http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=16248976&query_hl=2&itool=pubmed_docsum

J Biochem Mol Biol. 2002 Jan 31;35(1):116-26.

Nitric oxide as a pro-apoptotic as well as anti-apoptotic modulator.

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Nitric oxide (NO), synthesized from L-arginine by NO synthases, is a small, lipophilic, diffusible, highly reactive molecule with dichotomous regulatory roles in many biological events under physiological and pathological conditions. NO can promote apoptosis (pro-apoptosis) in some cells, whereas it inhibits apoptosis (anti-apoptosis) in other cells. This complexity is a consequence of the rate of NO production and the interaction with biological molecules such as metal ion, thiol, protein tyrosine, and reactive oxygen species. Long-lasting overproduction of NO acts as a pro-apoptotic modulator, activating caspase family proteases through the release of mitochondrial cytochrome c into cytosol, up-regulation of the p53 expression, and alterations in the expression of apoptosis-associated proteins, including the Bcl-2 family. However, low or physiological concentrations of NO prevent cells from apoptosis that is induced by the trophic factor withdrawal, Fas, TNFalpha/ActD, and LPS. The anti-apoptotic mechanism is understood on the basis of gene transcription of protective proteins. These include: heat shock protein, hemeoxygenase, or cyclooxygenase-2 and direct inhibition of the apoptotic executive effectors caspase family protease by S-nitrosylation of the cysteine thiol group in their catalytic site in a cell specific way. Our current understanding of the mechanisms by which NO exerts both pro- and anti-apototic action is discussed in this review article.

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http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=8899653&dopt=Abstract

J Neurol Sci. 1996 Aug;139 Suppl:16-26.

Role of SOD-1 and nitric oxide/cyclic GMP cascade on neurofilament aggregation in ALS/MND.

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A number of free radicals such as superoxide and nitric oxide may cause damage to motor neurons but the exact mechanism remains to be elucidated. A potent free radical, peroxynitrite, is readily formed from superoxide and nitric oxide, which captures superoxide three times faster than SOD-1. Peroxynitrite may nitrate tyrosine residues of light neurofilaments (NF-I), thereby altering NF assembly and causing NF accumulation in motor neurons. To test this hypothesis we have probed the massive NF aggregates which are histopathological hallmarks of ALS/MND with immunohistochemistry. We investigated localization of reaction products related to SOD-1, nitric oxide synthase (NOS) and cyclic GMP activities. Our studies show colocalization of NF aggregates with SOD-1/b-NOS/calmodulin /citrulline/cGMP and nitrotyrosine in upper motor neuron conglomerates (Cgl) and lower motor neutron axonal spheroids (Axs). This strongly supports the notion that peroxynitrite deranges NF phosphorylation and assembly, by nitrating tyrosine residues in NF-L. Impaired phosphorylation of NF subunits, either at NF-I or at NF-H, may affect the slow axonal transport culminating in proximo-distal accumulation of NF and slowly progressive motoneuron death.

PMID: 8899653 [PubMed - indexed for MEDLINE]

Nitric Oxide. 2002 Aug;7(1):18-23.

Nitric oxide mediates the effects of pulsed electromagnetic field stimulation on the osteoblast proliferation and differentiation.

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The purpose of this research was to investigate whether the effects of pulsed electromagnetic field (PEMF) stimulation on the osteoblast proliferation and differentiation are mediated by the increase in the nitric oxide (NO, nitrogen monoxide) synthesis. The osteoblasts (MC3T3-E1 cell line) were cultured in the absence (-NMMA group) or in the presence (+NMMA group) of the NO synthase inhibitor L-NMMA. First, osteoblasts were subjected to PEMF stimulation (15 Hz and 0.6 mT) up to 15 days. The DNA content and the NO concentration in the conditioned medium were determined on the 3rd, 7th, and 15th days of culture. Following, osteoblasts were stimulated in the proliferation (P-NMMA and P+NMMA groups) or in the differentiation (D-NMMA and D+NMMA groups) stages of maturation, and the alkaline phosphatase (AlPase) activity was determined on the 15th day of culture for all groups. PEMF stimulation increased significantly the nitrite concentration in the -NMMA group on the 3rd, 7th, and 15th days of culture. However, this effect was partially blocked in the +NMMA group. The DNA content in the -NMMA group, but not in the +NMMA group, increased significantly on the 3rd and 7th days of culture. The AlPase activity in the P-NMMA and D-NMMA groups, but not in the P+NMMA and D+NMMA groups, also increased significantly. In conclusion, the PEMF stimulatory effects on the osteoblasts proliferation and differentiation were mediated by the increase in the NO synthesis. Copyright 2002 Elsevier Science (USA)

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Neuroscience. 1999;93(2):597-603.

Nitric oxide transforms serotonin into an inactive form and this affects neuromodulation.

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Nitric oxide is a highly reactive molecule, diffusible and therefore ubiquitous in the central nervous system. Consequently, nitric oxide or nitric oxide-derived nitrogen oxides must enter into contact with neuromodulators and they can modify these molecules, especially monoamines, and thus change their regulatory action on synaptic transmission. We tested this possibility on a well-known, identified cholinergic synapse of Aplysia buccal ganglion, in which we have found that evoked acetylcholine release was decreased by extracellularly applied serotonin. We show that this modulatory effect of serotonin was largely reduced not only in the presence of 3-morpholinosydnonimine, a nitric oxide donor, but also when endogenous nitric oxide synthase was activated. We have shown that this decrease in the serotonin effect is due to the formation of chemical derivatives of serotonin, mainly a symmetric serotonin dimer, 4-nitroso-serotonin and 4-nitro-serotonin, which are ineffective in reproducing the modulatory effect of serotonin. Serotonin is involved in the regulation of several central functions, such as sleep-wake activity or mood. The consequences of chemical modifications of serotonin by nitric oxide must be taken into account in physiological as well as pathological situations. In addition, our results highlight the importance of the physiological implications of interactions between free radicals and neuromediators in the nervous system.

PMID: 10465443 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12415560&dopt=Abstract

Cell Biochem Funct. 2002 Dec;20(4):279-83.

Effects of electromagnetic radiation from a cellular telephone on the oxidant and antioxidant levels in rabbits.

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The number of reports on the effects induced by electromagnetic radiation (EMR) in various cellular systems is still increasing. Until now no satisfactory mechanism has been proposed to explain the biological effects of this radiation. Oxygen free radicals may play a role in mechanisms of adverse effects of EMR. This study was undertaken to investigate the influence of electromagnetic radiation of a digital GSM mobile telephone (900 MHz) on oxidant and antioxidant levels in rabbits. Adenosine deaminase, xanthine oxidase, catalase, myeloperoxidase, superoxide dismutase (SOD) and glutathione peroxidase activities as well as nitric oxide (NO) and malondialdehyde levels were measured in sera and brains of EMR-exposed and sham-exposed rabbits. Serum SOD activity increased, and serum NO levels decreased in EMR-exposed animals compared to the sham group. Other parameters were not changed in either group. This finding may indicate the possible role of increased oxidative stress in the pathophysiology of adverse effect of EMR. Decreased NO levels may also suggest a probable role of NO in the adverse effect.

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Regulation of apoptosis by nitrosative stress.

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Nitrosative stress can prevent or induce apoptosis. It occurs via S-nitrosylation by the interaction of nitric oxide (NO) with the biological thiols of proteins. Cellular redox potential and non-heme iron content determine S-nitrosylation. Apoptotic cell death is inhibited by S-nitrosylation of the redox-sensitive thiol in the catalytic site of caspase family proteases, which play an essential role in the apoptotic signal cascade. Nitrosative stress can also promote apoptosis by the activation of mitochondrial apoptotic pathways, such as the release of cytochrome c, an apoptosis-inducing factor, and endonuclease G from mitochondria, as well as the suppression of NF-kB activity. In this article we reviewed the mechanisms whereby S-nitrosylation and nitrosative stress regulate the apoptotic signal cascade.

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Exp Mol Med. 2002 Mar 31;34(1):53-9.

Enhanced expression of neuronal nitric oxide synthase and phospholipase C-gamma1 in regenerating murine neuronal cells by pulsed electromagnetic field.

Kim SS, **Shin HJ**, **Eom DW**, **Huh JR**, **Woo Y**, **Kim H**, **Ryu SH**, **Suh PG**, **Kim MJ**, **Kim JY**, **Koo TW**, **Cho YH**, **Chung SM**.

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Pulsed electromagnetic field (PEMF) has been shown to improve the rate of peripheral nerve regeneration. In the present study we investigated the expression of neuronal nitric oxide synthase (nNOS) and phospholipase C-gamma1 (PLC-gamma1) in regenerating rat laryngeal nerves during the exposure to PEMF after surgical transection and reanastomosis. Axons were found to regenerate into the distal stump nearly twice faster in PEMF-exposed animals than in the control. Consistently, motor function was better recovered in PEMF-treated rats. The expression of nNOS and PLC-gamma1 was highly enhanced in the regenerated nerves.

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http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=pubmed&cmd=Retrieve&dopt=AbstractPlus&list_uids=7688808&query_hl=19&itool=pubmed_docsum

J Physiol. 1993 Feb;461:513-24.

Increase in nitric oxide and cyclic GMP of rat cerebellum by radio frequency burst-type electromagnetic field radiation.

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1. Using rat cerebellum supernatant, the effects of radio frequency (RF) burst-type electromagnetic (EM) field radiation on the production of cyclic GMP were examined under various conditions. The radiation was generated by a generator coil, and set at a 10 MHz radiation frequency, a 50% burst time, a 10 kHz burst rate and a 5 V peak-to-peak generator voltage. 2. When the cerebellum supernatant was incubated with both exogenous L-arginine (nitric oxide (NO) donor) and NADPH, and irradiated by an RF burst-type EM field, the production of cyclic GMP was increased significantly from a level of 21-22 nmol min-1 (g tissue)-1 to 25-26 nmol min-1 (g tissue)-1. By contrast, such an effect was not found when the cerebellum supernatant was irradiated by an RF volley-type EM field. 3. When neither L-arginine nor NADPH were added to the cerebellum supernatant, the production of cyclic GMP was lowered to a level of 6 nmol min-1 (g tissue)-1 and the radiation effect was not found. When the cerebellum supernatant was chelated with EDTA, the production of cyclic GMP was lowered to a level of 7 nmol min-1 (g tissue)-1 and the radiation effect was not found. 4. Incubation with Methylene Blue, a guanylate cyclase inhibitor, lowered the production of cyclic GMP to a level of 10-12 nmol min-1 (g tissue)-1, and the radiation effect did not occur. On incubation with a NO synthase inhibitor, either NG-methyl-L-arginine or N omega-nitro-L-arginine methyl ester, the production of cyclic GMP was lowered to a level of 10-12 nmol min-1 (g tissue)-1 or 5-9 nmol min-1 (g tissue)-1 respectively, and the radiation effect was not observed. 5. Using electrochemical NO probes, the production of NO in the cerebellum supernatant was detected. The concentration of NO increased gradually after the onset of the EM field radiation. The radiation effect persisted, and reached a maximum after the cessation of the radiation. 6. In an in vivo study, the arterioles of the frog web were dilated by the radiation, and this radiation effect was almost completely abolished by the addition of a NO synthase inhibitor. This indicates that radiation activates NO synthase and ultimately induces vasodilatation.

PMID: 7688808 [PubMed - indexed for MEDLINE]

J Int Med Res. 2005 Sep-Oct;33(5):545-54.

Electromagnetic fields inhibit endothelin-1 production stimulated by thrombin in endothelial cells.

Morimoto S, **Takahashi T**, **Shimizu K**, **Kanda T**, **Okaishi K**, **Okuro M**, **Murai H**, **Nishimura Y**, **Nomura K**, **Tsuchiya H**, **Ohashi I**, **Matsumoto M**.

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Electromagnetic field (EMF) radiation has been found to induce arteriolar dilatation, but the mechanism of action remains largely unknown. This study investigated the effect of EMF radiation on the production of endothelin-1 (ET-1), a potent vasoconstrictor, by cultured endothelial cells. EMF radiation reduced ET-1 basal levels in human umbilical vein and microvascular endothelial cells, but failed to reduce ET-1 basal levels in bovine and human aortic endothelial cells. EMF radiation significantly inhibited thrombin-stimulated ET-1 production in all four endothelial cell types in a dose-dependent manner. EMF radiation significantly inhibited thrombin-induced endothelin-1 mRNA expression in all four cell types. The inhibitory effect of EMF radiation on ET-1 production was abolished by the nitric oxide synthase inhibitor NG-monomethyl-L-arginine (10(-3) mol/1). These results demonstrate that EMF radiation modulates ET-1 production in cultured vascular endothelial cells and the inhibitory effect of EMF radiation is, at least partly, mediated through a nitric oxide-related pathway.

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http://jpet.aspetjournals.org/cgi/content/full/295/1/314

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Nitric Oxide Is Involved in Acetylcholinesterase Inhibitor-Induced Myopathy in Rats

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Abstract

Excess activation of muscle nicotinic acetylcholine receptors due to genetic mutations, as seen in slow channel congenital myasthenic syndrome, or acetylcholinesterase (AChE) inhibition results in muscle cell degeneration. Our recent work showed that nitric oxide synthase (NOS) inhibitors prevent nicotine-induced muscle cell death in culture. In the present study, we examined the effects of NOS inhibition on nicotinic receptor-mediated myopathy in vivo. Rats injected with the AChE inhibitor paraoxon demonstrate a 90-fold increase in the number of dying muscle cells compared with control as evidenced histologically by centralized nuclei and the presence of degenerating profiles. Coadministration of the nonspecific NOS inhibitor nitro-L-arginine methyl ester or the neuronal NOS-specific inhibitor 7-nitroindazole dramatically reduced the presence of such degenerating profiles to ~20% of that seen with paraoxon alone. These results show that inhibition of NOS, as well as neuronal NOS, significantly reduces AChE inhibitor-induced muscle cell degeneration, suggesting that increased nitric oxide production mediates such myopathy.

(NDR : L'étude complète suit, pas chargée, trop longue)

http://www.asrim.ch/fr-ch/asrim/myopathie.html

La myopathie

Sous ce terme sont regroupées plusieurs maladies touchant l'unité motrice. L'unité motrice comprend le neurone moteur, localisé dans la moelle épinière, son prolongement ou axone dans le nerf périphérique, la jonction neuromusculaire et l'ensemble des fibres musculaires innervées par le neurone. L'unité motrice est l'unité anatomique et fonctionnelle terminale du système moteur. Toutes les maladies de l'unité motrice s'expriment pas un déficit moteur (parésie ou faiblesse musculaire ; plégie ou paralysie). Le déficit varie en sévérité et dans sa localisation (généralisée ou localisée à la face, au tronc, aux membres), selon la maladie.

Les processus d'atteinte

Dans les maladies de l'unité motrice, les processus d'atteinte aboutissent à la perte fonctionnelle des neurones moteurs (sclérose latérale amyotrophique, amyotrophies spinales, poliomyélite, etc...), des fibres nerveuses (neuropathies) ou du muscle (myopathies au sens propre).

Les processus d'atteinte sont acquis, dans le cadre le plus souvent d'une inflammation (myosites, polyneuropathies inflammatoires) ou d'une infection (poliomyélite, lèpre, myosites). Mais de nombreuses atteintes de l'unité motrice sont la conséquence d'un déficit de fonction du métabolisme ou de structure des cellules ; ces atteintes sont géniques ou génétiquement déterminées. Ces termes impliquent l'absence ou la présence d'une information tronquée dans le code génétique : l'information fait ainsi défaut à partir de codes régulant la vie cellulaire, localisés sur les chromosomes (ou gènes).

Les maladies génétiques

Les maladies dites géniques ont été reconnues depuis plusieurs décades par l'observation d'une transmission familiale de l'atteinte. Les mécanismes cellulaires à l'origine de la maladie sont compris depuis peu de temps, grâce à la biologie cellulaire (génie génétique). C'est en effet depuis 1986 qu'a été reconnu pour la première fois un déficit d'une protéine importante dans le maintien de la structure des fibres musculaires pour la forme la plus sévère de la myopathie, la dystrophie musculaire de Duchenne. Depuis lors, la médecine moléculaire a permis de reconnaître de nombreuses anomalies dans d'autres atteintes du nerf ou du muscle. Toutes ces maladies sont complexes et nécessitent un travail de regroupement clinique et une recherche laborieuse de laboratoire. En l'espace d'une quinzaine d'années, les connaissances ont explosé, mais le traitement du remplacement de la fonction cellulaire, par thérapie génique, fait encore défaut ou est expérimental. Son potentiel thérapeutique a été sous-estimé dans sa rapidité de mise en place, mais de nombreux efforts sont maintenus pour l'appliquer en pratique clinique.

Le diagnostic

La gravité et le profil évolutif varie beaucoup d'une maladie à l'autre. C'est les raisons pour lesquelles un diagnostic de maladie spécifique est indispensable à connaître. Le diagnostic nécessite des investigations qui sont idéalement réalisées dans un centre spécialisé, qui signifie que les médecins qui y travaillent ont l'expérience de ces maladies. Des informations spécifiques concernant les nombreuses maladies neuromusculaires connues peuvent être obtenues dans la documentation fournie gratuitement par l'ASRM ou par l'internet (voir liens de sites ou home pages).

Med Hypotheses. 2001 Aug;57(2):139-45.

Comment in: Med Hypotheses. 2005;65(3):631-3.

Common etiology of posttraumatic stress disorder, fibromyalgia, chronic fatigue syndrome and multiple chemical sensitivity via elevated nitric oxide/peroxynitrite.

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Three types of overlap occur among the disease states chronic fatigue syndrome (CFS), fibromyalgia (FM), multiple chemical sensitivity (MCS) and posttraumatic stress disorder (PTSD). They share common symptoms. Many patients meet the criteria for diagnosis for two or more of these disorders and each disorder appears to be often induced by a relatively short-term stress which is followed by a chronic pathology, suggesting that the stress may act by inducing a self-perpetuating vicious cycle. Such a vicious cycle mechanism has been proposed to explain the etiology of CFS and MCS, based on elevated levels of nitric oxide and its potent oxidant product, peroxynitrite. Six positive feedback loops were proposed to act such that when peroxynitrite levels are elevated, they may remain elevated. The biochemistry involved is not highly tissue-specific, so that variation in symptoms may be explained by a variation in nitric oxide/peroxynitrite tissue distribution. The evidence for the same biochemical mechanism in the etiology of PTSD and FM is discussed here, and while less extensive than in the case of CFS and MCS, it is nevertheless suggestive. Evidence supporting the role of elevated nitric oxide/peroxynitrite in these four disease states is summarized, including induction of nitric oxide by common apparent inducers of these disease states, markers of elevated nitric oxide/peroxynitrite in patients and evidence for an inductive role of elevated nitric oxide in animal models. This theory appears to be the first to provide a mechanistic explanation for the multiple overlaps of these disease states and it also explains the origin of many of their common symptoms and similarity to both Gulf War syndrome and chronic sequelae of carbon monoxide toxicity. This theory suggests multiple studies that should be performed to further test this proposed mechanism. If this mechanism proves central to the etiology of these four conditions, it may also be involved in other conditions of currently obscure etiology and criteria are suggested for identifying such conditions.

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Ann N Y Acad Sci. 2001 Mar;933:323-9.

Elevated nitric oxide/peroxynitrite mechanism for the common etiology of multiple chemical sensitivity, chronic fatigue syndrome, and posttraumatic stress disorder.

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Various types of evidence implicate nitric oxide and an oxidant, possibly peroxynitrite, in MCS and chemical intolerance (CI). The positive feedback loops proposed earlier for CFS may explain the chronic nature of MCS (CI) as well as several of its other reported properties. These observations raise the possibility that this proposed elevated nitric oxide/peroxynitrite mechanism may be the mechanism of a new disease paradigm, answering the question raised by Miller earlier: "Are we on the threshold of a new theory of disease?"

PMID: 12000033 [PubMed - indexed for MEDLINE]

Biochem J. 2002 Aug 15;366(Pt 1):203-9.

Selenium deficiency increases the expression of inducible nitric oxide synthase in RAW 264.7 macrophages: role of nuclear factor-kappaB in up-regulation.

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The inducible isoform of nitric oxide synthase (iNOS) is implicated in atherosclerosis, malignancy, rheumatoid arthritis, tissue and reperfusion injuries. A key determinant of the pro-oxidant versus protective effects of NO is the underlying redox status of the tissue. Selenoproteins, such as glutathione peroxidases (GPxs) and thioredoxin reductases, are key components of cellular defence and promote optimal antioxidant/oxidant balance. In this study, we have investigated the relationship between Se status, iNOS expression and NO production in Se-deficient and Se-supplemented RAW 264.7 macrophage cell lines. The cellular GPx activity, a measure of Se status, was 17-fold lower in Se-deficient RAW 264.7 cells and the total cellular oxidative tone, as assessed by flow cytometry with 2',7'-dichlorodihydrofluorescein diacetate, was higher in the Se-deficient cells than the Se-supplemented cells. Upon lipopolysaccharide (LPS) stimulation of these cells in culture, we found significantly higher iNOS transcript and protein expression levels with an increase in NO production in Se-deficient RAW 264.7 cells than the Se-supplemented cells. Electrophoretic mobility-shift assays, nuclear factor-kappaB (NF-kappaB)-luciferase reporter assays and Western blot analyses indicate that the increased expression of iNOS in Se deficiency could be due to an increased activation and consequent nuclear localization of the redox-sensitive transcription factor NF-kappaB.

These results suggest an inverse relationship between cellular Se status and iNOS expression in LPS-stimulated RAW 264.7 cells and provide evidence for the beneficial effects of dietary Se supplementation in the prevention and/or treatment of oxidative-stress-mediated inflammatory diseases.

PMID: 12006087 [PubMed - indexed for MEDLINE]

Otolaryngol Head Neck Surg. 2005 May;132(5):713-6.

Nitric oxide level in the nasal and sinus mucosa after exposure to electromagnetic field.

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OBJECTIVE: The purpose of this study was to examine the changes in nitric oxide (NO) level in the nasal and paranasal sinus mucosa after exposure radiofrequency electromagnetic fields (EMF).

STUDY DESIGN AND SETTING: Thirty male Sprague-Dawley rats were randomly grouped as follows: EMF group (group I; n, 10), EMF group in which melatonin received (group II; n, 10) and the control (sham operated) group (group III; n, 10). Groups I and II were exposed to a 900 MHz. Oral melatonin was given in group II. Control rats (group III) were also placed in the tube as the exposure groups, but without exposure to EMF. At the end of 2 weeks, the rats were sacrificed, and the nasal and paranasal sinus mucosa dissected. NO was measured in nasal and paranasal mucosa.

RESULTS: The nasal and paranasal sinus mucosa NO levels of group I were significantly higher than those of the control group (group III) ($P < 0.05$). However, there was no statistically significant difference between group II and the control group (group III) regarding NO output ($P > 0.05$).

CONCLUSION: Exposure to EMF released by mobile phones (900 MHz) increase NO levels in the sinus and nasal mucosa.

SIGNIFICANCE: Increased NO levels may act as a defense mechanism and presumably related to tissue damage. In addition, melatonin may have beneficial effect to prevent these changes in the mucosa.

PMID: 15886623 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10927193&dopt=Abstract

Pathophysiology. 2000 Jul;7(2):131-135.

Enhancement of nitric oxide generation by low frequency electromagnetic field.

Yoshikawa T, **Tanigawa M**, **Tanigawa T**, **Imai A**, **Hongo H**, **Kondo M**.

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Oxidative stress is implicated in the intracellular signal transduction pathways for nitric oxide synthase (NOS) induction. The electromagnetic field (EMF) is believed to increase the free radical lifespan [S. Roy, Y. Noda, V. Eckert, M.G. Traber, A. Mori, R. Liburdy, L. Packer, The phorbol 12-myristate 13-acetate (PMA)-induced oxidative burst in rat peritoneal neutrophils is increased by a 0.1 mT (60 Hz) magnetic field, FEBS Lett. 376 (1995) 164-6; F.S. Prato, M. Kavaliers, J.J. Carson, Behavioural evidence that magnetic field effects in the land snail, Cepaea nemoralis, might not depend on magnetite or induced electric currents, Bioelectromagnetics 17 (1996) 123-30; A.L. Hulbert, J. Metcalfe, R. Hesketh, Biological response to electromagnetic fields, FASEB 12 (1998) 395-420]. We tested the effects of EMF on endotoxin induced nitric oxide (NO) generation in vivo. Male BALB/C mice were injected with lipopolysaccharide (LPS) intraperitoneously (i.p.), followed by the exposure to EMF (0.1 mT, 60 Hz). Five hours and 30 min after the LPS administration, mice were administered with a NO spin trap, ferrous N-methyl-D-glucaminedithiocarbamate (MGD-Fe). Thirty minutes later, mice were sacrificed, and their livers were removed. The results were compared to three control groups: group A (LPS (-) EMF(-)); group B (LPS(-) EMF(+)); group C (LPS(+) EMF(-)). The ESR spectra of obtained livers were examined at room temperature. Three-line spectra of NO adducts were observed in the livers of all groups. In groups A and B very weak signals were observed, but in groups C and D strong spectra were observed. The signal intensity of the NO adducts in Group D was also significantly stronger than that in Group C. EMF itself did not induce NO generation, however, it enhanced LPS induced NO generation in vivo.

PMID: 10927193 [PubMed - as supplied by publisher]