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1: Antimicrob Agents Chemother. Related Articles, Cited
2005 Jun;49(6):2352-5. Articles, Books, LinkOut

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Intravesical nitric oxide delivery for prevention of catheter-associated urinary tract infections.

Carlsson S, Weitzberg E, Wiklund P, Lundberg JO.

Department of Surgery, Section of Urology, Karolinska Hospital, and Department of Physiology & Pharmacology, Karolinska Institute, 171 77 Stockholm, Sweden.

The use of indwelling urinary catheters is a major risk factor for urinary tract infection; and despite the availability of numerous preventive regimens, this condition is still extremely common. In earlier studies we have demonstrated the inhibitory effects of nitrite and ascorbic acid on bacterial growth in urine. When combined, these compounds generate antibacterial reactive nitrogen species, including the gas nitric oxide. We have now tested in a laboratory model of the urinary bladder whether filling of the catheter retention balloon with nitrite and ascorbic acid would generate measurable amounts of NO outside the membrane and whether this would affect bacterial growth in the surrounding urine. Two strains of Escherichia coli, one strain isolated from a patient (U1106024) and one reference strain (ATCC 25922), were tested. Nitric oxide gas was generated in the silicone balloon and readily diffused into the urine. When control catheters with ascorbic acid but without nitrite were used, bacterial counts increased from 9.0×10^5 to 2.0×10^8 CFU/ml (strain U1106024) and from 2.5×10^6 to 2.7×10^8 CFU/ml (strain ATCC 25922) after 24 h. In contrast, in test catheters with ascorbic

acid and nitrite, both strains tested were effectively killed. The NO donor {DETA NONOate, (Z)-1-[N-(2-aminoethyl)-N-(2-ammonioethyl)amino]diazene-1-ium-1,2-diolate} also showed antibacterial activity in the same model, thereby supporting a central role of NO in achieving the observed effects. Future clinical trials will reveal whether this novel approach for the intravesical delivery of an antibacterial gas could be used to prevent catheter-associated infections.

PMID: 15917532 [PubMed - indexed for MEDLINE]

□ 2: Am J Clin Nutr. 2005 Related Articles, Compound via MeSH, Apr;81(4):859-63. Substance via MeSH, Books, LinkOut

Full text article at
www.ajcn.org

Vitamin C supplementation to prevent premature rupture of the chorioamniotic membranes: a randomized trial.

Casanueva E, Ripoll C, Tolentino M, Morales RM, Pfeffer F, Vilchis P, Vadillo-Ortega F.

Public Health Research Branch, National Institute of Perinatology, Mexico City, Mexico. casanuev@servidor.unam.mx

BACKGROUND: Vitamin C is involved in the synthesis and degradation of collagen and is important for maintenance of the chorioamniotic membranes. Inadequate availability of ascorbic acid during pregnancy has been proposed as a risk factor for premature rupture of the chorioamniotic membranes (PROM). **OBJECTIVE:** The objective of the study was to evaluate the effectiveness of 100 mg vitamin C/d in preventing PROM. **DESIGN:** A controlled double-blind trial was performed. Pregnant women (n = 126) in their 20th wk of gestation were invited; 120 accepted and were randomly assigned to 2 groups (100 mg vitamin C/d or placebo). Every 4 wk, plasma and leukocyte vitamin C concentrations were measured, and each subject was evaluated for cervicovaginal infection. The incidence of PROM was recorded for each group as an indicator of the protective effect of vitamin C supplementation. **RESULTS:** One hundred nine patients finished the study. Mean plasma vitamin C concentrations decreased significantly throughout the pregnancy in both groups (P = 0.001), and there were no significant differences between groups. Between weeks 20 and 36, mean leukocyte vitamin C concentrations decreased from 17.5 to 15.23 microg/10(8) cells in the placebo group and increased from 17.26 to 22.17 microg/10(8) cells in the supplemented group (within- and between-group differences: P = 0.001). The incidence of PROM was 14 per 57 pregnancies (24.5%) in the placebo group and 4 per 52 pregnancies (7.69%) in the supplemented group (relative risk: 0.26; 95% CI: 0.078, 0.837). **CONCLUSION:** Daily supplementation with 100 mg vitamin C after 20 wk of gestation effectively lessens the incidence

of PROM.

Publication Types:
Clinical Trial
Randomized Controlled Trial

PMID: 15817864 [PubMed - indexed for MEDLINE]

3: Forum Nutr. 2003;56:42-5. [Related Articles, Books, LinkOut](#)

Vitamin C: from popular food supplement to specific drug.

Goldenberg H.

Institut f. Medizinische Chemie, Wien, Austria.
hans.goldenberg@univie.ac.at

The daily requirement of a human person for vitamin C (ascorbic acid) has now been established at 100 mg. This value was already on the map when Arnold Durig put together the most important needs of nutritional ingredients. The modern value rests on the saturating level of ascorbate in leukocytes, which is in the millimolar range. The mechanism of accumulation of ascorbate in these cells rests on the uptake of oxidized dehydroascorbic acid. It is very efficient and avoids loss of vitamin which occurs in vitro when ascorbate is oxidized because of the great instability of the dehydro form. Therefore and increased requirement in case of infection is very unlikely from the biochemical point of view. However, low concentrations of ascorbate are found in patients suffering from arterial diseases or diseases accompanied by arterial damage such as diabetes mellitus. Ascorbate is known as a protection factor for the arterial endothel, but it is not clear by what mechanism this protection is brought about. Moreover, under clinical conditions very high concentrations are needed, which are achieved only by intravenous infusion, and the protection is only observed when the disease is manifest, not in healthy people. Therefore, also in this respect an increase in daily intake seems of no prophylactic value. Thus, by using high concentrations of ascorbate as an i.v. drug, effects of this substance frequently observed in vitro, could be used for therapy. This includes not only treatment of arterial diseases, but also relates to the cytotoxic effects of the vitamin against certain tumor cells and may assist conventional chemotherapy.

Publication Types:
Review

PMID: 15806791 [PubMed - indexed for MEDLINE]

4: Eur J Epidemiol. [Related Articles, Books, LinkOut](#)

2005;20(1):67-71.



Effect modification by vitamin C on the relation between gastric cancer and *Helicobacter pylori*.

Kim DS, Lee MS, Kim YS, Kim DH, Bae JM, Shin MH, Ahn YO.

Department of Preventive Medicine, Seoul National University College of Medicine, Seoul, Korea.

A hospital-based case-control study of 295 cases with histologically confirmed gastric cancer and age and sex-matched controls was conducted to evaluate the effect of dietary vitamin C intake upon the relation between *Helicobacter pylori* infection and gastric cancer in Korea in 1997-1998. Anti-*H. pylori* IgG was detected by ELISA. A food frequency questionnaire, and a questionnaire on demographic factors, including past medical history, smoking, alcohol consumption, and life style was also administered. The prevalences of *H. pylori* IgG in cases and controls were 80.7% and 71.2%, respectively, and the odds ratio (OR) of *H. pylori* for gastric cancer was 1.68 (95% confidence interval (CI): 1.14, 2.44), after adjusting for age, sex, educational level, and a past medical history of gastritis or gastric ulcer. In a stratified analysis, *H. pylori* seropositivity was found to be a significant risk factor for gastric cancer in the low vitamin C intake group (OR = 4.68; 95% CI: 1.97, 11.1), but not in the high vitamin C intake group (OR = 0.72; 95% CI: 0.32, 1.65). Vitamin C intake was found to modify the relation between *H. pylori* and gastric cancer.

PMID: 15756906 [PubMed - indexed for MEDLINE]

5: Eur J Epidemiol. 2004;19(11):1061-2. Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut



Does vitamin C dietary intake modify the association between *Helicobacter pylori* infection and gastric cancer?

Lunet N, Barros H.

Publication Types:
Letter

PMID: 15648601 [PubMed - indexed for MEDLINE]

6: Roczn Akad Med Białymst. 2004;49:157-61. Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut

Antioxidants in the treatment of patients with renal failure.

Luciak M.

Department of Internal Medicine and Dialysis, Medical University of Lodz, Poland. rofe@toya.net.pl

Renal failure is accompanied by oxidative stress, which is caused by enhanced production of reactive oxygen species and impaired antioxidant defense. The suggested therapeutical interventions aimed at reducing oxidative stress in chronic renal failure patients are as follows: 1) the use of biocompatible membranes, ultrapure dialysate, and removal of endogenous foci of infection; 2) haemolipodialysis, and electrolysed reduced water for dialysate preparation; 3) administration of antioxidants (alpha-tocopherol, ascorbic acid, N-acetylcysteine, reduced glutathione); 4) substances possibly affecting oxidative stress indirectly (erythropoietin, sodium selenite). As currently available data have, as yet, provided rather limited evidence for the clinical benefit of antioxidant interventions, at present it is untimely to give practical recommendations with regard to antioxidant treatment of patients with renal failure.

Publication Types:

Review

Review, Tutorial

PMID: 15631333 [PubMed - indexed for MEDLINE]

7: Clin Pharmacol Ther. 2004 Dec;76(6):579-87. [Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut](#)

ELSEVIER
FULL-TEXT ARTICLE

The effect of clarithromycin, fluconazole, and rifabutin on dapsone hydroxylamine formation in individuals with human immunodeficiency virus infection (AACTG 283).

Winter HR, Trapnell CB, Slattery JT, Jacobson M, Greenspan DL, Hooton TM, Unadkat JD.

University of Washington, Seattle, WA 98195, USA.

BACKGROUND: Dapsone hydroxylamine formation is thought to be the cause of the high rates of adverse reactions to dapsone in human immunodeficiency virus (HIV)-infected individuals. Therefore we studied the effect of the commonly coadministered drugs fluconazole, clarithromycin, and rifabutin on hydroxylamine formation in individuals with HIV infection. **METHODS:** HIV-infected subjects (CD4 + > or =200 cells/mm³) were enrolled in a 2-part (A or B) open-label drug interaction study. In part A, subjects (n = 12) received dapsone (100-mg tablet once daily) alone for 2

weeks and then, in a randomly assigned order, received dapsone and either fluconazole (200 mg daily), rifabutin (300 mg daily), or fluconazole plus rifabutin, each for a 2-week period. Part B (n = 11) was identical to part A except that clarithromycin (500 mg twice daily) was substituted for rifabutin. On the last study day of each 2-week period, plasma and urine were collected over ascorbic acid for 24 hours. RESULTS: In part A, fluconazole decreased the area under the plasma concentration-time curve, percent of dose excreted in 24-hour urine, and formation clearance of the hydroxylamine by 49%, 53%, and 55% (n = 12, P < .05), respectively. This inhibition of in vivo hydroxylamine formation was quantitatively consistent with that predicted from human liver microsomal experiments. Rifabutin had no effect on hydroxylamine area under the plasma concentration-time curve or percent excreted in 24-hour urine but increased formation clearance of the hydroxylamine by 92% (n = 12, P < .05). Dapsone clearance was increased by rifabutin or rifabutin plus fluconazole (67% and 38%, respectively) (n = 12, P < .05) but was unaffected by fluconazole or clarithromycin. In part B, hydroxylamine production was unaffected by clarithromycin but was affected by fluconazole in a manner identical to that in part A. CONCLUSIONS: On the basis of these data and with the assumption that the exposure to the hydroxylamine is a determinant of dapsone toxicity, we predict that coadministration of fluconazole should decrease the rate of adverse reactions to dapsone in persons with HIV infection but that rifabutin and clarithromycin will have no effect. When dapsone is given in combination with rifabutin, dapsone dosage adjustment may be necessary in light of the increase in dapsone clearance.

Publication Types:

Clinical Trial

Randomized Controlled Trial

PMID: 15592329 [PubMed - indexed for MEDLINE]

8: Clin Evid. 2003 Dec;(10):1747-56. [Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut](#)

Update of:

Clin Evid. 2003 Jun;(9):1701-11.

Upper respiratory tract infection.

Del Mar C, Glasziou P.

Oxford, UK.

Publication Types:

Review

PMID: 15555175 [PubMed - indexed for MEDLINE]

9: Panminerva Med. 2004
Sep;46(3):165-9.

Related Articles, Books,
LinkOut

Is hepcidin the bridge linking *Helicobacter pylori* and anemia of chronic infection? A research proposal.

Pellicano R, Rizzetto M.

UOADU Gastro-Hepatology, Molinette Hospital, Turin, Italy.
rinaldo_pellican@hotmail.com

Since the last decade, several studies have reported on the link between chronic *Helicobacter pylori* (*H. pylori*) or *Helicobacter* species (*H. species*) infection and a variety of extragastric manifestations, comprising iron-deficiency anemia. A crucial question concerns which possible pathogenic mechanism of *H. pylori* infection may be involved in chronic anemia. Recent findings support the hypothesis that in subjects with *H. pylori*-positive gastritis, concomitant changes in intragastric pH and ascorbic acid are present that might play a role in impairing alimentary iron absorption with consequent sideropenic anemia. It has also been speculated that *H. pylori* infected antrum could act as a sequestering focus for iron. The bacterium enhances gastric lactoferrin, which captures iron from transferrin. The iron thus bound to lactoferrin is in turn picked up by the bacterium, by means of its outer membrane receptors, for its own growth. These models, however, are not able to answer why iron-deficiency anemia does not develop in all infected subjects. Recently, a new anti-microbial liver-made peptide, namely hepcidin, has been characterised. The link between hepcidin induction, inflammation and anemia both in humans and in animal models supports its key role as mediator of anemia of inflammation. In the present paper, we highlight the data available on the association between *H. pylori* and iron-deficiency anemia and, we propose to evaluate a possible mechanism involving hepcidin in a bridging role linking the infection to the anemia.

Publication Types:

Review

Review, Tutorial

PMID: 15510085 [PubMed - indexed for MEDLINE]

10: Expert Rev Anti Infect
Ther. 2003 Dec;1(4):619-26.

Related Articles, Compound via
MeSH, Substance via MeSH, Books,
LinkOut



Current and future therapeutic approaches to the common cold.

Mossad SB.

Department of Infectious Diseases Cleveland Clinic Foundation,
9500 Euclid avenue, S-32. Cleveland, OH, 44195, USA.
mossads@ccf.org

For decades, investigators have strived to elucidate the pathogenesis, and hence a treatment, for the common cold. Therapy has been tried with a variety of agents, ranging from anecdotal folk remedies, to well-designed medications. Measures primarily directed to relieve the symptoms of the common cold, rather than specific antimicrobial agents, are the current main stay of therapy. Millions of patients would benefit from an easy-to-perform diagnostic test and specific therapy that works quickly, shortening the duration of illness and preventing further spread of infection.

Publication Types:

Review

Review, Tutorial

PMID: 15482159 [PubMed - indexed for MEDLINE]

11: Clin Evid. 2003 Jun;(9):1701-11. Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut

Update in:

Clin Evid. 2003 Dec;(10):1747-56.

Update of:

Clin Evid. 2002 Jun;(7):1391-9.

Upper respiratory tract infection.

Del Mar C, Glasziou P.

University of Queensland, Brisbane, Australia.

Publication Types:

Review

PMID: 15366203 [PubMed - indexed for MEDLINE]

12: Biol Reprod. 2005 Jan;72(1):230-5. Related Articles, Books, Epub 2004 Sep 8. LinkOut

Full text article at
www.biolreprod.org

Reduced collagen and ascorbic acid concentrations and increased proteolytic susceptibility with prelabor fetal membrane rupture in

women.

Stuart EL, Evans GS, Lin YS, Powers HJ.

Human Nutrition Unit, University of Sheffield, Sheffield S5 7AU,
United Kingdom.

Prelabor rupture of the fetal membranes affects approximately 10% of women at term, resulting in an increased risk of maternal and neonatal infection. Evidence suggests that membrane rupture is related to biochemical processes involving the extracellular matrix of the membranes. We tested the hypothesis that prelabor ruptured membranes are characterized by reduced collagen concentrations, altered collagen cross-link profiles, and increased concentrations of biomarkers of oxidative damage. We also set out to determine whether these effects are modulated by ascorbic acid status. In a case-control study, we explored the role that ascorbic acid, oxidative stress, collagen, and collagen cross-links play in determining membrane integrity and developed a functional assay to assess membrane proteolytic susceptibility. Prelabor ruptured membrane had a reduced ascorbic acid concentration in comparison with controls while protein carbonyl and malondialdehyde concentrations were increased. Collagen concentrations were also reduced in prelabor ruptured membrane, and while the concentration of collagen cross-links was not significantly different between prelabor and timely ruptured membrane, there was a regional variation in cross-link ratio within the amniotic sac. Proteolytic resistance in vitro was reduced in prelabor ruptured membrane and also exhibited regional variation within the amniotic sac. Our findings are strongly supportive of a role for the enhanced degradation of membrane collagen in the determination of prelabor rupture of fetal membranes. The formation of the rupture initiation site is a function of a regional variation in collagen cross-link ratio. Tissue ascorbic acid status may be an important mediator of these processes.

PMID: 15355881 [PubMed - indexed for MEDLINE]

13: Asia Pac J Clin Nutr. 2004;13(3):226-30. [Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut](#)

Immunological response to antioxidant vitamin supplementation in rural Bangladeshi school children with group A streptococcal infection.

Ahmed J, Zaman MM, Ali SM.

National Centre for Control of Rheumatic Fever and Heart Diseases,
Sher-e-Bangla Nagar, Dhaka 1207, Bangladesh. jasim@bangla.net

Group A beta haemolytic streptococcal (GABHS) infection induce an abnormal immune response in a susceptible host. Micronutrient deficiency may affect the immune response of an individual. The aim of this study was to determine whether antioxidant vitamins could improve the abnormal immune response in GABHS infected children in rural Bangladesh. A total of 516 GABHS infected school children aged 5 to 15 years were randomly assigned to two groups. Group 1 (N=258) was treated with phenoxymethyl penicillin V and group 2 (N=258) was treated with penicillin V plus antioxidant vitamins (beta carotene, alpha tocopherol and ascorbic acid). From each group two blood samples were drawn; the first sample at the beginning of the study and another one after eight weeks. Streptococcal antibodies and immunoglobulin levels were compared between the two samples. The mean age of the study population was 10.6 years. Equal number of boys and girls were included in both groups. After treatment, antistreptolysin O (ASO) and antideoxyribonuclease B (ADNase B) titres were decreased in both groups. Serum alpha tocopherol and beta-carotene levels were increased significantly in group 2. In group 1 immunoglobulin M and A levels decreased significantly (P =0.0001) whereas immunoglobulin G showed no change. To the contrary, concentration of three immunoglobulins decreased significantly (P=0.0001) in group 2. Least-square means of between-group differences showed highly significant results for ASO, ADNase B, immunoglobulins M, A and G (P=0.0001). Our data indicate that treatment by antioxidant vitamins plus penicillin is more effective in decreasing immunological abnormalities in GABHS infected children then penicillin alone.

Publication Types:

Clinical Trial

Randomized Controlled Trial

PMID: 15331332 [PubMed - indexed for MEDLINE]

14: *Pediatr Nephrol.* 2004 Nov;19(11):1303; author reply 1304. Epub 2004 Aug 20. [Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut](#)

Comment on:

Pediatr Nephrol. 2004 Apr;19(4):378-83.



The effect of dietary factors on the risk of developing urinary tract infection.

Kalhoff H.

Publication Types:

Comment

Letter

PMID: 15322890 [PubMed - indexed for MEDLINE]

□ 15: Arch Dis Child Fetal Neonatal Ed. 2004 Sep;89(5):F412-6. Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut

ADC: Fetal Neonatal Ed.

Pulmonary antioxidant concentrations and oxidative damage in ventilated premature babies.

Collard KJ, Godeck S, Holley JE, Quinn MW.

Department of Medical Sciences, St Loye's School of Health Studies, Millbrook House, Millbrook Lane, Topsham Road, Exeter EX2 6ES, UK. k.j.collard@exeter.ac.uk

OBJECTIVE: To determine the relation between lipid peroxidation and the antioxidants ascorbate, urate, and glutathione in epithelial lining fluid in ventilated premature babies, and to relate the biochemical findings to clinical outcome. **DESIGN:** A cohort study conducted between January 1999 and June 2001. **SETTING:** A NHS neonatal intensive care unit. **PATIENTS:** An opportunity sample of 43 ventilated babies of less than 32 weeks gestation. **MAIN OUTCOME MEASURES:** The duration of supplementary oxygen according to the definition of bronchopulmonary dysplasia (BPD; oxygen dependency at 36 weeks gestational age). **METHODS:** Epithelial lining fluid was sampled by bronchoalveolar lavage. Ascorbate, urate, glutathione, and malondialdehyde (a marker of lipid peroxidation) were measured. **RESULTS:** Babies who developed BPD had significantly lower initial glutathione concentrations (mean (SEM) 1.89 (0.62) v 10.76 (2.79) microM; $p = 0.043$) and higher malondialdehyde concentrations (mean (SEM) 1.3 (0.31) v 0.345 (0.09) microM; $p < 0.05$) in the epithelial lining fluid than those who were not oxygen dependent. These variables were poor predictors of the development of BPD. Gestational age, endotracheal infection, and septicaemia had good predictive power. The level of oxidative damage was associated with the presence of endotracheal infection/septicaemia rather than inspired oxygen concentration. **CONCLUSIONS:** Endotracheal infection, septicaemia, and gestational age, rather than antioxidant concentrations, are the most powerful predictors of the development of BPD.

PMID: 15321959 [PubMed - indexed for MEDLINE]

□ 16: Mol Microbiol. 2004 Aug;53(3):807-20. Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut



Neisseria meningitidis accelerates ferritin degradation in host epithelial cells to yield an essential iron source.

Larson JA, Howie HL, So M.

Department of Molecular Microbiology and Immunology, Oregon Health and Science University, Portland, OR 97239, USA.
larsonja@ohsu.edu

In order to colonize humans and cause disease, pathogenic bacteria must assimilate iron from their host. The vast majority of non-haem iron in humans is localized intracellularly, within the storage molecule ferritin. Despite the vast reserves of iron within ferritin, no pathogen has been demonstrated previously to exploit this molecule as an iron source. Here, we show that the Gram-negative diplococcus *Neisseria meningitidis* can trigger rapid redistribution and degradation of cytosolic ferritin within infected epithelial cells. Indirect immunofluorescence microscopy revealed that cytosolic ferritin is aggregated and recruited to intracellular meningococci (MC). The half-life of ferritin within cultured epithelial cells was found to decrease from 20.1 to 5.3 h upon infection with MC. Supplementation of infected epithelial cells with ascorbic acid abolished ferritin redistribution and degradation and prevented intracellular MC from replicating. The lysosomal protease inhibitor leupeptin slowed ferritin turnover and also retarded MC replication. Our laboratory has shown recently that MC can interfere with transferrin uptake by infected cells (Bonnah R.A., et al., 2000, *Cell Microbiol* 2: 207-218) and that, perhaps as a result, the infected cells have a transcriptional profile indicative of iron starvation (Bonnah, R.A., et al., 2004, *Cell Microbiol* 6: 473-484). In view of these findings, we suggest that accelerated ferritin degradation occurs as a response to an iron starvation state induced by MC infection and that ferritin degradation provides intracellular MC with a critical source of iron. Copyright 2004 Blackwell Publishing Ltd

PMID: 15255894 [PubMed - indexed for MEDLINE]

17: *Curr Opin Ophthalmol*. 2004 Aug;15(4):333-41.



Related Articles, Books, LinkOut

Update on laser subepithelial keratectomy (LASEK).

Yee RW, Yee SB.

Hermann Eye Center, Department of Ophthalmology and Visual Science, University of Texas Health Science Center at Houston, Houston, Texas 77030, USA. richard.w.yee@uth.tmc.edu

PURPOSE OF REVIEW: This study reviews current concepts in laser subepithelial keratectomy (LASEK), variations in LASEK techniques, the role of pharmacology in LASEK, and optimizing outcomes in LASEK. **RECENT FINDINGS:** Recent studies continue to support the use of LASEK over that of LASIK in the correction of refractive error. In addition, the advent of pharmacological/biologic intervention, improved algorithms, and wavefront technology have expanded the armamentarium available to ophthalmologists in the maximization of LASEK outcomes. **SUMMARY:** LASEK offers an excellent profile in terms of both final outcome (uncorrected visual acuity) and safety (best corrected visual acuity). Untoward effects of LASEK are readily prevented/treated with a variety of agents. Postoperative pain can be ameliorated using topical and oral analgesia. Infection can be most effectively addressed with the fourth generation of fluoroquinolones. Haze may be treated or prevented using numerous remedies namely autologous serum, steroids, ascorbic acid, mitomycin-c, and NSAIDS. Wavefront combined with LASEK rather than with LASIK may offer the best refractive outcome.

Publication Types:
Evaluation Studies
Review
Review, Tutorial

PMID: 15232474 [PubMed - indexed for MEDLINE]

18: Int J Dermatol. 2004
Jul;43(7):494-7.

Related Articles, Books,
LinkOut



Plasma reactive oxygen species activity and antioxidant potential levels in rosacea patients: correlation with seropositivity to *Helicobacter pylori*.

Baz K, Cimen MY, Kokturk A, Aslan G, Ikizoglu G, Demirseren DD, Kanik A, Atik U.

Department of Dermatology, University of Mersin School of Medicine, Mersin, Turkey. drkbaz@hotmail.com

BACKGROUND: Recent studies have suggested that there might be an etiologic role for *Helicobacter pylori* (HP) in rosacea. HP is a Gram-negative bacterium that colonizes the gastric mucosa, increases the generation of reactive oxygen species (ROS), and decreases plasma antioxidants such as ascorbic acid. **AIM:** To investigate plasma ROS activities and antioxidant status, and their relationship with HP infection, in rosacea patients. **METHODS:** Twenty-nine rosacea patients and 20 age- and sex-matched healthy

controls were examined for specific immunoglobulin G (IgG) and IgM against HP, plasma malondialdehyde (MDA), and antioxidant potential (AOP) levels. **RESULTS:** Compared with controls, the seropositivity of HP for IgM was significantly higher ($P = 0.03$) and the seropositivity of HP for IgG was significantly lower ($P = 0.0001$) in patients with rosacea. Plasma MDA levels were higher ($P = 0.0001$) and AOP levels were lower ($P = 0.019$) in patients than in controls, regardless of the severity of the disease. Plasma MDA and AOP levels were not affected by the seropositivity of HP for IgM and/or IgG in either group. **CONCLUSION:** Our results suggest that rosacea is an oxidative stress condition, as reflected by the increased ROS activity and decreased AOP, regardless of HP infection.

PMID: 15230886 [PubMed - indexed for MEDLINE]

19: J Am Diet Assoc. 2004 Jul;104(7):1095-101. [Related Articles, Compound via MeSH, Substance via MeSH, Books, LinkOut](#)

Erratum in:

J Am Diet Assoc. 2004 Sep;104(9):1481.



US youths in the early stages of HIV disease have low intakes of some micronutrients important for optimal immune function.

Kruzich LA, Marquis GS, Carriquiry AL, Wilson CM, Stephensen CB.

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OBJECTIVE: We examined the association between micronutrient intakes and human immunodeficiency virus (HIV) infection in youths who were at increased nutritional risk because of the demands of growth and disease as well as poor dietary habits. **DESIGN:** This was a cross-sectional study to collect dietary intake data using the Block Food Frequency Questionnaire (98.2). Anthropometric, biochemical, clinical, and sociodemographic data were available. **Subjects/Setting** Participants included 264 HIV-infected and 127 HIV-uninfected adolescents and young adults from the Reaching for Excellence in Adolescent Care and Health network, a multisite observational study on HIV progression. **Statistical analyses** CD4(+) T cells were stratified for HIV-infected youths: ≥ 500 , 200 to 499, and < 200 cells/microL. Micronutrient intakes were compared by presence of HIV infection, using two-sample Student's t tests. Categorical analyses used chi(2) test. Generalized linear regression determined predictors of vitamins A, C, and E; iron; and zinc intakes. **RESULTS:** Almost half (49.0%) of the HIV-infected participants had CD4(+) T cells ≥ 500 cells/microL. After controlling for other factors, HIV-infected participants with CD4(+)

T cells ≥ 500 had decreased iron intake ($P < .05$) and tended to be associated with lower intakes of vitamins C and E ($P < .10$) compared with those with more advanced disease and HIV-uninfected youths. Among those youths with CD4(+) T cells between 200 and 499 cells/microL, a high anxiety score was associated with a sixfold increase in vitamin A intake as compared with those with a low score. Applications/conclusions Given the increased micronutrient requirements, nutrition counseling with HIV-infected youths should focus on early increase of intake of foods rich in micronutrients to improve growth, slow disease progression, and increase survival.

Publication Types:
Multicenter Study

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Low antioxidant vitamin intakes are associated with increases in adverse effects of chemotherapy in children with acute lymphoblastic leukemia.

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BACKGROUND: Chemotherapy leads to an increase in reactive oxygen species, which stresses the antioxidant defense system. Children with acute lymphoblastic leukemia rarely are overtly malnourished, which makes this population ideal for an investigation of the relations between dietary antioxidant consumption, plasma antioxidant concentrations, and chemotherapy-induced toxicity.

OBJECTIVE: This study was conducted to investigate the effect of therapy on antioxidant intakes in children with acute lymphoblastic leukemia, the relation between dietary antioxidant intakes and plasma antioxidant concentrations, and the relation between the incidence of side effects due to treatment and antioxidant intake.

DESIGN: We conducted a 6-mo observational study of 103 children with acute lymphoblastic leukemia. Plasma micronutrient concentrations, dietary intakes, and incidence of side effects of chemotherapy were ascertained at diagnosis and after 3 and 6 mo of therapy. **RESULTS:** Throughout the 6-mo study period, subjects ingested vitamin E, total carotenoid, beta-carotene, and vitamin A in amounts that were 66%, 30%, 59%, and 29%, respectively, of the US

recommended dietary allowance or of the amounts specified in the third National Health and Nutrition Examination Survey. Greater vitamin C intakes at 6 mo were associated with fewer therapy delays, less toxicity, and fewer days spent in the hospital. Greater vitamin E intakes at 3 mo were associated with a lower incidence of infection. Greater beta-carotene intakes at 6 mo were associated with a decreased risk of toxicity. CONCLUSION: A large percentage of children undergoing treatment for acute lymphoblastic leukemia have inadequate intakes of antioxidants and vitamin A. Lower intakes of antioxidants are associated with increases in the adverse side effects of chemotherapy.

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