SUMMARY OF PRODUCT CHARACTERISTICS

1 NAME OF THE MEDICINAL PRODUCT

Amiodarone Hydrochloride 200mg Tablets

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Each tablet contains 200mg Amiodarone Hydrochloride

For the full list of excipients, see section 6.1

3 PHARMACEUTICAL FORM

Tablet
Flat white tablets, marked “200”, with break line

4 CLINICAL PARTICULARS

4.1 Therapeutic indications
Amiodarone hydrochloride is indicated only for the treatment of severe rhythm disorders not responding to other therapies or when other treatments cannot be used. Treatment should be initiated and normally monitored only under hospital or specialist supervision.

Atrial flutter and fibrillation when other drugs cannot be used.

All types of tachyarrhythmias of paroxysmal nature including:
supraventricular, nodal and ventricular tachycardias, ventricular fibrillation; when other drugs cannot be used.

Tachyarrhythmias associated with Wolff-Parkinson-White Syndrome.

Tablets are used for stabilisation and long-term treatment.

4.2. Posology and Method of Administration

For oral administration

Adults:
It is particularly important that the minimum effective dose be used. In all cases the patient’s management must be judged on the individual response and well-being. The following dosage regimen is generally effective

Initial Stabilisation
Treatment should be started with 200 mg, three times a day and may be continued for one week. The dosage should then be reduced to 200mg, twice daily for a further week.

Maintenance
After the initial period the dosage should be reduced to 200mg daily, or less if appropriate. The scored 100mg tablet should be used to titrate the minimum dosage required to maintain control of the arrhythmia. The Maintenance dosage should be regularly reviewed, especially in rare cases where the patient may require a higher maintenance dose and where this exceeds 200mg daily.

General Considerations

Initial dosing
A high dose is needed in order to achieve adequate tissue levels rapidly

Maintenance
In patients with potentially lethal arrhythmias the long half-life is a valuable safeguard, as omission of occasional doses does not significantly influence the overall therapeutic effect. It is particularly important that the minimum effective dosage is used and the patient is monitored regularly to detect clinical signs of excess Amiodarone dosage. Therapy may then be adjusted accordingly. Too high a dose during maintenance therapy can cause side effects which are believed to be related to high tissue levels of amiodarone and its metabolites.

Sufficient time must be allowed for new distribution equilibrium to be achieved between dosage adjustments. Amiodarone is strongly protein bound and has an average plasma half-life of fifty days (reported range 20-100 days).

Dosage reduction/withdrawal
Side effects slowly disappear as tissue levels fall. Following drug withdrawal, residual tissue bound Amiodarone may protect a patient for up to one month. However, the likelihood of the occurrence of arrhythmia during this period should be considered.

Elderly
As with all patients it is important that the minimum effective dosage is used. Whilst there is no evidence that dosage requirements are different for the elderly, they may be more susceptible to bradycardia and conduction defects if too high a dose is employed. Particular attention should be paid to monitoring thyroid function. (See sections 4.3, 4.4, 4.8).

Paediatric population
The safety and efficacy of amiodarone in children has not been established.

Currently available data are described in sections 5.1 and 5.2.
4.3. **Contra-indications**

Sinus bradycardia and sino-atrial heart block. In patients with severe conduction disturbances (high grade AV block, bifascicular or trifascicular block) or sinus node disease, Amiodarone should be used only in conjunction with a pacemaker.

Where there is evidence or history of thyroid dysfunction. A thyroid function test should be performed prior to therapy in all patients.

Known hypersensitivity to iodine or to Amiodarone or to any of the excipients listed in section 6.1 (one 100 mg tablet contains approximately 37.5 mg iodine, one 200 mg tablet contains approximately 75 mg iodine).

The combination of Amiodarone with drugs which may prolong the QT interval and thereby induce Torsades de Pointes ventricular tachycardia is contra-indicated (see 4.5 section).

Pregnancy-except in exceptional circumstances (see section 4.6)

Amiodarone is contra-indicated in nursing mothers (see section 4.6).

4.4. **Special Warnings and Precautions for Use**

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

Amiodarone can cause serious adverse reactions affecting the eyes, heart, lung, liver, thyroid gland, skin and peripheral nervous system (see section 4.8). Because these reactions may be delayed, patients on long-term treatment should be carefully supervised. As undesirable effects are usually dose-related, the minimum effective maintenance dose should be given.

Before surgery, the anaesthetist should be informed that the patient is taking amiodarone (see sections 4.5 and 4.8).

**Cardiac disorders (see section 4.8):**

Too high a dosage may lead to severe bradycardia and to conduction disturbances with the appearance of an idioventricular rhythm, particularly in elderly patients or during digitalis therapy. In these circumstances, Amiodarone treatment should be withdrawn. If necessary beta-adrenostimulants or glucagon may be given. Because of the long half-life of amiodarone, if bradycardia is severe and symptomatic the insertion of a pacemaker should be considered.

Oral Amiodarone is not contra-indicated in patients with latent or manifest heart failure but caution should be exercised as, occasionally, existing heart failure may be worsened. In such cases, Amiodarone may be used with other appropriate therapies.

The pharmacological action of amiodarone induces ECG changes: QT prolongation (related to prolonged repolarisation) with the possible development of U-waves and deformed T-waves; these changes do not reflect toxicity.
In the elderly, heart rate may decrease markedly.

Treatment should be discontinued in case of onset of 2nd or 3rd degree A-V block, sino-atrial block, or bifascicular block.

Amiodarone has a low pro-arrhythmic effect. Onsets of new arrhythmias or worsening of treated arrhythmias, sometimes fatal, have been reported. It is important, but difficult, to differentiate a lack of efficacy of the drug from a proarrhythmic effect, whether or not this is associated with a worsening of the cardiac condition. Proarrhythmic effects generally occur in the context of drug interactions and/or electrolytic disorders (see sections 4.5. and 4.8).

Before starting amiodarone, it is recommended to perform an ECG and serum potassium measurement. Monitoring of ECG is recommended during treatment.

Amiodarone may increase the defibrillation threshold and/or pacing threshold in patients with an implantable cardioverter defibrillator or a pacemaker, which may adversely affect the efficacy of the device. Regular tests are recommended to ensure the proper function of the device after initiation of treatment or change in posology.

Endocrine disorders (see section 4.8)

Amiodarone may induce hypothyroidism or hyperthyroidism, particularly in patients with a personal history of thyroid disorders. Clinical and biological [including ultrasensitive TSH (usTSH)] monitoring should be performed prior to therapy in all patients. Monitoring should be carried out during treatment, at six-monthly intervals, and for several months following its discontinuation. This is particularly important in the elderly. In patients whose history indicates an increased risk of thyroid dysfunction, regular assessment is recommended. Serum usTSH level should be measured when thyroid dysfunction is suspected.

Amiodarone contains iodine and thus may interfere with radio-iodine uptake. However, thyroid function tests (free-T3, free-T4, usTSH) remain interpretable.

Amiodarone inhibits peripheral conversion of levothyroxine (T4) to triiodothyronine (T3) and may cause isolated biochemical changes (increase in serum free-T4, free-T3 being slightly decreased or even normal) in clinically euthyroid patients. There is no reason in such cases to discontinue amiodarone treatment if there is no clinical or further biological (usTSH) evidence of thyroid disease.

Hypothyroidism

Hypothyroidism should be suspected if the following clinical signs occur: weight gain, cold intolerance, reduced activity, excessive bradycardia. The diagnosis is supported by an increase in serum usTSH and an exaggerated TSH response to TRH. T3 and T4 levels may be low. Euthyroidism is usually obtained within 3 months following the discontinuation of treatment. In life-threatening situations, amiodarone therapy can be continued, in combination with levothyroxine. The dose of levothyroxine is adjusted according to TSH levels.

Hyperthyroidism

Hyperthyroidism may occur during amiodarone treatment, or, up to several months after discontinuation. Clinical features, such as weight loss, asthenia, restlessness,
increase in heart rate, onset of arrhythmia, angina, congestive heart failure should alert the physician. The diagnosis is supported by a decrease in serum usTSH level, an elevated T₃ and a reduced TSH response to thyrotropin releasing hormone. Elevation of reverse T₃ (rT₃) may also be found.

In the case of hyperthyroidism, therapy should be withdrawn. Clinical recovery usually occurs within a few months, although severe cases, sometimes resulting in fatalities, have been reported. Clinical recovery precedes the normalisation of thyroid function tests.

Courses of anti-thyroid drugs have been used for the treatment of severe thyroid hyperactivity; large doses may be required initially. These may not always be effective and concomitant high dose corticosteroid therapy (e.g. 1mg/kg prednisolone) may be required for several weeks.

Eye disorders (see section 4.8)

If blurred or decreased vision occurs, complete ophthalmologic examination including fundoscopy should be promptly performed. Appearance of optic neuropathy and/or optic neuritis requires amiodarone withdrawal due to the potential progression to blindness. Unless blurred or decreased vision occurs, ophthalmological examination is recommended annually.

Hepato-biliary disorders (see section 4.8):

Amiodarone may be associated with a variety of hepatic effects, including cirrhosis, hepatitis, jaundice and hepatic failure. Some fatalities have been reported, mainly following long-term therapy, although rarely they have occurred soon after starting treatment particularly after Amiodarone intravenous. It is advisable to monitor liver function particularly transaminases before treatment and six monthly thereafter.

At the beginning of therapy, elevation of serum transaminases which can be in isolation (1.5 to 3 times normal) may occur. These may return to normal with dose reduction, or sometimes spontaneously.

Isolated cases of acute liver disorders with elevated serum transaminases and/or jaundice may occur; in such cases treatment should be discontinued.

There have been reports of chronic liver disease. Alteration of laboratory tests which may be minimal (transaminases elevated 1.5 to 5 times normal) or clinical signs (possible hepatomegaly) during treatment for longer than 6 months should suggest this diagnosis. Routine monitoring of liver function tests is therefore advised. Abnormal clinical and laboratory test results usually regress upon cessation of treatment, but fatal cases have been reported. Histological findings may resemble pseudo-alcoholic hepatitis, but they can be variable and include cirrhosis.

Although there have been no literature reports on the potentiation of hepatic adverse effects of alcohol, patients should be advised to moderate their alcohol intake while taking Amiodarone.

Nervous system disorders (see section 4.8):
Amiodarone may induce peripheral sensorimotor neuropathy and/or myopathy. Both these conditions may be severe, although recovery usually occurs within several months after amiodarone withdrawal, but may sometimes be incomplete.

**Respiratory, thoracic and mediastinal disorders (see section 4.8):**

Onset of dyspnoea or non-productive cough may be related to pulmonary toxicity (hypersensitivity pneumonitis, alveolar/interstitial pneumonitis or fibrosis, pleuritis, bronchiolitis obliterans organising pneumonitis. Presenting features can include dyspnoea (which may be severe and unexplained by the current cardiac status), non-productive cough and deterioration in general health (fatigue, weight loss and fever). The onset is usually slow but may be rapidly progressive. Whilst the majority of cases have been reported with long term therapy, a few have occurred soon after starting treatment.

Patients should be carefully evaluated clinically and consideration given to chest X-rays before starting therapy. During treatment, if pulmonary toxicity is suspected, this should be repeated and associated with lung function testing including, where possible, measurement of transfer factor. Initial radiological changes may be difficult to distinguish from pulmonary venous congestion. Pulmonary toxicity has usually been reversible following early withdrawal of amiodarone therapy, with or without corticosteroid therapy. Clinical symptoms often resolve within a few weeks followed by slower radiological and lung function improvement. Some patients can deteriorate despite discontinuing Amiodarone.

**Skin and subcutaneous tissue disorders (see section 4.8)**

Patients should be instructed to avoid exposure to sun and to use protective measures during therapy as patients taking Amiodarone can become unduly sensitive to sunlight, which may persist after several months of discontinuation of Amiodarone. In most cases symptoms are limited to tingling, burning and erythema of sun-exposed skin but severe phototoxic reactions with blistering may be seen.

**Drug interactions (see section 4.5)**

Concomitant use of amiodarone is not recommended with the following drugs: beta-blockers, heart rate lowering calcium channel inhibitors (verapamil, diltiazem), stimulant laxative agents which may cause hypokalaemia. Increased plasma levels of flecainide have been reported with co-administration of amiodarone. The flecainide dose should be reduced accordingly and the patient closely monitored.

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

Some of the more important drugs that interact with Amiodarone include warfarin, digoxin, phenytoin and any drugs which prolong the QT interval.

Amiodarone raises the plasma concentrations of highly protein bound drugs, for example oral anticoagulants (warfarin) and phenytoin by inhibition of CYP 2C9. The dose of warfarin should be reduced accordingly. More frequent monitoring of prothrombin time both during and after Amiodarone treatment is recommended.
Phenytoin dosage should be reduced if signs of overdose appear, and plasma levels may be measured.

Administration of Amiodarone to a patient already receiving digoxin will bring about an increase in the plasma digoxin concentration and thus precipitate symptoms and signs associated with high digoxin levels. Clinical, ECG and biological monitoring is recommended and digoxin dosage should be halved. A synergistic effect on heart rate and atrioventricular conduction is also possible.

Fluoroquinolones

There have been rare reports of QTc interval prolongation, with or without torsades de pointes, in patients taking amiodrone with fluoroquinolones. Concomitant use of amiodarone with fluoroquinolones should be avoided (concomitant use with moxifloxacin is contra-indicated, see above).

Combined therapy with any drug known to prolong the QT interval is contra-indicated (see section 4.3) due to the increased risk of Torsade de Pointes. They include the following:

- Class Ia anti-arrhythmic drugs e.g. Quinidine, procainamide, disopyramide
- Class III anti-arrhythmic drugs e.g. sotalol, bretylium
- Intravenous erythromycin, co-trimoxazole or pentamidine injection
- Anti-psychotics e.g. Chlorpromazine, thioridazine, pimozide, haloperidol, fluphenazine, amisulpiride and sertindole
- Lithium and tricyclic anti-depressants e.g. Doxepin, maprotiline, amitriptyline
- Certain anti-histamines e.g. Terfenadine, astemizole, mizolastine
- Anti-malarials e.g. Quinine, mefloquine, halofantrine, chloroquine
- Moxifloxacin

Combined therapy with the following drugs is not recommended:
Beta blockers and certain calcium channel inhibitors (diltiazem, verapamil); potentiation of negative chronotropic properties and conduction slowing effects may occur.
Stimulant laxatives, which may cause hypokalaemia thus increasing the risk of torsades de pointes; other types of laxatives should be used.

Caution should be exercised over combined therapy with the following drugs which may cause hypokalemia: and/or hypomagnesaemia: diuretics, systemic corticosteroids, tetracosactide, intravenous amphotericin.

Hypokalemia: corrective action should be taken and QT interval monitored.

Torsades de Points: antiarrhythmic agents should not be given; pacing may be instituted and IV magnesium may be used.

General anaesthesia: caution is advised, also in patients receiving high dose oxygen therapy. The anaesthetist should be informed that the patient is taking Amiodarone. Potentially severe complications have been reported in patients taking Amiodarone undergoing general anaesthesia, such as bradycardia unresponsive to atropine, hypotension, disturbances of conduction, decreased cardiac output.
A few cases of adult respiratory distress syndrome, most often in the period immediately after surgery, have been observed. A possible interaction with the high oxygen concentration may be implicated.

Grapefruit juice inhibits cytochrome P450 3A4 and may increase the plasma concentration of amiodarone. Grapefruit juice should be avoided during treatment with oral amiodarone.

**Drugs metabolised by cytochrome P450 3A4**

When drugs are co-administered with amiodarone, an inhibitor of CYP 3A4, this may result in a higher level of their plasma concentrations, which may lead to a possible increase in their toxicity:

- **Ciclosporin**: plasma levels of ciclosporin may increase as much as 2-fold when used in combination. A reduction in the dose of ciclosporin may be necessary to maintain the plasma concentration within the therapeutic range.

- **Statins**: the risk of muscular toxicity is increased by concomitant administration of amiodarone with statins metabolised by CYP 3A4 such as simvastatin, atorvastatin and lovastatin. It is recommended to use a statin not metabolised by CYP 3A4 when given with amiodarone.

- **Other drugs metabolised by cytochrome P450 3A4**: examples of such drugs are lidocaine, tacrolimus, sildenafil, fentanyl, midazolam, triazolam, dihydroergotamine and ergotamine.

**Flecainide**

Given that flecainide is mainly metabolised by CYP 2D6, by inhibiting this isoenzyme, amiodarone may increase flecainide plasma levels; it is advised to reduce the flecainide dose by 50% and to monitor the patient closely for adverse effects. Monitoring of flecainide plasma levels is strongly recommended in such circumstances.

**Interaction with substrates of other CYP 450 isoenzymes**

In vitro studies show that amiodarone also has the potential to inhibit CYP 1A2, CYP 2C19 and CYP 2D6 through its main metabolite. When co-administered, amiodarone would be expected to increase the plasma concentration of drugs whose metabolism is dependent upon CYP 1A2, CYP 2C19 and CYP 2D6.

### 4.6 Fertility, pregnancy and lactation

**Pregnancy**

There are insufficient data on the use of Amiodarone during pregnancy in humans to judge any possible toxicity. In view of the pharmacological properties of the drug on the foetus and its effects on the foetal thyroid gland, its administration in pregnancy should be avoided, except in exceptional circumstances.
If, because of the long half-life of amiodarone, discontinuation of the drug is considered prior to planned conception, the real risk of reoccurrence of life threatening arrhythmias should be weighed against the possible hazard for the foetus.

**Breast-feeding**
Amiodarone is excreted into the breast milk in significant quantities and breast-feeding is contra-indicated.

### 4.7 Effects on ability to drive and use machines

Amiodarone may impair on the ability to drive and use machines in patients with clinical symptoms of amiodarone-induced eye disorders.

### 4.8 Undesirable effects

The following adverse reactions are classified by system organ class and ranked under heading of frequency using the following convention: very common (>= 10%), common (>= 1% and < 10%); uncommon (>= 0.1% and < 1%); rare (>= 0.01% and < 0.1%), very rare (< 0.01%), not known (cannot be estimated from the available data).

**Blood and lymphatic system disorders:**
- Very rare:
  - haemolytic anemia
  - aplastic anaemia
  - thrombocytopenia.

In patients taking amiodarone there have been incidental findings of bone marrow granulomas. The clinical significance of this is unknown.

**Cardiac disorders:**
- Common: bradycardia, generally moderate and dose-related.
- Uncommon:
  - onset or worsening of arrhythmia, sometimes followed by cardiac arrest (see sections 4.4 and 4.5.)
  - conduction disturbances (sinoatrial block, AV block of various degrees) (see section 4.4)
- Very rare: marked bradycardia or sinus arrest in patients with sinus node dysfunction and/or in elderly patients.
Endocrine disorders (see section 4.4):

- Common:
  - hypothyroidism
  - hyperthyroidism, sometimes fatal

- Very rare
  - syndrome of inappropriate antidiuretic hormone secretion (SIADH)

Eye disorders:

- Very common: corneal microdeposits usually limited to the area under the pupil, which are usually only discernable by slit-lamp examinations. They may be associated with colored halos in dazzling light or blurred vision. Corneal microdeposits consist of complex lipid deposits and are reversible following discontinuation of treatment. The deposits are considered essentially benign and do not require discontinuation of amiodarone.

- Very rare: optic neuropathy/neuritis that may progress to blindness (see section 4.4).

Gastrointestinal disorders:

- Very common: benign gastrointestinal disorders (nausea, vomiting, dysgeusia) usually occurring with loading dosage and resolving with dose reduction.

Hepato-biliary disorders: (see section 4.4).

- Very common: isolated increase in serum transaminases, which is usually moderate (1.5 to 3 times normal range), occurring at the beginning of therapy. It may return to normal with dose reduction or even spontaneously.

- Common: acute liver disorders with high serum transaminases and/or jaundice, including hepatic failure, which are sometimes fatal

- Very rare: chronic liver disease (pseudo alcoholic hepatitis, cirrhosis), sometimes fatal.

Immune system disorders:

Angioedema (there have been some reports of angioedema, although exact frequencies are not known)

Investigations:

- Very rare: increase in blood creatinine.

Nervous system disorders:

- Common:
- extrapyramidal tremor, for which regression usually occurs after reduction of dose or withdrawal
- nightmares
- sleep disorders.

• Uncommon: peripheral sensorimotor neuropathy and/or myopathy, usually reversible on withdrawal of the drug (see section 4.4).

• Very rare:

- cerebellar ataxia, for which regression usually occurs after reduction of dose or withdrawal
- benign intracranial hypertension (pseudo-tumor cerebri)
- headache
- vertigo.

Reproductive system and breast disorders:

• Very rare:

- epididymo-orchitis
- impotence.

Respiratory, thoracic and mediastinal disorders:

• Common: pulmonary toxicity [hypersensitivity pneumonitis, alveolar/interstitial pneumonitis or fibrosis, pleuritis, bronchiolitis obliterans organising pneumonia (BOOP)], sometimes fatal (see section 4.4).

• Very rare:

- bronchospasm in patients with severe respiratory failure and especially in asthmatic patients
- surgery (possible interaction with a high oxygen concentration) (see sections 4.4 and 4.5).

Pulmonary haemorrhage (there have been some reports of pulmonary haemorrhage, although exact frequencies are not known)

Skin and subcutaneous tissue disorders:

• Very common: photosensitivity (see section 4.4).
• Common: slate grey or bluish pigmentations of light-exposed skin, particularly the face, in case of prolonged treatment with high daily dosages; such pigmentations slowly disappear following treatment discontinuation.

• Very rare:
  - erythema during the course of radiotherapy
  - skin rashes, usually non-specific
  - exfoliative dermatitis
  - alopecia.

• Not known: urticaria

Vascular disorders:
• Very rare: vasculitis.

4.9 Overdose

Little information is available regarding acute overdosage with oral amiodarone. Few cases of sinus bradycardia, heart block, attacks of ventricular tachycardia, torsades de pointes, circulatory failure and hepatic injury have been reported.

In such an event of overdose treatment should be symptomatic, gastric lavage may be employed to reduce absorption in addition to general supportive measures. Neither Amiodarone nor its metabolites are dialysable. The patient should be monitored and if bradycardia occurs, beta-adrenostimulants or glucagon may be given. Spontaneously resolving attacks of ventricular tachycardia may also occur. Due to the pharmacokinetics of Amiodarone (long half-life), adequate and prolonged surveillance of the patient, particularly cardiac status, is recommended.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Antiarrhythmics, class III

ATC code: C01BD

No controlled paediatric studies have been undertaken.

In published studies the safety of amiodarone was evaluated in 1118 paediatric patients various arrhythmias. The following doses were used in paediatric clinical trials.

Oral
Loading dose: 10 to 20 mg/kg/day for 7 to 10 days (or 500 mg/m²/day if expressed per square meter)

Maintenance dose: the minimum effective dosage should be used; according to individual response, it may range between 5 to 10 mg/kg/day (or 250mg/m²/day if expressed per square meter)

5.2. Pharmacokinetic Properties

Amiodarone is strongly protein bound and has an average plasma half-life of 50 days. However there may be considered inter-patient variation with half-life values ranging from less than 20 days to more than 100 days having been reported. High initial doses of Amiodarone, for example 600 mg/day, should be given to achieve effective tissue levels as rapidly as possible. Owing to the long half-life of the drug, a maintenance dose of only 200 mg/day, or less is usually necessary. Sufficient time must be allowed for new distribution equilibrium to be achieved between adjustments of dose.

The long half-life is a valuable safeguard for patients with potentially lethal arrhythmias as omission of occasional doses does not significantly influence the protection afforded by an Amiodarone.

No controlled paediatric studies have been undertaken. In the limited published data available in paediatric patients, there were no differences noted compared to adults.

5.3 Preclinical safety data

In a 2-years carcinogenicity study in rats, amiodarone caused an increase in thyroid follicular tumours (adenomas and/or carcinomas) in both sexes at clinical relevant exposures. Since mutagenicity findings were negative, an epigenic rather than genotoxic mechanism is proposed for this type of tumour induction. In the mouse, carcinomas were not observed, but a dose-dependent thyroid follicular hyperplasia was seen. These effects on the thyroid in rats and mice are most likely due to effects of amiodarone on the synthesis and/or release of thyroid gland hormones. The relevance of these findings to man is low.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

The following inactive ingredients are used: Lactose Monohydrate, Pregelatinised Starch, Povidone, Colloidal Anhydrous Silica, Maize Starch, Magnesium Stearate.
6.2 Incompatibilities
Not applicable.

6.3 Shelf life
3 years.

6.4 Special precautions for storage
Do not store above 25°C. Store in the original package.

6.5 Nature and contents of container
Lithographed carton boxes containing PVC/Al blister strips of tablets. Each box contains a patient information leaflet.

Pack Size: 28 and 30 tablets.

6.6 Special precautions for disposal
Not applicable.

7 MARKETING AUTHORISATION HOLDER
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