

GB

„Vitamin E supplementation protects erythrocyte membranes from oxidative stress in healthy Chinese middle-aged and elderly people.“

DE

Vitamin-E-Supplementierung schützt bei gesunden Chinesen mittleren Alters und Älteren Erythrozytemembrane vor oxidativem Stress.

GR

„Η πρόσληψη Βιταμίνης Ε προστατεύει τις μεμβράνες των ερυθροκυττάρων από το οξειδωτικό στρες σε υγιείς Κινέζους μέσης και προχωρημένης ηλικίας.“

FR

En Chine, une supplémentation en vitamine E protège les membranes érythrocytaires contre le stress oxydatif chez les personnes d'âge moyen et les personnes âgées.

CZ

Suplementace vitamínem E chrání erytrocytární membrány před oxidačním stresem u zdravých čínských jedinců ve středním věku a starších jedinců.

[Nutr Res.](#) 2012 May;32(5):328-34. doi: 10.1016/j.nutres.2012.03.012. Epub 2012 Apr 27.

Vitamin E supplementation protects erythrocyte membranes from oxidative stress in healthy Chinese middle-aged and elderly people.

[Sun Y](#), [Ma A](#), [Li Y](#), [Han X](#), [Wang Q](#), [Liang H](#).

Source The Institute of Human Nutrition, Medical College of Qingdao University, 38 Dengzhou Road, Qingdao, China.

Abstract Elderly people are subject to higher levels of oxidative stress than are young people. Vitamin E, as a powerful antioxidant residing mainly in biomembranes, may provide effective protection against oxidative membrane damage and resultant age-related deterioration, especially in the elderly. We hypothesized that appropriate levels of vitamin E supplementation would protect erythrocyte membranes from oxidative stress and thus improve membrane fluidity in healthy middle-aged and elderly people. To test this, we conducted a 4-month double-blind, randomized trial in which 180 healthy subjects (55-70 years old) were randomly divided into 4 groups: group C (control), and 3 treatment groups in which daily doses of 100 mg (VE1), 200 mg (VE2), and 300 mg (VE3) dl- α -tocopheryl acetate were administered. We measured plasma α -tocopherol concentration, malondialdehyde, and superoxide dismutase levels, erythrocyte hemolysis, and erythrocyte membrane fluidity at the beginning and end of the trial. After 4 months supplementation, plasma α -tocopherol concentrations in the 3 treatment groups had increased by 71%, 78%, and 95%, respectively (all $P < .01$), and significant decreases in plasma malondialdehyde concentrations were observed in these groups (all $P < .05$). Erythrocyte hemolysis was decreased by 20% to 38% after vitamin E supplementation (all $P < .05$), and in addition, groups VE2 and VE3 showed dramatic improvements in erythrocyte membrane fluidity ($P < .01$). Surprisingly, superoxide dismutase activity also decreased significantly in the treatment groups (all $P < .05$). In summary, vitamin E supplementation apparently alleviates oxidative stress in healthy middle-aged to elderly people, at least in part by improving erythrocyte membrane fluidity and reducing erythrocyte hemolysis.

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PMID: 22652371

Ⓒ

„Effects of vitamin E on plasma lipid status and oxidative stress in Chinese women with metabolic syndrome.“

Ⓓ

Die Wirkung von Vitamin E auf den Plasmalipidstatus und oxidativen Stress bei chinesischen Frauen mit metabolischem Syndrom.

Ⓔ

Αποτελέσματα της Βιταμίνης Ε στην κατάσταση λιπιδίων στο πλάσμα και το οξειδωτικό στρες σε γυναίκες με μεταβολικό σύνδρομο.

Ⓕ

Effets de la vitamine E sur le statut des lipides plasmatiques et sur le stress oxydatif chez les Chinoises souffrant d'un syndrome métabolique.

Ⓖ

Účinky vitamínu E na hladinu plazmatických lipidů a oxidační stres u čínských žen s metabolickým syndromem.

[Int J Vitam Nutr Res.](#) 2010 Jun;80(3):178-87. doi: 10.1024/0300-9831/a000015.

Effects of vitamin E on plasma lipid status and oxidative stress in Chinese women with metabolic syndrome.

[Wang Q](#), [Sun Y](#), [Ma A](#), [Li Y](#), [Han X](#), [Liang H](#).

Source The Institute of Human Nutrition, Medical College of Qingdao University, Qingdao, China. kevin1971@yahoo.cn

Abstract Following the change of dietary structure and living style, metabolic syndrome (MetS) has become increasingly common in China, especially in women, who have abnormal plasma lipid profiles with increased levels of oxidative stress. Vitamin E (VitE) is a powerful chain-breaking antioxidant, which may be a protective factor against oxidative stress-related diseases. This study investigated the effects of three different dosages of tocopherol supplementation (100 IU /day, 200 IU /day, 300 IU /day) for 4 months in Chinese women with MetS. The plasma VitE concentrations increased significantly after the 4 months of supplementation ($p < 0.01$). The protective decreases in plasma total cholesterol were significant in 200 IU/day and 300 IU/day VitE groups ($p < 0.05$), but decreases in high-density lipoprotein cholesterol were also significant in all the supplementation groups ($p < 0.05$). Plasma triglycerides were unaltered ($p > 0.05$). The indicators of oxidative stress decreased substantially in all of the VitE supplementation groups: malondialdehyde (MDA) was reduced by nearly 50 percent (all groups, $p < 0.001$), erythrocyte hemolysis was decreased by nearly 40 percent (all groups, $p < 0.05$); among which the 300IU/day VitE group showed the most significant effect. However, the activity of superoxide dismutase (SOD) decreased after the trial ($p < 0.001$). VitE provided marked benefits in reducing oxidative stress levels and improving lipid status in women with MetS. Although no dose-effect relationship was observed, 300 IU VitE per day showed the optimal effect. Research is needed to identify potential protective mechanisms or utilization of vitamin E during MetS.

PMID: 21234859

GB

„Effect of vitamin E and C supplements on lipid peroxidation and GSH-dependent antioxidant enzyme status in the blood of women consuming oral contraceptives.“

DE

Die Wirkung von Vitamin-E- und -C-Supplementierung auf den Status von Lipidperoxidation und GSH-abhängigen antioxidativen Enzymen im Blut von Frauen, die orale Kontrazeptiva zu sich nehmen.

GR

„Αποτέλεσμα συμπληρωμάτων Βιταμίνης Ε και C στην υπεροξειδωση λιπιδίων και την κατάσταση GSH-εξαρτώμενου αντιοξειδωτικού ενζύμου στο αίμα γυναικών που λαμβάνουν αντισυλληπτικά χάπια.“

FR

Effet d'une supplémentation en vitamines E et C sur la peroxydation lipidique et sur le statut de l'enzyme anti-oxydante dépendante du GSH dans le sang des femmes consommant des contraceptifs oraux.

CZ

Účinek suplementace vitamínem E a C na lipidovou peroxidaci a hladinu antioxidantních enzymů závislých na GSH v krvi žen užívajících perorální kontraceptiva.

Effect of vitamin E and C supplements on lipid peroxidation and GSH-dependent antioxidant enzyme status in the blood of women consuming oral contraceptives.

[Zal F](#), [Mostafavi-Pour Z](#), [Amini F](#), [Heidari A](#).

Source Reproductive Biology Group, Graduate School of Biomedical Sciences, Shiraz University of Medical Sciences, Shiraz, Iran.

BACKGROUND: Oral contraceptives (OCs) may affect oxidative stress status. We aimed to assess whether supplementation with vitamins E and C reduced this OC effect.

STUDY DESIGN: One hundred twenty healthy female individuals were divided into three groups: A, control; B, untreated OCU (OC users); and C, treated OCU (OC users with vitamin E and C supplementation). In all cases, plasma glutathione peroxidase (GPx) and glutathione reductase (GR) activities and malondialdehyde (MDA) level were determined.

RESULTS: Significant increases were found in the plasma MDA level, and activities of GPx and GR in plasma were decreased in Group B compared to the control group. Supplementation with vitamin C and E significantly increased the activity of GPx and GR activity, and reduced plasma MDA levels in Group C ($p < .05$).

CONCLUSIONS: These data suggest that low-dose OCs, by enhancing the stress oxidative and lipid peroxidation, may represent a potential cardiovascular risk factor, and the use of vitamins E and C may be beneficial in ameliorating this side effect of OCs.

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PMID: 22494786

Ⓒ

„Impact of oral vitamin E supplementation on oxidative stress & lipid peroxidation in patients with polymorphous light eruption.“

Ⓓ

Die Auswirkung einer oralen Vitamin-E-Supplementierung auf oxidativen Stress und Lipidperoxidation bei Patienten mit polymorpher Lichteruption.

Ⓖ

Επίπτωση της λήψης συμπληρώματος βιταμίνης Ε από του στόματος στο οξειδωτικό στρες & και την υπεροξείδωση λιπιδίων σε ασθενείς με πολύμορφο εξάνθημα εκ φωτός.

Ⓕ

Impact de la supplémentation orale en vitamine E sur le stress oxydatif et la peroxydation lipidique chez les patients atteint d'éruptions polymorphes à la lumière.

Ⓒ

Vliv perorální suplementace vitamínem E na oxidační stres a lipidovou peroxidaci u pacientů s polymorfní světelnou erupcí.

[Indian J Med Res.](#) 2006 Jun;123(6):781-7.

Impact of oral vitamin E supplementation on oxidative stress & lipid peroxidation in patients with polymorphous light eruption.

[Ahmed RS](#), [Suke SG](#), [Seth V](#), [Jain A](#), [Bhattacharya SN](#), [Banerjee BD](#).

Source Environmental Biochemistry Laboratory, Department of Biochemistry , University of Delhi, Delhi, India.

BACKGROUND & OBJECTIVES: Polymorphous light eruption (PMLE) is a photo-induced disease which clinically manifests in the form of pruritic eruptions on sun/light exposed parts. Little is known about lipid peroxidation and free radical scavengers in patients during PMLE. The present study was therefore undertaken to evaluate oxidative stress and levels of antioxidant enzymes in patients of PMLE.

METHODS: The PMLE was diagnosed clinically by a consultant dermatologist and validated independently by another and through histopathologic findings. Blood samples were collected on day 1 and patients were given oral vitamin E supplementation (400 mg OD) along with topical sunscreen and advice for photo-protection. Samples were collected again after one week. The blood samples were evaluated for lipid peroxidation, oxygen free radical (OFR) scavenging enzymes, glutathione (GSH) and related enzymes such as glutathione reductase (GR), glutathione peroxidase (GPx), gamma glutamyl transpeptidase (GGT) and glutathione- S-transferase (GST) in erythrocytes and compared with healthy controls.

RESULTS: The serum malondialdehyde (MDA) level was higher and GSH level was lower in PMLE cases as compared to controls. There was a significant decrease in superoxide dismutase (SOD) activity while activities of catalase (CAT) and glutathione related enzymes were increased in PMLE cases. Administration of oral vitamin E for one week, along with photoprotection resulted in a significant decrease in MDA levels and activities of all others enzymes except SOD. The GSH was replenished and returned to normal.

INTERPRETATION & CONCLUSION: Oxidative stress and differential modulation of antioxidant enzymes in PMLE might play a pathogenic role in humans, which supports the incorporation of antioxidant drugs in the treatment protocol of the disease.

PMID: 16885600

GB

„Mitochondria as possible pharmaceutical targets for the effects of vitamin E and its homologues in oxidative stress-related diseases.“

DE

Mitochondrien als mögliche pharmazeutische Ziele für die Wirkung von Vitamin E und deren Homologe bei durch oxidativen Stress bedingten Erkrankungen.

GR

„Τα μιτοχόνδρια ως πιθανοί φαρμακευτικοί στόχοι για τα αποτελέσματα της Βιταμίνης Ε και των ομολόγων της σε ασθένειες που σχετίζονται με το οξειδωτικό στρες.“

FR

Les mitochondries, cibles pharmaceutiques possibles pour les effets de la vitamine E et de ses homologues dans les maladies liées au stress oxydatif.

CZ

Mitochondrie jako možné farmaceutické cíle pro účinky vitamínu E a jeho shodných skupin v chorobách souvisejících s oxidačním stresem.

[Curr Pharm Des.](#) 2011;17(21):2190-5.

Mitochondria as possible pharmaceutical targets for the effects of vitamin E and its homologues in oxidative stress-related diseases.

[Majima HJ](#), [Indo HP](#), [Suenaga S](#), [Matsui H](#), [Yen HC](#), [Ozawa T](#).

Source Department of Oncology, Kagoshima University Graduate School of Medical and Dental Sciences, Kagoshima 890-8544, Japan. hmajima@dent.kagoshima-u.ac.jp

It is well known that vitamin E functions as an antioxidant, and it is expected to exert an antioxidant effect when taken as a supplement. However, a number of cohort studies have shown that vitamin E does not alleviate oxidative stress and could even worsen it. Recently, Wang et al. investigated whether vitamin E intake was associated with amyotrophic lateral sclerosis (ALS) based on data from 5 cohort studies with 1,055,546 participants, of which 805 of them had developed ALS. They concluded in this large pooled prospective study, in which long-term vitamin E supplementation was associated with lower ALS rates, and therefore, a possible protective effect of vitamin E deserves further consideration. Performing further large cohort studies may reveal similar findings for other oxidative stress-related diseases. It is still controversial if antioxidants such as vitamin E provide a clinical therapeutic effect against oxidative stress-related diseases. If effective, the dose at which they should be administered and the duration of supplement exposure should be of interest. Vitamin E reduces production of reactive oxygen species by mitochondria and elicits further reactions in cells. It should be noted that mitochondria are important targets for vitamin E and its homologues. Therefore, a proper usage of vitamin E in subjects under high oxidative stress, due to its individually targeting property, will arise its importance in healthy life.

PMID: 21774784

Ⓒ

„Effects of vitamin E and carotenoid status on oxidative stress in health and disease. Evidence obtained from human intervention studies.“

Ⓓ

Die Wirkung von Vitamin-E- und Carotinoidstatus auf oxidativen Stress in Gesundheit und Krankheit. Evidenz aus Interventionsstudien am Menschen.

Ⓔ

„Αποτελέσματα της κατάστασης Βιταμίνη Ε και καροτονοειδών στο οξειδωτικό στρες στην υγεία και την ασθένεια. Στοιχεία που αποκομίστηκαν από μελέτες παρέμβασης σε ανθρώπους.“

Ⓕ

Effets de la vitamine E et statut du carotinoïde sur le stress oxydatif chez le sujet sain et malade. Indices obtenus à partir d'études d'intervention humaine.

Ⓖ

Účinky vitamínu E a hladiny karotenoidů na oxidační stres ve zdraví a v nemoci. Důkaz získaný z intervenčních studií provedených u lidí.

[Mol Aspects Med.](#) 2003 Dec;24(6):391-402.

Effects of vitamin E and carotenoid status on oxidative stress in health and disease. Evidence obtained from human intervention studies.

[Winklhofer-Roob BM](#), [Rock E](#), [Ribalta J](#), [Shmerling DH](#), [Roob JM](#).

Source Institute of Molecular Biology, Biochemistry and Microbiology (IMBM), University of Graz, Austria. brigitte.winklhoferroob@uni-graz.at

Abstract Vitamin E and carotenoids are known to act as antioxidants both in vitro and in vivo. In this review we present a series of studies in healthy subjects and in patients who exhibit either acute or chronic oxidative stress. In the EU-Commission funded VITAGE project we investigated the status and effects of vitamin E and carotenoids on oxidative stress in 300 healthy volunteers. Depletion studies limiting dietary vitamin E or carotenoid intake to approximately 25% of the dietary reference intakes and subsequent repletion by supplementation with either large doses of vitamin E or intermediate doses of carotenoids showed significant changes in ex vivo LDL oxidizability, total plasma peroxide concentrations and urinary 8-oxo-7,8-dihydro-2(')-deoxyguanosine excretion. Patients on chronic hemodialysis present with oxidative stress in the presence of normal vitamin E but impaired vitamin C status and, due to anemia, need to be treated with parenteral iron. We studied the effects of a single oral dose of vitamin E taken 6 h prior to intravenous infusion of 100 mg iron, which exceeded the iron-binding capacity of transferrin. Vitamin E significantly reduced and in combination with a single dose of vitamin C completely abrogated acute oxidative stress induced by the iron load. Patients with cystic fibrosis are exposed to chronic oxidative stress due to an overproduction of reactive oxygen species as a result of neutrophil-dominated lung inflammation and impaired antioxidant status. Biochemical vitamin E and carotenoid deficiencies could be fully corrected even in the presence of fat malabsorption using intermediate doses of either RRR alpha-tocopherol or all-rac alpha-tocopheryl acetate and water-miscible all-trans beta-carotene. Long-term supplementation reduced ex vivo LDL oxidizability, in vivo lipid peroxidation and lung inflammation.

PMID: 14585310

Ⓒ

Cellular protection and therapeutic potential of tocotrienols.

Ⓓ

Zellschutz und therapeutisches Potenzial von Tocotrienolen.

Ⓔ

Κυτταρική προστασία και θεραπευτικές δυνατότητες των τοκοτριενολών.

Ⓕ

Protection cellulaire et potentiel thérapeutique des tocotriénols.

Ⓖ

Buněčná ochrana a léčebný potenciál tokotrienolů.

[Curr Pharm Des.](#) 2011;17(21):2215-20.

Cellular protection and therapeutic potential of tocotrienols.

[Catalgol B](#), [Batirel S](#), [Ozer NK](#).

Source Department of Biochemistry, Faculty of Medicine, Marmara University, 34668 Haydarpasa, Istanbul, Turkey.

Abstract Tocotrienols, components belonging to vitamin E members, are used as potent therapeutics in the treatment of several diseases. Recent studies suggested tocotrienol to have better activity in many situations compared to tocopherols. Tocotrienols have been shown to lower the atherogenic apolipoprotein B and lipoprotein plasma levels. Additionally, tocotrienols with their anti-tumor effect together with anti-angiogenic and anti-thrombotic effects may serve as effective agents in cancer therapy. Besides these effects, some properties such as water insolubility and low stability limit the usage of tocotrienols in the clinic. However recent studies tried to increase the bioavailability with esterification and combination use. These efforts for the clinical usage of tocotrienols which may help them to take a wide place in the clinic and additional studies are needed to identify their therapeutical mechanisms.

PMID: 21774780

GB

„Dietary supplementation with α -tocopherol reduces neuroinflammation and neuronal degeneration in the rat brain after kainic acid-induced status epilepticus.“

DE

Dietätische Supplementierung mit α -Tocopherol reduziert Neuroinflammation und neuronale Degeneration im Rattenhirn nach Kainsäure-induziertem Status epilepticus.

GR

Η λήψη διατροφικών συμπληρωμάτων με α -τοκοφερόλη μειώνει τη νευροφλεγμονή και τον εκφυλισμό των νευρώνων στον εγκέφαλο αρουραίων μετά από επιληπτική κατάσταση που προκλήθηκε από καϊνικό οξύ.

FR

La supplémentation nutritionnelle avec de l' α -tocophérol réduit la neuro-inflammation et la neurodégénérescence cérébrale chez le rat après un état épileptique induit par l'acide kaïnique.

CZ

Potravinová suplementace α -tokoferolem snižuje neuroinflamaci a neuronovou degeneraci v mozku potkanů po epileptických stavech vyvolaných kainovou kyselinou.

Dietary supplementation with α -tocopherol reduces neuroinflammation and neuronal degeneration in the rat brain after kainic acid-induced status epilepticus.

[Betti M](#), [Minelli A](#), [Ambrogini P](#), [Ciuffoli S](#), [Viola V](#), [Galli F](#), [Canonica B](#), [Lattanzi D](#), [Colombo E](#), [Sestili P](#), [Cuppini R](#).

Source Department of Earth, Life and Environmental Sciences, University of Urbino Carlo Bo, I-61029 Urbino, Italy. CA126659

Abstract Vitamin E (as α -tocopherol, α -T) is proposed to alleviate glia-mediated inflammation in neurological diseases, but such a role in epilepsy is still elusive. This study investigated the effect of α -T supplementation on glial activation, neuronal cell death and oxidative stress of rat brain exposed to kainate-induced seizures. Animals were fed for 2 weeks with a α -T-enriched diet (estimated intake of 750 mg/kg/day) before undergoing status epilepticus. Compliance to supplementation was demonstrated by the remarkable increase in brain α -T. Four days after seizure, brain α -T returned to baseline and lipid peroxidation markers decreased as compared to non-supplemented rats. Status epilepticus induced a lower up-regulation of astrocytic and microglial antigens (GFAP and MHC II, respectively) and production of pro-inflammatory cytokines (IL-1 β and TNF- α) in supplemented than in non-supplemented animals. This anti-inflammatory effect was associated with a lower neuronal cell death. In conclusion, α -T dietary supplementation prevents oxidative stress, neuroglial over-activation and cell death occurring after kainate-induced seizures. This evidence paves the way to an anti-inflammatory and neuroprotective role of α -T interventions in epilepsy.

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PMID: 21749318

Ⓒ

Vitamin E in aging, dementia, and Alzheimer's disease.

Ⓓ

Vitamin E beim Alterungsprozess, bei Demenz und Alzheimer-Krankheit.

Ⓖ

Η Βιταμίνη Ε στη γήρανση, την άνοια και τη νόσο Αλτσχάιμερ.

Ⓕ

La vitamine E dans le vieillissement, la démence sénile et la maladie d'Alzheimer.

Ⓒ

Vitamín E při stárnutí, demenci a Alzheimerově chorobě.

[Biofactors](#). 2012 Mar-Apr;38(2):90-7. doi: 10.1002/biof.195. Epub 2012 Mar 16.

Vitamin E in aging, dementia, and Alzheimer's disease.

[Joshi YB](#), [Praticò D](#).

Source Department of Pharmacology, Temple University, School of Medicine, Philadelphia, PA, USA.

Abstract Since its discovery, vitamin E has been extensively researched by a large number of investigators in an attempt to fully understand its role in a variety of pathophysiological contexts. The vast majority of published work has focused on vitamin E's antioxidant properties, which is why it is well known as a lipophilic antioxidant that protects membranes from being oxidatively damaged by free radicals. However, several lines of investigation have recently revealed that vitamin E has biological roles unrelated to its antioxidant properties. Among these roles, vitamin E has been described as: a regulator of signal transduction, gene expression, and redox sensor. In parallel with the discovery of novel cellular functions of vitamin E, the introduction of the free radical theory of brain aging has propelled a renewed interest in this vitamin. Most of the resulting work has been based on the postulate that, by preventing and/or minimizing the oxidative stress-dependent brain damage, vitamin E could be used as a therapeutic approach. In this article, we will consider the existing literature regarding the biological properties of vitamin E and the potential therapeutic and/or preventative roles that this natural dietary factor plays in brain aging, cognition, and Alzheimer's dementia.

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PMID: 22422715

Ⓒ

„Aging brain: prevention of oxidative stress by vitamin E and exercise.“

Ⓓ

Das alternde Gehirn: Prävention von oxidativem Stress durch Vitamin E und sportliche Betätigung.

Ⓖ

Γηράσκων εγκέφαλος: πρόληψη του οξειδωτικού στρες με Βιταμίνη Ε και άσκηση.

Ⓕ

Le cerveau vieillissant : prévention du stress oxydatif par la vitamine E et l'exercice.

Ⓒ

Stárnoucí mozek: prevence oxidačního stresu vitamínem E a cvičením.

[ScientificWorldJournal](#). 2009 May 22;9:366-72. doi: 10.1100/tsw.2009.46.

Aging brain: prevention of oxidative stress by vitamin E and exercise.

[Asha Devi S.](#)

Source Laboratory of Gerontology, Department of Zoology, Bangalore University, Bangalore 560 056, India. asuba@blr.vsnl.net.in

Abstract

With aging, the brain undergoes neuronal loss in many areas. Although the loss of cells in the cerebral cortex, in particular the frontal cortex, has been recognized with aging, the influence of synaptic losses has a larger impact on cognitive decline. Much of the recent research on animals, as well as humans, has been aimed at slowing the cognitive decline through enrichment, and it has been found that the key factors are antioxidants and exercise. Several reports support the concept that regular supplementation of vitamin E and physical activity from as early as middle age can slow the cognitive decline observed during the later years. A few studies have also suggested that exercise is analogous to acetylcholine esterase inhibitors that are also used extensively to treat cognitive impairment and dementia in Alzheimer's disease. In addition, reports also support that vitamin E and exercise may act synergistically to overcome free radical injury and oxidative stress in the aging brain.

PMID:19468659

Free full text

Ⓒ

„Vitamin E in neurodegenerative disorders: Alzheimer's disease.“

Ⓓ

Vitamin E bei neurodegenerativen Störungen: Alzheimer-Krankheit.

Ⓖ

„Η Βιταμίνη Ε στις νευροεκφυλιστικές διαταραχές: νόσος Αλτσχάιμερ.“

Ⓕ

La vitamine E dans les troubles neurodégénératifs : la maladie d'Alzheimer.

Ⓒ

Vitamín E v neurodegenerativních poruchách: Alzheimerova choroba.

[Ann N Y Acad Sci.](#) 2004 Dec;1031:249-62.

Vitamin E in neurodegenerative disorders: Alzheimer's disease.

[Kontush K](#), [Schekatolina S](#).

Source INSERM Unité 551, Pavillon Benjamin Delessert, Hôpital de la Pitié, 83 boulevard de l'Hôpital, 75651 Paris Cedex 13, France. kontush@chups.jussieu.fr

Abstract Oxidative stress is important in the pathogenesis of Alzheimer's disease (AD). The brain contains high levels of oxidizable lipids that must be protected by antioxidants. Low concentrations of vitamin E, quantitatively the major lipophilic antioxidant in the brain, are frequently observed in cerebrospinal fluid (CSF) of AD patients, suggesting that supplementation with vitamin E might delay the development of AD. In a placebo-controlled trial, vitamin E (2000 IU/day, 2 years) slowed (-53%) functional deterioration in patients with moderate AD (Sano et al., *N. Engl. J. Med.* 336: 1216-1222, 1997). Recently, use of vitamin E and vitamin C supplements in combination was found to be associated with reduced prevalence (-78%) and incidence (-64%) of AD in elderly population (Zandi et al., *Arch. Neurol.* 61: 82-88, 2004). These results are consistent with the ability of the supplementation with vitamin E (400 IU/day, 1 month) to increase its levels in CSF (123%) and plasma (145%) of AD patients and, in combination with vitamin C (1000 g/day), to decrease the susceptibility of CSF lipoproteins (up to -32%) to in vitro oxidation (Kontush et al., *Free Radic. Biol. Med.* 31: 345-354, 2001). In addition, vitamin E reduced lipid peroxidation and amyloid deposition in a transgenic mice model of AD (Sung et al., *FASEB J.* 18: 323-325, 2004). Computer modeling of the influence of vitamin E on lipoprotein oxidation reveals that the vitamin develops antioxidative activity in CSF lipoproteins in the presence of physiologically relevant, low amounts of oxidants. By contrast, under similar conditions, vitamin E behaves as a pro-oxidant in plasma lipoproteins, consistent with the model of tocopherol-mediated peroxidation (Stocker, *Curr. Opin. Lipidol.* 5: 422-433, 1994). This distinction is related to major differences in the levels of vitamin E (50 nM vs. 30 microM) and oxidizable lipids (4 microM vs. 2.5 mM) between CSF and plasma, which result in major differences in oxidative conditions (per unit of vitamin E) between CSF and plasma in the presence of similar amounts of oxidants.

Altogether, these data suggest that vitamin E may be effective against in vivo oxidation of CSF lipoproteins and brain lipids, and offer new perspectives in the treatment of AD and other neurodegenerative disorders.

PMID: 15753151

GB

„A review of specific dietary antioxidants and the effects on biochemical mechanisms related to neurodegenerative processes.“

DE

Ein Review spezieller diätätischer Antioxidanzien und die Wirkung von biochemischen Mechanismen in Verbindung mit neurodegenerativen Prozessen.

GR

Μια αναθεώρηση συγκεκριμένων διατροφικών αντιοξειδωτικών και της δράσης τους στους βιοχημικούς μηχανισμούς που σχετίζονται με νευροεκφυλιστικές διαδικασίες.

FR

Passage en revue des anti-oxydants nutritionnels spécifiques et les effets sur des mécanismes biochimiques liés aux processus neurogénératifs.

CZ

Studie konkrétních potravinových antioxidantů a účinků na biochemické mechanismy související s neurodegenerativními procesy.

[Neurobiol Aging](#). 2002 Sep-Oct;23(5):719-35.

A review of specific dietary antioxidants and the effects on biochemical mechanisms related to neurodegenerative processes.

[Esposito E](#), [Rotilio D](#), [Di Matteo V](#), [Di Giulio C](#), [Cacchio M](#), [Algeri S](#).

Source Istituto di Ricerche Farmacologiche Mario Negri, Consorzio Mario Negri Sud, 66030 Santa Maria Imbaro, Chieti, Italy. esposito@cmns.mnegri.it

Abstract Aging is a major risk factor for neurodegenerative diseases including Alzheimer's disease (AD), Parkinson's disease (PD), and amyotrophic lateral sclerosis (ALS). An unbalanced overproduction of reactive oxygen species (ROS) may give rise to oxidative stress which can induce neuronal damage, ultimately leading to neuronal death by apoptosis or necrosis. A large body of evidence indicates that oxidative stress is involved in the pathogenesis of AD, PD, and ALS. An increasing number of studies show that nutritional antioxidants (especially Vitamin E and polyphenols) can block neuronal death in vitro, and may have therapeutic properties in animal models of neurodegenerative diseases including AD, PD, and ALS. Moreover, clinical data suggest that nutritional antioxidants might exert some protective effect against AD, PD, and ALS. In this paper, the biochemical mechanisms by which nutritional antioxidants can reduce or block neuronal death occurring in neurodegenerative disorders are reviewed. Particular emphasis will be given to the role played by the nuclear transcription factor-kappaB (NF-kappaB) in apoptosis, and in the pathogenesis of neurodegenerative disorders, such as AD, PD, and ALS. The effects of ROS and antioxidants on NF-kappaB function and their relevance in the pathophysiology of neurodegenerative diseases will also be examined.

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PMID: 12392777

Ⓒ

„Vitamin E protects neurons against oxidative cell death in vitro more effectively than 17-beta estradiol and induces the activity of the transcription factor NF-kappaB.“

Ⓓ

„Vitamin E schützt Neurone vor oxidativem Zelltod in vitro wirksamer als 17-beta-Östradiol und induziert die Aktivität des Transkriptionsfaktors NF-kappaB.“

Ⓖ

„Η Βιταμίνη Ε προστατεύει τους νευρώνες ενάντια στον οξειδωτικό θάνατο των κυττάρων in vitro πιο αποτελεσματικά έναντι της 17-βήτα οιστραδιόλης και προκαλεί τη ραστηριότητα του μεταγραφικού παράγοντα NF-kappaB.“

Ⓕ

La vitamine E protège les neurones contre la mort oxydative des cellules in vitro plus efficacement que le 17-bêta estradiol, et induit l'activité du facteur de transcription NF-kappaB.

Ⓒ

Vitamín E chrání neurony proti oxidačnímu odumření buněk in vitro efektivněji než 17-beta estradiol a vyvolává aktivitu transkripčního faktoru NF-kappaB.

[J Neural Transm.](#) 2000;107(4):393-407.

Vitamin E protects neurons against oxidative cell death in vitro more effectively than 17-beta estradiol and induces the activity of the transcription factor NF-kappaB.

[Behl C.](#)

Source Independent Research Group Neurodegeneration, Max Planck Institute of Psychiatry, Munich, Federal Republic of Germany. chris@mpipsykl.mpg.de

Abstract Antioxidants can function as powerful protectants for neurons in vitro. Here, the neuroprotective activity of lipophilic free radical scavengers synthetic (+/-) alpha-tocopherol (synthetic vitamin E) and natural (+) alpha-tocopherol (natural vitamin E) against oxidative stress was investigated and compared to the neuroprotective effect of the female sex hormone estradiol. Employing mouse clonal hippocampal HT22 cells and rat cerebellar granule neurons, we found that both types of alpha-tocopherol exerted a higher neuroprotective antioxidant activity than 17-beta estradiol. At concentrations as low as 100 nM, synthetic (+/-) alpha-tocopherol and natural (+) alpha-tocopherol protected neurons effectively against the oxidative cell death caused by the Alzheimer's disease-associated amyloid beta protein, hydrogen peroxide, and the excitatory amino acid glutamate. Moreover, vitamin E induced the activity of the redox-sensitive transcription factor NF-kappaB, which is involved in the control of nerve cell survival and, therefore, may play also a role in vitamin E-induced neuroprotection. These results may have implications regarding the prevention and treatment of oxidative stress-related degenerative disorders such as Alzheimer's disease.

PMID: 11215751

Ⓒ

„Role of antioxidants in generalised anxiety disorder and depression.“

Ⓓ

Die Rolle von Antioxidanzien bei generalisierter Angststörung und Depression.

Ⓖ

Ο ρόλος των αντιοξειδωτικών στη γενικευμένη αγχώδη διαταραχή και την κατάθλιψη.

Ⓕ

Rôle des anti-oxydants dans le trouble de l'anxiété généralisée et la dépression.

Ⓒ

Úloha antioxidantů při generalizované úzkostné poruše a depresi.

[Indian J Psychiatry](#). 2012 Jul;54(3):244-7. doi: 10.4103/0019-5545.102424.

Role of antioxidants in generalised anxiety disorder and depression.

[Gautam M](#), [Agrawal M](#), [Gautam M](#), [Sharma P](#), [Gautam AS](#), [Gautam S](#).

Source Santokba Durlabhji Memorial Hospital, Jaipur, Rajasthan, India.

BACKGROUND: Anxiety and depression form commonest stress-induced psychiatric disorders. To combat the biochemical changes which occur as a result of stress, there is antioxidant defence in the biological system. Secondary defence is by the nonenzymatic antioxidants like vitamins E (alphatocopherol), C (ascorbic acid), and β -carotene. Therefore, the authors interest was aroused to examine the status of these antioxidants in the biological system of patients suffering from stress-induced psychiatric disorders.

AIMS: This study was carried out to find out whether patients with generalized anxiety disorder (GAD) and depression have any difference in blood serum levels of vitamins A (β -carotene), C, and E in comparison to the normal healthy control group and whether supplementation of adequate doses of vitamins A (β -carotene), C, and E leads to improvement in anxiety and depression and reduction in scores of the patients.

MATERIALS AND METHODS: Eighty subjects in the age group of 20-60 years, who attended a psychiatric clinic of a private hospital and who met inclusion and exclusion criteria of the study and consented for psychological evaluation and blood screening to find out the serum levels of vitamins A, C, and E, were included in the study. Approval was sought from the institutional ethics committee for collecting the blood sample of these subjects before and after vitamins A, C, and E supplements given for a period of 6 weeks. **STATISTICS ANALYSIS:** It was observed that patients with GAD and depression had significantly lower levels of vitamins A, C, and E in comparison to healthy controls. After dietary supplementation of these vitamins for a period of 6 weeks, a significant reduction in anxiety and depression scores of patients was observed ($P < 0.001$). A significant increase in the blood levels of antioxidants was observed in patients ($P < 0.05$) except that of vitamin E in the group of depressed patients.

RESULTS AND CONCLUSION: The findings suggest that antioxidant supplement therapy as an adjuvant therapy is useful in patients with stress-induced psychiatric disorders and the results have been discussed.

PMID: 23226848

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