AOR CODE: AOR04182

Premium

**MCP**

$134.45 CAD

Detoxify and Maintain Healthy Cell Growth

- Contains a soluble source of modified citrus pectin
- Promotes detoxification
- Derived from oranges and free from colouring and masking agents

Gluten Free  Vegan  Non-GMO  Detoxification

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<tr>
<th>AOR Code</th>
<th>Variant</th>
<th>Price</th>
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<tbody>
<tr>
<td>AOR04182</td>
<td>450 G POWDER</td>
<td>$134.45</td>
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**Details**

MCP is modified citrus pectin, a carbohydrate derived from the soluble fibre of citrus peels which has been broken down into smaller fragments to be more easily absorbed in the digestive tract. AOR’s MCP is refined to match the specific size and weight of the pectin used in these studies.

**Label Info**

**Discussion**

MCP is modified citrus pectin, a carbohydrate derived from the soluble fibre of citrus peels that has been modified to be more easily absorbed in the digestive tract. MCP has been shown to help support detoxification.

**Product Variation**

<table>
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<tr>
<th>Product Code</th>
<th>Size</th>
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<tbody>
<tr>
<td>AOR04182</td>
<td>450 G POWDER</td>
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**Supplements Facts**

<table>
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<tr>
<th>Serving Size: 3 Scoops</th>
<th>Amount</th>
<th>% Daily</th>
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<tbody>
<tr>
<td>Modified Citrus Pectin</td>
<td>15 g*</td>
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</table>
Serving Size: 3 Scoops

*Contains 885 mg of sodium and up to 1350 mg of potassium per day.

Note: Herbal extracts will naturally vary in colour and taste from one batch to another.

Guarantees

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

Adult Dosage

Take 1 scoop mixed with water or juice 3 times daily with food or immediately after meals (to minimize gastrointestinal irritation and prevent too rapid absorption), or as directed by a qualified health care practitioner.

Cautions

Do not use if you are pregnant or breastfeeding. Diarrhea, stomach irritation, nausea, slowed heart rate and abnormal heart rhythm may occur. Discontinue use and contact your health care practitioner if you develop severe stomach pain, irregular heartbeat, chest pain, or for use beyond 4 weeks. Vomiting has been known to occur, in which case discontinue use and consult your health care practitioner.

Source

Pharmaceutical synthesis

Main Application

Methylation
Homocysteine
Detoxification
Cardiovascular health
Healthy blood pressure
Anti-aging

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

Citrus Pectin

Have you ever heard that the white pith of citrus fruits is good for you? Well it’s true! The white pith just under the rind of citrus fruits like grapefruits and oranges is full of healthy pectin. Unfortunately, the bitter taste and chalky texture makes it quite undesirable to eat. That’s where modified citrus pectin (MCP) comes in. MCP has been shown to be helpful in heavy metal toxicity and in promoting healthy cell growth.

Modified Citrus Pectin as a Heavy Metal Chelator

Heavy metal toxicity is known to be an important factor in declining general health. Adult case reports, one short study in healthy people and one small clinical study in young children have shown tremendous effectiveness for MCP in reducing heavy metal toxicity including lead, mercury, arsenic and cadmium. Larger clinical trials are required for greater conclusiveness, but results like heavy metal reductions by up to 560% are mind-blowing.

Why Citrus Pectin Needs to be Modified

Pectin from citrus fruits contains a significant number of residues of the sugar galactose. This pectin is present in the form of long, complex chains whose galactose residues are hidden deep within their intricate molecular branchings. To solve this problem, the carbohydrate chains can be split into shorter fragments under pH-controlled conditions, exposing more galactose residues and simplifying the complicated branchings of the molecule. Working with the fibers of lemons, grapefruits, or tangerines in this way creates pH-modified citrus pectin (MCP). This modification process maximizes the opportunities for the pectin’s galactose residues to interact with galectin-3, unleashing the potential of citrus pectin.

Lectins & Glycoconjugates: A Lock & Key Mechanism for the Spread of Unhealthy Cells

Galactose is the same kind of sugar residue to which galectin-3 binds. Galectin-3 is a type of lectin. Lectins are cellular communication molecules that allow similar types of cells to bind together (these are called glycoconjugates) and to remain attached to their appropriate tissues. Lectin-glycoconjugate interactions also play a key role in immunity, allowing immune cells to distinguish “self” from “non-self” (foreign) cells, and in other cell-to-cell interactions.

An Unfortunate Match

It’s been shown that many unhealthy cells exploit the lectin recognition system by covering themselves with “keys” that match the “locks” in distant, healthy tissues. Either the abnormal cell causes its surface to bristle with a lectin, which then binds to that lectin’s glycoconjugate on a healthy cell type, or vice-versa. Additionally, some unhealthy cells can coat themselves with lectins to allow them to bind to one another, ganging up into clumpings called emboli. This allows abnormal cells that are spreading to more easily escape from your immunological defenses, since the cells on the innermost layers of the emboli may survive even if the immune system destroys cells on the outer layer. By exploiting the match between lectin and glycoconjugate, unhealthy cells are more likely to launch an effective attack.

One lectin “lock” which many abnormal cells learn to “pick” is called galectin-3. Galectin-3 recognizes glycoconjugates, which have the sugar galactose as a key part of its structure (hence gal- (galactose) lectin). By sprouting galectin-3 – or its glycoconjugate – on their surfaces, these cells increase their
ability to spread, to take root, and to grow new masses of deadly unhealthy cells. Manipulative abuse of galectin-3 has been implicated in the spread of abnormal cells in the pancreas, colon, lung, ovaries, breast, and especially the prostate. By churning out galectin-3, abnormal prostate cells can easily bind to healthy lung tissue, which is vulnerable to this attack because of its high amount of the lectin’s glycoconjugate; alternatively, by creating more galectin-3 glycoconjugates, abnormal prostate cells find it easier to metastasize to bone tissue.

MCP: A Molecular Decoy Against Metastasis

The idea of using MCP is that the galactose residues on the pectin molecules can provide alternate binding sites for abnormal cells rather than other cells. Animal and clinical studies have certainly found that this is an effective means to reduce the spread of abnormal cells. By delaying abnormal cell growth, MCP provides a larger window of opportunity for other interventions to be more effective.

Other Mechanisms that Promote Normal Cell Growth

MCP can inhibit certain steps of galectin-3-induced angiogenesis (the growth of new blood vessels to feed abnormal cell masses). Galectin-3 can also inhibit abnormal cell anoikis (a defensive mechanism in which abnormal cells are forced to detach from the healthy tissue’s extracellular matrix, leading them to commit cellular “suicide” (apoptosis)), and MCP can block the anti-anoikis action of galectin-3.

Research

Heavy Metal Chelation

Healthy human subjects with no known heavy metal toxicities were given 15g of MCP for 5-days and 20 g on the sixth day. The amount of lead, arsenic and cadmium excreted in the urine was measured each day. After Day 1, arsenic excretion increased by 130%. At Day 6, cadmium excretion had increased by 150%. Over the course, lead excretion had increased by 560%! The chelation is thought to be attributed to the presence of “rhamnogalacturonan II,” which is found in MCP.

In 2007, it was reported that MCP helped reduce lead and mercury, toxic heavy metals, by 74% in five adult patients. This heavy metal reduction was reported to have assisted their return to good health.

Another small pilot clinical study was then published in 2008 in children hospitalized for lead toxicity. Seven children aged 5-12 years were given 15 g of MCP for a month. On average, serum lead levels were reduced by an average of a whopping 161% with no adverse effects.

Market Trends

MCP is known mostly for reducing the spread of abnormal cells. It is also known as a heavy metal chelator.

AOR Advantage

AOR’s MCP comes from oranges sourced from Florida and from France and not from grapefruits.
since grapefruit is known to interfere with certain medications. AOR’s MCP also does not contain any masking agents such as colours or flavours in order to maintain a pure product for the vulnerable populations who use this product. This means that the physical properties of the product like colour, taste and solubility are subject to change with environmental factors such as time of harvest, country of origin, soil conditions, etc.

References


Abstract

The role of modified citrus pectin as an effective chelator of lead in children hospitalized with toxic lead levels.


Zhao ZY, Liang L, Fan X, Yu Z, Hotchkiss AT, Wilk BJ, Eliaz I.

CONTEXT: Lead toxicity is an ongoing concern worldwide, and children, the most vulnerable to the long-lasting effects of lead exposure, are in urgent need of a safe and effective heavy metal chelating agent to overcome the heavy metals and lead exposure challenges they face day to day.

OBJECTIVE: This clinical study was performed to determine if the oral administration of modified citrus pectin (MCP) is effective at lowering lead toxicity in the blood of children between the ages of 5 and 12 years.

METHOD: Hospitalized children with a blood serum level greater than 20 microg/dL, as measured by graphite furnace atomic absorption spectrometry (GFAAS), who had not received any form of chelating and/or detoxification medication for 3 months prior were given 15 g of MCP (PectaSol) in 3 divided dosages a day. Blood serum and 24-hour urine excretion collection GFAAS analysis were performed on day 0, day 14, day 21, and day 28.

RESULT: This study showed a dramatic decrease in blood serum levels of lead (P = .0016; 161% average change) and a dramatic increase in 24-hour urine collection (P = .0007; 132% average change).

CONCLUSION: The need for a gentle, safe heavy metal-chelating agent, especially for children with high environmental chronic exposure, is great. The dramatic results and no observed adverse effects in this pilot study along with previous reports of the safe and effective use of MCP in adults indicate that MCP could be such an agent. Further studies to confirm its benefits are justified.

Integrative medicine and the role of modified citrus pectin/alginates in heavy metal chelation and detoxification–five case reports.

Forsch Komplementmed. 2007 Dec;14(6):358-64.

Eliaz I, Weil E, Wilk B.

Heavy metal body burden can contribute to chronic disease, as well as interfere with the body’s capacity to recover from illness. The five case studies presented here show that reduction in toxic heavy metals (74% average decrease) was achieved without side effects, with the use of PectaSol modified citrus pectin (MCP) (EcoNugenics; Santa Rosa, CA, USA) alone or with an MCP/alginates combination. The gradual decrease of total body heavy metal burden is believed to have played an important role in each patient’s recovery and health maintenance. This is the first known
documentation of evidence of such results in a clinical report of case studies with possible correlation between clinical outcome and a reduction in toxic heavy metal load in patients using MCP and/or an MCP/alginate complex.

The effect of modified citrus pectin on urinary excretion of toxic elements.


Eliaz I, Hotchkiss AT, Fishman ML, Rode D.

This study was undertaken to evaluate the effect of modified citrus pectin (MCP) on the urinary excretion of toxic elements in healthy individuals. MCP is a reduced molecular weight pectin (weight-average molar mass = 15400) that is mostly linear homogalacturonan with a 3.8% degree of esterification and approximately 10% rhamnogalacturonan II based on the presence of 2-keto-3-deoxy-octonic acid. Subjects ingested 15 g of MCP (PectaSol®, EcoNugenics® Inc., Santa Rosa, California 95407) each day for 5 days and 20 g on day 6. Twenty-four hour urine samples were collected on day 1 and day 6 for comparison with baseline. The urine samples were analysed for toxic and essential elements. In the first 24 h of MCP administration the urinary excretion of arsenic increased significantly (130%, p < 0.05). On day 6, urinary excretion was increased significantly for cadmium (150%, p < 0.05). In addition, lead showed a dramatic increase in excretion (560%, p < 0.08). This pilot trial provides the first evidence that oral administration of MCP increases significantly the urinary excretion of toxic metals in subjects with a ‘normal’ body load of metals. It is suggested that systemic chelation of toxic metals by MCP may in part be attributable to the presence of rhamnogalacturonan II, which has been shown previously to chelate metals.

Modified Citrus Pectin slows PSA doubling time: A Pilot Clinical Trial.


The purpose of this pilot clinical trial was to evaluate the ability of Modified Citrus Pectin to influence the PSA slope in men with prostate cancer. Patients who had either relapsed after or failed prior treatment for prostate cancer (PSA range 0.63 to 7.50) were given Modified Citrus Pectin (PectaSol®, EcoNugenics, Inc., San Rafael, CA 94901) at a dosage of 15 grams per day in three divided oral doses. PSA doubling time was calculated. A response (more than 30% lengthening of PSA doubling time) was seen in 4/7 patients (57%). One patient (1/7) had a partial response, one patient (1/7) had stable disease, and one patient (1/7) did not respond. Modified Citrus Pectin appears to lengthen the PSA doubling time in prostate cancer patients with low levels of PSA. Study responses are additionally compelling, as all study participants are still alive and evaluable for long-term follow-up almost three years after completion of this study. More research involving larger numbers of patients is needed to full), define the role of MCP in prostate cancer treatment.

Citrus pectin: characterization and inhibitory effect on fibroblast growth factor-receptor interaction.
This study was undertaken to characterize the pectin from four citrus species and to determine their in vitro inhibitory activities on the binding of fibroblast growth factor (FGF) to the FGF receptor (FGFR). Pectin from various parts of lemon, grapefruit, tangerine, and orange were isolated and characterized. Tangerine had the highest pectin content among the four citrus species. Segment membrane contained as much as or more pectin than flavedo/albedo. Anhydrogalacturonic content was highest in pectin from segment membrane of tangerine and flavedo/albedo of grapefruit. Lemon pectin contained the highest methoxyl content (MC), and grapefruit contained the largest proportion of lower molecular weight.

Changes in cell growth, cyclin/kinase, endogenous phosphoproteins and nm23 gene expression in human prostatic JCA-1 cells treated with modified citrus pectin.


Hsieh TC, Wu JM.

Modified citrus pectin (MCP) added to the media of cultured androgen-independent human prostatic JCA-1 cells reduced cell growth and correspondingly [3H]thymidine incorporation into DNA, which was correlated with the down-regulation of cyclin B and p34cdc2 MCP also induced distinct increases in specific endogenous phosphoproteins, including a cAMP-stimulated 52,000 (52-kDa) protein. Since metastasis has been inversely correlated with nm23 gene expression in some cancer cells and was reportedly inhibited by MCP in a rat prostate model, we investigated steady state level changes in the nm23 protein in JCA-1 cells and found it to be unexpectedly suppressed as a result of exposure to MCP.

Effects of natural complex carbohydrate (citrus pectin) on murine melanoma cell properties related to galectin-3 functions.


Inohara H, Raz A.

Citrus pectin (CP) and pH-modified citrus pectin (MCP) are highly branched and non-branched complex polysaccharides, respectively, rich in galactoside residues, capable of combining with the carbohydrate-binding domain of galectin-3. We reported previously that intravenous injection of B16-F1 murinemelanoma cells with CP or MCP into syngeneic mice resulted in a significant increase or decrease of lung colonization, respectively (Platt D, Raz A (1992) J Natl Cancer Inst 84:438-42). Here we studied the effects of these polysaccharides on cell-cell and cell-matrix interactions mediated by carbohydrate-recognition. MCP, but not CP, inhibited B16-F1 melanoma cells adhesion to laminin and asialofetuin-induced homotypic aggregation. Both polysaccharides inhibited anchorage-independent growth of B16-F1 cells in semisolid medium, i.e. agarose. These results indicate that carbohydrate-recognition by cell surface galectin-3 may be involved in cell-extracellular matrix interaction and play a
Galectin-3 induces endothelial cell morphogenesis and angiogenesis.

Increasing evidence suggests that carbohydrate-binding proteins play an essential role in tumor growth and metastasis. However, conflicting results on their function in the regulation of cell proliferation and differentiation during angiogenesis have been reported. We have examined the role of galectin-3 in the regulation of human umbilical vein endothelial cell proliferation, differentiation, migration, and neovascularization. Galectin-3, a carbohydrate-binding protein, with specificity for type 1 and 11 ABH blood group epitopes and polylactosamine glycan containing cell surface glycoproteins, is the major nonintegrin cellular laminin-binding protein. Because galectin-3 expression was shown to be associated in some tumor systems with metastasis, we questioned whether it induces endothelial cell morphogenesis. Here we show that galectin-3 affects chemotaxis and morphology and stimulates capillary tube formation of HUV-EC-C in vitro and angiogenesis in vivo. Endothelial cell morphogenesis is a carbohydrate-dependent process, as it is neutralized by specific sugars and antibodies. These findings demonstrate that endothelial cell surface carbohydrate recognition event(s) can induce a signaling cascade leading to the differentiation and angiogenesis of endothelial cells.