Efficacy of vitamin E in the conservative treatment of Peyronie’s disease: legend or reality?
A controlled study of 70 cases

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SUMMARY
The medical treatment is indicated in the development stage of Peyronie’s disease (PD) for at least 1 year after diagnosis and whenever in case of penile pain. This research was conducted to demonstrate the possible effectiveness of vitamin E in PD treatment, whereas in the scientific literature this topic is much discussed. A total of 70 patients (age:26–69 years, mean: 54.1 ± 9.71) diagnosed with PD were enrolled in a conservative treatment. In addition to medical histories and physical examinations all patients underwent the following tests: International Index of Erectile Function (IIEF) questionnaire, penile ultrasound and photographic documentation, pain evaluation by a conventional 10-point pain scale Visual analogue pain scale (VAS). All 70 patients were divided into two different treatment groups: A and B, with different combinations of drugs: A = vitamin E + verapamil (injection + iontophoresis) + blueberries + propolis + topical diclofenac; B = verapamil (injection + iontophoresis) + blueberries + propolis + topical diclofenac.

All patients were treated for 6 months after which they underwent the same follow-up tests as performed prior to the treatment. Intergroup analysis revealed statistically significant differences: in the vitamin E group the effective plaque size reduction was _50.2% whereas in the control group the reduction was _35.8% (p = 0.027). In group A the improvement of curvature occurred in 96.6% of the cases whereas in the control group B this occurred in 48.4% (p = 0.0001), moreover, the mean curvature decrease was respectively _12.25° and _6.73° (p = 0.01). IIEF score was significantly improved in group A patients with comorbidities and erectile dysfunction (p = 0.025). Increase in plaque size occurred only in the control group (17.1%) (p = 0.032). We can affirm that vitamin E can help to prevent the progression of PD. This study strongly supports the recommendation that the best approach for treating PD is multimodal therapy.

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Protective Effects of Haematococcus Astaxanthin on Oxidative Stress in Healthy Smokers

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Abstract
Free radicals induced by cigarette smoking have been strongly linked to increased oxidative stress in vivo, contributing to the pathobiology of various diseases. This study was performed to investigate the effects of Haematococcus astaxanthin (ASX), which has been known to be a potent antioxidant, on oxidative stress in smokers. Thirty-nine heavy smokers (‡ 20 cigarettes/day) and 39 non-smokers were enrolled in this study. Smokers were randomly divided into three dosage groups to receive ASX at doses of 5, 20, or 40 mg (n = 13, each) once daily for 3 weeks. Oxidative stress biomarkers such as malondialdehyde, isoprostane, supe-
Roxide dismutase, and total antioxidant capacity, and ASX levels in plasma were measured at baseline and after 1, 2, and 3 weeks of treatment. Compared with baseline, the plasma malondialdehyde and isoprostane levels decreased, whereas superoxide dismutase level and total antioxidant capacity increased in all ASX intervention groups over the 3-week period. In particular, isoprostane levels showed a significant dose-dependent decrease after ASX intake. The results suggest that ASX supplementation might prevent oxidative damage in smokers by suppressing lipid peroxidation and stimulating the activity of the antioxidant system in smokers.

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Astaxanthin inhibits inflammation and fibrosis in the liver and adipose tissue of mouse models of diet-induced obesity and nonalcoholic steatohepatitis

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Abstract

The objective of this study was to determine if astaxanthin (ASTX), a xanthophyll carotenoid, can prevent obesity-associated metabolic abnormalities, inflammation and fibrosis in diet-induced obesity (DIO) and nonalcoholic steatohepatitis (NASH) mouse models. Male C57BL/6J mice were fed a low-fat (6% fat, w/w), a high-fat/high-sucrose control (HF/HS; 35% fat, 35% sucrose, w/w), or a HF/HS containing ASTX (AHF/HS; 0.03% ASTX, w/w) for 30 weeks. To induce NASH, another set of mice was fed a HF/HS diet containing 2% cholesterol (HF/HS/HC) a HF/HS/HC with 0.015% ASTX (AHF/HS/HC) for 18 weeks. Compared to LF, HF/HS significantly increased plasma total cholesterol, triglyceride and glucose, which were lowered by ASTX. ASTX decreased hepatic mRNA levels of markers of macrophages and fibrosis in both models. The effect of ASTX was more prominent in NASH than DIO mice. In epididymal fat, ASTX also decreased macrophage infiltration and M1 macrophage marker expression, and inhibited hypoxia-inducible factor 1-α and its downstream fibrogenic genes in both mouse models. ASTX significantly decreased tumor necrosis factor α mRNA in the splenocytes from DIO mice upon lipopolysaccharides stimulation compared with those from control mice fed an HF/HS diet. Additionally, ASTX significantly elevated the levels of genes that regulate fatty acid β-oxidation and mitochondrial biogenesis in the skeletal muscle compared with control obese mice, whereas no differences were noted in adipose lipogenic genes. Our results indicate that ASTX inhibits inflammation and fibrosis in the liver and adipose tissue and enhances the skeletal muscle’s capacity for mitochondrial fatty acid oxidation in obese mice.