# Summary of Product Characteristics

**1. Name of the medicinal product**
NICARDIA RETARD 20
(Nifedipine Extended Release Tablets USP 20 mg)

**2. Qualitative and quantitative composition**
Each sustained release film coated tablet contains:
Nifedipine USP……..20 mg

For the full list of excipients, see section 6.1

**3. Pharmaceutical form**
Tablets

**4. Clinical particulars**
**4.1 Therapeutic indications**
Hypertension
Prophylaxis of chronic stable angina pectoris

**4.2 Posology and method of administration**
Posology
The recommended starting dose of Nicardia Retard 20 is 10 mg every 12 hours swallowed with water with subsequent titration of dosage according to response. Nicardia Retard 20 permit titration of the initial dosage, which may be adjusted to 40 mg every 12 hours, to a maximum daily dose of 80 mg.
Co-administration with CYP 3A4 inhibitors or CYP 3A4 inducers may result in the recommendation to adapt the nifedipine dose or not to use nifedipine at all (see section 4.5).
Method of administration
Oral use. As a rule, tablets are swallowed whole with a little liquid, either with or without food.
Nicardia Retard 20 should not be taken with grapefruit juice (see section 4.5).
Duration of treatment
Treatment may be continued indefinitely.
*Additional information on special populations*
*Older people (> 65 years)*
The pharmacokinetics of nifedipine are altered in older people so that lower maintenance doses of nifedipine may be required compared to younger patients.
*Patients with hepatic impairment*
Nifedipine is metabolised primarily by the liver and therefore patients with liver dysfunction should be carefully monitored and in severe cases, a dose reduction may be necessary.
*Patients with renal impairment*
Based on pharmacokinetic data, no dosage adjustment is required in patients with renal impairment (see Section 5.2).
*Paediatric population*
The safety and efficacy of nifedipine in children below 18 years of age has not been established. Currently available data for the use of nifedipine in hypertension are described in section 5.1.

**4.3 Contraindications**
• Hypersensitivity to the active substance or other dihydropyridines because of the theoretical risk of cross reactivity or to any of the excipients.

• Nicardia Retard 20 must not be used in cases of cardiogenic shock, clinically significant aortic stenosis, unstable angina, or during or within 4 weeks of a myocardial infarction.
• Nicardia Retard 20 should not be used for the treatment of acute attacks of angina.
• The safety of nifedipine in malignant hypertension has not been established.
• Nicardia Retard 20 should not be used for secondary prevention of myocardial infarction.
• Nicardia Retard 20 should not be administered concomitantly with rifampicin because no efficient plasma levels of nifedipine may be obtained due to enzyme induction (see section 4.5).

**4.4 Special warnings and precautions for use**

Nicardia Retard 20 is not a beta-blocker and therefore gives no protection against the dangers of abrupt beta-blocker withdrawal; any such withdrawal should be a gradual reduction of the dose of beta-blocker preferably over 8 - 10 days.

Nicardia Retard 20 may be used in combination with beta-blocking drugs and other antihypertensive agents but the possibility of an additive effect resulting in postural hypotension should be borne in mind. Nicardia Retard 20 will not prevent possible rebound effects after cessation of other antihypertensive therapy.

Care must be exercised in patients with very low blood pressure (severe hypotension with systolic pressure less than 90 mm HG), in cases of manifest heart failure and in the case of severe aortic stenosis.

Nicardia Retard 20 should not be used during pregnancy unless the clinical condition of the woman requires treatment with nifedipine. Nicardia Retard 20 should be reserved for women with severe hypertension who are unresponsive to standard therapy (see section 4.6).

Careful monitoring of blood pressure must be exercised when administering nifedipine with i.v. magnesium sulfate, owing to the possibility of an excessive fall in blood pressure, which could harm both mother and foetus. For further information regarding use in pregnancy, refer to section 4.6.

Nicardia Retard 20 is not recommended for use during breastfeeding because nifedipine has been reported to be excreted in human milk and the effects of oral absorption of small amounts of nifedipine exposure to the infant are not known (see section 4.6).

In patients with impaired liver function careful monitoring and, in severe cases, a dose reduction may be necessary.

Nicardia Retard 20 should be used with caution in patients whose cardiac reserve is poor.

Deterioration of heart failure has occasionally been observed with nifedipine.

The use of Nicardia Retard 20 in diabetic patients may require adjustment of their control.

In dialysis patients with malignant hypertension and hypovolaemia, a marked decrease in blood pressure can occur.

Nifedipine is metabolised via the cytochrome P450 3A4 system. Drugs that are known to either inhibit or to induce this enzyme system may therefore alter the first pass or the clearance of nifedipine (see section 4.5).

Drugs, which are known inhibitors of the cytochrome P450 3A4 system, and which may therefore lead to increased plasma concentrations of nifedipine are, for example:
• macrolide antibiotics (e.g., erythromycin)
• anti-HIV protease inhibitors (e.g., ritonavir)
• azole antimycotics (e.g., ketoconazole)
• the antidepressants, nefazodone and fluoxetine
• quinupristin/dalfopristin
• valproic acid
• cimetidine

Upon co-administration with these drugs, the blood pressure should be monitored and, if necessary, a reduction of the nifedipine dose should be considered.
Since this medicinal product contains lactose, patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.
For use in special populations see section 4.2.

**4.5 Interaction with other medicinal products and other forms of interaction**
Drugs that affect nifedipine
Nifedipine is metabolised via the cytochrome P450 3A4 system, located both in the intestinal mucosa and in the liver. Drugs that are known to either inhibit or to induce this enzyme system may therefore alter the first pass (after oral administration) or the clearance of nifedipine (see section 4.4).
The extent as well as the duration of interactions should be taken into account when administering nifedipine together with the following drugs:
**Rifampicin:** Rifampicin strongly induces the cytochrome P450 3A4 system. Upon coadministration with rifampicin, the bioavailability of nifedipine is distinctly reduced and thus its efficacy weakened. The use of nifedipine in combination with rifampicin is therefore contraindicated (see section 4.3).
Upon co-administration of the following weak to moderate inhibitors of the cytochrome P450 3A4 system, the blood pressure should be monitored and, if necessary, a reduction in the nifedipine dose considered (see sections 4.2 and 4.4).
In the majority of these cases, no formal studies to assess the potential for a drug interaction between nifedipine and the drug(s) listed have been undertaken, thus far.
**Macrolide antibiotics (e.g., erythromycin)**
No interaction studies have been carried out between nifedipine and macrolide antibiotics. Certain macrolide antibiotics are known to inhibit the cytochrome P450 3A4 mediated metabolism of other drugs. Therefore the potential for an increase of nifedipine plasma concentrations upon co-administration of both drugs cannot be excluded (see section 4.4).
Azithromycin, although structurally related to the class of macrolide antibiotics is void of CYP3A4 inhibition.
**Anti-HIV protease inhibitors (e.g. ritonavir)**
A clinical study investigating the potential of a drug interaction between nifedipine and certain anti-HIV protease inhibitors has not yet been performed. Drugs of this class are known to inhibit the cytochrome P450 3A4 system. In addition, drugs of this class have been shown to inhibit in vitro the cytochrome P450 3A4 mediated metabolism of nifedipine. When administered together with nifedipine, a substantial increase in plasma concentrations of nifedipine due to a decreased first pass metabolism and a decreased elimination cannot be excluded (see section 4.4).
**Azole anti-mycotics (e.g., ketoconazole)**
A formal interaction study investigating the potential of a drug interaction between nifedipine and certain azole anti-mycotics has not yet been performed. Drugs of this class are known to inhibit the cytochrome P450 3A4 system. When administered orally together with nifedipine, a substantial increase in systemic bioavailability of nifedipine due to a decreased first pass metabolism cannot be excluded (see section 4.4).
**Fluoxetine**
A clinical study investigating the potential of a drug interaction between nifedipine and fluoxetine has not yet been performed. Fluoxetine has been shown to inhibit in vitro the cytochrome P450 3A4 mediated metabolism of nifedipine. Therefore an increase of

nifedipine plasma concentrations upon co-administration of both drugs cannot be excluded (see section 4.4).

**Nefazodone**

A clinical study investigating the potential of a drug interaction between nifedipine and nefazodone has not yet been performed. Nefazodone is known to inhibit the cytochrome P450 3A4 mediated metabolism of other drugs. Therefore an increase of nifedipine plasma concentrations upon co-administration of both drugs cannot be excluded (see section 4.4).

**Quinupristin / Dalfopristin**

Simultaneous administration of quinupristin / dalfopristin and nifedipine may lead to increased plasma concentrations of nifedipine (see section 4.4).

**Valproic acid**

No formal studies have been performed to investigate the potential interaction between nifedipine and valproic acid. As valproic acid has been shown to increase the plasma concentrations of the structurally similar calcium channel blocker nimodipine due to enzyme inhibition, an increase in nifedipine plasma concentrations and hence an increase in efficacy cannot be excluded (see section 4.4).

**Cimetidine**

Due to its inhibition of cytochrome P450 3A4, cimetidine elevates the plasma concentrations of nifedipine and may potentiate the antihypertensive effect (see section 4.4).

Further studies

**Cisapride**

Simultaneous administration of cisapride and nifedipine may lead to increased plasma concentrations of nifedipine.

**Cytochrome P450 3A4 system inducing anti-epileptic drugs, such as phenytoin, carbamazepine and phenobarbitone**

Phenytoin induces the cytochrome P450 3A4 system. Upon co-administration with phenytoin, the bioavailability of nifedipine is reduced and thus its efficacy weakened. When both drugs are concomitantly administered, the clinical response to nifedipine should be monitored and, if necessary, an increase in the nifedipine dose considered. If the dose of nifedipine is increased during coadministration of both drugs, a reduction of the nifedipine dose should be considered when the treatment with phenytoin is discontinued.

No formal studies have been performed to investigate the potential interaction between nifedipine and carbamazepine or phenobarbitone. As both drugs have been shown to reduce the plasma concentrations of the structurally similar calcium channel blocker nimodipine due to enzyme induction, a decrease in nifedipine plasma concentrations and hence a decrease in efficacy cannot be excluded.

Effects of nifedipine on other drugs

**Blood pressure lowering drugs**

Nifedipine may increase the blood pressure lowering effect of concomitant applied antihypertensives, such as:

- diuretics,
- β-blockers,
- ACE-inhibitors,
- Angiotensin 1(AT1) receptor- antagonists,
- other calcium antagonists,
- α-adrenergic blocking agents,
- PDE5 inhibitors,
- α-methyldopa

When nifedipine is administered simultaneously with ß-receptor blockers the patient should be carefully monitored, since deterioration of heart failure is also known to develop in isolated cases.

**Digoxin**

The simultaneous administration of nifedipine and digoxin may lead to reduced digoxin clearance and, hence, an increase in the plasma concentrations of digoxin. The patient should therefore be checked for symptoms of digoxin overdosage as a precaution and, if necessary, the glycoside dose should be reduced taking account of the plasma concentration of digoxin.

**Quinidine**

When nifedipine and quinidine have been administered simultaneously, lowered quinidine or, after discontinuation of nifedipine, a distinct increase in plasma concentrations of quinidine has been observed in individual cases. For this reason, when nifedipine is either additionally administered or discontinued, monitoring of the quinidine plasma concentration and, if necessary, adjustment of the quinidine dose are recommended. Some authors reported increased plasma concentrations of nifedipine upon co-administration of both drugs, while others did not observe an alteration in the pharmacokinetics of nifedipine.

Therefore, the blood pressure should be carefully monitored, if quinidine is added to an existing therapy with nifedipine. If necessary, the dose of nifedipine should be decreased.

**Tacrolimus**

Tacrolimus has been shown to be metabolised via the cytochrome P450 3A4 system. Data recently published indicates that the dose of tacrolimus administered simultaneously with nifedipine may be reduced in individual cases. Upon co-administration of both drugs, the tacrolimus plasma concentrations should be monitored and, if necessary, a reduction in the tacrolimus dose considered.

Drug food interactions

**Grapefruit juice inhibits**

Grapefruit juice inhibits the cytochrome P450 3A4 system. Administration of nifedipine together with grapefruit juice thus results in elevated plasma concentrations and prolonged action of nifedipine due to a decreased first pass metabolism or reduced clearance. As a consequence, the blood pressure lowering effect of nifedipine may be increased. After regular intake of grapefruit juice, this effect may last for at least three days after the last ingestion of grapefruit juice.

Ingestion of grapefruit/grapefruit juice is therefore to be avoided while taking nifedipine (see section 4.2).

Other forms of interaction

Nifedipine may increase the spectrophotometric values of urinary vanillylmandelic acid, falsely. However, HPLC measurements are unaffected.

## 4.6 Fertility, pregnancy and breastfeeding

Nifedipine should not be used during pregnancy unless the clinical condition of the woman requires treatment with nifedipine. Nifedipine should be reserved for women with severe hypertension who are unresponsive to standard therapy (see section 4.4).

There are no adequate well controlled studies in pregnant women.

The available information is inadequate to rule out adverse drug effects on the unborn and newborn child. Therefore any use in pregnancy requires a very careful individual risk benefit assessment and should only be considered if all other treatment options are either not indicated or have failed to be efficacious.

In animal studies, nifedipine has been shown to produce embryotoxicity, foetotoxicity and teratogenicity (see section 5.3).

From the clinical evidence available a specific prenatal risk has not been identified. Although an increase in perinatal asphyxia, caesarean delivery, as well as prematurity and intrauterine growth retardation have been reported. It is unclear whether these reports are due to the underlying hypertension, its treatment, or to a specific drug effect.
Acute pulmonary oedema has been observed when calcium channel blockers, among others nifedipine, have been used as a tocolytic agent during pregnancy (see section 4.8), especially in cases of multiple pregnancy (twins or more), with the intravenous route and/or concomitant use of beta-2 agonists.

**Breast-feeding**

Nifedipine is excreted in the breast milk. The nifedipine concentration in the milk is almost comparable with mother serum concentration. For immediate release formulations, it is proposed to delay breastfeeding or milk expression for 3 to 4 hours after drug administration to decrease the nifedipine exposure to the infant (see section 4.4).

**Fertility**

In single cases of *in vitro* fertilisation calcium antagonists like nifedipine have been associated with reversible biochemical changes in the spermatozoa's head section that may result in impaired sperm function. In those men who are repeatedly unsuccessful in fathering a child by *in vitro* fertilisation, and where no other explanation can be found, calcium antagonists like nifedipine should be considered as possible causes.

## 4.7 Effects on ability to drive and use machines

Reactions to the drug, which vary in intensity from individual to individual, may impair the ability to drive or to operate machinery (see section 4.8). This applies particularly at the start of treatment, on changing the medication and in combination with alcohol.

## 4.8 Undesirable effects

Adverse drug reactions (ADRs) based on placebo-controlled studies with nifedipine sorted by CIOMS III categories of frequency (clinical trial data base: nifedipine n = 2,661; placebo n = 1,486; status: 22 Feb 2006 and the ACTION study: nifedipine n = 3,825; placebo n = 3,840) are listed below:
ADRs listed under "common" were observed with a frequency below 3% with the exception of oedema (9.9%) and headache (3.9%).
The frequencies of ADRs reported with nifedipine-containing products are summarised in the table below. Within each frequency grouping, undesirable effects are presented in order of decreasing seriousness. Frequencies are defined as common (≥1/100 to < 1/10), uncommon (≥ 1/1,000 to < 1/100) and rare (≥ 1/10,000 to < 1/1,000). The ADRs identified only during the ongoing postmarketing surveillance, and for which a frequency could not be estimated, are listed under “Not known”

| System Organ Class (MedDRA) | Common | Uncommon | Rare | Not known |
|---|---|---|---|---|
| **Blood and Lymphatic System Disorders** | | | | Agranulocytosis<br>Leucopenia |
| **Immune System Disorders** | | Allergic reaction<br>Allergic oedma /angioedema (incl. larynxoedema[1]) | Pruritus<br>Urticaria<br>Rash | Anaphylactic /anaphylactoid reaction |
| **Psychiatric Disorders** | | Anxiety reactions<br>Sleep disorders | | |

| | | | | |
|---|---|---|---|---|
| **Metabolism and Nutrition Disorders** | | | | Hyperglycaemia |
| **Nervous System Disorders** | Headache | Vertigo<br>Migraine<br>Dizziness<br>Tremor | Par-/Dysaesthesia | Hypoaesthesia<br>Somnolence |
| **Eye Disorders** | | Visual disturbances | | Eye pain |
| **Cardiac Disorders** | | Tachycardia<br>Palpitations | | Chest pain (Angina pectoris) |
| **Vascular Disorders** | Oedema (incl. peripheral oedema)<br>Vasodilatation | Hypotension<br>Syncope | | |
| **Respiratory, Thoracic and Mediastinal Disorders** | | Nosebleed<br>Nasal congestion | | Dyspnoea<br>Pulmonary oedema[2] |
| **Gastrointestinal Disorders** | Constipation | Gastrointestinal andabdominal pain<br>Nausea<br>Dyspepsia<br>Flatulence<br>Dry mouth | Gingival hyperplasia | Vomiting<br>Gastroesophagealsphincter insufficiency |
| **Hepatobiliary Disorders** | | Transient increase in liver enzymes | | Jaundice |
| **Skin and Subcutaneous Tissue Disorders** | | Erythema | | Toxic Epidermal Necrolysis<br>Photosensitivity allergic reaction<br>Palpable purpura |
| **Musculoskeletal and Connective Tissue Disorders** | | Muscle cramps<br>Joint swelling | | Arthralgia<br>Myalgia |
| **Renal and Urinary Disorders** | | Polyuria<br>Dysuria | | |
| **Reproductive System and Breast Disorders** | | Erectile dysfunction | | |
| **General Disorders and Administration Site Conditions** | Feeling unwell | Unspecific pain<br>Chills | | |

[1] may result in life-threatening outcome
[2] cases have been reported when used as tocolytic during pregnancy (see section 4.6)
In dialysis patients with malignant hypertension and hypovolaemia a distinct fall in blood pressure can occur as a result of vasodilation.

**4.9 Overdose**
Symptoms
The following symptoms are observed in cases of severe nifedipine intoxication:
Disturbances of consciousness to the point of coma, a drop in blood pressure, tachycardiac / bradycardiac heart rhythm disturbances, hyperglycaemia, metabolic acidosis, hypoxia, cardiogenic shock with pulmonary oedema.

Management
As far as treatment is concerned, elimination of nifedipine and the restoration of stable cardiovascular conditions have priority.
After oral ingestion thorough gastric lavage is indicated, if necessary in combination with irrigation of the small intestine.
Particularly in cases of intoxication with slow release nifedipine formulations, elimination must be as complete as possible, including the small intestine, to prevent the otherwise inevitable subsequent absorption of the active substance.
Haemodialysis serves no purpose as nifedipine is not dialysable, but plasmapheresis is advisable (high plasma protein binding, relatively low volume of distribution).
Hypotension as a result of cardiogenic shock and arterial vasodilatation can be treated with calcium (10-20 ml of a 10 % calcium gluconate solution administered slowly i.v. and repeated if necessary). As a result, the serum calcium can reach the upper normal range to slightly elevated levels. If an insufficient increase in blood pressure is achieved with calcium, vasoconstricting sympathomimetics such as dopamine or noradrenaline should be administered. The dosage of these drugs should be determined by the patient's response.
Symptomatic bradycardia may be treated with atropine, beta-sympathomimetics or a temporary cardiac pacemaker, as required.
Additional liquid or volume must be administered with caution because of the danger of overloading the heart.

**5. Pharmaceutical properties**
Pharmacotherapeutic group: Dihydropyridine derivatives

**5.1 Pharmacodynamic properties**
ATC code: C08CA05
Nifedipine is a specific and potent calcium antagonist of the 1, 4-dihydropyridine type. Calcium antagonists reduce the transmembranal influx of calcium ion inflow through the slow calcium channel into the cell. Nifedipine acts particularly on the cells of the myocardium and the smooth muscle cells of the coronary arteries and the peripheral resistance vessels.
In hypertension, the main action of nifedipine is to cause peripheral vasodilatation and thus reduce peripheral resistance.
In angina, nifedipine reduces peripheral and coronary vascular resistance, leading to an increase in coronary blood flow, cardiac output and stroke volume, whilst decreasing after-load.

Additionally, nifedipine dilates submaximally both clear and atherosclerotic coronary arteries, thus protecting the heart against coronary artery spasm and improving perfusion to the ischaemic myocardium.
Nifedipine reduces the frequency of painful attacks and the ischaemic ECG changes irrespective of the relative contribution from coronary artery spasm or atherosclerosis.
Nifedipine administered twice-daily provides 24-hour control of raised blood pressure. Nifedipine causes reduction in blood pressure such that the percentage lowering is directly related to its initial level. In normotensive individuals, Nifedipine has little or no effect on blood pressure.
Paediatric population
Limited information on comparison of nifedipine with other antihypertensives is available for both acute hypertension and long-term hypertension with different formulations in different dosages. Antihypertensive effects of nifedipine have been demonstrated but dose recommendations, long term safety and effect on cardiovascular outcome remain unestablished. Paediatric dosing forms are lacking

**5.2 Pharmacokinetic properties**
Absorption
The active substance nifedipine is rapidly and almost completely absorbed from the gastro-intestinal tract after oral administration on an empty stomach. Nifedipine is subject to a "first pass metabolism" in the liver, resulting in a systemic availability of orally administered nifedipine of between 50 to 70%. Following administration of a nifedipine-containing solution maximum serum concentrations are reported to occur after approx. 15 minutes. After the administration of other preparations having an immediate release peak serum concentrations are attained after 15 to 75 minutes.

Distribution
Nifedipine is about 95 % bound to plasma protein (albumin). The distribution half-life after intravenous administration was determined to be 5 to 6 minutes.

Biotransformation
Nifedipine is almost completely metabolised in the liver by oxidative and hydrolytic processes. These metabolites do not show any pharmacodynamic activity.
About 70 to 80% of a nifedipine dose is excreted in the urine in the form of its metabolites, the main metabolite (M-I) accounts for about 60 to 80% of the administered nifedipine dose. The rest is excreted in form of metabolites with the faeces. The unaltered substance is found only in traces (less than 0.1%) in the urine.

Elimination
The elimination half-life is about 2 to 5 hours.
No accumulation of the substance after the usual dose was reported during long-term treatment. In cases of impaired kidney function no substantial changes have been detected in comparison with healthy volunteers.
In cases of impaired liver function the elimination half-life is distinctly prolonged and the total clearance is reduced. A dose reduction may be necessary in severe cases.

Bioavailability
A bioavailability study with Nicardia Retard 20 made in the year 1991 with 24 volunteers showed the following results compared to the reference preparation:

| | Test preparation: | Reference preparation: |
|---|---|---|

| | | |
|---|---|---|
| Maximum steady-state plasma concentration (0- 12 h) (Css,max1 ) (ng/ml): | 36.3±12.1 | 39.8±15.9 |
| Maximum steady-state plasma concentration (12-24 h) (Css,max2 ) (ng/ml): | 39.1±15.4 | 50.3±19.6 |
| Area under the concentration-time-curve (24h) (AUCss ) (ng/ml*h): | 394.3±165.7 | 435.6±194.6 |
| Plateau time (0-24h) (h): | 3.67±1.37 | 3.68±1.97 |
| Peak-trough-fluctuation (0-12h) (PTF1) (%): | 182.1±40.3 | 204.6±66.7 |
| Peak-trough-fluctuation (12-24h) (PTF2) (%): | 206.4±48.2 | 246.6±85.6 |
| Values as mean values ± SD. | | |

**5.3 Preclinical safety data**

Preclinical data reveal no special hazard for humans based on conventional studies of single and repeated dose toxicity, genotoxicity and carcinogenic potential.

Reproduction toxicology

Nifedipine has been shown to produce teratogenic findings in rats, mice and rabbits, including digital anomalies, malformation of the extremities, cleft palates, cleft sternum, and malformation of the ribs. Digital anomalies and malformation of the extremities are possibly a result of compromised uterine blood flow, but have also been observed in animals treated with nifedipine solely after the end of the organogenesis period.

Nifedipine administration was associated with a variety of embryotoxic, placentotoxic and foetotoxic effects, including stunted foetuses (rats, mice, rabbits), small placentas and underdeveloped chorionic villi (monkeys), embryonic and foetal deaths (rats, mice, rabbits) and prolonged pregnancy/decreased neonatal survival (rats; not evaluated in other species). The risk to humans cannot be ruled out if a sufficiently high systemic exposure is achieved, however, all of the doses associated with the teratogenic, embryotoxic or foetotoxic effects in animals were maternally toxic and were several times the recommended maximum dose for humans (see section 4.6).

## 6. Pharmaceutical particulars

**6.1 List of excipients**

Hypromellose USP
Microcrystalline Cellulose NF
Lactose Monohydrate NF
Dibasic Calcium Phosphate Dihydrate USP
Corn Starch NF
Magnesium Stearate NF
Tabcoat TC Orange 2115
Purified Water USP

**6.2 Incompatibilities**

Not Applicable

**6.3 Shelf life**

3 years

**6.4 Special precautions for storage**

Store below 30ºC. Protect from light.

**6.5 Nature and content of container**

Alu-PVC blister of 10 tablets

**6.6 Special precautions for disposal and other handling**

Not Applicable

**7. Marketing authorization holder**

**UNIQUE PHARMACEUTICAL LABORATORIES**
(A Div. of J. B. Chemicals and Pharmaceuticals Ltd.)
Neelam Centre, B wing, 4th Floor, Hind Cycle Road,
Worli Mumbai – 400 030

**8. Marketing Authorization Number**

05126/07072/NMR/2018

**9. Date of First Authorization/Renewal of the Authorization**

20/04/2020

**10. Date of revision of the text**

27/07/2023