AOR CODE: AOR04004

Premium

**Curcumin-95**

**A Concentrated Extract of an Ancient Spice**
95% curcumin from turmeric

Traditionally used in Ayurveda

Natural antioxidant and joint protector

- Gluten Free
- Vegan
- Non-GMO
- Anti-Aging
- Antioxidant

**AOR Code**  | **Variant**
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AOR04004 | 90 VEGI-CAPS

**Details**
Turmeric root, a revered East Indian spice, contains several types of curcuminoids, of which curcumin is the most potent and clinically studied. Curcumin-95 is a curcuminoid extract standardized to 95% pure curcumin. Curcumin is a powerful antioxidant and anti-inflammatory and has been traditionally used to treat joint pain and various types of chronic pain, although it has many other benefits. It can help protect cells and prevent oxidation of cholesterol and cell membranes, has antibacterial, antifungal and antiviral effects, blocks abnormal cellular growth, promotes healthy cholesterol levels and supports cardiovascular health.

Curcumin-95 is best used by those looking for a great antioxidant in its most natural form that has not been altered by modern technologies. It is effective for joint pain and arthritis and beneficial for overall maintenance of good health. For an even more powerful anti-inflammatory, it is worth exploring Longvida Optimized Curcumin products which can be found here.

**Label Info**

**Discussion**
Curcumin is used in Herbal medicine to help relieve joint inflammation and provides antioxidants for the maintenance of good health.

**Product Variation**

**Product Code**  |  **Size**
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Supplements Facts

Serving Size: 1 Capsule

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Curcumin

Non-medical ingredients:
- potato starch, sodium stearyl fumarate. Capsule: hypromellose.

Guarantees

AOR™ guarantees that all ingredients have been declared on the label. Contains no wheat, gluten, corn, nuts, peanuts, sesame seeds, sulfites, mustard, soy, dairy, eggs, fish, shellfish or any animal byproduct.

Adult Dosage

Take 3 capsules daily with/without food, or as directed by a qualified health care practitioner.

Cautions

Consult a health care practitioner prior to use if you are pregnant, taking antiplatelet medication or blood thinners, have gallstones or a bile duct obstruction, have stomach ulcers or excess stomach acid, or if symptoms persist or worsen with use.

Source

Turmeric (Curcuma longa) rhizome

Main Application

- Anti-oxidant
- Anti-inflammatory
- Gastro-intestinal effects
- Cardiovascular effects
- Lipid metabolism
- Anti-bacterial/Anti-fungal
- Anti-viral
- Promotes normal cell activity
Disclaimer

The information and product descriptions appearing on this website are for information purposes only, and are not intended to provide or replace medical advice to individuals from a qualified health care professional. Consult with your physician if you have any health concerns, and before initiating any new diet, exercise, supplement, or other lifestyle changes.

Research

Background

*Curcuma longa* is a perennial herb that belongs to the ginger family. The rhizome is extensively used for imparting color and flavor to food including curries. As a powder, called turmeric, it is also used for medicinal and religious ceremonies. Curcumin is an extract of the herb turmeric (*Curcuma longa* Linn) which has wide-ranging health benefits. It is well known to have antioxidant effects, preventing cellular damage, and it reduces the production of inflammatory signals. It also promotes normal cell activity and growth.

Research

Pharmacological Properties of *Curcuma longa*

The benefits of *Curcuma longa* have been extensively researched, especially by Indian scientists.

1. Antioxidant Numerous studies have shown that the various constituents of *Curcuma longa* possess potent antioxidant properties. The ability of curcuminoids to reduce hydroxyl and peroxyl free radicals is well documented. Sharma reported curcumin to be an effective agent against lipid peroxidation.

2. Anti-inflammatory The Central Drug Research Institute in India found curcumin to be the major constituent responsible for the anti-inflammatory activity. The classical model for studying acute effects of anti-inflammatory agents is to test their inhibitory action on the development of rat paw edema - the exudative phase of inflammation - induced, for instance, by the local injection of carrageenan. Thus inflammation is thought to be in part due to the action of prostaglandin derivative from arachidonic acid metabolism. A detailed evaluation of curcumin as a potential non-steroidal anti-inflammatory agent by Srimal and Dhawan found curcumin to be highly effective after oral administration. Curcumin was effective in other models of inflammation including granuloma, pouch, cotton pellet, formalin-induced, and Freund's adjuvant. Many mechanisms of action have been attributed to curcumin. Some researchers found curcumin to be less effective in adrenalectomized rats, suggesting a participation of corticoidal steroids, while others did not observe any effect of curcumin salts on steroid release from the adrenal cortex. Recently, another, more specific in-vitro method has been developed which allows the study of the inhibitory mechanism of potential drugs. By using rat peritoneal neutrophilis, curcumin was tested for the direct effect on the 5-lipoxygenase activities. Another study found that curcumin was able to inhibit the production of pro-inflammatory mediators in microglial cells that had been stimulated to mount an inflammatory response. Microglial cells are activated after brain injuries and produce proinflammatory mediators and neurotoxic compounds. Curcumin decreased the production of these compounds, apparently by blocking NF-kB, a protein signal in the pathway that leads to their production. The overexpression of pro-inflammatory cytokines contributes to diseases such as Alzheimer's, cerebral ischemia and many degenerative and inflammatory conditions. The ability of curcumin to decrease inflammation presents an approach to
slow the progression of these diseases.

3. Gastro-intestinal effects Curcumin increases mucin content, thereby protecting the gastric mucosa against irritants. Controversial data exist regarding an anti-ulcerogenic activity of curcumin. Some researchers found a protective effect of curcumin against histamine-induced gastric ulceration, while others reported an ulcerogenic effect of curcumin. Curcumin also possesses anti-spasmodic properties. Curcumin showed liver protective effects against carbon tetrachloride, D-Galactosamine and peroxide induced cytotoxicity. Curcumin increased bile acid production in dogs and rats.

4. Cardiovascular effects A sharp and transient hypotensive effect of curcumin was reported in dogs. Curcumin also inhibited collagen and adrenaline-induced aggregation of platelets but did not affect prostacyclin (PGI2) synthesis.

5. Lipid metabolism Rao and co-workers reported that rats fed with curcumin and cholesterol in their diet had only half to one-third of the serum and liver cholesterol levels compared to the controlled groups receiving cholesterol alone.

6. Anti-bacterial/Anti-fungal Curcumin inhibited growth of most organisms including: Staph aureus, Streptococci, Lactobacilli, Corynebacterium, Baccilus aureus, and Micrococcus pyogenes. The crude ether and chloroform extracts of Curcuma longa showed fungistatic activity against several dermatophytes as well as anti-amoebic activity against Entamoeba histolytica.

7. Anti-viral A 1993 study showed curcumin as an effective ally in the treatment against HIV. Curcumin was effective in inhibiting the replication of HIV in both acutely infected and chronically infected cells.

8. Promotes Normal Cell Growth The ability of various extracts of Curcuma longa to promote normal cellular activity has been remarked by several researchers. Topical application of curcumin also significantly enhances normal cell activities. Curcumin was found to be a selective and non-competitive inhibitor of phosphorylase kinase.

Market Trends

Turmeric products have a longstanding reputation for being beneficial in the treatment and prevention of a variety of ailments. It is a substance that is considered very safe to use and that offers multiple health benefits. However, curcumin is the active ingredient in turmeric responsible for these effects, and it is the compound with which most of the clinical studies have been conducted, not turmeric.

AOR Advantage

AOR offers a standardized and potent curcumin extract, not a weak turmeric extract, that provides antioxidant, anti-inflammatory, cholesterol lowering, and antimicrobial benefits and promotes normal cell growth.

References

Abstract

A randomized, pilot study to assess the efficacy and safety of curcumin in patients with active rheumatoid arthritis.


Chandran B, Goel A.

Curcumin is known to possess potent antiinflammatory and antiarthritic properties. This pilot clinical study evaluated the safety and effectiveness of curcumin alone, and in combination with diclofenac sodium in patients with active rheumatoid arthritis (RA). Forty-five patients diagnosed with RA were randomized into three groups with patients receiving curcumin (500?mg) and diclofenac sodium (50? mg) alone or their combination. The primary endpoints were reduction in Disease Activity Score (DAS) 28. The secondary endpoints included American College of Rheumatology (ACR) criteria for reduction in tenderness and swelling of joint scores. Patients in all three treatment groups showed statistically significant changes in their DAS scores. Interestingly, the curcumin group showed the highest percentage of improvement in overall DAS and ACR scores (ACR 20, 50 and 70) and these scores were significantly better than the patients in the diclofenac sodium group. More importantly, curcumin treatment was found to be safe and did not relate with any adverse events. Our study provides the first evidence for the safety and superiority of curcumin treatment in patients with active
RA, and highlights the need for future large-scale trials to validate these findings in patients with RA and other arthritic conditions.

**Curcumin ingestion and exercise training improve vascular endothelial function in postmenopausal women.**


Vascular endothelial function is declines with aging and is associated with an increased risk of cardiovascular disease. Lifestyle modification, particularly aerobic exercise and dietary adjustment, has a favorable effect on vascular aging. Curcumin is a major component of turmeric with known anti-inflammatory and anti-oxidative effects. We investigated the effects of curcumin ingestion and aerobic exercise training on flow-mediated dilation as an indicator endothelial function in postmenopausal women. A total of 32 postmenopausal women were assigned to 3 groups: control, exercise, and curcumin groups. The curcumin group ingested curcumin orally for 8 weeks. The exercise group underwent moderate aerobic exercise training for 8 weeks. Before and after each intervention, flow-mediated dilation was measured. No difference in baseline flow-mediated dilation or other key dependent variables were detected among the groups. Flow-mediated dilation increased significantly and equally in the curcumin and exercise groups, whereas no changes were observed in the control group. Our results indicated that curcumin ingestion and aerobic exercise training can increase flow-mediated dilation in postmenopausal women, suggesting that both can potentially improve the age-related decline in endothelial function.

**Oral curcumin mitigates the clinical and neuropathologic phenotype of the Trembler-J mouse: a potential therapy for inherited neuropathy.**


Khajavi M, Shiga K, Wiszniewski W, He F, Shaw CA, Yan J, Wensel TG, Snipes GJ, Lupski JR.

Mutations in myelin genes cause inherited peripheral neuropathies that range in severity from adult-onset Charcot-Marie-Tooth disease type 1 to childhood-onset Dejerine-Sottas neuropathy and congenital hypomyelinating neuropathy. Many myelin gene mutants that cause severe disease, such as those in the myelin protein zero gene (MPZ) and the peripheral myelin protein 22 gene (PMP22), appear to make aberrant proteins that accumulate primarily within the endoplasmic reticulum (ER), resulting in Schwann cell death by apoptosis and, subsequently, peripheral neuropathy. We previously showed that curcumin supplementation could abrogate ER retention and aggregation-induced apoptosis associated with neuropathy-causing MPZ mutants. We now show reduced apoptosis after curcumin treatment of cells in tissue culture that express PMP22 mutants. Furthermore, we demonstrate that oral administration of curcumin partially mitigates the severe neuropathy phenotype of the Trembler-J mouse model in a dose-dependent manner. Administration of curcumin significantly decreases the percentage of apoptotic Schwann cells and results in increased number and size of myelinated axons in sciatic nerves, leading to improved motor performance. Our findings indicate that curcumin treatment is sufficient to relieve the toxic effect of mutant aggregation-induced apoptosis and improves the neuropathologic phenotype in an animal
model of human neuropathy, suggesting a potential therapeutic role in selected forms of inherited peripheral neuropathies.

Curcumin protects against acute liver damage in the rat by inhibiting NF-kappaB, proinflammatory cytokines production and oxidative stress.


Reyes-Gordillo K, Segovia J, Shibayama M, Vergara P, Moreno MG, Muriel P.

Curcumin, an anti-inflammatory and antioxidant compound, was evaluated for its ability to suppress acute carbon tetrachloride-induced liver damage. Acute hepatotoxicity was induced by oral administration of CCl(4) (4 g/kg, p.o.). Curcumin treatment (200 mg/kg, p.o.) was given before and 2 h after CCl(4) administration. Indicators of necrosis (alanine aminotransferase) and cholestasis (gamma-glutamyl transpeptidase and bilirubins) resulted in significant increases after CCl(4) intoxication, but these effects were prevented by curcumin treatment. As an indicator of oxidative stress, GSH was oxidized and the GSH/GSSG ratio decreased significantly by CCl(4), but was preserved within normal values by curcumin. In addition to its antioxidants properties, curcumin is capable of preventing NF-kappaB activation and therefore to prevent the secretion of proinflammatory cytokines. Therefore, in this study we determined the concentrations of tumor necrosis factor-alpha (TNF-alpha), interleukin-1beta (IL-1beta), and interleukin-6 (IL-6) mRNA, and NF-kappaB activation. CCl(4)-administered rats depicted significant increases in TNF-alpha, IL-1beta, and IL-6 production, while curcumin remarkably suppressed these mediators of inflammation in liver damage. These results were confirmed by measuring TNF-alpha, and IL-1beta protein production using Western Blot analysis. Accordingly, these proteins were increased by CCl(4) and this effect was abolished by curcumin. Administration of CCl(4) induced the translocation of NF-kappaB to the nucleus; CCl(4) induced NF-kappaB DNA binding activity was blocked by curcumin treatment. These findings suggest that curcumin prevents acute liver damage by at least two mechanisms: acting as an antioxidant and by inhibiting NF-kappaB activation and thus production of proinflammatory cytokines.

Curcumin downregulates homeobox gene NKX3.1 in prostate cancer cell LNCaP.


Zhang HN, Yu CX, Zhang PJ, Chen WW, Jiang AL, Kong F, Deng JT, Zhang JY, Young CY.

Aim: To elucidate the effect and the mechanisms of curcumin on the expression of the human homeobox gene NKX3.1 in the prostate cancer cell LNCaP.

Methods: The expression change of NKX3.1 in cells incubated with varying concentrations of curcumin was observed by Western blotting and RT-PCR. A dual luciferase reporter assay was used to test the effect of curcumin on the activity of the NKX3.1 1040 bp promoter. Curcumin-treated cells disposed to a designated amount of androgen analog R1881 and the androgen receptor (AR) antagonist flutamide, then the expression of NKX3.1 or the activity of the NKX3.1 promoter were investigated by Western blotting or reporter gene assay, respectively. Finally, Western blotting and electrophoretic mobility shift assay were performed to demonstrate the effect of curcumin on the expression of AR and its binding activity to the androgen response element (ARE).
Results: Curcumin downregulated the expression of NKX3.1 and the activity of the NKX3.1 1040 bp promoter in LNCaP cells. R1881 increased the expression of NKX3.1, and the AR antagonist flutamide decreased the expression of NKX3.1 in LNCaP cells, while curcumin could inhibit androgen-AR mediated induction of NKX3.1 expression. Curcumin decreased the expression of AR and the binding activity to ARE directly.

Conclusion: Curcumin could downregulate NKX3.1 expression in LNCaP cells. It could also inhibit the androgen-AR mediated induction of NKX3.1 expression by downregulating AR expression and blocking its DNA binding activity.

Phase I Clinical Trial of Oral Curcumin.


Curcumin, a polyphenolic antioxidant derived from a dietary spice, exhibits anticancer activity in rodents and in humans. Its efficacy appears to be related to induction of glutathione S-transferase enzymes, inhibition of prostaglandin E2 (PGE2) production, or suppression of oxidative DNA adduct (M1G) formation. We designed a dose-escalation study to explore the pharmacology of curcumin in humans. Fifteen patients with advanced colorectal cancer refractory to standard chemotherapies consumed capsules compatible with curcumin doses between 0.45 and 3.6 g daily for up to 4 months. Levels of curcumin and its metabolites in plasma, urine, and feces were analyzed by high-pressure liquid chromatography and mass spectrometry. Three biomarkers of the potential activity of curcumin were translated from preclinical models and measured in patient blood leukocytes: glutathione S-transferase activity, levels of M1G, and PGE2 production induced ex vivo. Dose-limiting toxicity was not observed. Curcumin and its glucuronide and sulfate metabolites were detected in plasma in the 10 nmol/L range and in urine. A daily dose of 3.6 g curcumin engendered 62% and 57% decreases in inducible PGE2 production in blood samples taken 1 hour after dose on days 1 and 29, respectively, of treatment compared with levels observed immediately predose (P < 0.05). A daily oral dose of 3.6 g of curcumin is advocated for Phase II evaluation in the prevention or treatment of cancers outside the gastrointestinal tract. PGE2 production in blood and target tissue may indicate biological activity. Levels of curcumin and its metabolites in the urine can be used to assess general compliance.

Phase I clinical trial of curcumin, a chemopreventive agent, in patients with high-risk or pre-malignant lesions.


Curcumin (diferuloylmethane), a yellow substance from the root of the plant Curcuma longa Linn., has been demonstrated to inhibit carcinogenesis of murine skin, stomach, intestine and liver. However, the toxicology, pharmacokinetics and biologically effective dose of curcumin in humans have not been reported. This prospective phase-I study evaluated these issues of curcumin in patients with one of
the following five high-risk conditions: 1) recently resected urinary bladder cancer; 2) arsenic Bowen's disease of the skin; 3) uterine cervical intraepithelial neoplasm (CIN); 4) oral leucoplakia; and 5) intestinal metaplasia of the stomach. Curcumin was taken orally for 3 months. Biopsy of the lesion sites was done immediately before and 3 months after starting curcumin treatment. The starting dose was 500 mg/day. If no toxicity > or = grade II was noted in at least 3 successive patients, the dose was then escalated to another level in the order of 1,000, 2,000, 4,000, 8,000, and 12,000 mg/day. The concentration of curcumin in serum and urine was determined by high pressure liquid chromatography (HPLC). A total of 25 patients were enrolled in this study. There was no treatment-related toxicity up to 8,000 mg/day. Beyond 8,000 mg/day, the bulky volume of the drug was unacceptable to the patients. The serum concentration of curcumin usually peaked at 1 to 2 hours after oral intake of curcumin and gradually declined within 12 hours. The average peak serum concentrations after taking 4,000 mg, 6,000 mg and 8,000 mg of curcumin were 0.51 /- 0.11 microM, 0.63 /- 0.06 microM and 1.77 /- 1.87 microM, respectively. Urinary excretion of curcumin was undetectable. One of 4 patients with CIN and 1 of 7 patients with oral leucoplakia proceeded to develop frank malignancies in spite of curcumin treatment. In contrast, histologic improvement of precancerous lesions was seen in 1 out of 2 patients with recently resected bladder cancer, 2 out of 7 patients of oral leucoplakia, 1 out of 6 patients of intestinal metaplasia of the stomach, 1 out of 4 patients with CIN and 2 out of 6 patients with Bowen’s disease. In conclusion, this study demonstrated that curcumin is not toxic to humans up to 8,000 mg/day when taken by mouth for 3 months. Our results also suggest a biologic effect of curcumin in the chemoprevention of cancer.

Inhibitory effect of dietary curcumin on skin carcinogenesis in mice.


Limtrakul P, Lipigorngoson S, Namwong O, Apisariyakul A, Dunn FW.

Laboratory animal model studies have suggested that curcumin may play an important role in inhibiting the process of carcinogenesis. Curcumin, the yellow pigment that is obtained from rhizomes of the plant Curcuma longa Linn (Family Zingiberaceae), is commonly used as a spice and food coloring agent. The present study was designed to investigate the chemopreventive action of dietary curcumin on 7,12-dimethylbenz[a]anthracene (DMBA)-initiated and 12-O-tetradecanoylphorbol-13-acetate (TPA)-promoted skin tumor formation in male Swiss albino mice. At 6 weeks of age, groups of animals were fed the standard (modified AIN-76 A) diet or a diet containing 1% curcumin. At 8 weeks of age, all animals, except those in the vehicle (acetone)-treated groups, received 100 microg of DMBA dissolved in 100 microl of acetone in a single application to the skin of the back. From 1 week after DMBA application, tumor promoter (2.5 microg of TPA dissolved in 100 microl of acetone) was applied to the same areas on mouse skin twice a week for 26 weeks. All groups continued on their respective dietary regimen until the termination of the experiment. The results indicate that dietary administration of curcumin significantly inhibited the number of tumors per mouse (P < 0.05) and the tumor volume (P < 0.01). The percentage of tumor-bearing mice tended to be lower in the mice on the curcumin diet than those on the standard diet. There was no difference in growth between mice of the standard and 1% curcumin groups. The results indicate the safety and the anti-carcinogenic effect of curcumin in mice.

Turmeric: A Brief Review of Medicinal Properties
Turmeric has been attributed a number of medicinal properties in the traditional system of medicine and its internal as well local use has been advocated. The major claims have been for use as antiseptic, cure for poisoning, eliminating body waste products, for dyspepsia, respiratory disorders and cure for a number of skin diseases including promotion of wound healing. Recent studies have confirmed some of the older claims and brought out several new useful properties. Curcumin, curcuminoids and essential oils are the major active constituents. The main activities have been found to be anti-inflammatory, hepatoprotective, antimicrobial, wound healing, anticancer, antitumor and antiviral. Discovery of antiviral properties in curcumin, particularly against HIV, is interesting and needs proper evaluation. The review highlights some of the newer researchers, which may explain the multifaceted activity of this natural product. Different extracts of turmeric and also curcumin have been tried clinically in several diseased conditions with gratifying results.

Mechanism of antiinflammatory actions of curcumine and boswellic acids.


Ammon HP, Safayhi H, Mack T, Sabieraj J.

Curcumine from Curcuma longa and the gum resin of Boswellia serrata, which were demonstrated to act as anti-inflammatories in in vivo animal models, were studied in a set of in vitro experiments in order to elucidate the mechanism of their beneficial effects. Curcumine inhibited the 5-lipoxygenase activity in rat peritoneal neutrophils as well as the 12-lipoxygenase and the cyclooxygenase activities in human platelets. In a cell free peroxidation system curcumine exerted strong antioxidative activity. Thus, its effects on the dioxygenases are probably due to its reducing capacity. Boswellic acids were isolated from the gum resin of Boswellia serrata and identified as the active principles. Boswellic acids inhibited the leukotriene synthesis via 5-lipoxygenase, but did not affect the 12-lipoxygenase and the cyclooxygenase activities. Additionally, boswellic acids did not impair the peroxidation of arachidonic acid by iron and ascorbate. The data suggest that boswellic acids are specific, non-redox inhibitors of leukotriene synthesis either interacting directly with 5-lipoxygenase or blocking its translocation.