Brown U. '99
- Brown University '03
- Naturopathic Medical Doctor '07
- Fellowship in Stem Cell Therapeutics '12
- American Board of Anti-Aging Health Practitioners '12
- Owner and founder of Wdowin NMD and Cover Three

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Nutritional Terms

- Docosahexaenoic Acid (DHA) - omega-3 fatty acid that is a primary structural component of the human brain and cerebral cortex. Antioxidant, etc.
- Eicosapentaenoic Acid (EPA) - omega-3 fatty acid and structural components in cell membrane phospholipid bilayers and synaptic membranes in the brain
- Curcumin - bright yellow polyphenol produced by some plants, principal curcuminoid of turmeric, anti-inflammatory among other things.
- Resveratrol - polyphenol, antioxidant and induces mitochondrial biogenesis
- Alpha-glycerophosphocholine (Alpha GPC) - choline containing molecule, cognitive-promoting properties and improves cell membrane health
- Uridine Monophosphate - nucleotide base, increases synthesis of cellular membranes and synergistic with choline and omega 3 fatty acids
- Anthocyanins - universal plant colorants (red, purple, and blue hues) evident in many fruits, vegetables. Powerful antioxidants.

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Concussion

comes from the Latin word **concutere**, which means "to shake violently"

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Signs and Symptoms

Concussion Signs Observed	Concussion Symptoms Reported
• Can't recall events *prior to* or *after* a hit or fall. • Appears dazed or stunned. • Forgets an instruction, is confused about an assignment or position, or is unsure of the game, score, or opponent. • Moves clumsily. • Answers questions slowly. • Loses consciousness *(even briefly)*. • Shows mood, behavior, or personality changes.	• Headache or "pressure" in head. • Nausea or vomiting. • Balance problems or dizziness, or double or blurry vision. • Bothered by light or noise. • Feeling sluggish, hazy, foggy, or groggy. • Confusion, or concentration or memory problems. • Just not "feeling right," or "feeling down".

https://www.cdc.gov/headsup/basics/concussion_symptoms.html

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Concussion Pathology

What is happening in the brain?

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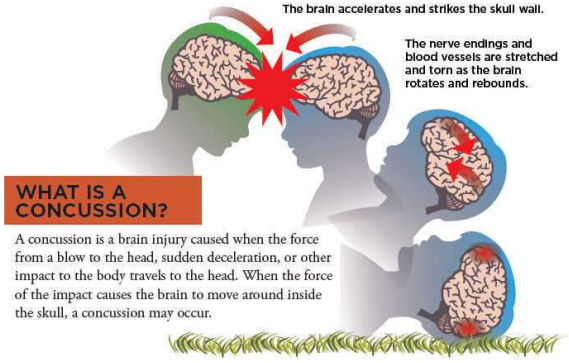
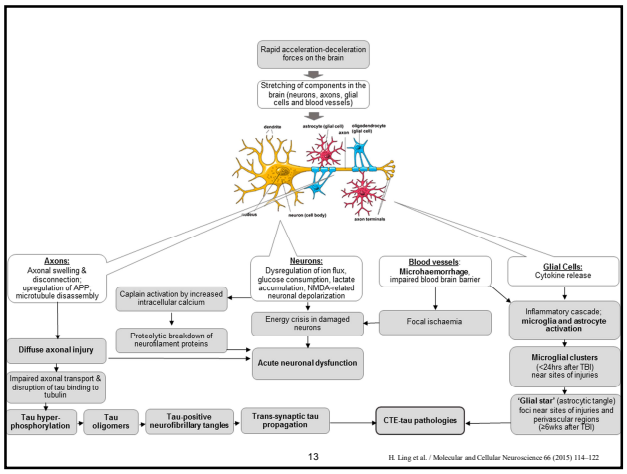


12 https://www.childrenshospitaloakland.org/main/concussion-guide2.aspx



- Physical Damage
- Membrane Excitotoxicity - Ion Flux
- Hypermetabolism - Oxidative Stress
- Inflammation - Cytokine Release
- Brain Repair

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With Nutrition

- How do we make stronger brains?
- How do we decrease membrane excitotoxicity?
- How do we decrease oxidative stress?
- How do we improve the function of the blood brain barrier?
- How do we decrease neuroinflammation?
- How do we stimulate repair?

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Physical Damage

How do we make stronger brains?

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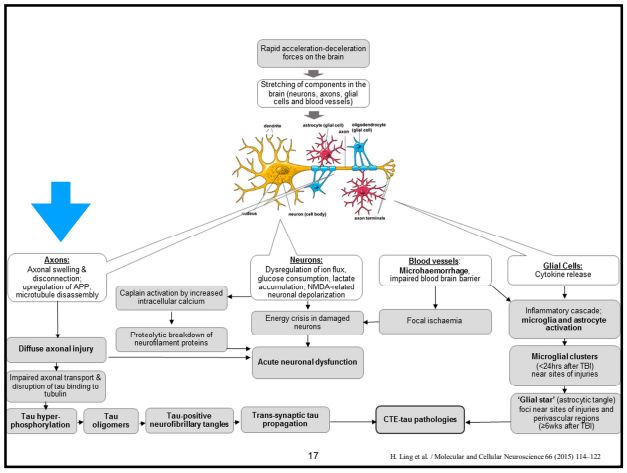

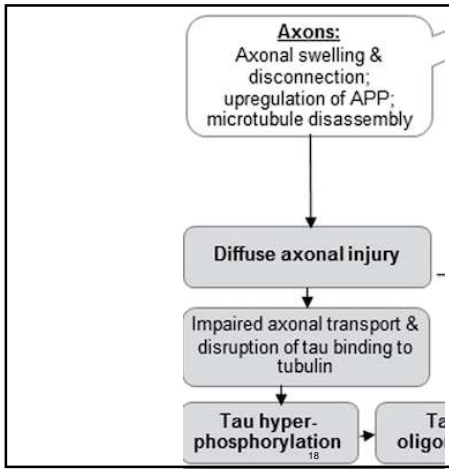

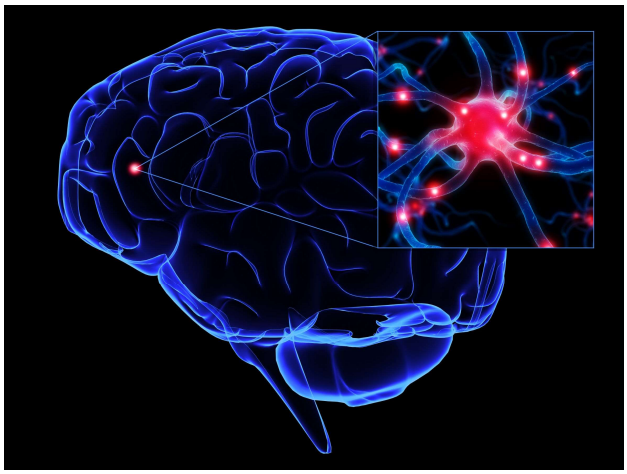

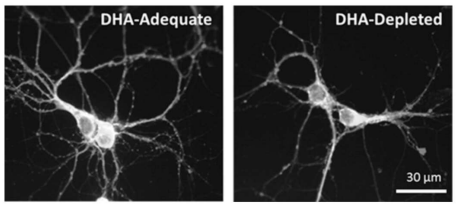

FIGURE 2. Hippocampal neurite growth and synaptogenesis (evaluated by synapsin puncta) impaired by DHA depletion.[24]

Neuroprotection by Docosahexanoic Acid in Brain Injury

Mil Med. 2014;179(suppl_11):106-111. doi:10.7205/MILMED-D-14-00162

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US National Library of Medicine National Institutes of Health

PubMed 24763080[uid]
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Nutrients. 2014 Apr 23;6(4):1701-10. doi: 10.3390/nu6041701.

A nutrient combination that can affect synapse formation.

Wurtman RJ[1].

Author information

Abstract

Brain neurons form synapses throughout the life span. This process is initiated by neuronal depolarization, however the numbers of synapses thus formed depend on brain levels of three key nutrients-uridine, the omega-3 fatty acid DHA, and choline. Given together, these nutrients accelerate formation of synaptic membrane, the major component of synapses. In infants, when synaptogenesis is maximal, relatively large amounts of all three nutrients are provided in bioavailable forms (e.g., uridine in the UMP of mothers' milk and infant formulas). However, in adults the uridine in foods, mostly present at RNA, is not bioavailable, and no food has ever been compelling demonstrated to elevate plasma uridine levels. Moreover, the quantities of DHA and choline in regular foods can be insufficient for raising their blood levels enough to promote optimal synaptogenesis. In Alzheimer's disease (AD) the need for extra quantities of the three nutrients is enhanced, both because their basal plasma levels may be subnormal (reflecting impaired hepatic synthesis), and because especially high brain levels are needed for correcting the disease-related deficiencies in synaptic membrane and synapses.

PMID: 24763080 PMCID: PMC4011061 DOI: 10.3390/nu6041701

[Indexed for MEDLINE] Free PMC Article

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US National Library of Medicine National Institutes of Health

PubMed 22128646[uid]
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Mil Med. 2011 Oct;176(10):1120-7.

Neuroprotection for the warrior: dietary supplementation with omega-3 fatty acids.

Lewis MD[1], Bailes J.

Author information

Abstract

Nutrition has traditionally involved in supplying energy and hydration. An emerging concept developed by the authors is the concept of using omega-3 fatty acids (n-3 FAs) to increase the resilience of the brain. The n-3 FAs have numerous proven benefits including support of cardiovascular and psychiatric health. Docosahexaenoic acid in particular, is found in high concentrations in the brain. N-3 FAs provide benefits by exerting a protective mechanism at the cellular and neuronal levels including the modulation of inflammatory cascade following traumatic brain injury. Promising research and evolving clinical experience now indicate that n-3 FA is useful and effective for recovery following traumatic brain injury. More exciting is that new laboratory research shows the beneficial effects extend to when n-3 FA is given before injury. Given the safety profile, availability, and affordability of n-3 FA, Generally Recognized As Safe amounts of eicosapentaenoic acid and docosahexaenoic acid (up to 3,000 mg daily) should be considered for the athlete and soldier, not only for its general health benefits, but particularly also for those at risk or high exposure to brain impacts. A comprehensive, coordinated research program to evaluate the multiple uses of n-3 FA should be a high priority for the Department of Defense.

PMID: 22128646

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Med Sci Sports Exerc. 2016 Jun;48(6):974-82. doi: 10.1249/MSS.0000000000000875.

Effect of Docosahexaenoic Acid on a Biomarker of Head Trauma in American Football.

Oliver JM[1], Jones MT, Kirk KM, Gable DA, Repshas JT, Johnson TA, Andréasson U, Norgren N, Blennow K, Zetterberg H.

Author information

Abstract

PURPOSE: American football athletes are exposed to subconcussive impacts over the course of the season resulting in elevations in serum neurofilament light (NFL), a biomarker of axonal injury. Docosahexaenoic acid (DHA) has been reported to reduce axonal trauma associated with traumatic brain injury in rodent models. However, the optimal dose in American football athletes is unknown. This study examined the effect of differing doses of DHA on serum NFL over the course of a season of American football.

METHODS: In a randomized, double-blind, placebo-controlled, parallel design, 81 National Collegiate Athletic Association Division I American football athletes were assigned to ingest either 2, 4, 6 g·d of DHA or placebo. Blood was sampled at specific times over the course of 189 d, coincident with changes in intensity, hours of contact, and likely changes in head impacts. Standardized magnitude-based inference was used to define outcomes.

RESULTS: DHA supplementation increased plasma DHA in a dose-dependent manner (2 g·d: mean difference from baseline; ±90% CL; 2 g·d: 1.3; ±0.6; 4 g·d: 1.6; ±0.7%; 6 g·d: 2.8; ±1.2%). Serum NFL increased to a greater extent in starters (area under the curve, 1995 ± 1383 pg·mL) versus nonstarters (1398 ± 581 pg·mL; P = 0.024). Irrespective of dose, supplemental DHA likely attenuated serum NFL coincident with increases in serum NFL by likely small and moderate magnitude (effect size = 0.4-0.7).

CONCLUSIONS: Findings from this study, the first large-scale study examining potential prophylactic use of DHA in American football athletes, include identification of optimal dose of DHA, suggesting a neuroprotective effect of DHA supplementation.

PMID: 26765633 DOI: 10.1249/MSS.0000000000000875

[Indexed for MEDLINE]

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J Biol Chem. 2013 Feb 8;288(6):4056-65. doi: 10.1074/jbc.M112.393751. Epub 2012 Dec 21.

Curcumin suppresses soluble tau dimers and corrects molecular chaperone, synaptic, and behavioral deficits in aged human tau transgenic mice.

Ma QL[1], Zuo X, Yang F, Ubeda OJ, Gant DJ, Alaverdyan M, Teng E, Hu S, Chen PP, Maiti P, Teter B, Cole GM, Frautschy SA.

Author information

Abstract

The mechanisms underlying Tau-related synaptic and cognitive deficits and the interrelationships between Tau species, their clearance pathways, and synaptic impairments remain poorly understood. To gain insight into these mechanisms, we examined these interrelationships in aged non-mutant genomic human Tau mice, with established Tau pathology and neuron loss. We also examined how these interrelationships changed with an intervention by feeding mice either a control diet or one containing the brain permeable beta-amyloid and Tau aggregate binding molecule curcumin. Transgene-dependent elevations in soluble and insoluble phospho-Tau monomer and soluble Tau dimers accompanied deficits in behavior, hippocampal excitatory synaptic markers, and molecular chaperones (heat shock proteins (HSPs)) involved in Tau degradation and microtubule stability. In human Tau mice but not control mice, HSP70, HSP70/HSP72, and HSP90 were reduced in membrane-enriched fractions but not in cytosolic fractions. The synaptic proteins PSD95 and NR2B were reduced in dendritic fields and redistributed into perikarya, corresponding to changes observed by immunoblot. Curcumin selectively suppressed levels of soluble Tau dimers, but not of insoluble and monomeric phospho-Tau, while correcting behavioral, synaptic, and HSP deficits. Treatment increased PSD95 co-immunoprecipitating with NR2B and, independent of transgene, increased HSPs implicated in Tau clearance. It elevated HSP90 and HSC70 without increasing HSP mRNAs; that is, without induction of the heat shock response. Instead curcumin differentially impacted HSP90 client kinases, reducing Fyn without reducing Akt. In summary, curcumin reduced soluble Tau and elevated HSPs involved in Tau clearance, showing that even after tangles have formed, Tau-dependent behavioral and synaptic deficits can be corrected.

PMID: 23264626 PMCID: PMC3567657 DOI: 10.1074/jbc.M112.393751

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Membrane Excitotoxicity - Ion Flux

How do we decrease membrane excitotoxicity?

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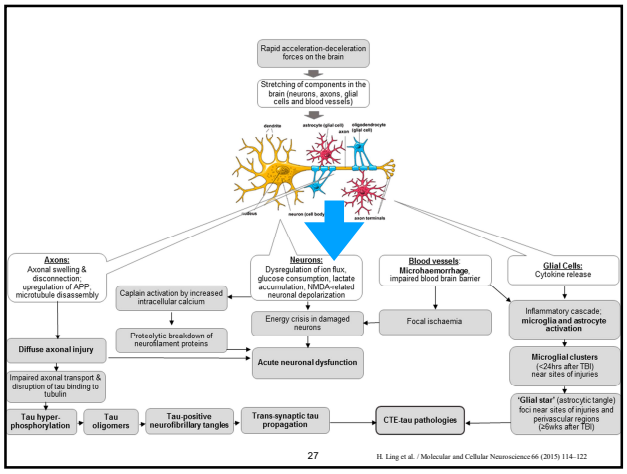
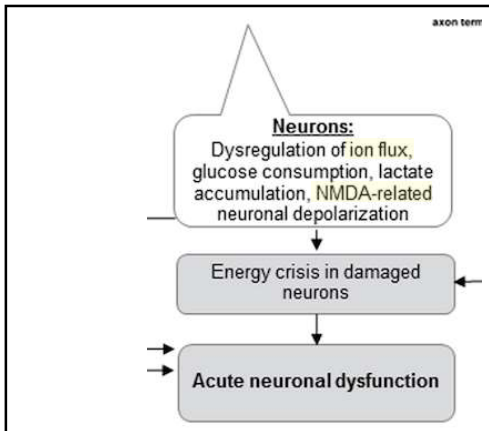


27 H. Ling et al. / Molecular and Cellular Neuroscience 66 (2015) 114–122



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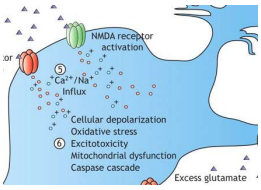

Curcumin… protected both retinal and hippocampal neurons against NMDA-induced cell death, confirming its anti-excitotoxic property.

Curcumin Protects against NMDA-Induced Toxicity: A Possible Role for NR2A Subunit.

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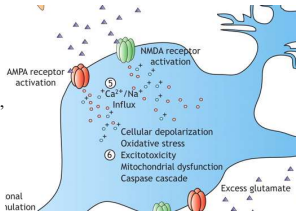
Oxidative stress mediated by NMDA, AMPA/KA channels in acute hippocampal slices: Neuroprotective effect of resveratrol.



Highlights

- Glutamate toxicity induces oxidative stress and cellular damage.
- Resveratrol prevents oxidative stress mediated by NMDA, AMPA/KA receptors.
- Resveratrol modulates $\Delta\Psi_m$, Na^+K^+-ATPase and glutamine synthetase activity.
- Resveratrol presents neuroprotective effect against glutamate toxicity.

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Hypermetabolism - Oxidative Stress

How do we decrease oxidative stress?

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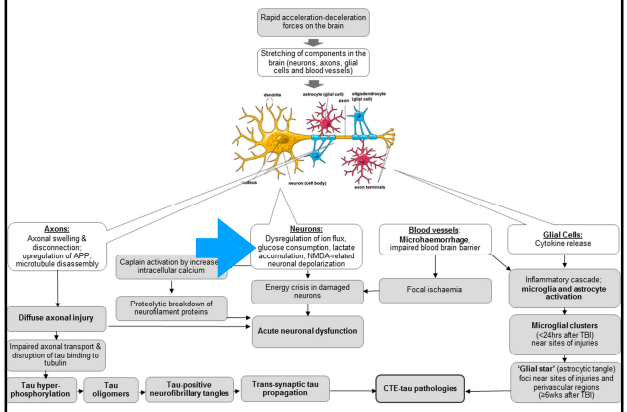

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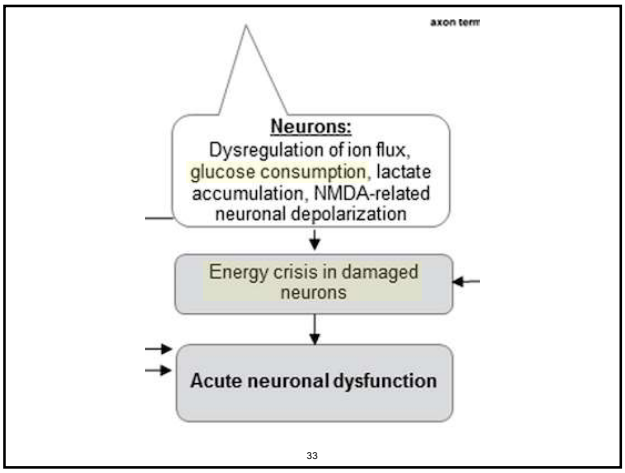

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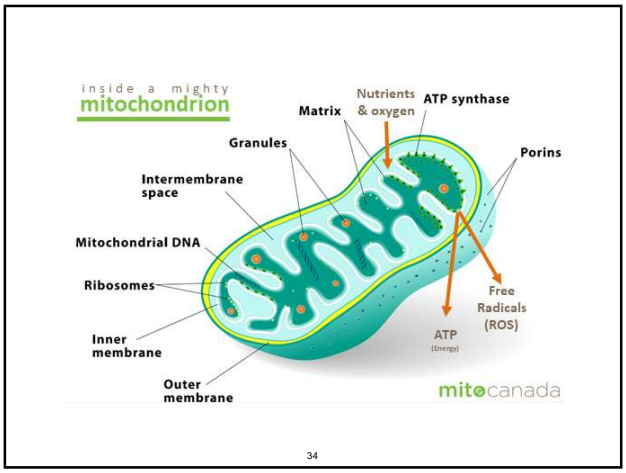

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Antioxidants

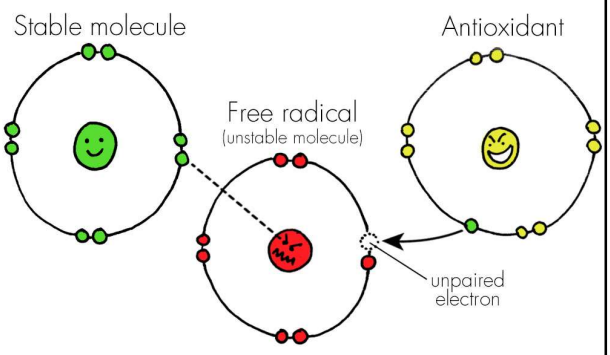
Eat your fruits and vegetables

35

What is an Antioxidant?

- a substance that inhibits oxidation
- a substance such as vitamin C or E that removes potentially damaging oxidizing agents in a living organism

https://www.dictionary.com/browse/antioxidant?s=t



37 https://www.haleo.co.uk/the-body/antioxidants/

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Appl Physiol Nutr Metab. 2011 Dec;36(6):976-84. doi: 10.1139/h11-120. Epub 2011 Nov 23.

Effect of blueberry ingestion on natural killer cell counts, oxidative stress, and inflammation prior to and after 2.5 h of running.

McAnulty LS[1], Nieman DC, Dumke CL, Shooter LA, Henson DA, Utter AC, Milne G, McAnulty SR.

Author information

Abstract

Blueberries are rich in antioxidants known as anthocyanins, which may exhibit significant health benefits. Strenous exercise is known to acutely generate oxidative stress and an inflammatory state, and serves as an on-demand model to test antioxidant and anti-inflammatory compounds. The purpose of this study was to examine whether 250 g of blueberries per day for 6 weeks and 375 g given 1 h prior to 2.5 h of running at ~72% maximal oxygen consumption counters oxidative stress, inflammation, and immune changes. Twenty-five well-trained subjects were recruited and randomized into blueberry (BB) (N = 13) or control (CON) (N = 12) groups. Blood, muscle, and urine samples were obtained pre-exercise and immediately postexercise, and blood and urine 1 h postexercise. Blood was examined for F_2-isoprostanes for oxidative stress, cortisol, cytokines, homocysteine, leukocytes, T-cell function, natural killer (NK), and lymphocyte cell counts for inflammation and immune system activation, and ferric reducing ability of plasma for antioxidant capacity. Muscle biopsies were examined for glycogen and NFkB expression to evaluate stress and inflammation. Urine was tested for modification of DNA (8-OHDG) and RNA (5-OHMU) as markers of nucleic acid oxidation. A 2 (treatment) × 3 (time) repeated measures ANOVA was used for statistical analysis. Increases in F_2-isoprostanes and 5-OHMU were significantly less in BB and plasma IL-10 and NK cell counts were significantly greater in BB vs. CON. Changes in all other markers did not differ. This study indicates that daily blueberry consumption for 6 weeks increases NK cell counts, and acute ingestion reduces oxidative stress and increases anti-inflammatory cytokines.

PMID: 22111516 DOI: 10.1139/h11-120

[Indexed for MEDLINE]

Blood Brain Barrier

How do we protect the function?

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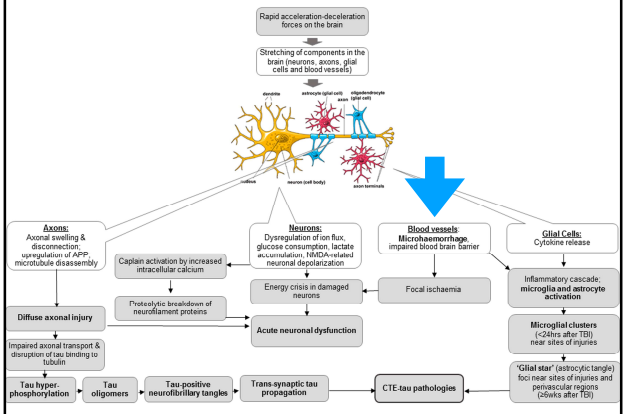
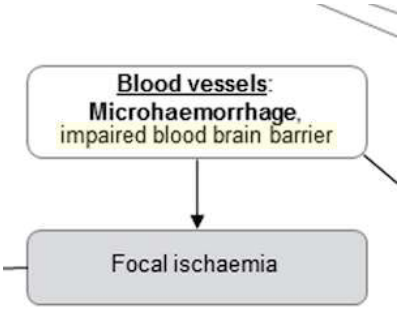


40 H. Ling et al. / Molecular and Cellular Neuroscience 66 (2015) 114–122



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Ann N Y Acad Sci. 2017 Sep;1403(1):142-149. doi: 10.1111/nyas.13431. Epub 2017 Aug 16.

Resveratrol for Alzheimer's disease.

Sawda C[1], Moussa C[1,2], Turner RS[1,2].

Author information

Abstract

The amyloid hypothesis suggests that the progressive accumulation and deposition of central nervous system (CNS) amyloid with aging is the proximate cause of Alzheimer's disease (AD). Thus, targeting molecular mechanisms of aging may be a viable treatment approach. Caloric restriction prevents diseases of aging, including AD, in animal models, perhaps by activation of sirtuins. The sirtuins (e.g., mammalian SIRT1) are deacetylases that link energy balance (NAD^+ /NADH) to regulation of gene transcription. Resveratrol is a potent activator of SIRT1, and thus may mimic caloric restriction to prevent diseases of aging. We conducted a randomized, double-blind, placebo-controlled, phase II trial of resveratrol for individuals with mild-to-moderate AD. Resveratrol (1) is detectable in cerebrospinal fluid (at low nanomolar levels), (2) is safe and well tolerated, (3) alters AD biomarker trajectories, (4) preserves blood-brain barrier integrity, and (5) modulates the CNS immune response. Further studies are needed to determine the safety and efficacy of resveratrol and the validity of this approach in the treatment and prevention of AD and other diseases of aging.

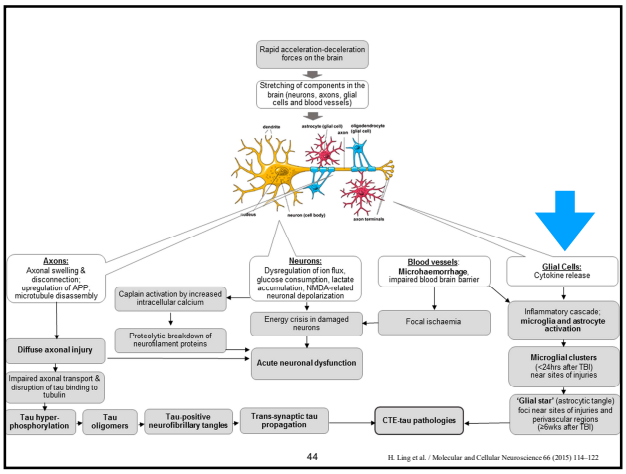
KEYWORDS: Alzheimer's disease; amyloid; polyphenol; resveratrol; sirtuin

PMID: 28815614 PMCID: PMC5664214 [Available on 2018-09-01] DOI: 10.1111/nyas.13431

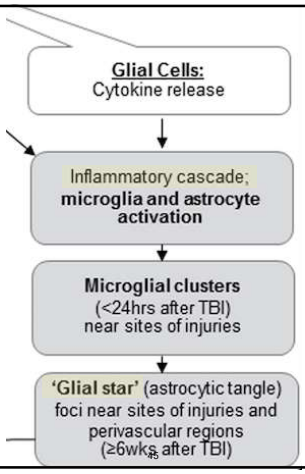
[Indexed for MEDLINE]

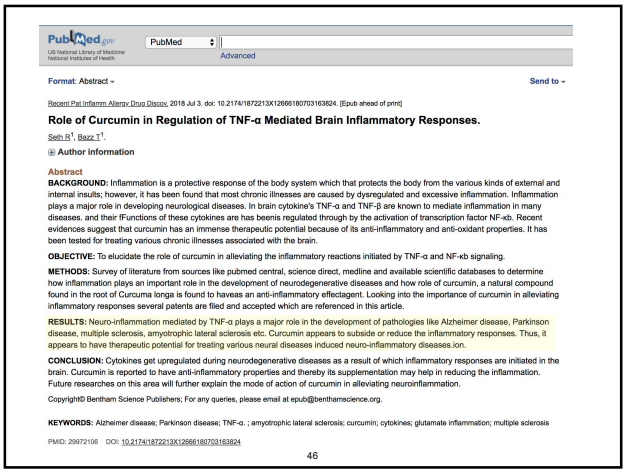
Inflammation - Cytokine Release

How do we decrease neuroinflammation?



H. Ling et al. / Molecular and Cellular Neuroscience 66 (2015) 114–122





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Recent Pat Inflamm Allergy Drug Discov. 2018 Jul 3. doi: 10.2174/1872213X12666180703163824. [Epub ahead of print]

Role of Curcumin in Regulation of TNF-α Mediated Brain Inflammatory Responses.

Seth R[1], Bazz T[1].

Author information

Abstract

BACKGROUND: Inflammation is a protective response of the body system which that protects the body from the various kinds of external and internal insults; however, it has been found that most chronic illnesses are caused by dysregulated and excessive inflammation. Inflammation plays a major role in developing neurological diseases. In brain cytokine's TNF-α and TNF-β are known to mediate inflammation in many diseases. and their IFunctions of these cytokines are has beenis regulated through by the activation of transcription factor NF-κb. Recent evidences suggest that curcumin has an immense therapeutic potential because of its anti-inflammatory and anti-oxidant properties. It has been tested for treating various chronic illnesses associated with the brain.

OBJECTIVE: To elucidate the role of curcumin in alleviating the inflammatory reactions initiated by TNF-α and NF-κb signaling.

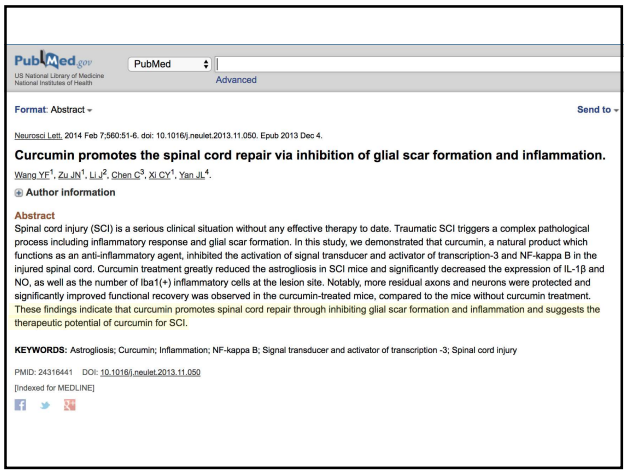
METHODS: Survey of literature from sources like pubmed central, science direct, medline and available scientific databases to determine how inflammation plays an important role in the development of neurodegenerative diseases and how role of curcumin, a natural compound found in the root of Curcuma longa is found to haveas an anti-inflammatory effectagent. Looking into the importance of curcumin in alleviating inflammatory responses several patents are filed and accepted which are referenced in this article.

RESULTS: Neuro-inflammation mediated by TNF-α plays a major role in the development of pathologies like Alzheimer disease, Parkinson disease, multiple sclerosis, amyotrophic lateral sclerosis etc. Curcumin appears to subside or reduce the inflammatory responses. Thus, it appears to have therapeutic potential for treating various neural diseases induced neuro-inflammatory diseases.ion.

CONCLUSION: Cytokines get upregulated during neurodegenerative diseases as a result of which inflammatory responses are initiated in the brain. Curcumin is reported to have anti-inflammatory properties and thereby its supplementation may help in reducing the inflammation. Future researches on this area will further explain the mode of action of curcumin in alleviating neuroinflammation.

KEYWORDS: Alzheimer disease; Parkinson disease; TNF-α. ; amyotrophic lateral sclerosis; curcumin; cytokines; glutamate inflammation; multiple sclerosis

PMID: 29972106 DOI: 10.2174/1872213X12666180703163824



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Neurosci Lett. 2014 Feb 7;560:51-6. doi: 10.1016/j.neulet.2013.11.050. Epub 2013 Dec 4.

Curcumin promotes the spinal cord repair via inhibition of glial scar formation and inflammation.

Wang YF[1], Zu JN[1], Li J[2], Chen C[3], Xi CY[1], Yan JL[4].

Author information

Abstract

Spinal cord injury (SCI) is a serious clinical situation without any effective therapy to date. Traumatic SCI triggers a complex pathological process including inflammatory response and glial scar formation. In this study, we demonstrated that curcumin, a natural product which functions as an anti-inflammatory agent, inhibited the activation of signal transducer and activator of transcription-3 and NF-kappa B in the injured spinal cord. Curcumin treatment greatly reduced the astrogliosis in SCI mice and significantly decreased the expression of IL-1β and NO, as well as the number of Iba1(+) inflammatory cells at the lesion site. Notably, more residual axons and neurons were protected and significantly improved functional recovery was observed in the curcumin-treated mice, compared to the mice without curcumin treatment. These findings indicate that curcumin promotes spinal cord repair through inhibiting glial scar formation and inflammation and suggests the therapeutic potential of curcumin for SCI.

KEYWORDS: Astrogliosis; Curcumin; Inflammation; NF-kappa B; Signal transducer and activator of transcription -3; Spinal cord injury

PMID: 24316441 DOI: 10.1016/j.neulet.2013.11.050

[Indexed for MEDLINE]

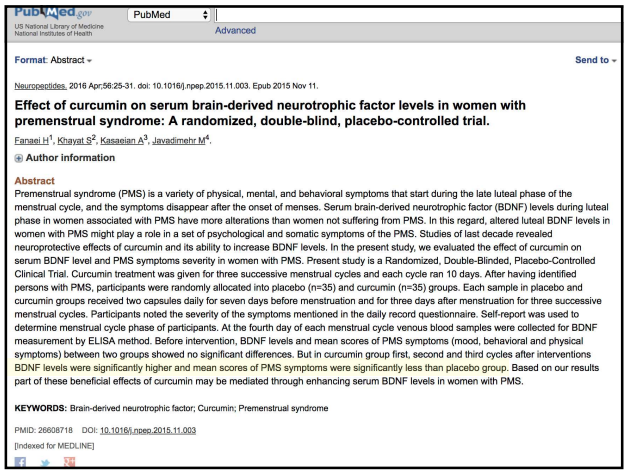
Repair

How do we stimulate repair?

Brain-Derived Neurotrophic Factor (BDNF)

- plays a significant role in neurogenesis (the growth and development of nervous tissue)
- enhances synaptogenesis (formation of synapses)
- enhances dendridogenesis (formation of neural dendrites)
- promotes protective pathways by inhibiting damage
- enhances cell survival
- etc

49 https://en.wikipedia.org/wiki/Brain-derived_neurotrophic_factor



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Neuropeptides. 2016 Apr;56:25-31. doi: 10.1016/j.npep.2015.11.003. Epub 2015 Nov 11.

Effect of curcumin on serum brain-derived neurotrophic factor levels in women with premenstrual syndrome: A randomized, double-blind, placebo-controlled trial.

Fanaei H[1], Khayat S[2], Kasaeian A[3], Javadimehr M[4].

Author information

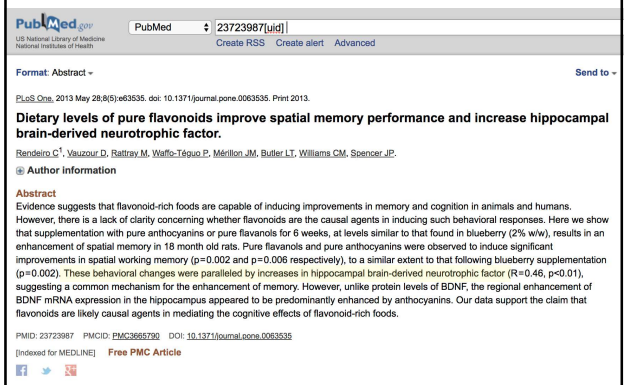
Abstract

Premenstrual syndrome (PMS) is a variety of physical, mental, and behavioral symptoms that start during the late luteal phase of the menstrual cycle, and the symptoms disappear after the onset of menses. Serum brain-derived neurotrophic factor (BDNF) levels during luteal phase in women associated with PMS have more alterations than women not suffering from PMS. In this regard, altered luteal BDNF levels in women with PMS might play a role in a set of psychological and somatic symptoms of the PMS. Studies of last decade revealed neuroprotective effects of curcumin and its ability to increase BDNF levels. In the present study, we evaluated the effect of curcumin on serum BDNF level and PMS symptoms severity in women with PMS. Present study is a Randomized, Double-Blinded, Placebo-Controlled Clinical Trial. Curcumin treatment was given for three successive menstrual cycles and each cycle ran 10 days. After having identified persons with PMS, participants were randomly allocated into placebo (n=35) and curcumin (n=35) groups. Each sample in placebo and curcumin groups received two capsules daily for seven days before menstruation and for three days after menstruation for three successive menstrual cycles. Participants noted the severity of the symptoms mentioned in the daily record questionnaire. Self-report was used to determine menstrual cycle phase of participants. At the fourth day of each menstrual cycle venous blood samples were collected for BDNF measurement by ELISA method. Before intervention, BDNF levels and mean scores of PMS symptoms (mood, behavioral and physical symptoms) between two groups showed no significant differences. But in curcumin group first, second and third cycles after interventions BDNF levels were significantly higher and mean scores of PMS symptoms were significantly less than placebo group. Based on our results part of these beneficial effects of curcumin may be mediated through enhancing serum BDNF levels in women with PMS.

KEYWORDS: Brain-derived neurotrophic factor; Curcumin; Premenstrual syndrome

PMID: 26608718 DOI: 10.1016/j.npep.2015.11.003

[Indexed for MEDLINE]



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PLoS One. 2013 May 28;8(5):e63535. doi: 10.1371/journal.pone.0063535. Print 2013.

Dietary levels of pure flavonoids improve spatial memory performance and increase hippocampal brain-derived neurotrophic factor.

Rendeiro C[1], Vauzour D, Rattray M, Waffo-Téguo P, Mérillon JM, Butler LT, Williams CM, Spencer JP.

Author information

Abstract

Evidence suggests that flavonoid-rich foods are capable of inducing improvements in memory and cognition in animals and humans. However, there is a lack of clarity concerning whether flavonoids are the causal agents in inducing such behavioral responses. Here we show that supplementation with pure anthocyanins or pure flavanols for 6 weeks, at levels similar to that found in blueberry (2% w/w), results in an enhancement of spatial memory in 18 month old rats. Pure flavanols and pure anthocyanins were observed to induce significant improvements in spatial working memory ($p=0.002$ and $p=0.006$ respectively), to a similar extent to that following blueberry supplementation ($p=0.002$). These behavioral changes were paralleled by increases in hippocampal brain-derived neurotrophic factor ($R=0.46$, $p<0.01$), suggesting a common mechanism for the enhancement of memory. However, unlike protein levels of BDNF, the regional enhancement of BDNF mRNA expression in the hippocampus appeared to be predominantly enhanced by anthocyanins. Our data support the claim that flavonoids are likely causal agents in mediating the cognitive effects of flavonoid-rich foods.

PMID: 23723987 PMCID: PMC3665790 DOI: 10.1371/journal.pone.0063535

[Indexed for MEDLINE] **Free PMC Article**

What Should we Eat?

52

Pop Quiz

53

A or B?

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A or B?

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A or B?

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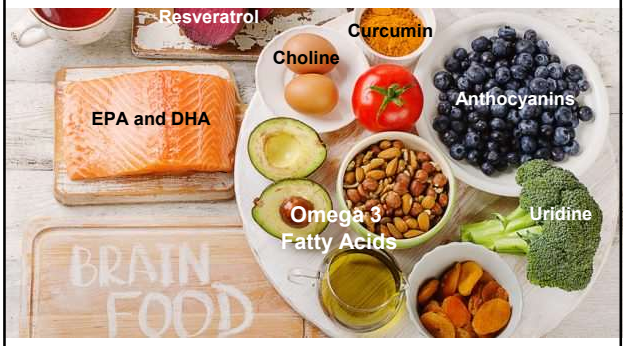

57 https://diabetickitchen.com/foods-and-recipes-for-brain-health/

58 https://medium.com/hallhang/11-brain-foods-to-boost-focus-and-memory-55604da8044b

Brain Healthy Diet

- Eat as organic as possible
- Eat as fresh as possible
- Avoid fried and processed foods
- Eat oily fish, wild caught salmon and trout
- Eat meat and eggs that are free range, grass finished
- Eat lots of plants of color, (blue violet, red) and anthocyanins
- Eat more curry or curcumin and spices
- Eat more red grapes and blueberries
- Eat as fresh as possible

59

In Summary

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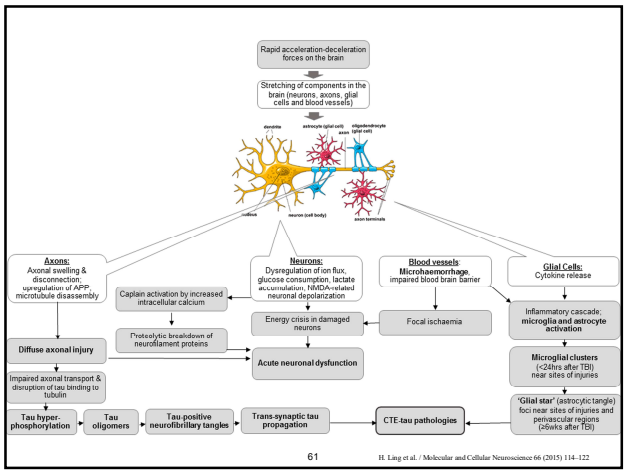

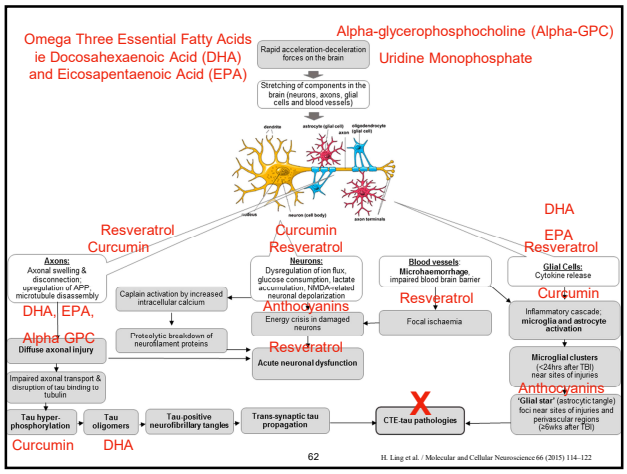

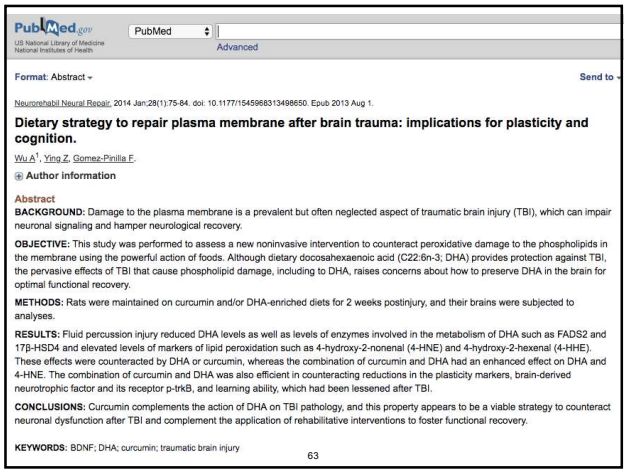

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Neurorehabil Neural Repair. 2014 Jan;28(1):75-84. doi: 10.1177/1545968313498650. Epub 2013 Aug 1.

Dietary strategy to repair plasma membrane after brain trauma: implications for plasticity and cognition.

Wu A[1], Ying Z, Gomez-Pinilla F.

Author information

Abstract

BACKGROUND: Damage to the plasma membrane is a prevalent but often neglected aspect of traumatic brain injury (TBI), which can impair neuronal signaling and hamper neurological recovery.

OBJECTIVE: This study was performed to assess a new noninvasive intervention to counteract peroxidative damage to the phospholipids in the membrane using the powerful action of foods. Although dietary docosahexaenoic acid (C22:6n-3; DHA) provides protection against TBI, the pervasive effects of TBI that cause phospholipid damage, including to DHA, raises concerns about how to preserve DHA in the brain for optimal functional recovery.

METHODS: Rats were maintained on curcumin and/or DHA-enriched diets for 2 weeks postinjury, and their brains were subjected to analyses.

RESULTS: Fluid percussion injury reduced DHA levels as well as levels of enzymes involved in the metabolism of DHA such as FADS2 and 17β-HSD4 and elevated levels of markers of lipid peroxidation such as 4-hydroxy-2-nonenal (4-HNE) and 4-hydroxy-2-hexenal (4-HHE). These effects were counteracted by DHA or curcumin, whereas the combination of curcumin and DHA had an enhanced effect on DHA and 4-HNE. The combination of curcumin and DHA was also efficient in counteracting reductions in the plasticity markers, brain-derived neurotrophic factor and its receptor p-trkB, and learning ability, which had been lessened after TBI.

CONCLUSIONS: Curcumin complements the action of DHA on TBI pathology, and this property appears to be a viable strategy to counteract neuronal dysfunction after TBI and complement the application of rehabilitative interventions to foster functional recovery.

KEYWORDS: BDNF; DHA; curcumin; traumatic brain injury

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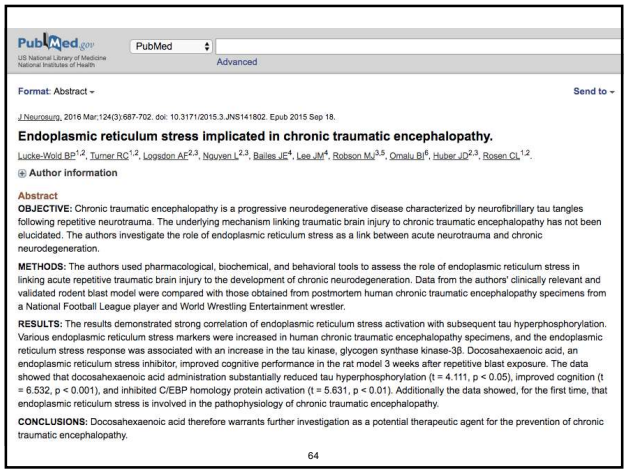

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Endoplasmic reticulum stress implicated in chronic traumatic encephalopathy.

Lucke-Wold BP[1,2], Turner RC[1,2], Logsdon AF[2,3], Nguyen L[2,3], Bailes JE[4], Lee JM[4], Robson MJ[3,5], Omalu BI[6], Huber JD[2,3], Rosen CL[1,2].

Author information

Abstract

OBJECTIVE: Chronic traumatic encephalopathy is a progressive neurodegenerative disease characterized by neurofibrillary tau tangles following repetitive neurotrauma. The underlying mechanism linking traumatic brain injury to chronic traumatic encephalopathy has not been elucidated. The authors investigate the role of endoplasmic reticulum stress as a link between acute neurotrauma and chronic neurodegeneration.

METHODS: The authors used pharmacological, biochemical, and behavioral tools to assess the role of endoplasmic reticulum stress in linking acute repetitive traumatic brain injury to the development of chronic neurodegeneration. Data from the authors' clinically relevant and validated rodent blast model were compared with those obtained from postmortem human chronic traumatic encephalopathy specimens from a National Football League player and World Wrestling Entertainment wrestler.

RESULTS: The results demonstrated strong correlation of endoplasmic reticulum stress activation with subsequent tau hyperphosphorylation. Various endoplasmic reticulum stress markers were increased in human chronic traumatic encephalopathy specimens, and the endoplasmic reticulum stress response was associated with an increase in the tau kinase, glycogen synthase kinase-3β. Docosahexaenoic acid, an endoplasmic reticulum stress inhibitor, improved cognitive performance in the rat model 3 weeks after repetitive blast exposure. The data showed that docosahexaenoic acid administration substantially reduced tau hyperphosphorylation ($t = 4.111$, $p < 0.05$), improved cognition ($t = 6.532$, $p < 0.001$), and inhibited C/EBP homology protein activation ($t = 5.631$, $p < 0.01$). Additionally the data showed, for the first time, that endoplasmic reticulum stress is involved in the pathophysiology of chronic traumatic encephalopathy.

CONCLUSIONS: Docosahexaenoic acid therefore warrants further investigation as a potential therapeutic agent for the prevention of chronic traumatic encephalopathy.

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"Knowing is not enough; we must apply.
Willing is not enough; we must do."

- Goethe

65

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Thank You