SUMMARY OF THE PRODUCT CHARACTERISTICS (SmPC)

1. NAME OF THE FINISHED PHARMACEUTICAL PRODUCTS

LISIOFIL 5 (LISINOPRIL TABLETS USP 5 mg)

1.1. Strength: Each Uncoated Tablet Contains:-

Lisinopril anhydrous 5 mg

(as Lisinopril USP)

1.2 Pharmaceutical form: Oral Solids – Uncoated Tablets

2. QUALITY AND QUANTITATIVE COMPOSITION

S.No	Ingredients	Specification	Qty/Tablet (mg)	Reason for inclusion
1	Lisinopril	USP	5.44	Active Ingredient
2	Maize Starch (mixing)	BP	4.00	Diluent
3	Maize Starch (for paste)	BP	3.20	Binder
4	Maize Starch (for lubrication)	BP	6.80	Disintegrant
5	Mannitol	BP	51.00	Diluent
6	Anhydrous Calcium Hydrogen Phosphate	BP	67.98	Diluent
7	Magnesium Stearate	BP	1.40	Lubricant
8	Sunset yellow FCF	IHS	0.18	Colour
9	Purified water	BP	q.s	Solvent

3. PHARMACEUTICAL FORM

Orange coloured, circular, uncoated tablets with breakline 'L and 5' debossed on one side and 'U' on the other side.

4. CLINICAL PARTICULARS

4.1 Therapeutic indications

Hypertension

Lisinopril is indicated in the treatment of essential hypertension. It may be used alone as initial therapy or concomitantly with other classes of antihypertensive agents.

Heart Failure:

Lisinopril is indicated as adjunctive therapy in the management of heart failure in patients who are nor responding adequately to diuretics and digitalis.

Acute Myocardial Infarction

Lisinopril is indicated for the treatment of haemodynamically stable patients within 24 hours of an acute myocardial infarction improve survival. Patients should receive, as appropriate, the standard recommended treatments such as thrombolytics, aspirin and beta-blocker.

In using Lisinopril, consideration should be given to the fact that another angiotensin-converting enzyme inhibitor, captopril, has caused agranulocytosis, particularly in patients with renal impairment or collagen vascular disease, and that available data are insufficient to show that Lisinopril does not have a similar risk.

4.2 Posology and method of administration

Hypertension: Initial Therapy: In patients with uncomplicated essential hypertension not on diuretic therapy, the recommended initial dose is 10 mg once a day. Dosage should be adjusted according to blood pressure response. The usual dosage range is 20 to 40 mg per day administered in a single daily dose. The antihypertensive effect may diminish toward the end of the dosing interval regardless of the administered dose, but most commonly with a dose of 10 mg daily. This can be evaluated by measuring blood pressure just prior to dosing to determine whether satisfactory control is being maintained for 24 hours. If it is not, an

increase in dose should be considered. Doses up to 80 mg have been used but do not appear to give a greater effect. If blood pressure is not controlled with lisinopril alone, a low dose of a diuretic may be added. Hydrochlorothiazide 12.5 mg has been shown to provide an additive effect. After the addition of a diuretic, it may be possible to reduce the dose of lisinopril. Diuretic Treated Patients: In hypertensive patients who are currently being treated with a diuretic, symptomatic hypotension may occur occasionally following the initial dose of lisinopril. The diuretic should be discontinued, if possible, for two to three days before beginning therapy with lisinopril to reduce the likelihood of hypotension. The dosage of lisinopril should be adjusted according to blood pressure response. If the patient's blood pressure is not controlled with lisinopril alone, diuretic therapy may be resumed as described above. If the diuretic cannot be discontinued, an initial dose of 5 mg should be used under medical supervision for at least two hours and until blood pressure has stabilized for at least an additional hour. Concomitant administration of lisinopril with potassium supplements, potassium salt substitutes, or potassium-sparing diuretics may lead to increases of serum potassium. Dosage Adjustment in Renal Impairment: The usual dose of lisinopril (10 mg) is recommended for patients with a creatinine clearance >30 mL/min (serum creatinine of up to approximately 3 mg/dL). For patients with creatinine clearance =10 mL/min = 30 mL/min (serum creatinine =3 mg/dL), the first dose is 5 mg once daily. For patients with creatinine clearance <10 mL/min (usually on hemodialysis) the recommended initial dose is 2.5 mg. The dosage may be titrated upward until blood pressure is controlled or to a maximum of 40 mg daily.

Renal Status	**Creatinine Clearance mL/min**	**Initial Dose mg/day**
Normal Renal Function to Mild Impairment	> 30	10
Moderate to Severe Impairment	= 10 Â£ 30	5
Dialysis Patients*	< 10	2.5**

* Check with warnings, Anaphylactoid Reactions During Membrane Exposure.

** Dosage or dosing interval should be adjusted depending on the blood pressure response.

Heart Failure: Lisinopril is indicated as adjunctive therapy with diuretics and (usually) digitalis. The recommended starting dose is 5 mg once a day. When initiating treatment with lisinopril in patients with heart failure, the initial dose should be administered under medical observation,

especially in those patients with low blood pressure (systolic blood pressure below 100mmHg). The mean peak blood pressure lowering occurs six to eight hours after dosing. Observation should continue until blood pressure is stable. The concomitant diuretic dose should be reduced, if possible, to help minimize hypovolemia which may contribute to hypotension.The usual effective dosage range is 5 to 40 mg per day administered as a single daily dose. The dose of Lisinopril can be increased by increments of no greater than 10 mg, at intervals of no less than 2 weeks to the highest tolerated dose, up to a maximum of 40 mg daily. Dose adjustment should be based on the clinical response of individual patients.Dosage Adjustment in Patients with Heart Failure and Renal Impairment or Hyponatremia: In patients with heart failure who have hyponatremia (serum sodium < 130 mEq/L) or moderate to severe renal impairment (creatinine clearance Â£ 30 mL/min or serum creatinine > 3 mg/dL), therapy with Lisinopril should be initiated at a dose of 2.5 mg once a day under close medical supervision. Acute Myocardial Infarction: In hemodynamically stable patients within 24 hours of the onset of symptoms of acute myocardial infarction, the first dose of Lisinopril is 5 mg given orally, followed by 5 mg after 24 hours, 10 mg after 48 hours and then 10 mg of Lisinopril once daily. Dosing should continue for six weeks. Patients should receive, as appropriate, the standard recommended treatments such as thrombolytics, aspirin, and beta-blockers.Patients with a low systolic blood pressure (Â£120 mmHg) when treatment is started or during the first 3 days after the infarct should be given a lower 2.5 mg oral dose of lsinopril. If hypotension occurs (systolic blood pressure Â£100 mmHg) a daily maintenance dose of 5 mg may be given with temporary reductions to 2.5 mg if needed. If prolonged hypotension occurs (systolic blood pressure < 90 mmHg for more than 1 hour) lisinopril should be withdrawn. Dosage Adjustment in Patients With Myocardial Infarction with Renal Impairment: In acute myocardial infarction, treatment with lisinopril should be initiated with caution in patients with evidence of renal dysfunction, defined as serum creatinine concentration exceeding 2 mg/dL. No evaluation of dosing adjustments in myocardial infarction patients with severe renal impairment has been performed. Use in Elderly: In general, the clinical response was similar in younger and older patients given similar doses of lisinopril. Pharmacokinetic studies, however indicate that maximum blood levels and area under the plasma concentration time curve (AUC) are doubled in older patients, so that dosage adjustments should be made with particular caution.

Precautions: Aortic Stenosis/Hypertrophic Cardiomyopathy, Impaired Renal Function: Evaluation of patients with hypertension, heart failure or myocardial infarction should always include assessment of renal function. Hyperkalemia, Cough & Surgery/Anesthesia.

Use in Pregnancy:

	Pregnancy Category	Explanation
1st Trimester	C	Animal studies have shown an adverse effect and there are no adequate studies in pregnant women OR no animal studies have been conducted and there are no adequate studies in pregnant woman.
2nd Trimester	D	Studies in pregnant women have demonstrated as risk to the fetus. However, the benefits of therapy in a life threatening situation or a serious disease, may outweigh the potential risk.
3rd Trimester	D	Studies in pregnant women have demonstrated as risk to the fetus. However, the benefits of therapy in a life threatening situation or a serious disease, may outweigh the potential risk.

Lactation: It is not known whether Lisinopril is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised if Lisinopril is given to women who are breast feeding.

Pediatric Use : Antihypertensive effects of Lisinopril have been established in hypertensive pediatric patients aged 6 to 16 years. There are no data on the effect of Lisinopril on blood pressure in pediatric patients under the age of 6 or in pediatric patients with glomerular filtration rate <30 mL/min/1.73 m2.

4.3 Method of administration

Oral

4.4 Contraindications

Lisinopril is contraindicated in patients who are hypersensitive to this product and in patients with a history of angioedema related to previous treatment with an angiotensin converting enzyme inhibitor and in patients with hereditary or idiopathic angioedema. Lisinopril should not be given to patients with aortic stenosis or hypertrophic cardiomyopathy

4.5 Special warnings and precautions for use

Anaphylactoid and Possibly Related Reactions; Head and Neck Angioedema; Intestinal Angioedema Hypotension: Leukopenia/Neutropenia/Agranulocytosis; Hepatic Failure & Fetal/Neonatal Morbidity and Mortality.

4.6 Paediatric population

Antihypertensive effects of Lisinopril have been established in hypertensive pediatric patients aged 6 to 16 years. There are no data on the effect of Lisinopril on blood pressure in pediatric patients under the age of 6 or in pediatric patients with glomerular filtration rate <30 mL/min/1.73 m2.

4.7 Interaction with other medicinal products and other forms of interaction

Drug interactions : Hypotension - Patients on Diuretic Therapy: Patients on diuretics, and especially those in whom diuretic therapy was recently instituted, may occasionally experience an excessive reduction of blood pressure after initiation of therapy with lisinopirl. The possibility of hypotensive effects with lisinopirl can be minimized by either discontinuing the diuretic or increasing the salt intake prior to initiation of treatment with lisinopirl. If it is necessary to continue the diuretic, initiate therapy with lisinopirl at a dose of 5 mg daily, and provide close medical supervision after the initial dose until blood pressure has stabilized. When a diuretic is added to the therapy of a patient receiving lisinopirl, an additional antihypertensive effect is usually observed. Antidiabetics: Epidemiological studies have suggested that concomitant administration of ACE inhibitors and antidiabetic medicines (insulins, oral hypoglycemic agents) may cause an increased blood-glucose-lowering effect with risk of hypoglycemia. Non-steroidal

Anti-inflammatory Agents: In some patients with compromised renal function who are being treated with non-steroidal anti-inflammatory drugs, the co-administration of lisinopril may result in a further deterioration of renal function. These effects are usually reversible. Other Agents: Lisinopril has been used concomitantly with nitrates and/or digoxin without evidence of clinically significant adverse interactions. This included post myocardial infarction patients who were receiving intravenous or transdermal nitroglycerin. No clinically important pharmacokinetic interactions occurred when lisinopril was used concomitantly with propranolol or hydrochlorothiazide. The presence of food in the stomach does not alter the bioavailability of lisinopril. Agents Increasing Serum Potassium: Lisinopril attenuates potassium loss caused by thiazide-type diuretics. Use of lisinopril with potassium-sparing diuretics (e.g., spironolactone, eplerenone, triamterene or amiloride), potassium supplements, or potassium-containing salt substitutes may lead to significant increases in serum potassium. Lithium: Gold: Nitritoid reactions (symptoms include facial flushing, nausea, vomiting and hypotension) have been reported rarely in patients on therapy with injectable gold (sodium aurothiomalate) and concomitant ACE inhibitor therapy including lisinopril

4.8 Additional information on special populations

Renal Status	**Creatinine Clearance mL/min**	**Initial Dose mg/day**
Normal Renal Function to Mild Impairment	> 30	10
Moderate to Severe Impairment	= 10 Â£ 30	5
Dialysis Patients*	< 10	2.5**

* Check with warnings, Anaphylactoid Reactions During Membrane Exposure.

** Dosage or dosing interval should be adjusted depending on the blood pressure response.

4.9 Pregnancy and lactation

	Pregnancy Category	Explanation
1st Trimester	C	Animal studies have shown an adverse effect and there are no adequate studies in pregnant

		women OR no animal studies have been conducted and there are no adequate studies in pregnant woman.
2nd Trimester	D	Studies in pregnant women have demonstrated as risk to the fetus. However, the benefits of therapy in a life threatening situation or a serious disease, may outweigh the potential risk.
3rd Trimester	D	Studies in pregnant women have demonstrated as risk to the fetus. However, the benefits of therapy in a life threatening situation or a serious disease, may outweigh the potential risk.

Lactation: It is not known whether Lisinopril is secreted in human milk. Because many drugs are secreted in human milk, caution should be exercised if Lisinopril is given to women who are breast feeding.

4.10 Effects on ability to drive and use machines

No specific precautions but see section . Possible Adverse Drug Reactions.

4.11 Undesirable effects

Lisinopril Tablets has been found in controlled clinical trials to be generally well tolerated.
For the most part, side effects were mild and transient in nature.
The most frequent clinical side effects of Lisinopril Tablets in controlled trials were:
dizziness, headache, diarrhoea, fatigue, cough and nausea. Other side effects occurring less frequently were: orthostatic effects (including hypotension), rash and asthenia.
Side effects which occurred rarely, either during controlled clinical trials or after the drug was marketed, include:

Cardiovascular

Myocardial infarction or cerebrovascular accident possibly secondary to excessive hypotension in high risk patients ,palpitations , tachycardia.

Digestive

abdominal pain and indigestion, dry mouth ,hepatitis (hepatocellular or cholestatic) jaundice ,pancreatitis , vomiting.

Nervous System

mental confusion , mood alterations, paraesthesia , vertigo as with other angiotensin converting enzyme inhibitors, taste disturbance, and sleep disturbances, have been reported.

Respiratory

Bronchospasm, rhinitis, sinusitis.

Skin

Alopecia , diaphoresis, pruritus , urticaria , psoriasis and severe skin disorders have been reported, including pemphigus, toxic epidermal necrolysis, Stevens-Johnson Syndrome and erythema multiforme.

Urogenital

Impotence, oliguria/anuria, acute renal failure, renal dysfunction, uraemia

A symptom complex has been reported which may include one or more of the following: fever, vasculitis, myalgia, arthralgia/arthritis, a positive ANA, elevated ESR, eosinophilia and leukocytosis, rash, photosensitivity or other dermatological manifestations may occur.

4.12 Overdose

Following a single oral dose of 20 g/kg, no lethality occurred in rats and death occurred in one of 20 mice receiving the same dose. The most likely manifestation of over dosage would be hypotension, for which the usual treatment would be intravenous infusion of normal saline solution. Treatment of over dosage: Lisinopril can be removed by hemodialysis.

5. PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Angiotensin converting enzyme inhibitors, ATC code: C09A A03

Mechanism of action

Lisinopril is a peptidyl dipeptidase inhibitor. It inhibits the angiotensin converting enzyme (ACE) that catalyses the conversion of angiotensin I to the vasoconstrictor peptide, angiotensin II. Angiotensin II also stimulates aldosterone secretion by the adrenal cortex. Inhibition of ACE results in decreased concentrations of angiotensin II which results in decreased vasopressor activity and reduced aldosterone secretion. The latter decrease may result in an increase in serum potassium concentration.

Whilst the mechanism through which lisinopril lowers blood pressure is believed to be primarily suppression of the renin-angiotensin-aldosterone system, lisinopril is antihypertensive even in patients with low renin hypertension. ACE is identical to kininase II, an enzyme that degrades bradykinin. Whether increased levels of bradykinin, a potent vasodilatory peptide, play a role in the therapeutic effects of lisinopril remains to be elucidated.

The effect of lisinopril on mortality and morbidity in heart failure has been studied by comparing a high dose (32.5mg or 35mg once daily) with a low dose (2.5mg or 5mg once daily). In a study of 3l64 patients, with a median follow up period of 46 months for surviving patients, high dose lisinopril produced a 12% risk reduction in the combined endpoint of all-cause mortality and all-cause hospitalisation ($p = 0.002$) and an 8% risk reduction in all-cause mortality and cardiovascular hospitalisation ($p = 0.036$) compared with low dose. Risk reductions for all-cause mortality (8%; $p = 0.128$) and cardiovascular mortality (10%; $p = 0.073$) were observed.

In a post-hoc analysis, the number of hospitalisations for heart failure was reduced by 24% ($p=0.002$) in patients treated with high-dose lisinopril compared with low dose. Symptomatic benefits were similar in patients treated with high and low doses of lisinopril.

The results of the study showed that the overall adverse event profiles for patients treated with high or low dose lisinopril were similar in both nature and number. Predictable events resulting from ACE inhibition, such as hypotension or altered renal function, were manageable and rarely led to treatment withdrawal. Cough was less frequent in patients treated with high dose lisinopril compared with low dose.

In the GISSI-3 trial, which used a 2x2 factorial design to compare the effects of lisinopril and glyceryl trinitrate given alone or in combination for 6 weeks versus control in 19,394, patients who were administered the treatment within 24 hours of an acute myocardial infarction, lisinopril produced a statistically significant risk reduction in mortality of 11% versus control (2p=0.03). The risk reduction with glyceryl trinitrate was not significant but the combination of lisinopril and glyceryl trinitrate produced a significant risk reduction in mortality of 17% versus control (2p=0.02). In the sub-groups of elderly (age > 70 years) and females, pre-defined as patients at high risk of mortality, significant benefit was observed for a combined endpoint of mortality and cardiac function. The combined endpoint for all patients, as well as the high-risk sub-groups, at 6 months also showed significant benefit for those treated with lisinopril or lisinopril plus glyceryl trinitrate for 6 weeks, indicating a prevention effect for lisinopril. As would be expected from any vasodilator treatment, increased incidences of hypotension and renal dysfunction were associated with lisinopril treatment but these were not associated with a proportional increase in mortality.

In a double-blind, randomised, multicentre trial which compared lisinopril with a calcium channel blocker in 335 hypertensive Type 2 diabetes mellitus subjects with incipient nephropathy characterized by micro albuminuria, lisinopril 10mg to 20mg administered once daily for 12 months, reduced systolic/diastolic blood pressure by 13/10 mmHg and urinary albumin excretion rate by 40%.

When compared with the calcium channel blocker, which produced a similar reduction in blood pressure, those treated with lisinopril showed a significantly greater reduction in urinary albumin excretion rate, providing evidence that the ACE inhibitory action of lisinopril reduced microalbuminuria by a direct mechanism on renal tissues in addition to its blood pressure lowering effect.

Lisinopril treatment does not affect glycaemic control as shown by a lack of significant effect on levels of glycated haemoglobin (HbA1c).

In a clinical study involving 115 paediatric patients with hypertension, aged 6-16 years, patients who weighed less than 50 kg received either 0.625 mg, 2.5 mg or 20 mg of lisinopril once a day, and patients who weighed 50 kg or more received either 1.25 mg, 5 mg or 40 mg of lisinopril once a day. At the end of 2 weeks, lisinopril administered once daily lowered trough blood

pressure in a dose-dependent manner with a consistent antihypertensive efficacy demonstrated at doses greater than 1.25 mg. This effect was confirmed in a withdrawal phase, where the diastolic pressure rose by about 9 mm Hg more in patients randomized to placebo than it did in patients who were randomized to remain on the middle and high doses of lisinopril. The dose-dependent antihypertensive effect of lisinopril was consistent across several demographic subgroups: age, Tanner stage, gender, and race.

Two large randomised, controlled trials (ONTARGET (ONgoing Telmisartan Alone and in combination with Ramipril Global Endpoint Trial) and VA NEPHRON-D (The Veterans Affairs Nephropathy in Diabetes)) have examined the use of the combination of an ACE-inhibitor with an angiotensin II receptor blocker.

ONTARGET was a study conducted in patients with a history of cardiovascular or cerebrovascular disease, or type 2 diabetes mellitus accompanied by evidence of end-organ damage. VA NEPHRON-D was a study in patients with type 2 diabetes mellitus and diabetic nephropathy.

These studies have shown no significant beneficial effect on renal and/or cardiovascular outcomes and mortality, while an increased risk of hyperkalaemia, acute kidney injury and/or hypotension as compared to monotherapy was observed. Given their similar pharmacodynamic properties, these results are also relevant for other ACE-inhibitors and angiotensin II receptor blockers.

ACE-inhibitors and angiotensin II receptor blockers should therefore not be used concomitantly in patients with diabetic nephropathy.

ALTITUDE (Aliskiren Trial in Type 2 Diabetes Using Cardiovascular and Renal Disease Endpoints) was a study designed to test the benefit of adding aliskiren to a standard therapy of an ACE-inhibitor or an angiotensin II receptor blocker in patients with type 2 diabetes mellitus and chronic kidney disease, cardiovascular disease, or both. The study was terminated early because of an increased risk of adverse outcomes. Cardiovascular death and stroke were both numerically more frequent in the aliskiren group than in the placebo group and adverse events and serious adverse events of interest (hyperkalaemia, hypotension and renal dysfunction) were more frequently reported in the aliskiren group than in the placebo group.

5.2 Pharmacokinetic properties

Lisinopril is an orally active non-sulphydryl-containing ACE inhibitor.

• Absorption

Following oral administration of lisinopril, peak serum concentrations occur within about 7 hours, although there was a trend to a small delay in time taken to reach peak serum concentrations in acute myocardial infarction patients. Based on urinary recovery, the mean extent of absorption of lisinopril is approximately 25% with inter-patient variability of 6-60% over the dose range studied (5-80mg).

The absolute bioavailability is reduced approximately 16% in patients with heart failure. Lisinopril absorption is not affected by the presence of food.

• Distribution

Lisinopril does not appear to be bound to serum proteins other than to circulating angiotensin converting enzyme (ACE). Studies in rats indicate that lisinopril crosses the blood-brain barrier poorly.

• Elimination

Lisinopril does not undergo metabolism and is excreted entirely unchanged into the urine. On multiple dosing lisinopril has an effective half-life of accumulation of 12.6 hours. The clearance of lisinopril in healthy subjects is approximately 50 ml/min. Declining serum concentrations exhibit a prolonged terminal phase, which does not contribute to drug accumulation. This terminal phase probably represents saturable binding to ACE and is not proportional to dose.

• Hepatic impairment

Impairment of hepatic function in cirrhotic patients resulted in a decrease in lisinopril absorption (about 30% as determined by urinary recovery) but an increase in exposure (approximately 50%) compared to healthy subjects due to decreased clearance.

• Renal impairment

Impaired renal function decreases elimination of lisinopril, which is excreted via the kidneys, but this decrease becomes clinically important only when the glomerular filtration rate is below 30ml/min. In mild to moderate renal impairment (creatinine clearance 30-80ml/min) mean AVC

was increased by 13% only, while a 4.5-fold increase in mean AVC was observed in severe renal impairment (creatinine clearance 5-30 ml/min).

Lisinopril can be removed by dialysis. During 4 hours of haemodialysis, plasma lisinopril concentrations decreased on average by 60%, with a dialysis clearance between 40 and 55ml/min.

• Heart failure

Patients with heart failure have a greater exposure of lisinopril when compared to healthy subjects (an increase in AUC on average of 125%), but based on the urinary recovery of lisinopril, there is reduced absorption of approximately 16% compared to healthy subjects.

• Elderly

Older patients have higher blood levels and higher values for the area under the plasma concentration time curve (increased approximately 60%) compared with younger subjects.

• Paediatric population

The pharmacokinetic profile of lisinopril was studied in 29 paediatric hypertensive patients, aged between 6 and 16 years, with a GFR above 30 ml/min/1.73m2.

After doses of 0.1 to 0.2 mg/kg, steady state peak plasma concentrations of lisinopril occurredwithin 6 hours, and the extent of absorption based on urinary recovery was about 28%. These values are similar to those obtained previously in adults.

AUC and Cmax values in children in this study were consistent with those observed in adults.

5.3 Preclinical safety data

Preclinical data reveal no special hazard for humans based on conventional studies of general pharmacology, repeated dose toxicity, genotoxicity, and carcinogenic potential. Angiotensin converting enzyme inhibitors, as a class, have been shown to induce adverse effects on the late foetal development, resulting in foetal death and congenital effects, in particular affecting the skull.

Foetotoxicity, intrauterine growth retardation and patent ductus arteriosus have also been reported.

These developmental anomalies are thought to be partly due to a direct action of ACE inhibitors on the foetal renin -angiotensin system and partly due to ischaemia resulting from maternal hypotension and decreases in foetal-placental blood flow and oxygen/nutrients delivery to the foetus.

6. PHARMACEUTICAL PARTICULARS

6.1 List of Excipients

S. No	Name of the Ingredients	Reference
1.	Maize Starch	BP
2.	Mannitol	BP
3.	Anhydrous Calcium Hydrogen Phosphate	BP
4.	Magnesium Stearate	BP
5.	Sunset yellow FCF	In-House
6.	Purified water	BP

6.2 Incompatibilities

Not Applicable

6.3 Shelf Life

36 months

6.4 Special Precautions for storage

Store below 30°C in a dry place. Protect from light.

6.5 Nature and contents of container

Nature: 14 tablets in a blister strip and packed in a Calendar pack. 2 such packs packed in a carton along with packing insert.

Contents of container: 2 x 14’s

6.6 Special precautions for disposal

No special requirements.

7 Registrant

M/s. FOURRTS (INDIA) LABORATORIES PVT LIMITED

Address : Plot No.1, Fourrts Avenue, Annai Indira Nagar,
Okkiyam Thoraipakkam, Chennai - 600 097 . India

Phone : 91-44-4344 1880

Fax : 91-44-4282 9592

E-mail :export@fourrts.com

8 Manufacturer

M/s. FOURRTS (INDIA) LABORATORIES PVT LIMITED

Address : Vandalur Road, Kelambakkam - 603103, Tamil Nadu, India

Phone : 91-44-4740 4310

Fax : 91-44-2747 5083

9 Date of revision of the text

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