FESTPROPRA
(PROPRANOLOL TABLETS BP 40 MG)

Summary of Product Characteristics (SmPC)

1. Name of the medicinal product

1.1 (Invented) name of the medicinal product

FESTPROPRA

INN (GENERIC NAME)

PROPRANOLOL TABLETS BP 40 MG

1.2 Strength :- 40 mg per tablet

1.3 Pharmaceutical form :- Tablets

FESTPROPRA
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2. QUALITATIVE AND QUANTITATIVE COMPOSITION

FESTPROPRA (PROPRANOLOL TABLETS BP 40 MG)

Each Film coated Tablets Contains:
- Propranolol Hydrochloride BP (40 mg)
- Approved colour used. (-)
- Excipients: (0 QS)

Batch Size: 20,20,000 Tablets

SR NO	INGREDIENTS	Spec.	Mg/Tab	Overages (%)	Reason for Inclusion
DRY MIXING INGREDIENT					
1.	Propranolol Hydrochloride	BP	40.00		Active
2	Diabasic Calcium Phosphate	BP	60.00		Diluent
3.	Maize starch (Drying loss compensate)	BP	69.280	10%	Diluent
BINDER INGREDIENTS					
4.	Maize starch (paste)	BP	13.500		Binder
5.	Methyl paraben	BP	0.2		Preservative
6.	Propyl paraben	BP	0.02		Preservative
7.	Gelatin	BP	4.00		Binder
LUBRICANTS					
8	Sodium starch glycolate	BP	5.0		Disintegrant
9	Purified Talcum	BP	5.0		Lubricant
10	Magnesium stearate	BP	3.0		Lubricant

AVERAGE WEIGHT OF TABLETS :- 200MG/TABS
10% ADDITIONAL QUANTITY OF STARCH TO COMPENSATE THE LOSS ON DRYING.

SR NO	INGREDIENTS	Spec.	Mg/Tab	STD QTY (KG)
FILM COATING				
1	Hydroxy Propyl Methyl Cellulose E15CPS	BP	5.357	Film coating
2	Titanium Dioxide	BP	0.357	Opacifier
3	Purified Talc	BP	0.893	Glidant
4	Poly Ethylene Glycol 4000	BP	0.714	Plasticiser
5	Propylene Glycol	BP	0.714	Plasticiser
6	Col. Erythrosine Lake	IHS	0.0356	Colour

7	Col. Sunset Yellow Lake	IHS	0.00347	Colour
8	Methylene Chloride	BP	0.0535	Solvent
9	Isopropyl Alcohol	BP	0.0535	Solvent

AVERAGE WEIGHT OF COATED TABLETS :- 207 MG/TABS we are Applying
Total 4% solution for weight gaining(3.5%) & 0.5% evaporation lose.

#Solvents evaporate during manufacturing process. Hence, not a part of final product.
BP = British Pharmacopoeia
IHS = In- House Specification

3. PHARMACEUTICAL FORM. :

Pink coloured, Circular, film coated tablets, having embossing "PRO 40" on one side of each tablet.

4. CLINICAL PARTICULARS

4.1 Therapeutic indications:

• Angina pectoris.

• Hypertension.

• Long-term prophylaxis against myocardial reinfarction after recovery from acute myocardial infarction

• Hypertrophic obstructive cardiomyopathy.

• Essential tremor.

• Supraventricular cardiac arrhythmia.

• Ventricular cardiac arrythmias.

• Hyperthyroidism and thyrotoxicosis

• Phaeochromocytoma (with an alpha-blocker).

• Migraine.

• Prophylaxis of upper gastrointestinal bleeding in patients with portal hypertension and oesophageal varices.

4.2 Posology and method of administration:

Adults:

Hypertension

Initially 40 mg two or three times daily, which may be increased by 80 mg per day at weekly intervals according to response. The usual dose range is 160 to 320 mg per day. With concurrent diuretic or other antihypertensive drugs a further reduction of blood pressure is obtained.

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Angina, migraine and essential tremor

The starting dose is 40 mg two to three times daily, increasing by the same amount at weekly intervals according to the patient response. An adequate response in migraine is usually seen in the range 80 to 160 mg/day and in angina and essential tremor in the range 120 to 240 mg/day.

Arrhythmias, , hypertrophic obstructive cardiomyopathy and thyrotoxicosis

A dosage range of 10 to 40 mg three or four times a day usually achieves the required response.

Post myocardial infarction:

Treatment should start between days 5 and 21 after myocardial infarction, with an initial dose of 40mg four times a day for two or three days. In order to improve compliance, the total daily dosage may thereafter be given as 80mg twice a day.

Hyperthyroidism

The dose is adjusted according to clinical response

Portal Hypertension:

Dosage should be titrated to achieve approximately 25% reduction in heart rate at rest. Dosing should begin with 40mg twice daily, increasing to 80mg twice daily depending on heart rate response. If necessary, the dose may be increased incrementally to a maximum of 160mg twice daily.

Phaeochromocytoma

(Used only with an alpha-receptor blocking drug).

Pre-operative: 60 mg daily for 3 days is recommended. Non-operable malignant cases: 30 mg daily.

Hepatic impairment:

The bioavailability of propranolol may be increased in patients with hepatic impairment and dose adjustments may be required. In patients with severe liver disease (e.g. cirrhosis) a low initial dose is recommended (not exceeding 20mg three times a day) with close monitoring of the response to treatment (such as the effect on heart rate).

Renal impairment:

Concentrations of propranolol may increase in patients with significant renal impairment and haemodialysis. Caution should be exercised when starting treatment and selecting the initial dose.

As with other beta-adrenoceptor blocking agents, treatment should not be discontinued abruptly. The dosage should be withdrawn gradually over a period of 7 to 14 days. Either the equivalent dosage of another beta-adrenoceptor blocker may be substituted or the withdrawal of propranolol should be gradual. Patients should be followed during withdrawal especially those with ischaemic heart disease. The risk/benefit of stopping beta blockade should be made for each patient.

Elderly:

Evidence concerning the relationship between blood level and age is conflicting. Propranolol should be used to treat older people with caution. It is suggested that treatment should start with the lowest dose. The optimum dose should be individually determined according to clinical response.

Paediatric population

Arrhythmias

Dosage should be determined according to the cardiac status of the patient and the circumstances necessitating treatment. The dose should be adjusted individually and the following is a guide: Children and adolescents: 0.25-0.5 mg / kg 3-4 times daily, adjusted according to clinical response.

Migraine

Oral: Under the age of 12: 20 mg two or three times daily. Over the age of 12: The adult dose.

Method of administration

For oral administration.

4.3 Contraindications:

• Hypersensitivity to the active substance(s) or to any of the excipients.

• Cardiac decompensation which is not adequately treated.

• Sick sinus syndrome/SA-block.

• History of bronchospasm or bronchial asthma, chronic obstructive pulmonary disease.

• Metabolic acidosis.

• Second and third-degree heart block.

• Patients prone to hypoglycaemia, e.g. due to prolonged fasting or restricted counter regulatory reserve.

• Cardiogenic shock.

• Untreated phaeochromocytoma.

• Severe bradycardia.

• Severe hypotension

• Severe peripheral arterial disturbances

• Prinzmetal's angina

4.4 Special warnings and precautions for use:

Propranolol as with other beta-blockers:

- although contraindicated in uncontrolled heart failure, may be used in patients whose signs of heart failure have been controlled. Caution must be exercised in patients whose cardiac reserve is poor.

- should not be used in combination with calcium channel blockers with negative inotropic effects (e.g. verapamil, diltiazem), as it can lead to an exaggeration of these effects particularly in patients with impaired ventricular function and/or SA or AV conduction abnormalities. This may result in severe hypotension, bradycardia and cardiac failure. Neither the beta-blocker nor the calcium channel blocker should be administered intravenously within 48 hours of discontinuing the other.

- although contraindicated in severe peripheral arterial circulatory disturbances, may also aggravate less severe peripheral arterial circulatory disturbances.

- due to its negative effect on conduction time, caution must be exercised if it is given to patients with first degree heart block.

- may block/modify the signs and symptoms of the hypoglycaemia (especially tachycardia). Propranolol occasionally causes hypoglycaemia, even in non-diabetic patients, e.g. neonates, infants, children, elderly patients, patients on haemodialysis or patients suffering from chronic liver disease and patients suffering from overdose. Severe hypoglycaemia associated with Propranolol has rarely presented with seizures and/or coma in isolated patients. Caution must be exercised in the concurrent use of Propranolol and hypoglycaemic therapy in diabetic patients. Propranolol may prolong the hypoglycaemic response to insulin.

- may mask the signs of thyrotoxicosis.

- should not be used in untreated phaeochromocytoma. However, in patients with phaeochromocytoma, an alpha-blocker may be given concomitantly.

- will reduce heart rate as a result of its pharmacological action. In the rare instances when a treated patient develops symptoms which may be attributable to a slow heart rate, the dose may be reduced.

- may cause a more severe reaction to a variety of allergens when given to patients with a history of anaphylactic reaction to such allergens. Such patients may be unresponsive to the usual doses of adrenaline used to treat the allergic reactions.

Abrupt withdrawal of beta-blockers is to be avoided. The dosage should be withdrawn gradually over a period of 7 to 14 days. Patients should be followed during withdrawal especially those with ischaemic heart disease.

When a patient is scheduled for surgery and a decision is made to discontinue beta-blocker therapy, this should be done at least 48 hours prior to the procedure. The risk/benefit of stopping beta blockade should be made for each patient.

Since the half-life may be increased in patients with significant hepatic or renal impairment, caution must be exercised when starting treatment and selecting the initial dose.

Propranolol must be used with caution in patients with decompensated cirrhosis . In patients with portal hypertension, liver function may deteriorate and hepatic encephalopathy may develop. There have been reports suggesting that treatment with propranolol may increase the risk of developing hepatic encephalopathy.

In patients with chronic obstructive pulmonary disease, non-selective beta blockers such as propranolol may aggravate the obstructive condition. Therefore propranolol should not be used in this condition.

Bronchospasm can usually be reversed by beta2 agonist bronchodilators such as salbutamol. Large doses of the beta bronchodilator may be required to overcome the beta blockade produced by propranolol and the dose should be titrated according to the clinical response; both intravenous and inhalational administration should be considered. The use of intravenous aminophylline and/or the use of ipratropium (given by nebuliser) may also be considered. Glucagon (1 to 2 mg given intravenously) has also been reported to produce a bronchodilator effect in asthmatic patients. Oxygen or artificial ventilation may be required in severe cases.

Isolated reports of myasthenia gravis like syndrome or exacerbation of myasthenia gravis have been reported in patients administered propranolol.

Interference with laboratory tests:

Propranolol has been reported to interfere with the estimation of serum bilirubin by the diazo method and with the determination of catecholamines by methods using fluorescence.

Lactose:

This medicine contains lactose. Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.

4.5 Interaction with other medicinal products and other forms of interaction:

Combination not recommended:

Combined use of beta-blockers and calcium channel blockers with negative inotropic effects (e.g., verapamil, diltiazem) can lead to an exaggeration of the negative AV conduction and sinus node function particularly in patients with impaired ventricular function and/or SA or AV conduction abnormalities. This may result in severe hypotension and bradycardia. The combination with proproanolol should be avoided, especially in patients with cardiac decompensation.

Concomitant use of sympathomimetic agents e.g., adrenaline, may counteract the effect of beta-blockers. Caution must be exercised in the parenteral administration of preparations containing adrenaline to patients taking beta-blockers as, in rare cases, vasoconstriction, hypertension and bradycardia may result.

Beta-agonist bronchodilators :

Non-cardioselective beta-blockers oppose the bronchodilator effects of beta-agonist bronchodilators, propranolol is contraindicated in patients with asthma.

Fingolimod:

Potentiation of bradycardia effects with possible fatal outcomes. Treatment with Fingolimod should not be initiated in patients receiving beta blockers. In case of combination, appropriate monitoring for treatment initiation, at least overnight monitoring is recommended.

Barbiturates:

The plasma levels and the effects of beta-blockers are reduced by the barbiturates. Barbiturates are potent liver enzyme inducers which may increase the metabolism of propranolol.

Propafenone:

Plasma propranolol levels can be raised up to 100% by propafenone. This probably was because propranolol is partially metabolized by the same enzyme like propafenone (CYP2D6). This combination is also not advisable because propafenone has negative inotropic effects.

Warfarin:

Propranolol may cause a reduction in clearance and an increase in plasma concentrations of warfarin.

MAO inhibitors:

Concomitant use of MAO inhibitors (except MAO-B inhibitors) with antihypertensive agents may diminish the antihypertensive effect and lead to hypertensive reactions.

Glycosides:

Digitalis glycosides, in association with beta-blockers, may increase atrio-ventricular conduction time.

Combination to be used with caution, dose adjustment may be required

Amiodarone:

A few case reports suggest that patients treated with amiodarone can have severe sinus bradycardia when treated concomitantly with propranolol. Amiodarone has an extremely long half-life (about 50 days), which means that interactions may occur long after discontinuation of therapy.

Class I antiarrhythmic drugs (disopyramide, quinidine):

Class I antiarrhythmic drugs and beta-blockers have additive negative inotropic effects which may result in hypotension and severe hemodynamic side effects in patients with impaired left ventricular function.

Non-steroidal anti-inflammatory / anti-rheumatic drugs (NSAIDs):

Anti-inflammatory drugs of NSAID-type counter the antihypertensive effect of beta-blockers. It has been studied mainly in indomethacin. In a study on diclofenac no such interaction could be detected. Data for COX-2 inhibitors are missing.

Cimetidine:

Cimetidine increases levels of propranolol in plasma, probably by inhibiting its first pass metabolism. There may be a risk of eg bradycardia with oral dosing.

Alcohol:

Concomitant use of alcohol may increase the plasma levels of propranolol.

Anaesthetics:

Concomitant use of beta-adrenergic antagonists and anaesthetics may attenuate reflex tachycardia and increase the risk of hypotension. As a general rule, avoid sudden withdrawal

of beta-blocker treatment. The anaesthesiologist should be informed when the patient is receiving beta-adrenergic antagonists. Anaesthetic agents causing myocardial depression are best avoided.

Epinephrine (adrenaline):

A number of reports are available for severe hypertension and bradycardia in patients treated with propranolol and epinephrine. These clinical observations have been confirmed by studies in healthy volunteers. It has also been suggested that the intravascular administration of epinephrine may trigger these reactions.

Fluvoxamine:

Fluvoxamine inhibits oxidative metabolism and increases plasma concentrations of propranolol. This may result in severe bradycardia.

Centrally-acting antihypertensives (clonidine, moxonidine, methyldopa):

Concomitant use of centrally acting antihypertensive drugs may worsen heart failure by a decrease in the central sympathetic tonus (reduction of heart rate and cardiac output, vasodilation). Abrupt withdrawal, particularly if prior to beta-blocker discontinuation, may increase risk of "rebound hypertension".

If the two drugs are co administered, the beta-blocker should be withdrawn several days before discontinuing clonidine. If replacing clonidine by beta blocker therapy, the introduction of beta-blockers should be delayed for several days after clonidine administration has stopped.

Rifampicin:

The metabolism of propranolol may be increased by potent liver enzyme inducer rifampicin.

Alpha blockers:

Concomittant use with alpha blockers increases the risk of hypotension, especially orthostatic hypotension, and tachycardia and palpitations.

Dihydropyridine calcium channel blockers: e.g nifedipine:

Concomitant use may increase the risk of hypotension, and cardiac failure may occur with latent cardiac insufficiency.

Chlorpromazine:

The concurrent use of chlorpromazine with propranolol can result in a marked rise in plasma levels of both drugs, and thereby enhance its effects on heart rate and blood pressure as well as an enhanced antipsychotic effect for chlorpramazine and an increased antihypertensive effect for propranolol.

Lidocaine:

Administration of propranolol during infusion of lidocaine may increase the plasma concentration of lidocaine by approximately 30%. Patients already receiving propranolol tend to have higher lidocaine levels than controls. The combination should be avoided.

Antimigraine drugs:

During concomitant treatment with propranolol it inhibited the first-pass metabolism of rizatriptan whose AUC increases by 70-80%. A dose of 5 mg of rizatriptan is recommended

for combination therapy. Ergotamine with propranolol has resulted in reports of vasospastic reactions in some patients.

Theophylline:

Propranolol reduces the metabolic clearance of theophylline by about 30% at a dosage of 120 mg / day and 50% at doses of 720 mg / day.

Insulin and oral antidiabetic drugs:

Concomitant use may mask certain symptoms of hypoglycaemia (palpitations, tachycardia). Propranolol may prolong the hypoglycaemic response to insulin.

Tobacco:

Tobacco smoking can reduce the beneficial effects of the beta-blockers on heart rate and blood pressure.

Laboratory tests:

Interference with laboratory tests - Propranolol has been reported to interfere with the estimation of serum bilirubin by the diazo method and with the determination of catecholamines by methods using fluorescence.

4.6 Pregnancy and lactation:

Pregnancy:

As with all drugs Propranolol should not be given during pregnancy unless its use is essential. There is no evidence of teratogenicity with propranolol.However beta-blockers reduce placental perfusion, which may result in intra uterine foetal death, immature and premature deliveries. In addition, adverse effects (especially hypoglycaemia and bradycardia in the neonate and bradycardia in the foetus) may occur. There is an increased risk of cardiac and pulmonary complications in the neonate in the post-natal period.

Breast feeding:

Most beta-adrenoceptor blocking drugs, particularly lipophilic compounds, will pass into breast milk although to a variable extent. Breast feeding is therefore not recommended following administration of these compounds.

Fertility: No relevant data on effect of fertility in humans is available.

4.7 Effects on ability to drive and use machines:

Propranolol has no or negligible influence on the ability to drive and use machines. It should be taken into account that occasionally dizziness or fatigue may occur.

4.8 UNDESIRABLE EFFECTS:

Propranolol is usually well tolerated. In clinical studies the undesired events reported are usually attributable to the pharmacological actions of propranolol.

Adverse reactions related to propranolol are listed below by system organ class and frequency. Frequencies are defined as:

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); Frequency not known (cannot be estimated from the available data).

System Organ Class	Common	Uncommon	Rare	Very Rare	Not known
Blood and lymphatic system disorders			Thrombocytopaenia		Agranulocytosis
Immune system disorders			Angioedema		
Metabolism and nutrition disorders				Hypoglycaemia in neonates, infants, children, elderly patients, patients on haemodialysis, patients on concomitant antidiabetic therapy, patients with prolonged fasting and patients with chronic liver disease has been reported. Changes in lipid metabolism(changes in blood concentrations of triglycerides and cholesterol). Severe hypoglycemia may rarely lead to seizures or coma.	
Psychiatric disorders	Sleep disturbances, nightmares		Hallucinations, psychoses, mood changes		Depression
Nervous system			Confusion, memory	Isolated reports	Headache,

disorders			loss, paraesthesia, dizziness	of myasthenia gravis like syndrome or exacerbation of myasthenia gravis have been reported	seizure linked to hypoglycaemia
Eye disorders			Dry eyes, visual disturbances		Conjunctivitis
Cardiac disorders	Bradycardia, cold extremities		Heart failure deterioration, precipitation of heart block, postural hypotension, which may be associated with syncope		Worsening of attacks of angina pectoris
Vascular disorders	Raynaud's phenomenon		Exacerbation of intermittent claudication		
Respiratory, thoracic and mediastinal disorders	Breathlessness		Bronchospasm may occur in patients with bronchial asthma or a history of asthmatic complaints, sometimes with fatal outcome		Dyspnoea
Gastrointestinal disorders		Gastrointestinal disturbance, such as nausea, vomiting, diarrhoea			Constipation, dry mouth
Skin and subcutaneous tissue disorders			Purpura, alopecia, psoriasiform skin reactions, exacerbation of psoriasis, skin rashes	Isolated cases of hyperhidrosis has been reported	
Musculoskeletal and connective tissue disorders					Arthralgia
Renal and urinary disorders					Reduced renal blood flow and GFR
Reproductive					Impotence

system and breast disorders					
General disorders and administration site conditions	Fatigue and/or lassitude (often transient)		Dizziness		
Investigations			An increase in ANA (Antinuclear Antibodies) has been observed, however the clinical relevance of this is not clear		

Discontinuance of the drug should be considered if, according to clinical judgement, the wellbeing of the patient is adversely affected by any of the above reactions. Cessation of therapy with a beta-blocker should be gradual. In the rare event of intolerance manifested as bradycardia and hypotension, the drug should be withdrawn and, if necessary, treatment for overdosage instituted.

4.9 OVERDOSE:

Propranolol is known to cause severe toxicity when used in overdose. Patients should be informed of the signs of overdose and advised to seek urgent medical assistance if an overdose of propranolol has been taken.

Clinical features:

Cardiac

Bradycardia, hypotension, pulmonary oedema, syncope and cardiogenic shock may develop. QRS complex prolongation, ventricular tachycardia, first to third degree AV block, ventricular fibrillation or asystole may also occur. Development of cardiovascular complications is more likely if other cardioactive drugs, especially calcium channel blockers, digoxin, cyclic antidepressants or neuroleptics have also been ingested. Older patients and those with underlying ischaemic heart disease are at risk of developing severe cardiovascular compromise.

CNS

Drowsiness, confusion, seizures, hallucinations, dilated pupils and in severe cases coma may occur. Neurological signs such as coma or absence of pupil reactivity are unreliable prognostic indicators during resuscitation.

Other features

Bronchospasm, hyperkalaemia and occasionally CNS-mediated respiratory depression may occur.

Management

In cases of overdose or extreme falls in heart rate or blood pressure, treatment with propranolol must be stopped. Management should include general symptomatic and

supportive measures including a clear airway and monitoring of vital signs until stable. In symptomatic patients, or patients with an abnormal ECG, early discussion with critical care should be considered.

Consult national clinical guidance for further information on the management of overdose.

5 Pharmacological Properties:

5.1 Pharmacodynamic properties:

Pharmacotherapeutic group: Beta blocking agents, non-selective (beta blocker) ATC code: C07AA05

Propranolol is a competitive antagonist at both the beta1- and beta2 adrenoceptors. It has no agonist activity at the beta adrenoceptor, but has membrane stabilising activity at concentrations exceeding 1 to 3 mg/litre, though such concentrations are rarely achieved during oral therapy.

Competitive beta blockade has been demonstrated in man by a parallel shift to the right in the dose-heart rate response curve to beta agonists such as isoprenaline.

Propranolol as with other beta-blockers, has negative inotropic effects, and is therefore contraindicated in uncontrolled heart failure.

Propranolol is a racemic mixture and the active form is the S (-) isomer of propranolol. With the exception of inhibition of the conversion of thyroxine to triiodothyronine, it is unlikely that any additional ancillary properties possessed by R (+) propranolol, in comparison with the racemic mixture, will give rise to different therapeutic effects.

Propranolol is effective and well tolerated in most ethnic populations, although the response may be less in black patients.

5.2 Pharmacokinetic properties:

Following intravenous administration the plasma half-life of propranolol is about 2 hours and the ratio of metabolites to parent drug in the blood is lower than after oral administration. In particular 4-hydroxypropranolol is not present after intravenous administration. Propranolol is completely absorbed after oral administration and peak plasma concentrations occur 1 to 2 hours after dosing in fasting patients. The liver removes up to 90% of an oral dose with an elimination half-life of 3 to 6 hours. Propranolol is widely and rapidly distributed throughout the body with highest levels occurring in the lungs, liver, kidney, brain and heart. Propranolol is highly protein bound (80 to 95%).

5.3 Preclinical safety data :

Non-clinical data reveal no special hazard for humans based on conventional studies of safety pharmacology, repeated dose toxicity, local tolerance, genotoxicity, carcinogenic potential and toxicity to reproduction.

6 Pharmaceutical Particulars

6.1 List of Excipients.

Sr. No.	Excipients	Quality standard	Overages (%)
1.	Propranolol Hydrochloride	BP	0%
2.	Dibasic Calcium Phosphate	BP	0%
3.	Maize Starch*	BP	0%
4.	Maize Starch (for Paste)*	BP	10%
5.	Gelatin	BP	0%
6.	Methyl Paraben	BP	0%
7.	Propyl Paraben	BP	0%
8	Purified Talcum	BP	0%
9	Magnesium Stearate	BP	0%
10	Sodium Starch Glycolate	BP	0%
11	HPMC E15CPS	BP	0%
12	Titanium Dioxide	BP	0%
13.	Purified Talc	BP	0%
14.	Polyethylene Glycol 4000	BP	0%
15.	Propylene Glycol	BP	0%
16.	Colour Erythrosine Lake	IHS	0%
17.	Colour Sunset Yellow Lake	IHS	0%
18.	Methylene Chloride#	BP	0%
19.	Isopropyl Alcohol#	BP	0%

6.2 Incompatibilities

Not applicable.

6.3 Shelf life

3 Years

6.4 Special precautions for storage

Do not store above 30°C.

6.5 Nature and contents of container

Jar pack of 1000 Tablets.

1.3.2 Labelling (outer & inner labels)

It is a Tablets.

Not Applicable.

1.3.3 Package Insert (also known as patient information PIL)

Enclosed with the sample.