

Efficacy of high-dose glycine in the treatment of enduring negative symptoms of schizophrenia.

Heresco-Levy U, Javitt DC, Ermilov M, et al. *Arch Gen Psychiatry* 1999;56:29-36.

BACKGROUND: Disturbances of N-methyl-D-aspartate (NMDA) receptor-mediated glutamatergic neurotransmission may play an important role in the pathophysiology of negative symptoms of schizophrenia. Glycine, a small nonessential amino acid, functions as an obligatory coagonist at NMDA receptors through its action at a strychnine-insensitive binding site on the NMDA receptor complex. Glycine-induced augmentation of NMDA receptor-mediated neurotransmission may thus offer a potentially safe and feasible approach for ameliorating persistent negative symptoms of schizophrenia. **METHODS:** Twenty-two treatment-resistant schizophrenic patients participated in a double-blind, placebo-controlled, 6-week, crossover treatment trial with 0.8 g/kg per day of glycine added to their ongoing antipsychotic medication. Clinical assessments, including the Brief Psychiatric Rating Scale (BPRS), the Positive and Negative Syndrome Scale (PANSS), the Simpson-Angus Scale for Extrapyramidal Symptoms, and the Abnormal Involuntary Movement Scale, were performed biweekly throughout the study. Clinical laboratory values and amino acid serum levels were monitored. **RESULTS:** Glycine treatment was well tolerated and induced increased glycine ($P=.001$) and serine ($P=.001$) serum levels. Glycine administration resulted in (1) a significant ($P<.001$) 30% \pm 16% reduction in negative symptoms, as measured by the PANSS, and (2) a significant ($P<.001$) 30% \pm 18% improvement in the BPRS total scores. The improvement in negative symptoms was unrelated to alterations in extrapyramidal effects or symptoms of depression. Low pretreatment glycine serum levels significantly predicted ($r=0.80$) clinical response. **CONCLUSION:** These findings support hypoglutamatergic hypotheses of schizophrenia and suggest a novel approach for the pharmacotherapy of negative symptoms associated with this illness.

Abstracts

Recently Published Abstracts

Diet and skin disease in dogs and cats.

Watson TD. *J Nutr*
1998;128:2783S-2789S.

Dietary factors have a major role in the maintenance of healthy coat and skin, and are significant in the etiology and therapy of certain skin diseases. Nutritional deficiencies are now uncommon as a result of the widespread feeding of complete and balanced pet foods. Deficiencies of (n-6) polyunsaturated fatty acids, zinc and vitamins, however, do arise in certain animal- or product-related instances. Supraphysiologic doses of vitamin A have been used in the management of vitamin A-responsive dermatosis in Cocker spaniels; other keratinization defects and seborrheic conditions may respond to retinoid therapy. Much interest has been paid to the therapeutic value of polyunsaturated fatty acid supplements in the management of dermatologic conditions associated with hypersensitivity reactions or keratinization defects. These studies have generally yielded disappointing results, which may reflect shortcomings in the design of some trials. Nevertheless, a placebo-controlled, double-blind, cross-over study has demonstrated a clear benefit of high dose (n-3) fatty acids in the management of pruritic skin disease. There is also preliminary experimental evidence that specific dietary (n-6):(n-3) fatty ratios are useful in the dietary management of inflammatory diseases. Although results of controlled clinical trials are awaited, the argument exists that it is the absolute amount of (n-3) fatty acid intake rather than ratio that is responsible for potential health benefits.

Diabetes mellitus and serum carotenoids: findings from the Third National Health and Nutrition Examination Survey.

Ford ES, Will JC, Bowman BA, Narayan KM. *Am J Epidemiol* 1999;149:168-176.

Little is known about carotenoids, a diverse group of plant compounds with antioxidant activity, and their association with diabetes, a condition characterized by oxidative stress. Data from phase I of the Third National Health and Nutrition Examination Survey (1988-1991) were used to examine concentrations of alpha-carotene, beta-carotene, cryptoxanthin, lutein/zeaxanthin, and lycopene in 40- to 74-year-old persons with a normal glucose tolerance (n = 1,010), impaired glucose tolerance (n = 277), newly diagnosed diabetes (n = 148), and previously diagnosed diabetes (n = 230) based on World Health Organization criteria. After adjustment for age, sex, race, education, serum cotinine, serum cholesterol, body mass index, physical activity, alcohol consumption, vitamin use, and carotene and energy intake, geometric means of beta-carotene were 0.363, 0.316, and 0.290 micromol/liter for persons with a normal glucose tolerance, impaired glucose tolerance, and newly diagnosed diabetes, respectively (p = 0.004 for linear trend), and geometric means for serum lycopene were 0.277, 0.259, and 0.231 micromol/liter, respectively (p = 0.044 for linear trend). All serum carotenoids were inversely related to fasting serum insulin after adjustment for confounders (p < 0.05 for each carotenoid). If confirmed, these data suggest new opportunities for research that include exploring a possible role for carotenoids in the pathogenesis of insulin resistance and diabetes.

Abstracts

Recently Published Abstracts

Chronic pancreatitis at Manchester, UK. Focus on therapy.

McCloy R. *Digestion*
1998;59:S36-S48.

The Manchester 'oxidant stress' hypothesis for the development of pancreatitis accommodates published information on both chronic pancreatitis and acute pancreatitis. Oxidant stress, mainly from reactive xenobiotic metabolites, is perceived as the pivotal pre-morbid problem in chronic pancreatitis and, by depleting glutathione, targets the exocytosis mechanism of the pancreatic acinar cell. Inhalation exposure to petrochemical products is identified as an independent risk factor in patients at Manchester Royal Infirmary, where some 50% of patients referred have non-alcoholic disease. This paper describes the development of antioxidant therapy, using supplements of methionine, vitamin C and selenium, and its validation in a placebo-controlled trial, followed by a retrospective cross-sectional study in 94 consecutive patients for an average of 30 months. Antioxidant therapy emerges as a safe and effective medical alternative to surgery for painful chronic pancreatitis.

Interventional nutrition for cardiac disease.

Freeman LM. *Clin Tech Small Anim Pract*
1998;13:232-237.

Animals with cardiac disease can have a variety of nutritional alterations for which interventional nutrition can be beneficial. Deviation from optimal body weight, both obesity and cachexia, is a common problem in cardiac patients and adversely affects the animal. Methods for maintaining optimal weight are important for good quality of life in dogs and cats with cardiac disease. Providing proper diets to prevent excess intake of sodium and chloride also is important, but severe salt restriction may not be necessary until later stages of disease. Certain nutrient deficiencies may play a role in the pathogenesis or complications of cardiac disease, but nutrients also may have effects on cardiac disease which are above and beyond their nutritional effects (nutritional pharmacology). Supplementation of nutrients such as taurine, carnitine, coenzyme Q10, and omega-3 polyunsaturated fatty acids may have benefits in dogs or cats with cardiac disease through a number of different mechanisms. By addressing each of these areas maintaining optimal weight, avoiding nutritional deficiencies and excesses, and providing the benefits of nutritional pharmacology, optimal patient management can be achieved.

Vitamin E and vitamin C supplement use and risk of incident Alzheimer disease.

Morris MC, Beckett LA, Scherr PA, et al. *Alzheimer Dis Assoc Disord* 1998;12:121-126.

Oxidative stress may play a role in neurologic disease. The present study examined the relation between use of vitamin E and vitamin C and incident Alzheimer disease in a prospective study of 633 persons 65 years and older. A stratified random sample was selected from a disease-free population. At baseline, all vitamin supplements taken in the previous 2 weeks were identified by direct inspection. After an average follow-up period of 4.3 years, 91 of the sample participants with vitamin information met accepted criteria for the clinical diagnosis of Alzheimer disease. None of the 27 vitamin E supplement users had Alzheimer disease compared with 3.9 predicted based on the crude observed incidence among nonusers ($p = 0.04$) and 2.5 predicted based on age, sex, years of education, and length of follow-up interval ($p = 0.23$). None of the 23 vitamin C supplement users had Alzheimer disease compared with 3.3 predicted based on the crude observed incidence among nonusers ($p = 0.10$) and 3.2 predicted adjusted for age, sex, education, and follow-up interval ($p = 0.04$). There was no relation between Alzheimer disease and use of multivitamins. These data suggest that use of the higher-dose vitamin E and vitamin C supplements may lower the risk of Alzheimer disease.

Changes in thyroid hormone concentrations after administration of ashwagandha root extract to adult male mice.

Panda S, Kar A. *J Pharm Pharmacol* 1998;50:1065-1068.

The importance of ashwagandha root extract in the regulation of thyroid function with special reference to type-I iodothyronine 5'-monodeiodinase activity in mice liver has been investigated. Although the root extract (1.4 g kg⁻¹) administered daily for 20 days by gastric intubation increased serum 3,3',5'-triiodothyronine (T3) and tetraiodothyronine (T4) concentrations and hepatic glucose-6-phosphatase activity, hepatic iodothyronine 5'-monodeiodinase activity did not change significantly. Furthermore, ashwagandha root extract significantly reduced hepatic lipid peroxidation, whereas the activity of antioxidant enzymes such as superoxide dismutase and catalase were increased. These findings reveal that the ashwagandha root extract stimulates thyroidal activity and also enhances the antiperoxidation of hepatic tissue.

Dehydroepiandrosterone: an inexpensive steroid hormone that decreases the mortality due to sepsis following trauma-induced hemorrhage.

Angele MK, Catania RA, Ayala A, et al. *Arch Surg* 1998;133:1281-1288.

BACKGROUND: Recent studies suggest that male sex steroids play a role in producing immunodepression following trauma-hemorrhage. This notion is supported by studies showing that castration of male mice before trauma-hemorrhage or the administration of the androgen receptor blocker flutamide following trauma-hemorrhage in noncastrated animals prevents immunodepression and improves the survival rate of animals subjected to subsequent sepsis. However, it remains unknown whether the most abundant steroid hormone, dehydroepiandrosterone (DHEA), protects or depresses immune functions following trauma-hemorrhage. In this regard, DHEA has been reported to have estrogenic and androgenic properties, depending on the hormonal milieu. **OBJECTIVE:** To determine whether administration of DHEA after trauma-hemorrhage has any salutary or deleterious effects on immune responses, and whether it improves the survival of animals subjected to subsequent sepsis. **DESIGN:** Male C3H/HeN mice underwent laparotomy (ie, trauma-induced) and hemorrhagic shock (blood pressure, 35±5 mm Hg for 90 minutes) followed by fluid resuscitation, or sham operation. The animals then received 100 mg of DHEA (4 mg/kg) or propylene glycol (hereafter referred to as vehicle). At 24 hours after trauma-hemorrhage and resuscitation, the animals were killed and blood, spleens, and peritoneal macrophages were harvested. Splenocyte proliferation and interleukin (IL) 2 release and splenic and peritoneal macrophage IL-1 and IL-6 release were determined. In a separate set of experiments, sepsis was induced by cecal ligation and puncture at 48 hours after trauma-hemorrhage and resuscitation. For those studies, the animals received vehicle, a single 100-microg dose of DHEA, or 100 microg/d DHEA for 3 days following hemorrhage and resuscitation. Survival was monitored for 10 days after the induction of sepsis. **RESULTS:** Administration of DHEA restored the depressed splenocyte and macrophage functions at 24 hours after trauma-hemorrhage. Moreover, daily administration of DHEA for 3 days significantly increased the survival of animals subjected to subsequent sepsis ($P=.01$). **CONCLUSION:** The finding that DHEA markedly improves the depressed immune functions and survival of animals subjected to subsequent sepsis suggests that short-term treatment with DHEA after trauma-hemorrhage is a safe and novel approach for preventing immunodepression and for decreasing the mortality rate due to subsequent sepsis.

Selenium depletion in patients with gastrointestinal diseases: are there any predictive factors?

Rannem T, Ladefoged K, Hylander E, et al. *Scand J Gastroenterol* 1998;33:1057-1061.

BACKGROUND: Patients with intestinal disease are at risk of developing selenium deficiency due to impaired intestinal absorption. The aim of the present study was to evaluate selenium status and to identify predictive factors of selenium depletion in patients with gastrointestinal disease. **METHODS:** The concentration of selenium and the activity of glutathione peroxidase in plasma and erythrocytes were measured by fluorometry and by spectrophotometry. Eighty-six patients with Crohn's disease, 40 patients with ulcerative colitis, and 39 patients with various other gastrointestinal diseases were studied. Twenty-seven patients (16%) received home parenteral nutrition. Stool mass, faecal fat, and vitamin B12 absorption were analysed in 100 patients. **RESULTS:** The plasma selenium concentration was decreased in 85% of the patients receiving supplementary parenteral nutrition and in 20% of the patients receiving oral nutrition, among them in 26% of the patients with Crohn's disease. Almost all patients with ulcerative colitis had normal selenium levels. A statistically significant correlation was found between plasma selenium and vitamin B12 absorption, stool mass, faecal fat excretion, body mass index, P-albumin, P-zinc, and the length of the remaining small bowel. Stepwise regression analyses showed that the strongest predictors of selenium deficiency were stool mass, vitamin B12 absorption, and the length of the small-bowel resection. **CONCLUSION:** Selenium deficiency is common in patients with severe gastrointestinal disorders. The deficiency is mainly related to malabsorption, and a low selenium level was almost invariably present in patients who needed parenteral supplementation due to gut failure.

Effect of selenium deficiency on cardiac function of individuals with severe disabilities under long-term tube feeding.

Saito Y, Hashimoto T, Sasaki M, et al. *Dev Med Child Neurol* 1998;40:743-748.

Eleven orally fed (group A) and 14 tube-fed patients (group B) with severe disabilities and neurological disorders were compared to determine whether selenium (Se) deficiency is present in patients undergoing long-term tube feeding, and to examine if Se supplementation has beneficial effects on their cardiac function. Assessments of Se intake, serum levels of Se, and cardiac-function analysis including chest X-ray, electrocardiography (ECG), and M-mode echocardiography (UCG) were made. For group B, the effect of Se supplementation on cardiac function was assessed. Statistical significance was determined using Mann-Whitney U test and Wilcoxon signed-rank test. Deficiencies of Se were found in group B. ECG abnormalities were more common in group B than group A. UCG showed a lower ejection fraction, a lower mean rate of circumferential fiber shortening corrected by heart rate, and a lower diastolic velocity of the left-ventricular posterior walls in group B than in group A. In group B, Se supplementation resulted in normalized serum Se levels, partial improvement of ECG abnormalities, and an increase of cardiac functions on UCG. It is thought that Se has beneficial effects on the myocardium of chronically tube-fed patients. Se supplementation is recommended in this population.

Chemoprevention of colorectal cancer in inflammatory bowel disease? A potential role for folate.

Mouzas IA, Papavassiliou E, Koutroubakis I. *Ital J Gastroenterol Hepatol* 1998;30:421-425.

Patients with ulcerative colitis have an increased risk for developing colon cancer compared to the general population. The risk is related to the extension of the disease and its duration. This risk is the same for Crohn's colitis patients of equal extension and duration. By chemoprevention we mean the use of specific natural or synthetic chemical agents to reverse, suppress or prevent progression to invasive cancer. The chemopreventive agents for colon cancer are either of natural origin (vitamins, minerals, food constituents) or synthetic chemicals (difluoromethyl ornithine) and pharmaceutical agents (aspirin, oltipraz). Apart from folate, no other agent has so far been used in vivo for the prevention of colon cancer in long-standing inflammatory bowel disease. The use of folate was, however, not primarily intended to prevent cancer but to enhance folate absorption in ulcerative colitis. From retrospective studies, within the framework of cancer surveillance programmes, it became evident that folate supplementation may play a positive role as a chemopreventive agent against colorectal cancer in patients with long-standing, extensive ulcerative colitis. There is also evidence suggesting that folate supplementation may contribute to regulation of rectal cell proliferation in ulcerative colitis patients. There is a real need for multicentre, randomized, prospective clinical studies in order to evaluate the promising role of folate in preventing colorectal cancer in patients with long-standing inflammatory bowel disease.

A new mixed micellar preparation for oral vitamin K prophylaxis: randomised controlled comparison with an intramuscular formulation in breast fed infants.

Greer FR, Marshall SP, Severson RR, et al. *Arch Dis Child* 1998;79:300-305.

OBJECTIVE: To compare a new oral preparation of vitamin K1 (Konakion MM) containing lecithin and glycocholic acid with a standard intramuscular (IM) preparation during the first 8 weeks of life in exclusively breast fed infants. **METHODS:** Infants were randomised at birth to the IM group (1 mg vitamin K) or the oral group (2 mg given at birth and repeated at 7 and 30 days of life). Prothrombin time (INR), plasma vitamin K1, and PIVKA II (undercarboxylated prothrombin) were monitored at 14, 30, and 56 days of age. **RESULTS:** Seventy nine infants were randomised to the oral group and 77 to the IM group. Sixty seven infants in each group completed eight weeks of the study. Prothrombin times did not differ between the two groups. Mean (SD) plasma vitamin K1 values (in ng/ml) decreased in both groups over time, but were higher in the oral group at 14 and 56 days: 2.0 (1.6) v 1.3 (1.1) at 14 days; 0.5 (0.3) v 0.5 (0.7) at 30 days; and 0.5 (0.8) v 0.2 (0.2) at 56 days of life. PIVKA II was raised (\geq 0.1 AU/ml) in cord blood in 47% of the infants. By 14 days, only one infant in each group had a raised PIVKA II value and both of these initially had high concentrations of PIVKA II in cord blood. At 30 days, there were no raised PIVKA II values. At 56 days, there were no raised PIVKA II values in the oral group, although three infants in the IM group had raised values. **CONCLUSIONS:** Plasma vitamin K concentrations were at least equal or significantly higher in babies given oral vitamin K supplements compared with IM treated babies at the time points measured. Through the first 8 weeks of life, multiple doses of the new oral preparation maintain haemostasis and vitamin K status in breast fed infants at least equal to that of the intramuscular preparation.

Reversal of defective nerve conduction with vitamin E supplementation in type 2 diabetes: a preliminary study.

Tutuncu NB, Bayraktar M, Varli K. *Diabetes Care* 1998;21:1915-1918.

OBJECTIVE: The present study has examined the effect of vitamin E, the principal modulator of free radical activity, on electrophysiological parameters in patients with diabetic peripheral sensorimotor polyneuropathy, matched for duration of disease and metabolic control. **RESEARCH DESIGN AND METHODS:** A total of 21 subjects with type 2 diabetes were enrolled in this double-blind randomized placebo-controlled study (vitamin E, 11 patients; placebo, 10 patients). Patients were randomly assigned to receive either 900 mg vitamin E or placebo for 6 months. The average dietary vitamin E consumption of the subjects was similar during the study. The main outcome measure was the electrophysiological tests assessing nerve conduction. Fasting plasma glucose, HbA1c, postprandial plasma glucose, and electrophysiological parameters in the basal state and after 6 months of treatment were studied. **RESULTS:** Glycemic indexes did not show any significant changes during the study, whereas nerve conduction improved significantly in 2 of the 12 studied electrophysiological parameters after 6 months in patients on vitamin E supplementation. The changes in the electrophysiological parameters were obvious in the median motor nerve fibers and tibial motor nerve fibers. Nerve conduction velocity in the median motor nerve fibers ($P = 0.0019$) and tibial motor nerve distal latency ($P = 0.0284$) improved significantly after 6 months of vitamin E supplementation. **CONCLUSIONS:** This study shows that defective nerve conduction in diabetic subjects with mild-to-moderate peripheral neuropathy may be improved by pharmacological doses of vitamin E supplementation. Further studies with a larger number of patients for longer periods of time are needed.

Abstracts

Recently Published Abstracts

Enhancement of wound healing by curcumin in animals.

Sidhu GS, Singh AK, Thaloor D, et al. *Wound Repair Regen* 1998;6:167-177.

Tissue repair and wound healing are complex processes that involve inflammation, granulation, and remodeling of the tissue. In this study, we evaluated the in vivo effects of curcumin (diferuloylmethane), a natural product obtained from the rhizomes of *Curcuma longa* on wound healing in rats and guinea pigs. We observed faster wound closure of punch wounds in curcumin-treated animals in comparison with untreated controls. Biopsies of the wound showed reepithelialization of the epidermis and increased migration of various cells including myofibroblasts, fibroblasts, and macrophages in the wound bed. Multiple areas within the dermis showed extensive neovascularization, and Masson's Trichrome staining showed greater collagen deposition in curcumin-treated wounds. Immunohistochemical localization of transforming growth factor-beta1 showed an increase in curcumin-treated wounds as compared with untreated wounds. In situ hybridization and polymerase chain reaction analysis also showed an increase in the mRNA transcripts of transforming growth factor-beta1 and fibronectin in curcumin-treated wounds. Because transforming growth factor-beta1 is known to enhance wound healing, it may be possible that transforming growth factor-beta1 plays an important role in the enhancement of wound healing by curcumin.

Prevention of precancerous colonic lesions in rats by soy flakes, soy flour, genistein, and calcium.

Thiagarajan DG, Bennink MR, Bourquin LD, Kavas FA. *Am J Clin Nutr* 1998;68:1394S-1399S.

The main purpose of this research was to determine whether diets containing soy products would inhibit the early stages of azoxymethane-induced colon cancer in F344 rats. Additional objectives were to determine whether feeding starch instead of sucrose, feeding additional calcium (0.5% compared with 0.1%), or feeding a low-fiber powdered enteral formula would influence early colon carcinogenesis. Colon cancer was initiated with 2 injections of azoxymethane (15 mg/kg body wt) and a 12-wk dietary treatment period was started 1 wk after the second injection. Precancerous colon lesions were assessed as foci with aberrant crypts (FAC). The mean numbers of FAC were 133 [soy concentrate (low concentration of phytochemicals)], 111 (starch substituted for sucrose), 98 [full-fat soy flakes (whole soybeans)], 87 (defatted soy flour), 77 (0.015% genistein), and 70 (0.5% Ca). The soy flour and full-fat soy flake diets contained 0.049% genistein derivatives (primarily glycosides), but were less effective in inhibiting the formation of FAC than the diet containing 0.015% genistein (as the aglycone). Eating soybeans and soy flour may reduce the early stages of colon cancer.

Reduced serum dehydroepiandrosterone levels in diabetic patients with hyperinsulinaemia.

Yamaguchi Y, Tanaka S, Yamakawa T, et al. *Clin Endocrinol (Oxf)* 1998;49:377-383.

OBJECTIVE: To elucidate the interaction between insulin and dehydroepiandrosterone (DHEA) concentrations, we evaluated serum DHEA and DHEA-sulphate (DHEA-S) levels in diabetic patients with hyperinsulinaemia. **PATIENTS AND DESIGN:** Twenty-four subjects with non-insulin dependent diabetes mellitus, 12 hyperinsulinaemic subjects (fasting serum insulin concentrations ≥ 10 mU/ml (71.8 pmol/l)) and 12 non-hyperinsulinaemic subjects, and 10 normal control subjects were studied. Serum DHEA, DHEA-S, cortisol and ACTH levels were investigated in these subjects. Moreover, their serum DHEA levels were compared during hyperinsulinaemic-euglycaemic clamp and after ACTH stimulation. **MEASUREMENTS:** Serum insulin, cortisol, ACTH, DHEA and DHEA-S concentrations were evaluated by RIA. Serum glucose was determined by the glucose oxidase method. **RESULTS:** Diabetic patients with hyperinsulinaemia showed significantly lower levels of serum DHEA and DHEA-S than controls. After ACTH stimulation, these patients also showed significantly lower DHEA levels. During the hyperinsulinaemic-euglycaemic clamp, serum DHEA concentrations of diabetic patients with hyperinsulinaemia remained low and did not decline further, although those of control subjects and non-hyperinsulinaemic diabetic patients showed a significant decline of serum DHEA levels. Even after ACTH stimulation during the clamp, serum DHEA in hyperinsulinaemic patients was still significantly lower than in controls. **CONCLUSIONS:** In diabetic patients with hyperinsulinaemia, baseline DHEA levels are chronically and maximally suppressed compared to control subjects and non-hyperinsulinaemic diabetic patients, and thus not decreased further by exogenous insulin infusion during hyperinsulinaemic-euglycaemic clamp

Immune function in aged women is improved by ingestion of vitamins C and E.

de la Fuente M, Ferrandez MD, Burgos MS, et al. *Can J Physiol Pharmacol* 1998;76:373-380.

We have investigated the effects of supplementation of the diet with the antioxidant vitamins C and E on several functions of the immune response of aged women. Ten healthy women and 20 women (72 +/- 6 years old) suffering two diseases often associated with age (10 with major depression disorders, MDD, and 10 with coronary heart disease, CHD) were administered 1 g of vitamin C and 200 mg of vitamin E daily for 16 weeks. Blood samples were collected before and after treatment for measurement of several immunological functions, namely proliferative response of lymphocytes to the mitogen phytohemagglutinin (20 mg/L) and phagocytic functions of polymorphonuclear (PMN) neutrophils, i.e., adherence to vascular endothelium, chemotaxis, phagocytosis of latex beads, and superoxide anion production. In addition, we also determined the levels of serum cortisol and lipid peroxides. Intake of vitamins resulted in a significant increase in the lymphoproliferative capacity and in the phagocytic functions of PMN neutrophils as well as in a significant decrease of serum levels of lipid peroxides and cortisol, both in the healthy aged women and in the aged women with MDD or CHD. These findings suggest an important role of antioxidant supplementation in the improvement of immune function in aged females as well as in the prevention and treatment of specific diseases associated with age that are quite prevalent in the developed countries.

Abstracts

Recently Published Abstracts

Vitamin E (alpha-tocopherol) in the treatment of tardive dyskinesia: a statistical meta-analysis.

Barak Y, Swartz M, Shamir E, et al. *Ann Clin Psychiatry* 1998;10:101-105.

Tardive dyskinesia is an involuntary movement disorder developing following treatment with neuroleptics. As many as 50% of chronic psychotic patients develop this disabling condition. No treatment has been found effective for tardive dyskinesia. This study was undertaken to meta-analyze the effects of vitamin E (alpha-tocopherol) reported in the last decade. All studies published since 1987, focusing on vitamin E and tardive dyskinesia are reviewed. Double-blind studies are analyzed using measures of effect and variance as described by secondary analysis of magnitude of effects in pooled data. A total of 223 patients received vitamin E treatment (400-1600 IU/day) for tardive dyskinesia, in 12 studies. A significant subgroup (28.3%) showed a modest improvement. Vitamin E was well tolerated, and only rarely did side effects occur-of no clinical significance. Vitamin E is a safe, well-tolerated compound that may provide some beneficial effects in patients suffering from neuroleptic-induced tardive dyskinesia.

Metabolic changes in patients with mitochondrial myopathies and effects of coenzyme Q10 therapy.

Chan A, Reichmann H, Kogel A, et al. *J Neurol* 1998;245:681-685.

We used a standardized bicycle ergometry protocol with a stepwise increasing workload (30-100 W) to evaluate various metabolic factors for the diagnosis and metabolic monitoring of mitochondrial encephalomyopathies. All patients (n = 9) showed pathological venous lactate/pyruvate (L/P) ratios, which normalized in three patients after 6 months of coenzyme Q10 (CoQ) therapy. Thus, the L/P ratio proved to be the clinically most useful parameter in the evaluation and monitoring of mitochondrial diseases, showing higher sensitivity than lactate measurements only. CoQ may exert a favourable effect in some patients with mitochondrial diseases.

Protective action of plant polyphenols on radiation-induced chromatid breaks in cultured human cells.

Parshad R, Sanford KK, Price FM, et al. *Anticancer Res* 1998;5A:3263-3266.

The present study was performed to determine whether plant polyphenols can protect human cells against radiation-induced DNA damage manifested as chromatid breaks. Since each chromatid contains a single continuous molecule of double stranded DNA, chromatid breaks represent unrepaired DNA strand breaks. The addition of green or black tea extracts, their polyphenols or curcumin to cultures of human skin fibroblasts or PHA-stimulated blood lymphocytes significantly reduced the frequencies of radiation-induced chromatid breaks. An exception to this general finding was that the green tea polyphenol, (-)epigallocatechin gallate, had no effect. The protective action of these plant polyphenols seems to result from their known antioxidant properties, particularly the scavaging of hydroxyl free radicals. Frequencies of chromatid breaks in cells arrested immediately after irradiation or 0.5 to 1.5 hours post-irradiation in the presence or absence of a DNA repair inhibitor, provide a measure of DNA damage. The results of the present study show that tea and other plant polyphenols can protect human cells against radiation-induced DNA damage.