For the use of only a Registered Medical Practitioner or a Hospital or a Laboratory

Not to be sold by retail without the prescription of a Registered Medical Practitioner

Prescribing Information

1. Generic Name

Methylcobalamin, Alpha Lipoic Acid, Pyridoxine Hydrochloride, and Folic Acid Capsules

(Brand Name: MEGO®-XL Capsules)

2. Qualitative and Quantitative Composition

Each hard gelatin capsule contains:

Methylcobalamin IP …….. 1500 mcg.

Alpha Lipoic Acid USP ……..…… 100 mg.

Pyridoxine Hydrochloride IP ……. 3 mg.

Folic Acid IP ……... 1.5 mg.

Excipients ……. q.s.

Containing Folic Acid and Methylcobalamin Tablets IP (as film coated).

Colour: Titanium Dioxide and Red Oxide of Iron.

Colour used in capsule shell: Ponceau 4R, Carmoisine, Titanium Dioxide.

Methylparaben and Propylparaben used as antimicrobial preservative.

Overages are added to compensate loss on storage.

3. Dosage Form and Strength

Dosage Form: Capsule.

Dosage Strength: Methylcobalamin 1500 mcg, alpha lipoic acid 100 mg, pyridoxine hydrochloride 3 mg, and folic acid 1.5 mg per capsules.

4. Clinical Particulars

4.1 Therapeutic Indication

For therapeutic use.

MEGO-XL Capsules are indicated for the treatment of peripheral neuropathies including diabetic neuropathy in adults.

4.2Posology and Method of Administration

For oral administration in adults.

Usual dose is one capsule of MEGO-XL to be administered once daily. Therapy is usually given for 8 to 12 consecutive weeks.

Or, as prescribed by the physician.

4.3Contraindications

MEGO-XL Capsules are contraindicated in the following:

- Hypersensitivity to methylcobalamin, alpha lipoic acid, pyridoxine, folic acid or to any component of the formulation.
- An existing hypervitaminosis.

4.4Special Warnings and Precautions for Use

Methylcobalamin

Methylcobalamin is susceptible to photolysis. Thus, MEGO-XL Capsules should not be exposed to light/moisture.

The prolonged use of larger doses of methylcobalamin is not recommended for patients whose occupation requires the handling of mercury or mercury compounds.

Those with early Leber's disease (hereditary optic nerve atrophy) when treated with vitamin B12 can develop swift, and severe optic atrophy. MEGO-XL Capsules must be advocated with caution in such patients.

Alpha Lipoic Acid

Alpha lipoic acid should be avoided in alcoholics, thiamine deficiency, thyroid disease, and 2 weeks prior to surgery. Blood glucose must be regularly monitored while taking alpha lipoic acid due to its hypoglycemic action.

Pyridoxine

Pyridoxine may decrease the efficiency of levodopa. Pyridoxine should be advocated cautiously with drugs such as oral hypoglycemics, anticonvulsants, furosemide, isoniazid, penicillamine, hydralazine, and oral contraceptives.

Folic Acid

Taking folic acid might mask anemia caused by vitamin B12 deficiency and delay appropriate treatment. Taking folic acid supplements might make seizures worse in people with seizure disorders, particularly in high doses.

4.5Drug Interactions

Methylcobalamin

Oral Contraceptives: Serum concentrations of methylcobalamin may be decreased by use of oral contraceptives.

Chloramphenicol: Chloramphenicol should not be used with methylcobalamin. Parenteral chloramphenicol may attenuate the effect of vitamin B12 in anemia.

Other Drugs: Excessive alcohol consumption, anti-acne drugs, anti-retrovirals, anti-gout drugs (colchicine), antihypertensives, anti-tubercular drugs (aminosalicylic acid), anti-ulcer drugs, biguanides (metformin), H2 antagonists (cimetidine, ranitidine), proton pump inhibitors, sulfonamides, tetracyclines, aminoglycosides and antiepileptic drugs can reduce vitamin B12 levels. Thus, caution should be exercised while co-administering vitamin B12 with these drugs.
Drug/Laboratory Test Interactions: Use of antibiotics, methotrexate, and pyrimethamine may invalidate vitamin B12 diagnostic blood assays.

Alpha Lipoic Acid

There is some concern that antioxidants might decrease the effectiveness of some medications used for cancers. Alpha lipoic acid might decrease blood sugar levels. Taking alpha lipoic acid along with antidiabetic medications such as insulin, sulphonylureas, and glitazones might cause decrease in blood sugar level.

Pyridoxine

Pyridoxine reduces the effects of levodopa and activity of altretamine. It also decreases serum concentrations of phenobarbital and phenytoin. Pyridoxine may decrease antibiotic activities of erythromycin, kanamycin, streptomycin, doxycycline, and lincomycin. Drugs such as hydralazine, isoniazid, penicillamine, and oral contraceptives may increase the requirements for pyridoxine.

Folic Acid

Phenytoin: Folic acid may increase phenytoin metabolism and lower the serum concentration of phenytoin resulting in increased seizure activity. Also, phenytoin may decrease serum folic acid concentrations.
Methotrexate: Folic acid may decrease a patient's response to methotrexate therapy.
Barbiturates: Folate reduces serum barbiturate concentrations.
Other Drugs and Alcohol: Folate deficiency states may be produced by drugs such as antiepileptics, oral contraceptives, anti-tuberculosis drugs, alcohol, and folic acid antagonists such as methotrexate, pyrimethamine, triamterene, trimethoprim, and sulfonamides.
Drug/Laboratory Test Interactions: Most of the antibiotics, methotrexate and pyrimethamine may invalidate folic acid diagnostic blood assays.

4.6Use in Special Populations

Pregnant Women

There are no adequate and well controlled studies of this combination therapy in pregnant women. MEGO-XL Capsules should be used during pregnancy only if the potential benefit justifies the possible risk to the fetus.

Lactating Women

It is not known whether components of MEGO-XL Capsule are excreted in human milk. Caution should be exercised when this product is administered to a nursing woman. Nursing mothers should not use this preparation unless clearly needed and recommended by physician.

Paediatric Patients
The safety and efficacy of this formulation has not been established in the paediatric population. MEGO-XL Capsules are not intended for use in children.

Geriatric Patients
Generally, dose adjustment is not required in the geriatric population. Elderly patients with normal renal and hepatic function may be given the same dose as recommended for adults.

Renal and Hepatic Impairment Patients
This formulation has not been studied in patents with hepatic and renal impairment. Caution should be exercised when MEGO-XL Capsules are administered to these patients and it is recommended to monitor renal and hepatic functions.

4.7Effect on Ability to Drive and Use Machines
MEGO-XL Capsules are unlikely to cause any impact on the ability to drive and use machines.

4.8Undesirable Effects
Methylcobalamin
Rash, anorexia, nausea, vomiting, diarrhea, headache has been reported with methylcobalamin therapy. Other reported adverse reactions include anaphylactic reaction such as decrease in blood pressure or dyspnea, hot sensation, diaphoresis.

Alpha Lipoic Acid
Side effects from using alpha lipoic acid appear to be rare and mild, such as nausea or skin rash.

Pyridoxine
Pyridoxine usually has no side effects when used in recommended doses. Pyridoxine can cause side effects such as headache, nausea, drowsiness, numbness/tingling of arms/legs when taken in large doses and for a longer period of time.

Folic Acid
Folic acid is generally well tolerated. Gastrointestinal disturbances and allergic reactions have been reported rarely with use of folic acid.

4.9Overdose
Methylcobalamin

Data regarding overdose with methylcobalamin is limited. Methylcobalamin has excellent tolerability and no known toxicity. In the event of overdose, treatment should be symptomatic and supportive.

Alpha Lipoic Acid

No overdose has been reported with alpha lipoic acid. Symptoms of overdose may include hypoglycemia, headache, feeling hungry, weakness, sweating, confusion, irritability, dizziness, tachycardia, or feeling jittery. If overdose occurs, treatment should be symptomatic and supportive.

Pyridoxine

Pyridoxine can cause neurological disorders, such as loss of sensation in legs and lack of balance/coordination, when taken in high doses (200 mg or more per day) over a long period of time. Pyridoxine/vitamin B6 toxicity can damage sensory nerves, leading to numbness in the hands and feet as well as difficulty in walking. Symptoms of a pyridoxine overdose may include poor coordination, staggering, numbness, decreased sensation to touch, temperature, vibration and tiredness for up to 6 months.

Folic Acid

Toxicity from excessive folic acid intake does not normally occur as folic acid is water soluble and regularly excreted by the body. High levels of folic acid can provoke seizures in patients taking anticonvulsant medications.

5. Pharmacological Properties

5.1 Mechanism of Action

Methylcobalamin

Methylcobalamin regulates nerve function and reduces plasma homocysteine levels by following mechanisms:

1. Methylcobalamin promotes myelination (phospholipid synthesis): Methylcobalamin promotes the synthesis of lecithin, the main constituent of medullary sheath lipid and increases myelination of neurons in rat tissue culture more than cobamamide does.

2. Methylcobalamin promotes axonal transport and axonal regeneration: Methylcobalamin normalizes axonal skeletal protein transport in sciatic nerve cells from rat models with streptozotocin-induced diabetes mellitus. It exhibits neuropathologically and electrophysiologically inhibitory effects on nerve degeneration in neuropathies induced by drugs, such as adriamycin, acrylamide, and vincristine (in rats and rabbits), models of axonal degeneration in mice and neuropathies in rats with spontaneous diabetes mellitus.

3. Methylcobalamin is a kind of endogenous coenzyme B12: Methylcobalamin plays an important role in transmethylation as a coenzyme of methionine synthetase in the synthesis of methionine from homocysteine.

4. Methylcobalamin is well transported to nerve cell organelles, and promotes nucleic acid and protein synthesis: Methylcobalamin is better transported to nerve cell organelles than cyanocobalamin in rats. Also, methylcobalamin promotes nucleic acid and protein synthesis in rats more than cobamamide does.

Alpha Lipoic Acid

As an antioxidant, alpha lipoic acid (ALA) directly terminates free radicals, chelates transition metal ions, increases cytosolic glutathione and vitamin C levels, and prevents toxicities associated with their loss. These diverse actions suggest that ALA acts by multiple mechanisms both physiologically and pharmacologically.

Pyridoxine

Pyridoxine/vitamin B6 is a water soluble vitamin required for amino acid, carbohydrate, and fat metabolism. Pyridoxine have role as a coenzyme in a wide variety of enzymes involved in cell growth and cell division.

High homocysteine level in the blood (hyperhomocysteinemia) is a risk factor for cardiovascular disease, blood clotting abnormalities, myocardial infarction (heart attack), and ischemic stroke. Pyridoxine alone or in combination with folic acid has been shown to be effective for lowering homocysteine levels.

Folic Acid

Folic acid is reduced in the body to tetrahydofolate, which is a coenzyme for various metabolic processes including the synthesis of purine and pyrimidine nucleotides, and hence the synthesis of DNA.

5.2Pharmacodynamic Properties

Methylcobalamin

Methylcobalamin is the neurologically active form of vitamin B12. Methylcobalamin is useful to treat or correct various neurological defects such as neuropathies. In many cases, liver does not convert cyanocobalamin, the commonly available form of vitamin B12, into adequate amounts of methylcobalamin. Nutritional inadequacies, enzyme defects, and pathological changes to tissues can all contribute to a reduced ability of the body to accomplish the synthesis of the active forms of vitamin B12 from cyanocobalamin. MEGO-XL Capsule provides readymade form of vitamin B12 i.e., methylcobalamin to treat various types of neuropathies including diabetic neuropathy.

Alpha Lipoic Acid

Alpha-lipoic acid is an antioxidant also known as ALA, Acetate Replacing Factor, Biletan, Lipoicin or Thioctic Acid. Alpha lipoic acid is a naturally occurring fatty acid that can be found in many foods. Alpha lipoic acid seems to delay or reverse peripheral diabetic neuropathy through its multiple antioxidant properties. ALA improves glycemic control and polyneuropathies

associated with diabetes mellitus. ALA enhances glucose uptake (thus, lowers plasma glucose levels) in type 2 diabetes patients. ALA has been useful to provide relief from symptoms caused by diabetic neuropathy such as the pain, burning, tingling, and numbing. Its biosynthesis decreases as people age and is reduced in people with compromised health, suggesting a supplementation of ALA in such cases.

Pyridoxine

Pyridoxine is essential for cell growth and cell division. Pyridoxine also involves in carbohydrate, protein, and fat metabolism. Pyridoxine also reduces homocysteine levels in the blood.

Folic Acid

Folic acid is used in the treatment and prevention of the folate deficiency state. It is also involved in some amino-acid conversions, and in the formation and utilization of formate.

5.3Pharmacokinetic Properties

Methylcobalamin

Absorption: Vitamin B12 binds to intrinsic factor, a glycoprotein secreted by the gastric mucosa, and is then actively absorbed from the gastrointestinal tract. A small amount of vitamin B12 can also be absorbed from the gastrointestinal tract by passive diffusion. When methylcobalamin was administered orally to healthy adult male volunteers at single doses of 120 mcg and 1500 mcg during fasting, the peak serum total vitamin B12 concentration was reached after 3 hours for both doses, and this was dose-dependent.

Distribution: Vitamin B12 is extensively bound to specific plasma proteins called transcobalamins; transcobalamin II appears to be involved in the rapid transport of the cobalamins to tissues. Vitamin B12 diffuses across the placenta and also appears in breast milk.

Metabolism and Excretion: Vitamin B12 is stored in the liver and excreted in the bile. It undergoes extensive enterohepatic recycling; a part of the dose is excreted in the urine, most of it in the first 8 hours. About 40 to 80% of the cumulative amount of total B12 is excreted in the urine within 24 hours. Elimination half-life of methylcobalamin is 12.5 hours after a single-dose oral administration.

Alpha Lipoic Acid

After oral administration, alpha lipoic acid is readily and nearly completely absorbed with a limited absolute bioavailability of about 30% caused by high hepatic extraction. The primary metabolic pathways of alpha lipoic acid are s-methylation and beta-oxidation. Major circulating metabolites were the s-methylated beta-oxidation products 4, 6-bismethylthio-hexanoic acid and 2,4-bismethylthio-butanoic acid, whereas its conjugated forms accounted for the major portion excreted in urine. Despite the prolonged half-lives of the major metabolites compared to the parent drug, no evidence of accumulation was found. Mean values of 12.4% of the administered dose were recovered in the urine after 24 hours as the sum of alpha lipoic acid and its metabolites.

Pyridoxine

Pyridoxine hydrochloride is absorbed rapidly from the upper intestine regardless of the size of the dose given. Absorption may also occur from ileum and to a small extent from the colon. Pyridoxine is rapidly converted in the liver to pyridoxine phosphate, pyridoxal phosphate and pyridoxamine phosphate via oxidation. This causes the release of pyridoxal and some pyridoxal phosphate to the general circulation where it reaches other organs chiefly as circulating pyridoxal. Pyridoxine and its metabolites are stored mainly in the liver where there is oxidation to 4-pyridoxic acid and other inactive metabolites. Pyridoxal crosses the placenta and is also distributed into breast milk. Pyridoxine is excreted primarily unchanged in the urine; with a small amount in the form of metabolite, most likely 4-pyridoxic acid. As the dose increases, proportionally greater amounts are excreted unchanged in the urine.

Folic Acid

Folic acid is rapidly absorbed from the gastrointestinal tract, mainly from the duodenum and jejunum. The naturally occurring folate polyglutamates are largely deconjugated, and then reduced by dihydrofolate reductase in the intestines to form 5-methyltetrahydrofolate, which appears in the portal circulation, where it is extensively bound to plasma proteins. Folic acid given therapeutically enters the portal circulation largely unchanged, since it is a poor substrate for reduction by dihydrofolate reductase. Folic acid is converted to the metabolically active form 5-methyltetrahydrofolate in the plasma and liver. Folate is distributed into breast milk. The principal storage site of folate is the liver; it is also actively concentrated in the cerebrospinal fluid (CSF). Folate undergoes enterohepatic circulation. Folate metabolites are eliminated in the urine and folate in excess of body requirements is excreted unchanged in the urine. Folic acid is removed by haemodialysis.

6. Nonclinical Properties

6.1 Animal Toxicology

Methylcobalamin

Repeated dose toxicity: A total of 48 Sprague-Dawley rats were randomly assigned to receive, intravenously, 1, 5, 25 or 100 mg/kg of cyanocobalamin (6 males and 6 females in each group) three times per week until completion of the study at 182 days (26 weeks). The animals were weekly examined and blood samples were taken at days 1, 85 and 182 for cyanocobalamin determination. Finally, all animals survived throughout the study period and had similar growth rates. No evidence of toxicity was detected by a detailed weekly examination of animals during the study period. Therefore the no-observed-adverse-effect-level (NOAEL) can be established at least at 100 mg/kg under the test conditions.

Carcinogenicity: Vitamin B12 (as cyanocobalamin or hydroxocobalamin) has a long history of safe use even at high doses. A tumour promoting effect of vitamin B12 has been reported in one study in rats. Rats kept on a methionine deficient diet supplemented with 5 µg/100 g vitamin B12

and treated with the carcinogen p-dimethylaminobenzene (DAB) had a higher incidence of hepatomas compared to the group without supplemental vitamin B12. A control group receiving the supplemented diet without DAB showed no hepatic tumours. In another study, the effect of methylcobalamin and cyanocobalamin on the growth of Walker's carcinosarcoma and on the longevity of rats with implanted Zajdela ascites hepatoma cells has been studied. Study reported reduced survival of rats upon treatment with both compounds.

Alpha Lipoic Acid

In acute toxicity studies, the cat was the most sensitive species, followed by the monkey, dog, mouse and rat. Route of administration impacts toxicity (intravenous was most toxic, followed by intraperitoneal, subcutaneous, and oral). The liver and kidney were targets of toxicity in short term studies in rats and cats. No toxicity was seen following chronic exposure in dogs. No data are available for reproductive/developmental toxicity. ALA is not mutagenic in the Ames and micronucleus genotoxicity assays. Carcinogenicity assessment was negative.

Ant-mutagenic effects: Study investigated both the mutagenicity and anti-mutagenicity of alpha-lipoic acid (ALA) in the bone marrow cells of mice using a chromosomal aberration assay. Cyclophosphamide (CP) 40 mg/kg was used as a clastogen in the positive control, and a vehicle-treated negative control group was also included. Multiple dose levels (15, 30, and 100 mg/kg of ALA) were given by intraperitoneal injection (IP) alone and in combination with CP (CP was administered 1 h prior to ALA). Bone marrow samples were collected 12 and 24 h after drug administration. The results demonstrated a significant increase in the frequency of chromosomal aberrations (CA) in bone marrow cells with depressions in the mitotic index (MI) of the positive control group of mice. However, in the groups of mice treated with different doses of ALA in the presence of CP, the percentages of CA decreased significantly with increases in mitotic activity. The results also indicate that ALA given alone in different doses had no mutagenic effect on mouse bone marrow cells. ALA has a dose and time-dependent protective effect against the mutagenicity induced by CP.

Pyridoxine

Acute oral toxicity: The acute oral toxicity of ten batches of pyridoxine hydrochloride was tested in mice. After 10 days observation period, the LD50 was found to be 6994 mg/kg body weight.

Repeated dose toxicity - oral: Rats and mice were orally exposed to pyridoxine hydrochloride for 10 days. Only at the lowest test doses (2000 mg/kg/day (mice) and 500 mg/kg/day (rats)) no mortality was seen. Rats and mice dosed at the highest dose (16000 mg/ kg/day) died on the first day. At 8000 mg/kg/day, exposed mice and rats died after second dosing. Pyridoxine influenced body weight gain of mice and rats in a dose-dependent way.

Mutagenicity: An Ames test was performed with pyridoxine. All bacterial strains showed negative responses up to 5000 ug/plate, i.e. no significant dose-related increase in the number of revertants with or without metabolic activation was seen. No cytotoxicity and/or precipitation of the test substance was observed. The negative and strain-specific positive control values were within the

laboratory historical control data ranges indicating that the test conditions were adequate and that the metabolic activation system functioned properly. Based on the results of this study it is concluded that pyridoxine is not mutagenic in the *Salmonella typhimurium* reverse mutation assay and in the *Escherichia coli* reverse mutation assay with or without metabolic activation.

Folic Acid

Animal studies have shown that folic acid can be a neurotoxin, and can cause convulsions in laboratory animals. This evidence is in part based upon in vitro tissue and cell culture studies, and/or using very high dose levels (i.v. dosages 60-90 mg). There is however no clear evidence for a folic acid-induced neurotoxicity in humans.

Toxicity in different strains of mice showed that toxic doses of folic acid may lead to convulsions, ataxia and weakness. Histopathological studies in some strains of mice showed acute renal tubular necrosis.

In another study with experimentally (diet) induced vitamin B12 deficiency in rhesus monkeys, three of the nine monkeys received 5 mg/week of supplemental folic acid intramuscularly, followed by 5 mg in the drinking water (5 days/week). Five animals developed visual impairment and optic atrophy, including the 3 monkeys that received supplemental folic acid. Apparently, the optical nerve lesions occurred earlier (by 10-11months) in the folic acid-treated animals. It should be noted that the visual lesions observed in these monkeys are only rarely noted in human disease. Spastic paralysis of hind legs and tail was found in 3 animals, including 2 animals receiving folic acid. Other lesions in cranial and peripheral nerves and in the white matter of the spinal cord were observed in some animals, but were apparently not affected by supplemental folic acid.

7. Description

MEGO-XL Capsules are scarlet size 1 hard gelatin capsule containing light yellow coloured granular powder with a round, biconvex, brown coloured film coated tablet (Folic Acid and Methylcobalamin Tablets IP).

Each capsule of MEGO-XL contains 1500 mcg of methylcobalamin, 100 mg of alpha lipoic acid, 3 mg of pyridoxine hydrochloride, and 1.5 mg of folic acid for oral administration.

Methylcobalamin

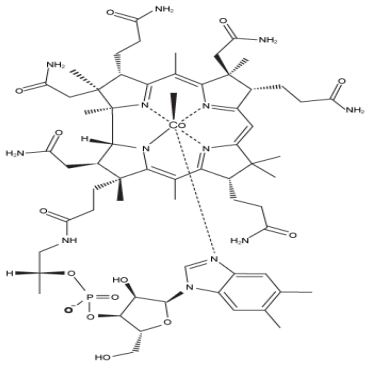
Methylcobalamin appears as dark red crystals or crystalline powder. It is sparingly soluble in water, slightly soluble in ethanol and practically insoluble in acetonitrile.

Molecular Weight: 1344.38 g/mol.

Molecular Formula: C63H91CoN13O14P.

Chemical Name: Methyl-5, 6-dimethylbenzimidazolylcobalamin.

Structural Formula:

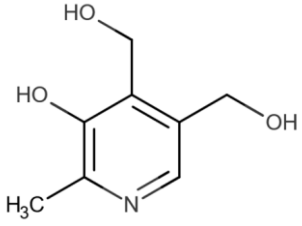


Alpha Lipoic Acid

Alpha lipoic acid is a dithiol compound derived from octanoic acid. Alpha lipoic acid appears as yellow crystalline powder which is soluble in methanol, ethanol, diethyl ether and chloroform.
Molecular Weight: 206.3 g/mol.
Molecular Formula: C8H14O2S2.
Chemical Name: 5-(dithiolan-3-yl) pentanoic acid.
Structural Formula:

Pyridoxine

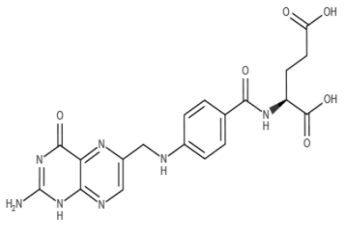
Pyridoxine hydrochloride is a white or practically white crystals or crystalline powder, soluble in water and insoluble in ether.
Molecular Weight: 169.18 g/mol.
Molecular Formula: C8H11NO3.
Chemical Name: 4,5-bis(hydroxymethyl)-2-methylpyridin-3-ol.
Structural Formula:



Folic Acid

Folic acid is a yellow, yellow-brownish, or yellowish orange, odourless crystalline powder. Very slightly soluble in water; insoluble in alcohol, in acetone, in chloroform, and in ether.
Molecular Weight: 441.40 g/mol.

Molecular Formula: C19H19N7O6.
Chemical Name: (2S)-2-[(4-{[(2-amino-4-oxo-1,4-dihydropteridin-6-yl)methyl]amino} phenyl) formamido] pentanedioic acid.
Structural Formula:



Inactive ingredients (excipients) of MEGO-XL Capsules contain Microcrystalline Cellulose, Polyvinyl Pyrrolidone, Colloidal Silicon Dioxide, Magnesium Stearate, Sodium Starch Glycolate, Dibasic Calcium Phosphate, Ethyl Cellulose, Stearic Acid, Talcum, Starch, Lactose, and Hard Gelatin Capsule Shell.

8. Pharmaceutical Particulars

8.1 Incompatibilities

None known.

8.2Shelf-life

18 months.

8.3Packaging Information

10 capsules per strip.

8.4Storage and Handling Instructions

Store at temperature not exceeding 25°C and protect from light and moisture.
Keep out of reach of children.

9. Patient Counseling Information

Instructions to Patients

- Instruct patients not to change their medication dose or schedule without consulting doctor or pharmacist. Do not exceed the recommended dose or duration of treatment.
- Pregnant women and nursing mothers can use this medicine only in consultation with their doctor.
- This medicine is not recommended for use in children.
- Instruct patients not to share their medication with others even though it has been prescribed for same disease/condition. Also, not to use medication prescribed for others.

- MEGO-XL Capsules should not be exposed to light/moisture because methylcobalamin is susceptible to degradation by photolysis.

10. Details of Manufacturer

Akums Drugs & Pharmaceuticals Ltd.
Plot No. 26A, 27-30, Sector-8A, I.I.E., SIDCUL,
Ranipur, Haridwar – 249 403, Uttarakhand.

11. Details of Permission or License Number with Date

Mfg. Lic. No.: 8/UA/LL/SC/P-2014; Date of FDA Product Permission: 15/09/2015.

12. Date of Revision

April 2021.

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