Luma TC Tablets
MEDICAL FOOD, No Rx Required, 08225-951-90 (90 Tablets)

Dye Free - Gluten Free · Bovine Free · Soy Free · Sugar Free · Casein Free - Yeast Free · Glucose Free · Lactose Free

DESCRIPTION: Luma TC Tablets is a "no prescription required" form of medical food to be used under medical supervision that contains TC(Theracurmin) sub-micron particle curcumin, nutritional micro-dose lithium, pre metabolized B vitamin coenzymes and NAC in the amounts and forms that cannot be achieved by the modification of normal diet alone. Luma TC circumvents genetic polymorphisms, poor aging, environmental confounders, and faulty central nervous system neuronal enzyme inhibition or production to lower homocysteine, prevent neuronal oxidative stress, reduce CNS inflammation, prevent beta amyloid protein plaques and tau tangle generated nerve cell apoptosis, and promote neurogenesis to restore brain mass and connectivity.

- Luma TC can be authorized by a licensed medical practitioner.
- Luma TC can be recommended by a healthcare professional.
- Luma TC can be ordered without a prescription by consumers.
- Luma TC should be used under medical supervision or consultation.

Please call Direct Value Dispense at 985-629-5742 for assistance.

Ingredients:

TC(Theracurmin) sub-micron particle curcumin …….180mg.
Lithium Orotate …….....5mg.
N-Acetylcysteine …….....300mg.
L-Methylfolate Calcium(B9) ……500mcg.
Methylcobalamin(B12) ……50mcg.
Pyridoxal-5-Phosphate(B6) ……5mg.

Inactive ingredients: Microcrystalline Cellulose, Croscarmellose Sodium, Silicon Dioxide, Magnesium Stearate,Carnauba Wax,Hypomellose,PolyetheleneGlycol

Medical foods are intended for patients who have a limited or impaired capacity to ingest, digest, absorb, or metabolize ordinary foodstuffs or certain nutrients, or who have other special medically determined nutrient requirements, the dietary management of which cannot be achieved by the modification of the normal diet alone. [1,2]

PHARMACOLOGY

TC SUB-MICRON PARTICLE CURCUMIN 4-hydroxy-3-methoxyphenyl1,6-heptadeine-2,5dione is the Indian herb used in curry powder and is a polyphenolic compound derived from turmeric, the dried rhizome of Curcuma Longa. Curcumin readily crosses the blood brain barrier and is easily absorbed into neurons, but generally has poor absorption into the blood. Human clinical bioavailability trials have demonstrated that TC Sub-Micron Particle Curcumin has a higher absorptive capacity which was 28 times higher t han ordinary curcumin powder. Curcumin has potent CNS anti-inflammatory activity. Curcumin is found to inhibit (COX2), IL-1, IL-6, TNF, phospholipases, transcription factor, and enzymes involved in metabolizing the membrane phospholipids into prostaglandins and regulate NF-kB. Curcumin modulates the levels of norepinephrine, dopamine, and serotonin in the brain and inhibits the (MAO)-A and (MAO)-B enzyme that decomposes dopamine and serotonin. Curcumin inhibits the formation and promotes the disaggregation of amyloid-Beta plaques and facilitates macrophage uptake and ingestion of plaques. Curcumin attenuates the hyperphosphorylation of tau enhancing destructive tau protein and tangle clearance. In a recently published 18-month RCT human trial Theracurmin TC Sub-Micron particle 180mg. per day in divided doses, a bioavailable form of curcumin, led to significant memory and attention benefits compared to placebo. FDDNP-Pet scans performed pre and post-treatment suggested that behavioral and cognitive benefits are associated with decreases in plaque and tangle accumulation in brain regions modulating mood and memory compared to placebo. Curcumin has powerful antioxidant properties demonstrated by the inhibition of the formation of free radicals. It decreases the low-density lipoprotein oxidation and the free radicals that cause deterioration of neurons not only in Alzheimer's Disease but also in other neuron degenerative disorders such as Parkinson's Disease, and TBI. Curcumin is a strong binder of neurotoxic heavy metals such as lead, cadmium, copper, zinc. Curcumin suppresses inflammatory damage by preventing metal induction of NF-kappa B.Curcumin is known to modulate the cholinergic system and protect from neurodegeneration. [11,12,13,14,25]

NUTRITIONAL MICRO-DOSE LITHIUM OROTATE is a highly bioavailable orotate chelated form of lithium. Lithium Orotate is the stable, intact unionized chelate and is believed to transport lithium efficiently through the neuron cell membrane and to the various sites of action within the cell. Lithium Orotate is three times more effective at raising brain concentrations of the mineral than lithium carbonate. Lithium directly inhibits hyperactive GSK-beta 3 enzyme by binding to the magnesium-sensitive sites of the enzyme in neurons. This action prevents aggregation of amyloid-Beta-Plaques and Tau protein destruction and resulting neurofibrillary tangles. Lithium

directly inhibits the hyperactive B-APP-cleaving enzyme BACE1(Beta Secretase 1) enzyme which also prevents the aggregation of amyloid-Beta-Plaques and Tau protein destruction and resulting neurofibrillary tangles. This greatly reduces self-apoptosis mechanisms resulting in brain tissue destruction. The inhibition of GSK-beta 3 and BACE1(Beta Secretase 1) are two of the most relevant mechanisms of action of lithium and substantiates its putative role in the prevention and management of the Alzheimer's Disease process. Lithium also inhibits the enzyme inositol monophosphatase (IMP) and suppresses the formation of (IP3). IP3 is a neuronal intracellular messenger implicated in the regulation of many intracellular pathways relevant to neuropsychiatric disorders, including autophagy as well as p53 and BAX the pro-apoptotic molecules. Lithium demonstrates the ability to activate the Wnt-Beta Catenin signaling pathway which plays a role in neural development and adult neurogenesis and the AKT intra-neuronal pathway which also regulates growth factor/neurotrophin signaling. Lithium inhibits the influx of calcium into neurons. In the brain, apoptosis pathways are promoted by signaling pathways that are activated when calcium enters the cell, so blocking this process improves cell survival. Calcium influx mediates the activity of N-methyl-D-asparate (NMDA) receptors, which contribute to glutamate-induced excitotoxicity in neurons. Lithium has been shown to enhance mitochondrial respiratory rate and reduce oxidative stress, protect DNA against damage from oxidative stress, and modulates calcium influx in the mitochondria. Lithium is also important for enhancing transport of two other critically important brain nutrients, folate and vitamin B12 into cells. Since vitamin B12 and folate also affect mood-associated parameters, the stimulation of the transport of these vitamins into brain cells by lithium may be cited as yet another mechanism of the anti-depressive mood elevating and anti-aggressive actions of lithium at nutritional dose levels particularly in the presence of B vitamin polymorphisms such as MTHFR SNP. Lithium was found to be protective against oxidative stress in dopaminergic N27 cells which over express A53T alpha-synuclein. Lithium prevents/degrades alpha-synuclein protein aggregation as seen in Lewy Bodies associated with Parkinson's Disease. Lithium also in vivo and in vitro increases tyrosine hydroxylase (dopamine precursor) in the frontal cortex, hippocampus and striatum. Lithium has the important neuroprotective effect of stimulating synthesis and release of the neurotrophic factors, brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), vascular endothelial growth factor (VEGF), and fibroblast growth factor21 (FGF-21). Increased availability of these factors protects neurons against neurotoxic insults, stimulates hippocampal neurogenesis, increases synaptic plasticity, and long-term potentiation, lessening pro inflammatory responses. Lithium can also attenuate arachidonic acid production, an essential feature of the innate inflammatory response. Lithium stimulates proliferation of stem cells, including bone marrow and neural stem cells in the subventricular zone, striatum, and forebrain and increases brain cell density and volume. Neural stem cells restrict damage and promote repair of damaged spinal cord neurons. Lithium has shown to be instrumental in axon sprouting, improved white and gray matter structure and functional integrity in multiple brain areas, as well as increased newly generated oligodendrocytes and survival. This demonstrates value of lithium to rescue, repair, and even regenerate white matter as well as neuron myelination. Lithium has also been shown to be a longevity ("anti-aging") nutrient. It has been observed that populations who consume lithium amounts in their water show reduced all-cause mortality. [16,17,18,19,20,21,24,26]

N-ACETYLCYSTEINE is a precursor of L-cysteine, which in turn is a component of the endogenous antioxidant glutathione. Glutathione plays an important role in antioxidant activities, redox-regulated cell signaling and immune response. Because cysteine availability is a limiting factor for glutathione synthesis, N-acetyl cysteine helps counter oxidative stress by maintaining or increasing Glutathione levels. Several studies have highlighted the imbalance in glutathione redox system as a feature of Alzheimer's disease onset and progression. Another study shows that glutathione levels decrease with age, but glutathione levels are significantly depleted in Alzheimer's disease. Combinations of methylfolate, methylcobalamin, P 5 P, and NAC in clinical studies have been shown to reduce homocysteine 30-50% which according to NIH based on the Framingham homocysteine cohort reduces risk of Alzheimer's disease by 50%. [15,16]

PHARMACOLOGY: L-METHYLFOLATE is the primary biologically active diastereomer of folate. It is the primary form of folate in circulation and is also the form transported across membranes – particularly across the blood brain barrier – into peripheral tissues. In the cell, L-methylfolate is used in the re-methylation of homocysteine to form methionine and tetrahydrofolate (THF). About 70% of food folate and cellular folate is comprised of L-methylfolate. Folic acid, the synthetic form of folate, must undergo enzymatic reduction by methylenetetrahdyrofolate reductase (MTHFR) to be biologically active. Genetic mutations of MTHFR result in a cell's inability to convert folic acid to L-methylfolate. The inability for a cell to reduce homocysteine to methionine may result in hyperhomocysteinemia , low glutathione and low CH3/methylator availability which is considered to be the IEM responsible for faulty methionine synthesis. Elevated homocysteine is associated with cognitive decline, white matter damage, brain atrophy, neurofibrillary tangles, and dementia and according to an International Consensus statement elevated homocysteine is a modifiable risk factor for the development of cognitive decline, dementia and Alzheimer's disease in older persons. **Luma TC Tablets** are specifically formulated to help patients meet medically determined nutrient requirements, the dietary management of which cannot be achieved by the modification of diet alone. FOLATE has been reported to enhance synthesis and/or regeneration of tetrahydrobiopterin (BH4), which is an essential cofactor in the biosynthesis of monoamine neurotransmitters serotonin, dopamine and norepinephrine. **Luma TC Tablets** contain L-methylfolate calcium. L-methylfolate is converted into functional, metabolically active coenzyme forms for use in the body, and supplies the active folate substrate, THF for use in transformylation and methylation biochemistry. It has also been shown that patients with hyperhomocysteinemia can be associated with the C677T, A1298C SNPs, and some 40 others generally untested of the MTHFR gene polymorphisms. Elevated homocysteine is associated with cognitive decline, white matter damage, brain atrophy, neurofibrillary tangles, and dementia. [3,4,5,6,7,8,9,10]

VITAMIN B12 coenzyme (Methylcobalamin) along with methylfolate is required for the remethylation of homocysteine to methionine, a reaction in

which the methyl group of L-methylfolate is donated to re-methylate homocysteine. Lack of methylcobalamin can cause the "Methyl Trap". Methylcobalamin can circumvent B12 vitamin polymorphisms such as Transcobalamin 1 and 2 and FUT2 and FUT6. There are 59 known vitamin B12 related polymorphisms. Homocysteine has been reported to stimulate or alter transcription factors involved in inflammation, with an important ancillary consequence of BH4 depletion. [3,4,7,22]

VITAMIN B6 coenzyme (Pyridoxal-5-Phosphate) plays a critical role in the generation of glutathione from cystathionine and cysteine, as well as for the recycle of other B-vitamins into reduced folate forms. P-5-P can circumvent known B6 polymorphisms of the ALPL, CBS, MTRR, and MTR genes. The aromatic amino acid decarboxylase considered to decarboxylate both dihydroxyphenylalanine (DOPA) and 5-hydroxytryptophan (5-HTP) requires pyridoxal-5'-phosphate as coenzyme. P-5-P is instrumental in glutathione production in transulfuration. [3,4,7,23]

In the FDA publication "Frequently Asked Question About Medical Foods"; Second Edition, Guidance for Industry, Food and Drug Administration 2016 on page 9 question 20 the question is asked, Does FDA generally consider inborn errors of metabolism (IEMs) to be diseases or conditions that a medical food could be used to manage? [2]

Yes. FDA generally considers IEMs to be diseases or conditions that a medical food could be used to manage. IEMs include inherited biochemical disorders in which a specific enzyme defect interferes with the normal metabolism of protein, fat, or carbohydrate.

As a result of diminished or absent enzyme activity in these disorders certain compounds accumulate in the body to toxic levels, and levels of other compounds that the body normally may become deficient. Without appropriate and accessible management, these metabolic disturbances can lead to a host of medical and developmental consequences, ranging from INTELLECTUAL DISABILITY TO SEVERE COGNITIVE IMPAIRMENT AND EVEN DEATH. Management may include one or a combination of the following: drug therapy, modification of normal diet, or use of a medical food. For these IEMs, a medical food is required in addition to a specific dietary modification in order to obtain adequate levels of essential nutrients (e.g., essential amino acids, essential fatty acids) that are restricted by modifying diet. Medical foods become INDESPINSABLE for individuals with these IEMs in order to meet the daily requirements of essential nutrients and to limit metabolic disturbances associated with those particular IEMs. [2]

The specific ingredients in LUMA TC address multiple IEMs that affect the disease processes listed in the usage section.

Usage: LUMA TC Tablets can be used for patients with the distinct nutritional requirements for the dietary management of certain neuronal metabolic processes (Inborn Errors of Metabolism) of a genetic, poor aging or environmental nature identified with hyperhomocysteinemia, oxidative stress, CNS inflammation, heavy metal toxicity, beta amyloid protein plaque production apoptosis, tau protein tangle production apoptosis, and to promote the positive production of the Neurotrophins, Brain Derived Neurotrophic Factor, Nerve Growth Factor, Vascular Endothelial Growth Factor, and Fibroblast Growth Factor, resulting in neurogenesis and restoration of neurons for the prevention and or management of Mild Cognitive Impairment and Major Depressive Disorder and to Prevent, Manage and Restore Neuronal Tissue, and to Treat Early Dementia associated with the Alzheimer's and Parkinson's Disease process and Traumatic Brain Injury. [1-27]

ADVERSE REACTIONS: Allergic reactions have been reported following the use of the ingredients in Luma TC. Mild transient diarrhea, polycythemia vera, itching, transitory exanthema, and the feeling of swelling, acne, skin reactions, photosensitivity, nausea, vomiting, abdominal pain, loss of appetite, paresthesia, somnolence, nausea, and headaches have been reported with the ingredients in Luma TC. In general, side effects associated with the ingredients in Luma TC have occurred at much higher doses than those found in Luma TC. Luma TC is a natural coenzyme Medical Food not a drug and can be considered generally safe.

The safety of nutritional micro-dose lithium orotate is comparable to low dose forms of other nutrients such as zinc. In fact, nutritional micro-dose lithium orotate has a much wider therapeutic and biologically compatible (non-toxic) window than zinc. The U.S. Environmental Protection Agency has estimated that the daily lithium intake of an average adult ranges from about 0.65mg. to 3mg naturally with intake of food and water. Nutritional micro-dose lithium has been officially added to the World Health organization's list of nutritionally essential trace elements alongside zinc, iodine, and others. The provisional RDA for nutritional micro-dose lithium is 1mg per day. Doses up to 40mgs a day are very safe with a low incidence of side effects. After 30 years of nutritional micro-dose use of lithium orotate internationally it has been shown to be completely free of negative side effects and without toxicity to the brain, heart, kidneys and liver. Nutritional micro-dose lithium orotate 2.5 mgs will not cause weight gain sedation or sleepiness. Nutritional micro-dose lithium orotate does not require blood tests to establish a therapeutic level as prescription forms do, nor is it toxic to the kidneys like lithium Rx pharmaceuticals. It is important to understand that most side effects and reactions are dose dependent and none should be expected at nutritional micro-dose lithium orotate 2.5 mg doses such as in Luma TC. A typical dosage of the drug form lithium carbonate for Rx bipolar depression is 337 mgs. of elemental lithium that contrasts with the 2.5mgs in Luma TC. Nutritional micro-dose lithium orotate is highly bioavailable orotate chelate and functions as a targeted delivery system to the brain. Therefore, the nutritional micro-dose lithium orotate can be used with proven effectiveness in the Luma TC indicated IEM claims in much lower servings with remarkable results with no side effects. [18,3,4,15,11,27]

DRUG INTERACTIONS/PRECAUTIONS: This product is contraindicated in patients with a known hypersensitivity to any of the ingredients contained in this product. The chronic use of curcumin could cause liver toxicity at high doses. For this reason, Luma TC should be used with caution in individuals with liver disease, heavy drinkers or alcoholics and those who take prescription medications that are metabolized in the liver. Curcumin was found to be safe in human clinical trials with doses up to 10 grams a day. Curcumin may interact with "blood thinning" drugs NSAIDs, reserpine. Curcumin is not recommended for persons with biliary tract obstruction. The amount of oxacylic acid in LUMA TC nano particle curcumin is 0.005 mg. which is about 25 parts per million and carries an extremely low risk of kidney stone development even in patients with kidney stone propensity.

Consult your medical professional before use during pregnancy.
Talk to your licensed medical practitioner, healthcare practitioner, personal physician, and/or pharmacist before taking or using any prescription, over-the-counter medicines. Call your medical practitioner about side effects. You may report side effects by calling (866) 289-5961.

DOSAGE AND ADMINISTRATION: The usual adult dose for prevention and or management is one (1) tablet daily, or as directed by a licensed medical practitioner.

HOW SUPPLIED: Luma TC is supplied as a beige oblong pill in bottles of 90(08225-951-90) and can be purchased at JayMac's exclusive distributer DVD at phone # 985-629-5742. For more information please see the LumaTC website at lumaforlife.com

STORAGE: Store at Controlled Room Temperature 15°-30° C (59°-86°F). [See USP]. Protect from light and moisture. Dispense in a tight, light-resistant container.

MANUFACTURED FOR:
JAYMAC PHARMACEUTICALS, LLC.
Sunset, Louisiana MADE IN USA

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