Summary of Product Characteristics

1. Name of the medicinal product

MELOPRES 50 (Metoprolol Tartrate tablets USP 50 mg)

2. Qualitative and quantitative composition

Each uncoated tablets contains:
Metoprolol Tartrate USP 50 mg

For the full list of excipients, see section 6.1.

3. Pharmaceutical form

Uncoated tablet
White, circular flat beveled edges uncoated tablets, having break line on one side and plain on other side.

4. Clinical particulars

4.1 Therapeutic indications

Metoprolol tartrate is indicated in adults for:

- Hypertension.
- Angina pectoris.
- Tachycardiac arrhythmia, particularly supraventricular tachycardia.
- Prevention of cardiac death and re-infarction after the acute phase of myocardial infarction.
- Prophylaxis of migraine.

4.2 Posology and method of administration

Posology

The dose must always be adjusted to the individual requirements of the patient but should not exceed 400 mg/day. The following are guidelines:

Adults:

Hypertension: Initially 100 mg daily. This may be increased, if necessary, to 200 mg daily in single or divided doses. Combination therapy with another antihypertensive agent may also be considered to further reduce blood pressure.

Angina pectoris: Usually 50-100 mg twice daily. The dose may be further increased or combined with nitrates.
Tachycardiac arrhythmias: A daily dose of 100 -200 mg is usually sufficient. If necessary the dose may be increased.
After acute intravenous treatment of myocardial infarction: Orally, therapy should commence 15 minutes after the last intravenous injection with 50 mg every 6 hours for 48 hours.
Prophylaxis after myocardial infarction: Maintenance dose is 100 mg twice daily.
Prophylaxis of migraine: 50-100 mg twice daily.

Patients with renal impairment

The rate of elimination is insignificantly affected by renal function and therefore no dose adjustment is needed.

Patients with hepatic impairment

Usually metoprolol can be given at the same dose to patients with cirrhosis of the liver as to patients with normal hepatic function. A dose reduction should only be considered when there are signs of severely impaired hepatic function (i.e. shunt operated patients)

(see Section 5.2).

Elderly patients
There are no adequate data from the use in patients above the age of 80. Take special precautions when increasing the dose. However, caution is advised in elderly patients as a fall in blood pressure or excessive bradycardia may have more pronounced effects.

Paediatric population:
There is limited data on the use of metoprolol in children and adolescents, therefore the use of Metoprolol tartrate is not recommended.

Method of administration
The tablets should be taken on an empty stomach (see section 5.2).

4.3 Contraindications

- Hypersensitivity to metoprolol, other beta blockers or to any of the excipients listed in section 6.1.
- Grade II or III atrioventricular block.
- Patients with unstable or acute decompensated heart failure (pulmonary oedema, hypoperfusion or hypotension), in which case continuous or periodical intravenous inotropic β receptor agonist therapy is indicated.
- Manifest and clinically significant sinus bradycardia (heart frequency < 50/min.).
- Sick sinus syndrome.
- Cardiogenic shock.
- Severe peripheral arterial disease.
- Hypotension (systolic < 90 mmHg).
- Metabolic acidosis.
- Severe bronchial asthma or chronic obstructive pulmonary disease.
- Higher grade sinoatrial block

Metoprolol may not be administered to patients with suspected acute myocardial infarction and a heart rate of < 50 beats/min., PQ interval > 0.24 seconds or systolic blood pressure < 100 mmHg.
Concomitant intravenous administration of calcium blockers of the type verapamil or diltiazem or other antiarrhythmics (such as disopyramide) is contraindicated (exception: intensive care unit) (see section 4.5). Untreated phaeochromocytoma.

4.4 Special warnings and precautions for use

Beta blockers must be administered with caution to asthmatics. If an asthmatic uses a beta-2 agonist (as tablets or by inhalation) when initiating metoprolol treatment, the dose of the beta-2 agonist must be controlled and increased if necessary.
Metoprolol may reduce the effect of diabetes treatment and mask the symptoms of hypoglycaemia AV conduction disorders may be aggravated in rare cases in connection with metoprolol treatment (possible atrioventricular block). Beta-blockers should be given only with caution to patients with first degree atrioventricular block (see section 4.3). Metoprolol may exacerbate the symptoms of peripheral vascular disorders due to its antihypertensive effect. When prescribing metoprolol to patients with a pheochromocytoma, an alpha blocker must be used before initiating treatment and during the metoprolol treatment. In patients with Prinzmetal's angina β1 selective agents should be used with care because may increase the number and duration of angina attacks.
Metoprolol treatment may possibly mask the symptoms of thyreotoxicosis. Therefore, metoprolol should be administered with caution to patients having or suspected of developing thyreotoxicosis and both thyroid and cardiac functions should be monitored closely. Before surgery, the anaesthesiologist must be informed that the patient takes beta blockers. It is not recommended to discontinue beta blocker treatment during a surgical

procedure. Beta blocker treatment must not be suddenly discontinued. If the treatment is to be discontinued, it must, where possible, be gradually reduced over a period of at least two weeks during which the dose is withdrawn gradually, the doses diminishing to 25 mg for the last 6 days before the treatment is discontinued. If the patient presents with any symptoms, the dose should be reduced at a lower rate. Sudden discontinuation of beta blockers may possibly exacerbate heart failure and increase the risk of myocardial infarction and sudden death.

Like other beta blockers, metoprolol may also increase both the sensitivity to allergens and the severity of anaphylactic reactions. Adrenalin treatment does not always give the desired therapeutic effect in individuals receiving beta blockers (see also section 4.5). Beta blockers may trigger or exacerbate psoriasis.

Up to the present, there is insufficient data from the use of metoprolol in patients with heart failure and the following accompanying factors:

- Unstable heart failure (NYHA IV).
- Acute myocardial infarction or unstable angina pectoris in the preceding 28 days.
- Impaired renal function.
- Impaired hepatic function.
- Patients above the age of 80.
- Patients under the age of 40.
- Haemodynamically significant valve diseases.
- Hypertrophic obstructive cardiomyopathy.
- During or after cardiac surgery within the last four months before treatment with metoprolol.

In the case of increasing bradycardia the dosage should be reduced, or treatment gradually discontinued.
Metoprolol tartrate may not be administered to patients with untreated congestive heart failure. The congestive heart failure needs to be brought under control first of all. If concomitant digoxin treatment is taking place, it must be borne in mind that both medicinal products slow AV conduction and that there is therefore a risk of AV dissociation. In addition, mild cardiovascular complications may occur, manifesting as dizziness, bradycardia, and a tendency to collapse.

Dry eyes either alone or, occasionally, with skin rashes has occurred. In most cases the symptoms cleared when metoprolol treatment was withdrawn. Patients should be observed carefully for potential ocular effects. If such effects occur, discontinuation of metoprolol should be considered.

This medicine contains less than 1 mmol sodium (23 mg) per tablets, that is to say essentially 'sodium-free'.

4.5 Interaction with other medicinal products and other forms of interaction

The following combinations with metoprolol should be avoided:

Barbituric acid derivatives Barbiturates (studied for pentobarbital) induce the metabolism of metoprolol through enzyme induction.

Propafenon When propafenon was commenced in four patients, who were then treated with metoprolol, the plasma concentrations of metoprolol increased 2-5-fold and two patients suffered typical metoprolol side effects. The interaction was confirmed in a study involving eight healthy research subjects. The interaction is probably due to the fact that propafenon, like quinidine, inhibits the metabolism of metoprolol via

cytochrome P450 2D6. The combination is probably difficult to manage due to the fact that propafenon also has beta-receptor blocking properties.

Calcium antagonists In the case of the concomitant use of calcium antagonists of the verapamil or diltiazem types, an increase in negative inotropic and chronotropic effects can occur. Calcium antagonists of the verapamil type should not be administered intravenously to patients who are being treated with beta blockers, due to the risk of hypotension, AV conduction disturbances, and left ventricular insufficiency (see section 4.3). In patients with impaired cardiac function, the combination is contraindicated. As with other beta-blockers, concomitant therapy with dihydropyridines (such as nifedipine and amlodipine), may increase the risk of hypotension, and cardiac failure may occur in patients with latent cardiac insufficiency.

The following combinations with metoprolol may require dose adjustment:

Amiodarone
One case history indicates that patients treated with amiodarone can develop severe sinus bradycardia during concomitant treatment with metoprolol. Amiodarone has an extremely long half-life (approximately 50 days), which means that interactions can occur a long time after discontinuation of the preparation.

Class I-antiarrhythmics
Class I-antiarrhythmics and beta-receptor blockers have additive negative inotropic effects, which can result in serious haemodynamic adverse reactions in patients with impaired left-ventricular function. The combination should be avoided in "sick sinus syndrome" and pathological AV-conduction. The interaction is best documented for disopyramide.

Non-steroidal anti-inflammatory drugs/antirheumatic agents (NSAID)
NSAID-type antiphlogistics counteract the antihypertensive effect of beta-receptor blocking agents. Studies have primarily been performed on indomethacin. This interaction is not believed to occur with sulindac. It has not been possible to demonstrate such an interaction in a study relating to diclofenac.

CYP2D6 inhibitors
Metoprolol is a CYP2D6-substrate. Drugs which inhibit this enzyme may increase the plasma concentration of metoprolol. Examples of clinically significant inhibitors of CYP2D6 are antidepressants such as fluoxetine, paroxetine or bupropion, antipsychotics such as thioridazine, antiarrhythmics such as propafenone, antiretrovirals such as ritonavir, antihistamines such as diphenhydramine, antimalarials such as hydroxychloroquine or quinidine, antifungals such as terbinafine and medications for stomach ulcers such as cimetidine. On commencement of treatment with these medicinal products in patients being treated with metoprolol the dose of metoprolol may need to be reduced.

Diphenhydramine
Diphenhydramine reduces (2.5 times) clearance of metoprolol to alpha-hydroxymetoprolol in fast hydroxylaters via CYP 2 D6, at the same time as the effects of metoprolol are increased.

Digitalis glycosides
Digitalis glycosides in connection with beta-receptor blockers, can increase the atrioventricular conduction time and induce bradycardia.
Epinephrine A dozen reports exist in respect of severe hypertension and bradycardia in

patients treated with non- selective beta-receptor blockers (including pindolol and propanalol), who were administered epinephrine (adrenaline). These clinical observations have been confirmed in studies on healthy research subjects. It has also been suggested that epinephrine, administered as local anaesthesia, may give rise to these reactions on intravasal administration. The risk should be considerably less with cardioselective beta-receptor blockers.

Phenylpropanolamine
Phenylpropanolamine (norephedrine) in single doses of 50 mg may increase the diastolic blood pressure to pathological levels in healthy research subjects. In general, propanolol counteracts the rise in blood pressure triggered by phenylpropanolamine. Beta-receptor blockers may, however, trigger paradoxical hypertensive reactions in patients taking high doses of phenylpropanolamine. Hypertensive crises during treatment solely with phenylpropanolamine have been described in a couple of cases.

Quinidine
Quinidine inhibits the metabolism of metoprolol in so-called "fast hydroxylaters" (just over 90% in Sweden), with significantly increased plasma values and resultant increase in beta blockade. Similar reaction might be expected to occur with other beta-blockers which are metabolized by the same enzyme (cytochrome P450 2 D6).

Sympathetic ganglion blockers, or other beta blockers
Patients who are concomitantly receiving sympathetic ganglion blockers, or other beta blockers (including in the form of eye drops) must continue being monitored.

MAO inhibitors
MAO inhibitors should be used with caution as concomitant administration with beta-blockers may result in bradycardia and an enhanced hypotensive effect. Monitoring of blood pressure and heart rate are recommended during initial use.

Centrally-acting antihypertensives (clonidine, guanfacin, moxonidine, methyldopa, rilmenidine)
Abrupt withdrawal, particularly if prior to beta-blocker discontinuation, may increase risk of "rebound hypertension".

The concomitant use of clonidine with a non-selective beta blocker, and possibly also with a selective beta blocker, medication needs to be continued for some time after beta-blocker therapy is discontinued. Paroxetine may increase plasma levels of metoprolol resulting in increased beta-blocking effects Ergotamine As beta-blockers may affect the peripheral circulation, care should be exercised when drugs with similar activity, e.g. ergotamine are given concurrently Nitrates may enhance the hypotensive effect of metoprolol Parasympathomimetics Concurrent use of parasympathomimetics may result prolonged bradycardia.

Sympathomimetics
Metoprolol will antagonize the β1 effect of sympathomimetic agent but should have little influence on the bronchodilator effects of β2 agonists at normal therapeutic dose.

General anaesthetics
An increase in the cardio-depressive effect due to the concomitant administration of inhalational anaesthetics is possible; however, since beta blockade can prevent excessive fluctuations in blood pressure whilst the patient is intubated and is rapidly antagonised with beta sympathomimetics, concomitant use is not contraindicated (see section 4.4).

Insulin and oral antidiabetic agents
The blood glucose-reducing effect of insulin and oral blood glucose-reducing drugs can be intensified by beta blockers, in particular non-selective beta blockers. In this case, the dosage of the oral blood glucose-reducing drug must be adjusted.

Alpha blockers such as prazosine, tamsulosin, terazosine, doxazosine
Increased risk of hypotension, especially severe orthostatic hypotension.

Floctafenine:
Beta blockers may impede the compensatory cardiovascular reactions associated with hypotension or shock that may be induced by floctafenine

Skeletal muscle relaxant
Curare muscle relaxant with metoprolol enhanced neuromuscular blockade. Blood pressure should be monitored and dosage adjustment of the antihypertensive be made if necessary. Lidocaine Metoprolol can reduce the clearance of lidocaine.

Hepatic enzyme inducers
Enzyme inducing agents (e.g. rifampicin) may reduce plasma concentrations of metoprolol. Mefloquine Increased risk of bradycardia.

Antacid
An increase in the plasma concentrations of metoprolol has been observed when the drug was coadministered with an antacid.

Alcohol
During concomitant ingestion of alcohol and metoprolol the concentration of blood alcohol may reach higher levels and may decrease more slowly.

The effects of metoprolol and other antihypertensive drugs on blood pressure are usually additive. Care should be taken when combining with other antihypertensive drugs or drugs that might reduce blood pressure, such as tricyclic antidepressants, barbiturates and phenothiazines. However, combinations of antihypertensive drugs may often be used with benefits to improve control of hypertension.

4.6 Fertility, pregnancy and lactation

Pregnancy:
Since there are no well-controlled studies of the use of metoprolol in pregnant women, metoprolol may only be used during pregnancy if the benefits to the mother outweigh the risk to the embryo/foetus.
Beta blockers reduce placental perfusion and may cause foetal death and premature birth. Intrauterine growth retardation has been observed after long term treatment of pregnant women with mild to moderate hypertension. Beta Blockers have been reported to cause prolonged delivery and bradycardia in the foetus and the newborn child. There are also reports of hypoglycaemia, hypotension, increased bilirubinaemia and inhibited response to anoxia in newborn children. Therefore the lowest possible dose should be used, and treatment should be discontinued 48-72 hours before the calculated birth date. If this is not possible, the newborn child should be monitored for 48-72 hours post-partum for signs and symptoms of beta blocking (e.g. cardiac and pulmonary complications).
Beta blockers have not shown potential teratogenic activity in animals, but reduced blood flow in the umbilical cord, growth retardation, reduced ossification and increased numbers of foetal and post-natal deaths.

Breast-feeding:
The concentration of metoprolol in breast milk is approximately three times higher than the one in the mother's plasma. Even though the risk of adverse effects in the breastfeeding baby would appear to be low after administration of therapeutic doses of the medicinal product (except in individuals with poor metabolic capacity), breastfeeding babies should be monitored for signs of beta blocking.

4.7 Effects on ability to drive and use machines

As with all beta-blockers, metoprolol may affect patients' ability to drive and operate machinery. It should be taken into account that occasionally dizziness or fatigue may occur. Patients should be warned accordingly. These effects may possibly be enhanced in case of concomitant ingestion of alcohol or after changing to another medicinal product.

4.8 Undesirable effects

Metoprolol is well tolerated, and the undesirable effects are generally mild and reversible. The most commonly reported adverse reactions during treatment is fatigue. Gangrene (in patients with severe peripheral circulatory disorder), thrombocytopenia and agranulocytosis may occur very rarely (less than 1 case per 10,000 patients).The following undesirable effects have been reported during the course of clinical studies or have been reported after routine use. In many cases, a link with the use of metoprolol (tartrate) has not been firmly established.

	Very Common (≥ 1/10)	Common (≥ 1/100 to < 1/10)	Uncommon (≥ 1/1,000 to < 1/100)	Rare (≥ 1/10,000 to < 1/1,000)	Very rare (< 1/10,000)
Blood and lymphatic system					Thrombocytopenia, leukopenia
Endocrine disorders				Deterioration of latent	
Metabolism and nutrition disorders			Weight gain.		
Psychiatric disorders			Depression, concentration problems, drowsiness or insomnia, nightmares.	Nervousness, anxiety.	Forgetfulness or memory impairment, confusion, hallucinations, personality changes (e.g.
Nervous system disorders		Dizziness, headache.	Paresthesia.		

Eye disorders				Visual disturbances, dry or	
Ear and labyrinth disorders					Tinnitus, hearing problems.
Cardiac disorders		Bradycardia, balance disturbances (very rarely with associated syncope), palpitations.	Temporary exacerbation of symptoms of heart failure, first- degree atrioventricular block, precordial pain.	Functional heart symptoms, heart arrhythmia, conductivity disturbances.	
Vascular disorders	Pronounced blood pressure drop and orthostatic hypotension, very rarely with syncope.	Cold hands and feet.			Necrosis in patients with severe peripheral vascular disorders prior to treatment, exacerbation of claudication intermittent or Raynaud's syndrome.
Respiratory, thoracic and mediastinal disorders		Functional dyspnea.	Bronchospasms.	Rhinitis.	
Gastrointestinal disorders		Nausea, abdominal pain, diarrhoea, constipation.	Vomiting.	Dryness of mouth.	Taste disturbances.
Hepatobiliary disorders				Abnormal LFT values.	Hepatitis.

Skin and subcutaneous tissue disorders			Rash (psoriasis like urticaria and dystrophic cutaneous lesions), increased perspiration.	Hair loss.	Light hypersensitivity reactions, exacerbation of psoriasis, new psoriasis manifestation, psoriasis-like dermatological changes.
Musculoskeletal and connective tissue disorders			Muscle spasms.		Arthralgia, muscle weakness.
Reproductive system and breast disorders				Impotence and other sexual dysfunctions, induration penis plastica (Peyronie's syndrome).	
General disorders and administration site	Fatigue.		Oedema.		

Reporting of suspected adverse reactions

Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions via company website www.zimlab.in.

4.9 Overdose

Toxicity:

7.5 g to an adult resulted in a lethal intoxication. 100 mg to a 5-year-old did not result in any symptoms after gastriclavage. 450 mg to a 12-year-old and 1.4 g to an adult resulted in moderate intoxication. 2.5 g to an adult resulted in a serious intoxication and 7.5 g to an adult resulted in a very serious intoxication.

Symptoms:

An overdose of metoprolol may cause severe hypotension, sinus bradycardia, atrioventricular block, heart failure, cardiogenic shock, cardiac arrest, asystole, QT-prolongation (isolated cases), poor peripheral perfusion, bronchospasms, loss of consciousness (even coma), nausea, vomiting or cyanosis. Respiratory depression, apnea,

fatigue, fine tremor, seizures, sweating, paraesthesias, possible oesophageal spasm, hypoglycaemia (especially in children) or hyperglycaemia, hyperkalaemia, renal effects, transient symptoms of myasthenia.

In certain cases, especially among children and adolescents, CNS-symptoms and respiratory depression may predominate.
The symptoms may be exacerbated by concomitant ingestion of alcohol, antihypertensive agents, chinidine or barbiturates.
The first signs of an overdose present within 20 minutes to 2 hours after taking the medicinal product. The effects of massive overdose may persist for several days, despite declining plasma concentrations.

Management:
Patients should be admitted to hospital and, generally, should be managed in an intensive care setting, with continuousmonitoring of cardiac function, blood gases, and blood biochemistry. Emergency supportive measures such as artificial ventilation or cardiac pacing should be instituted if appropriate. Even apparently well patients who have taken a small overdose should be closely observed for signs of poisoning for at least 4 hours.
Active charcoal, gastric lavage if necessary. NOTE! Atropine (0.25-0.5 mg i.v. to adults, 10-20 micrograms/kg to children) should be administered prior to gastric lavage (due to the risk of vagal stimulation). Intubation and assisted ventilation should occur based on a very wide indication. Adequate volume substitution. Glucose infusion. ECG monitoring. Atropine sulphate may be administered (0.5 - 2.0 mg intravenously) for blocking the vagus nerve. This can be repeated. In case of severe hypotension, bradycardia or in risk of heart failure, the patient could be given a beta-1 agonist (e.g. prenalterol or isoprenaline) intravenously at intervals of 2-5 minutes or as continuous infusion until achieving the desired effect. If a selective beta-1 agonist is unavailable, dopamine may be used. If the desired effect is not achieved, another sympathomimetic agent may be used, e.g. dobutamine or noradrenaline.
The patient may also be given 1-10 mg glucagon. It may be necessary to use a pacemaker. A beta-2 agonist may be administered intravenously to prevent bronchospasms in the patient, the patients should be monitored for evidence of cardiac arrhythmias during and after administration of the bronchodilator. Note! The doses required for managing overdoses are much higher than the therapeutic doses usually applied as the beta blocker has blocked the beta receptors.
Note! In case of cardiac arrest after overdosage with a beta-blocker, cardiopulmonary resuscitation during several hours may be required.

5. Pharmacological properties

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Beta blockers, selective.
ATC code: C 07 AB 02.

Mechanism of action
Metoprolol is a beta – 1 Selective blocker.
It has relatively greater blocking effect on beta receptors (i.e. those mediating adrenergic stimulation of heart and contractility and release of the fatty acids from fat stores) than on beta receptors which are chiefly involved in broncho and vasodilation.
Metoprolol only exhibits insignificant membrane stabilising effect and has no agonist effect.

Metoprolol reduces or blocks the stimulating effect of catecholamines (particularly released in case of physical or mental stress) on the heart. Metoprolol reduces tachycardia, decreases the cardiac output and the contractility, and lowers the blood pressure.
If required, metoprolol may be administered concomitantly with a beta-2 agonist to patients with symptoms of obstructive pulmonary disease.

5.2 Pharmacokinetic properties

Absorption and distribution:

Metoprolol is completely absorbed after an oral dose, peak plasma concentrations occurring 1.5 – 2 hours after dosing. Due to a pronounced first passage metabolism for metoprolol, the bioavailability of a single oral dose is approx. 50 %. Concomitant intake of food increases bioavailability to approximately 70% only a small fraction of metoprolol (approx. 5- 10 %) binds to plasma proteins. Metoprolol crosses the placenta, and is found in breast milk.

Biotransformation and elimination:

Metoprolol is metabolised by hepatic oxidation. The three known main metabolites have been shown not to have a clinically significant beta blocking effect. Metoprolol is metabolised primarily, but not solely, by the hepatic enzyme cytochrome (CYP) 2D6. Due to the polymorphy of the CYP 2D6 gene, the turnover rates vary with the individual. Individuals with poor metabolic capacity (approx. 7-8 %) exhibit higher plasma concentrations and slower elimination than individuals with good metabolic capacity. The plasma concentrations are stable and repeatable in the individuals, however. More than 95 % of an oral dose is excreted in urine. Approximately 5 % of the dose is excreted in unchanged form; in single cases up to an entire 30 %. The elimination half-life of metoprolol in plasma is 3.5 hours on average (interval 1-9 hours). Total clearance is approx. 1 L/min.

The pharmacokinetics of metoprolol in the elderly is not significantly different from that in younger populations. The systemic bioavailability and elimination of metoprolol is normal in renal failure patients. The elimination of metabolites is slower than normal, however. Significant accumulation of metabolites has been observed in patients with a glomerular filtration rate of less than 5 mL/min. The metabolite accumulation does not potentiate the beta blocking action of metoprolol.

Patients with hepatic cirrhosis may experience an increase in the bioavailability of metoprolol and a decline in totalclearance. However, the exposure increase only has clinical relevance in patients with severely impaired hepatic functionor portocaval shunt. In patients with portocaval shunt, the total clearance is approx. 0.3 L/min, and the AUC values areapprox. six times larger than in healthy individuals.

5.3 Preclinical safety data

There are no other relevant preclinical data than those already mentioned in other sections of this summary of product characteristics.

6. Pharmaceutical particulars

6.1 List of excipients

Lactose
Povidone
Microcrystalline cellulose

Magnesium Stearate
Croscarmellose Sodium
Purified talc
Isopropyl Alcohol

6.2 Incompatibilitieses

Not applicable

6.3 Shelf life

3 years

6.4 Special precautions for storage

Store at a temperature not exceeding 30°C, protect from light and moisture.

6.5 Nature and contents of container

10 x 10 Tablets Alu- Alu Blister Pack

6.6 Special precautions for disposal and other handling

Any unused product or waste material should be disposed of in accordance with local requirements.

7. Marketing authorisation holder

Zim Laboratories Limited.
Sadoday Gyan (Ground Floor),
Opp. NADT, Nelson Square,
Nagpur – 440013
India.

8. Marketing authorisation number(s)

06256/07878/REN/2021

9. Date of first authorisation/renewal of the authorization

24/07/2021

10. Date of revision of the text

02/07/2023