ALTLIX™

Alcachofra diferenciada

**DESCRIÇÃO**

O Altilix™ é um extrato de uma subespécie diferenciada de alcachofra (*Cynara cardunculus* L. var. *altiis*), padronizado em 1% a 12% de ácido clorogênico e 2 a 4% de luteína.

**MECANISMO DE AÇÃO**

O Altilix™ aumenta a produção de bile e alcaliniza o intestino, melhorando a motilidade intestinal (ação colerética). A luteína, presente no Altilix™, atenua a atividade da HMG-Coa redutase e assim reduz a biossíntese de colesterol. Sua ação antioxidante hepatoprotetora é conferida pela redução da produção de fatores pro oxidantes como NRF-1 e NRF-2.

**INDICAÇÕES**

- Diurético e detoxificante;
- Hepatoprotetor;
- Colerético e anticolestático.

**DOSE USUAL**

Recomendação oral de 100 a 200mg de Altilix™ ao dia.

**SUGESTÕES DE FÓRMULAS**

<table>
<thead>
<tr>
<th>Fórmula</th>
<th>Quantidade</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Altilix™</strong></td>
<td>200mg</td>
</tr>
<tr>
<td><strong>Cacti-Nea™</strong></td>
<td>1 g</td>
</tr>
<tr>
<td>Sachê qsp.</td>
<td>1 dose</td>
</tr>
</tbody>
</table>

**Modo de uso:** dissolver 1 dose em 1 copo com água ou suco, 1 vez ao dia.

**Indicação:** detoxificação no gerenciamento do peso.

<table>
<thead>
<tr>
<th>Fórmula</th>
<th>Quantidade</th>
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<tbody>
<tr>
<td><strong>Altilix™</strong></td>
<td>200mg</td>
</tr>
<tr>
<td>Vitamina B12</td>
<td>1mg</td>
</tr>
<tr>
<td>NAC...</td>
<td>100mg</td>
</tr>
<tr>
<td>MSM...</td>
<td>200mg</td>
</tr>
<tr>
<td>Zinco...</td>
<td>10mg</td>
</tr>
</tbody>
</table>

**Modo de uso:** 1 dose ao dia.

**Indicação:** detoxificação completa.

**PRINCIPAIS REFERÊNCIAS**


Protective effects of luteolin-7-glucoside against liver injury caused by carbon tetrachloride in rats.

Ixeris chinensis (Thunb.) Nakai has been used as a Chinese folk medicine; the information on the physiological and biochemical functions of the compounds extracted from I. chinensis is still scanty. We investigated the effects of luteolin-7-glucoside (LUTG) isolated from I. chinensis against liver injury caused by carbon tetrachloride (CCl4). CCl4 significantly increased the enzyme activities of glutamic pyruvic transaminase (GPT) and glutamic oxaloacetic transaminase (GOT) in blood serum, as well as the level of malondialdehyde (MDA) and 8-hydroxydeoxyguanosine (8-OHdG) in liver tissue, and decreased the levels of reduced glutathione (GSH). Pretreatment with LUTG was not only able to suppress the elevation of GPT, GOT, MDA and 8-OHdG, and inhibit the reduction of GSH in a dose-dependent manner in vivo, but also reduce the damage of hepatocytes in vitro. On the other hand, we also found LUTG has strong antioxidant activity against reactive oxygen species (ROS) in vitro in a concentration-dependent manner. The hepatoprotective activity of LUTG was possibly due to its antioxidant properties, acting as scavengers of ROS. These results obtained in vivo and in vitro suggest that LUTG had protective effects against hepatic oxidative injury induced by chemicals. Further studies on the pharmaceutical functions and immunological responses of LUTG may help in the development of a clinical application.

The Effect of Ginger (Zingiber officinalis) and Artichoke (Cynara cardunculus) Extract Supplementation on Functional Dyspepsia: A Randomised, Double-Blind, and Placebo-Controlled Clinical Trial.

Objective. Functional dyspepsia (FD) is a frequent clinical finding in western world. The aim of this study is to compare the efficacy of a ginger and artichoke supplementation versus placebo in the treatment of FD. Methods. A prospective multicentre, double blind, randomized, placebo controlled, parallel-group comparison of the supplement and placebo over a period of 4 weeks was performed. Two capsules/day were supplied (before lunch and dinner) to 126 FD patients (supplementation/placebo: 65/61). Results. After 14 days of treatment, only supplementation group (SG) showed a significant amelioration (SG: α S = +1.195 MCA score units (u), P = 0.017; placebo: α P = +0.347 u, P = 0.513). The intercept (α) resulted to be significantly higher in SG than in placebo (α S - α P = +0.848 u, P < 0.001). At the end of the study, the advantage of SG versus placebo persists without variation (β S - β P = +0.077 u, P = 0.542). In SG, a significant advantage is observed for nausea (β S - β P = -0.398 u, P < 0.001), epigastric fullness (β S - β P = -0.241, P < 0.001), epigastric pain (β S - β P = -0.173 u, P = 0.002), and bloating (β S - β P = -0.167 u, P = 0.017). Conclusions. The association between ginger and artichoke leaf extracts appears safe and efficacious in the treatment of FD and could represent a promising treatment for this disease.

Anti-diabetic effects of luteolin and luteolin-7-O-glucoside on KK-A(y) mice.

Anti-diabetic potential of luteolin (LU) and luteolin-7-O-glucoside (LUG) were investigated in the amount of equimolar on KK-A(y) mice. The results showed that both of LU and LUG significantly improved blood glucose, HbA1c, insulin, and HOMR-IR levels. Anti-inflammatory and anti-oxidative effects of the LU and LUG were also proved. Furthermore, TGs in serum and liver were significantly decreased in the LU and LUG groups, as well as the mRNA expression of fat acid expression-related genes (SREBP-1c), compared to the basal diet group (CON). When compared the effects between the LU and LUG groups, TGs of the LU group were lower than those of the LUG group, accompanied with significantly decreased FAS activity and SREBP-1c expression in liver. These results suggested that both LU and LUG had positive effects of anti-diabetes on KK-A(y) mice, but LU more potently ameliorated diabetes than LUG, which might be attributed to the inhibitory of lipid synthesis.
Antigenotoxic effect of extract from *Cynara cardunculus* L.

The extract of artichoke *Cynara cardunculus* L. (CCE) was investigated for its potential antigenotoxic and antioxidant effects using four experimental model systems. In the Saccharomyces cerevisiae mutagenicity/antimutagenicity assay, CCE significantly reduced the frequency of 4-nitroquinoline-N-oxide-induced revertants at the ilv1 locus and mitotic gene convertants at the trp5 locus in the diploid Saccharomyces cerevisiae tester strain D7. In the simultaneous toxicity and clastogenicity/anticlastogenicity assay, it exerted an anticlastogenic effect against N-nitroso-N'-methylurea-induced clastogenicity in the plant species Vicia sativa L. On the contrary, despite CCE not being mutagenic itself, in the preincubation Ames assay with metabolic activation, it significantly increased the mutagenic effect of 2-aminofluorene in the bacterial strain Salmonella typhimurium TA98. In the 1,1-diphenyl-2-picrylhydrazyl (DPPH) free radical scavenging assay, CCE exhibited considerable antioxidant activity. The SC50 value representing 0.0054% CCE corresponds to an antioxidant activity of 216.8 microm ascorbic acid which was used as a reference compound. Although the mechanism of CCE action still remains to be elucidated, different possible mechanisms are probably involved in the CCE antigenotoxic effects. It could be concluded that CCE is of particular interest as a suitable candidate for an effective chemopreventive agent.

Protective effects of chlorogenic acid on acute hepatotoxicity induced by lipopolysaccharide in mice.

OBJECTIVE AND DESIGN: To investigate the potential protective effects of chlorogenic acid (CGA) on acute liver injury caused by lipopolysaccharide (LPS) in mice. MATERIALS AND METHODS: C57BL/6J mice were pretreated with CGA (50 mg/kg, intraperitoneally) once per day for 5 days before an overnight LPS challenge (30 mg/kg, intraperitoneally). Severity of liver injury was assessed by histological analysis and determination of serum ALT and AST levels. Expression and activation of key regulators involved in the inflammatory response were determined, respectively, by real-time RT-PCR and western blotting. RESULTS: In contrast to the yellow color of the liver in LPS-treated mice, CGA maintained the normal reddish appearance of the liver. Histological analysis indicated that CGA attenuated the infiltration of neutrophil cells and the necrosis of hepatocytes. CGA also decreased the elevated plasma levels of ALT and AST. At the transcriptional level, CGA pretreatment suppressed hepatic mRNA expression of toll-like receptor 4 (TLR4), TNF-alpha and NF-kappaB p65 subunit. In contrast, mRNA level of the transcriptional coactivator PGC-1alpha was restored by CGA. Finally, CGA reduced the phosphorylation of NF-kappaB p65 subunit in the liver. CONCLUSION: Our data suggest that CGA has remarkable hepatoprotective effects on LPS-induced liver injury and that the possible mechanism is related to its anti-inflammatory action.

Inhibition of cholesterol biosynthesis in primary cultured rat hepatocytes by artichoke (*Cynara scolymus* L.) extracts.

High-dose aqueous extracts from artichoke leaves were found to inhibit cholesterol biosynthesis from 14C-acetate in primary cultured rat hepatocytes in a concentration-dependent biphasic manner with moderate inhibition (approximately 20%) between 0.007 and 0.1 mg/ml and more strong inhibition at 1 mg/ml. Cytotoxic effects detected by lactate dehydrogenase leakage and the 3-[4, 5-dimethylthiazol-2-yl]-2,5-dephenyl tetrazolium bromide-assay were restricted to higher concentrations. Replacement of 14C-acetate by 14C-mevalonate largely omitted the inhibiting effect of artichoke extracts indicating an inhibition at the level of hydroxymethylglutaryl-CoA-reductase. However, no direct inhibition of this enzyme could be detected and no other enzymic steps later in the biosynthetic pathway for cholesterol seemed to be affected. Instead, inhibition was found to occur in a time-dependent manner, to last for several hours even after washing out the extracts by fresh medium and to be fully reversible within 20 hr after removal of the extracts. In addition, the stimulation of HMGCoA-reductase activity by insulin was efficiently blocked by the extracts, although other insulin-independent phenomena, such as increased lactate production, were not influenced. These results suggest an indirect modulation of hydroxymethylglutaryl-CoA-reductase activity as the most likely inhibitory mechanism of the artichoke extracts. Screening of several known constituents of artichoke extracts revealed that cynaroside and particularly its aglycone luteolin were mainly responsible for inhibition, whereas chlorogenic acid was much less effective and caffeic acid, cynarin and other dicaffeoylquinic acids were without significant influence. Indeed, luteolin also efficiently blocked the insulin effect on cholesterol biosynthesis. In conclusion, these results demonstrate that artichoke extracts may inhibit hepatic cholesterol biosynthesis in an indirect but efficient manner and, thus, may contribute via this action to the recently confirmed hypolipidemic influence of this phytopharmacon in man.
Metabolic control of mitochondrial biogenesis through the PGC-1 family regulatory network.

The PGC-1 family of regulated coactivators, consisting of PGC-1α, PGC-1β and PRC, plays a central role in a regulatory network governing the transcriptional control of mitochondrial biogenesis and respiratory function. These coactivators target multiple transcription factors including NRF-1, NRF-2 and the orphan nuclear hormone receptor, ERRα, among others. In addition, they themselves are the targets of coactivator and co-repressor complexes that regulate gene expression through chromatin remodeling. The expression of PGC-1 family members is modulated by extracellular signals controlling metabolism, differentiation or cell growth and in some cases their activities are known to be regulated by post-translational modification by the energy sensors, AMPK and SIRT1. Recent gene knockout and silencing studies of many members of the PGC-1 network have revealed phenotypes of wide ranging severity suggestive of complex compensatory interactions or broadly integrative functions that are not exclusive to mitochondrial biogenesis. The results point to a central role for the PGC-1 family in integrating mitochondrial biogenesis and energy production with many diverse cellular functions. This article is part of a Special Issue entitled: Mitochondria and Cardioprotection.

REFERÊNCIAS


