capacity (31 ± 14% vs. 40 ± 14%, p = 0.02).We found a statistically no significant difference for the other functional features or HDL properties between the groups. Conclusions: Small HDL particles, low HDL phospholipid content and decreased cholesterol efflux capacity were related to magnitude of subclinical atherosclerosis and hypoalphalipoproteinemia in a primary care population.

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A40880

Peri/epicellular protein disulfide isomerase reshapes vascular architecture to counteracts constrictive remodeling

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Whole-vessel remodeling critically determines lumen caliber in vascular (patho)physiology and is reportedly redox-dependent. We hypothesized that cell-surface pool of the endoplasmic reticulum (ER) redox chaperone Protein Disulfide Isomerase-A1 (peri/epicellular = pecPDI), known to support thrombosis, also regulates disease-associated vascular architecture. In human coronary atheromas, PDI expression inversely correlated with constrictive remodeling and plaque stability. In rabbit iliac artery overdistension model, there was unusually high PDI upregulation (~25-fold vs. basal 14 days post-injury), involving both intracellular and pecPDI. Silencing PDI by siRNA in vitro induced ER stress markers upregulation and apoptosis (assessed by TUNEL assay). PDI knockdown also upregulated proliferation marker PCNA and decreased differentiation marker calponin-C. In contrast, pecPDI neutralization with anti-PDI antibodies (PDIAb) did not enhance ER stress or apoptosis. In vivo pecPDI neutralization with PDIAb-containing perivascular gel from days 12–14 post-injury promoted ca. 25% decrease in maximally dilated arteriographic vascular caliber and corresponding whole-vessel circumference loss at optical coherence tomography, without changing neointima, suggesting constrictive remodeling. This was accompanied by decreased oxidant generation and nitrogen oxide production. Constrictive remodeling was corroborated by marked changes in collagen organization, switching from circumferential to radial fiber orientation and to more rigid fiber type. Cytoskeleton architecture was also disrupted, with loss of stress fiber coherent organization and switch from thin to medium-thickness actin fibers, all leading to impaired viscoelastic ductility. Total and PDI-associated expressions of beta1-integrin, as well as cell-surface reduced beta1-integrin levels, were diminished after PDIAb treatment, implicating beta1-integrin as a likely pecPDI target during vessel repair. Integrin signaling is a master regulator of mechanobiology connecting the extracellular matrix environment to focal adhesion and actin-cytoskeleton. Indeed, FAK phosphorylation, a downstream beta1-integrin effector, was decreased by PDIAb. Thus, PDI is highly upregulated after injury and reshapes matrix and cytoskeleton architecture to support an anticonstrictive remodeling effect. Such findings suggest an important role for PDI in lumen maintenance during vascular remodeling by regulation of mechano-adaptive mechanisms.

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A40897

Biochemical and histopathological parameters analyzed in rabbits fed a diet enriched with fat/sucrose/cholesterol and treated with vitamin D

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Introduction: Low levels of Vitamin D increase risk of cardiovascular diseases. This study aims to assess the effects of vitamin D in an experimental model of rabbits fed a diet enriched with fat/sucrose/cholesterol (FSC). Methods: The rabbits were fed a chow enriched with fat 10%/sucrose 40%/cholesterol 0.5% for three months and thereafter, the chow were substituted for FSC with cholesterol 0.1% or standard chow for more 3 months. The following groups were formed: I – FSC 0.5/0.1%; GII – FSC 0.5/0.1% + Vit D 1000 IU/day; GIII – FSC 0.5%/ standard chow (SC) and GIV – FSC 0.5%/ standard chow + 1000 IU of Vit D/day. In periods of 0, 3 and 6 months the following parameters were evaluated: weight, lipid profile and serum glucose. After 6 months the animals were euthanized and the aortas were removed for atheroma plaques analysis. Statistical analysis was performed by Kruskal–Wallis non-parametric tests followed by Dunn's test. Results: After 6 months, the following values were observed, respectively, in groups I, II, III and IV: total cholesterol (mg/dL):420 ± 128; 363 ± 119; 149 ± 136; 162 ± 132. Triglycerides(mg/dL): 172 ± 58; 202 ± 79; 74 ± 24; 63 ± 13. Glucose (mg/dL):97 ± 5; 92 ± 13; 100 ± 18; 106 ± 11. Plaques in aorta(%): 83 ± 24,62; 84,64 ± 23,22; 15,38 ± 5,95; 24,11 ± 8,81. Intima/media ratio in arch aorta: 2.98 ± 2.16; 2.57 ± 1.06; 2.74 ± 1.22; 1.35 ± 0.39*. Thoracic aorta: 2.13 ± 1.15; 1.80 ± 1.05; 1.54 ± 0.52; 1.48 ± 0.42. Abdominal aorta: 1.73 ± 0.55; 2.06 ± 1.23; 1.59 ± 0.41; 1.15 ± 0.47. The histopathological aspects of aortas in groups III and IV were more fibrous than in groups I and II, independently of vitamin D treatment, however, lower intima/media rate was observed in the group IV that had received the standard diet and vitamin D. Conclusion: The normalization of the diet improved the lipid profile and atherosclerotic plaques in aorta, with beneficial effect of vitamin D.

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A40924

Comparison of coenzyme Q10 and herbal *Withania somnifera* supplements on fatigue parameters and biochemical profile in dyslipidemic patients with statin in chronic use

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Introduction: The statin class of drugs is the first choice in dyslipidemic patients, to reduction of plasma cholesterol levels, especially LDL (low density lipoprotein) for the prevention of cardiovascular disease (CVD). However, fatigue is a side effect in some patients, possibly by inhibiting melovanato conversion to coenzyme Q10 (CoQ10), a cofactor in the electron transport chain for the energy formation, in the same cascade of cholesterol formation. CoQ10 supplementation helps reduce the effects caused by continuous use of statins. In traditional Chinese Medicine, the *Withania somnifera* (or Ashwagandha), is considered an adaptogen herbal and indicated for fatigue and hipocholesterolemic use. Methods: In this

study of case, a young patient with dyslipidemia in statin use only, with reports of chronic fatigue, was selected to receive supplementation of CoQ10 (100 mg) and *Withania somnifera* (300 mg dry extract), in capsules, blindly for 30 days each, in addition to answering validated questionnaire on fatigue (Piper Fatigue Scale-Revised [PFSR]), usual dietary recall and perform biochemical tests after each period. Results: Both supplementation shows improved their symptoms of fatigue reported (PFSR basal 69,1% versus CoQ10 50,0% and *Withania somnifera* 43,2%), but there was a change of biochemical parameters for total cholesterol (185 mg/dL to 313 mg/dL), high density lipoprotein (HDL) (43 mg/dL to 55 mg/dL), LDL (124 mg/dL to 226 mg/dL) and triglycerides (91 to 159 mg/dL) after use of *Withania somnifera*, in comparison with CoQ10, without alter in your habitual dietary. Conclusion: Therefore, the use of herbal medicine can be a viable alternative and effective supplementation in patients reporting fatigue, but further studies are needed for the population in continuous use of statins, to assess whether there is a hypocholesterolemic action of competition between them.

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A40934

Isolated hypercholesterolemia and consumer of cardioprotective foods

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Introduction: Diet is one of the changes in lifestyle recommended for dyslipidemic patients, such as prevention of cardiovascular events. Nutrition aims to stimulate the consumption of cardioprotective food, the functional bioactive compounds: fiber, omega 3–6–9 and polyphenols. Objective: Reduction of plasma levels of total cholesterol and fractions, in a hyperlipidemic patient without medication. Methodology: hyperlipidemic patient received an orientation cardioprotective foods (olive oil, yogurt, green tea, flaxseed, dark chocolate, fish, oats, soybeans and by-products, grape juice, nut, avocado), with the recommendation for daily consumption to reduce hypercholesterolemia, as the IV Guideline on Dyslipidemia. The inclusion in the food routine was free choice. A food frequency questionnaire of these foods and biochemical tests were obtained before and after a follow-up three months. We selected a woman of 44 years without use of medications in primary prevention. Results: Over the frequency of cardioprotective foods, the patient reported using in the initial evaluation, only olive oil and fish, in the recommendations. During treatment, the patient included, besides the two already earlier: yogurt, linseed, dark chocolate, oats, whole grape juice and oilseeds. Other foods (green tea, soybeans and by-products and avocado) were included, but at a lower frequency than the one suggested. The plasma levels decreased, in basal versus 90 days, 31.41% LDL-cholesterol, 8.19% HDL-cholesterol and 11.00% triglyceride. The anthropometric measurements were reduced too: weight (61.1 to 59.0 kg), body mass index (23.59 to 22.77 kg/m^2) and waist circumference (84.5 to 82.5 cm). Conclusion: A frequent use of cardioprotective foods, can reduce the plasma levels of LDL-cholesterol, with low interference in HDL-cholesterol.

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A40952

Cyclo-oxygenase gene expression in acute myocardial infarction: A case report

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Introduction: The cyclo oxygenase (COX) enzymes are directly involved in inflammation. Arachidonic acid is converted by COX to prostaglandins (PG). PG participates in platelet activation, vasoconstriction, gastrointestinal protection and bronchodilation. These processes show an important association of COX-2 levels with major adverse cardiovascular events. This effect suggests protection of COX-2 in individuals who do not have it inhibited artificially. The objective of this report is to demonstrate the association between high COX-2 expression during the course of a patient with acute myocardial infarction (AMI) undergoing coronarography. Case report: A 54 year-old female patient, African descent, hypertensive and former user of cocaine was admitted through the emergency room with chest pain and anterior wall ischemia in the EKG. About 45 days before admission, she had been treated with angioplasty with conventional stenting in the anterior descending artery. Serial blood samples of each 6 h were made to assess the gene expression of COX-2 and NF-kB activity. Our results showed a rise of COX-2 gene expression and NF-kB after AMI diagnosis and coronarography during time. Conclusion: Atherosclerosis is the major cause behind major adverse cardiovascular events. The COX-2 has increased its expression in symptomatic atherosclerotic plaques however plate regions have fat cells that have no expression, suggesting that the mechanism behind its regulation is more complex. Areas with higher expression of COX-2 with great macrophage activity are subject to instability and rupture, triggering the cascade of events culminating in necrosis. In contrast, blocking the COX-2 action aggravates ischemia because of its vasoconstrictor effect with consequent increase in myocardial oxygen consumption. The balance between the intensity of the inflammatory reaction and plaque instability seems to be the scales between the concentration of PG E2 which is pro instability of the plaque, and PG I2 a natural pro angiogenesis stimulator

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