Summary of Product Characteristics

1 NAME OF THE MEDICINAL PRODUCT

Lansoprazole Teva 30 mg Gastro-resistant Capsules

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

One gastro-resistant capsule, hard contains 30 mg of lansoprazole.

Excipients:
One gastro-resistant capsule, hard contains 166 mg of sucrose.
For a full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Gastro-resistant capsule, hard.
A hard gelatin size 1 capsule with a light grey opaque cap and flesh opaque body, filled with granules.
Body and cap imprinting: 93
7351

4 CLINICAL PARTICULARS

4.1 Therapeutic Indications

- Treatment of duodenal and gastric ulcer
- Treatment of reflux oesophagitis
- Eradication of *Helicobacter pylori* concurrently given with appropriate antibiotic therapy for treatment of *H. pylori*-associated ulcers
- Treatment of NSAID-associated gastric and duodenal ulcers in patients requiring continuous NSAID treatment
- Zollinger-Ellison syndrome

The 30 mg strength should be used for the above indications with few exceptions (see section 4.2 and impaired hepatic function)

- Long-term prophylaxis of reflux oesophagitis
- Symptomatic gastro-oesophageal reflux disease
- Prophylaxis of nonsteroidal anti-inflammatory drugs (NSAID)-associated gastric and duodenal ulcer in patients at risk (see section 4.2) requiring continuous NSAID treatment

The 15 mg strength should be used for the above indications.

4.2 Posology and method of administration

For optimal effect, Lansoprazole capsules should be taken once daily in the morning, except when used for *H. pylori* eradication when treatment should be twice a day, once in the morning and once in the evening. The capsules are swallowed whole with liquid. The capsules may be emptied, but the contents may not be chewed or ground. The intake of food reduces the bioavailability of lansoprazole: it is recommended to take lansoprazole at least 30 minutes before the meal.

For patients with difficulty swallowing; studies and clinical practice suggest that the capsules may be opened and the granules mixed with a small amount of water, apple/tomato juice or sprinkled onto a small amount of soft food (e.g. yoghurt, apple puree) to ease administration. Capsules may also be opened and granules mixed with a 40 mL of apple juice for administration through nasogastric tube (see section 5.2).

After preparing the suspension or mixture, the drug should be administered immediately.

Treatment of duodenal ulcer:
The recommended dose is 30 mg once daily for 2 weeks. In patients not fully healed within this time, the medication should be continued at the same dose for another two weeks.

Treatment of gastric ulcer:
The recommended dose is 30 mg once daily for 4 weeks. The ulcer usually heals within 4 weeks, but in patients not fully healed within this time, the medication should be continued at the same dose for another 4 weeks.

Treatment of reflux oesophagitis:
The recommended dose of lansoprazole is 30 mg once daily for 4 weeks. In patients not fully healed within this time, the treatment may be continued at the same dose for another 4 weeks.

Prophylaxis of reflux oesophagitis:
15 mg once daily. The dose may be increased up to 30 mg daily as necessary.

Treatment of NSAID-associated benign gastric and duodenal ulcers in patients requiring continued NSAID treatment:
30 mg once daily for four weeks. In patients not fully healed the treatment may be continued for another four weeks. For patients at risk or with ulcers that are difficult to heal, a longer course of treatment and/or a higher dose should be considered.

Prophylaxis of NSAID-associated gastric and duodenal ulcers in patients at risk (such as age > 65 or history of gastric or duodenal cancer) requiring prolonged NSAID treatment:
15 mg once daily. If the treatment fails the dose 30 mg once daily should be used.

Symptomatic gastro-oesophageal reflux disease:
The recommended dose is 15 mg or 30 mg once daily. Relief of symptoms is obtained rapidly. Individual adjustment of dosage should be considered. If the symptoms are not relieved within 4 weeks with a daily dose of 30 mg, further examinations are recommended.

Eradication of *Helicobacter pylori*:
30 mg lansoprazole 2 times daily for one week in combination with one of the following three combinations:
a) amoxicillin 1 g twice daily + clarithromycin 500 mg twice daily
b) clarithromycin 250 mg twice daily + metronidazole 400-500 mg twice daily
c) amoxicillin 1 g twice daily + metronidazole 400-500 mg twice daily. Lower eradication rates were seen using this combination than in regimens involving clarithromycin. It may be suitable for those who are unable to take clarithromycin as part of an eradication therapy, when local resistance rates to metronidazole are low

Six months after successful eradication treatment, the risk of re-infection is low and relapse is therefore unlikely.

Consideration should be given to official local guidance (e.g. national recommendations) regarding bacterial resistance, duration of treatment and the appropriate use and prescription of antibacterial agents.

Zollinger-Ellison syndrome:
The recommended initial dose is 60 mg once daily. The dose should be individually adjusted and the treatment should be continued for as long as necessary. Daily doses of up to 180 mg have been used. If the required daily dose exceeds 120 mg, it should be given in two divided doses.

Impaired hepatic or renal function:
There is no need to change the dose in patients with impaired renal function. The normal daily dose of 30 mg should not be exceeded in these patients, however. Care should be exercised in the administration of lansoprazole in patients with mildly to moderately impaired hepatic function. In mildly impaired patients, the dose should not exceed 30 mg. In patients with moderately impaired hepatic function, the dose should be restricted to 15 mg daily. Due to the lack of data in patients with severely impaired hepatic function, these patients should not be treated with lansoprazole (see section 4.4).

Children:
Lansoprazole is not recommended in children as safety and efficacy have not been established in this population. Treatment of small children below one year of age should be avoided as available data have not shown beneficial effects in the treatment of gastro-oesophageal reflux disease.

Elderly:
Due to delayed elimination of lansoprazole in the elderly it may be necessary to administer the treatment in doses of 15-30 mg adjusted to individual requirements. However, the daily dose in the elderly should not exceed 30 mg.

4.3 Contraindications

Hypersensitivity to the active substance or to any of the excipients.

Co-administration of atazanavir with proton pump inhibitors such as lansoprazole is not recommended (see section 4.5). If the combination of atazanavir with a proton pump inhibitor is judged unavoidable, close clinical monitoring is recommended in combination with an increase in the dose of atazanavir to 400 mg with 100 mg of ritonavir; doses of proton pump inhibitors comparable to omeprazole 20 mg should not be exceeded.

4.4 Special warnings and precautions for use

The diagnosis of gastroduodenal ulcers and reflux oesophagitis should be confirmed by endoscopy or other appropriate diagnostic means. Reflux oesophagitis may not present as ulceration and/or visual damage, therefore in certain cases endoscopy alone may not be sufficient.

In common with other anti-ulcer therapies, the possibility of malignant gastric tumour should be excluded before initiating treatment of gastric ulcer with lansoprazole, because lansoprazole can mask the symptoms and delay the diagnosis.

Lansoprazole should be used with caution in patients with moderate and severe hepatic dysfunction (see section 4.2).

Lansoprazole has a similar mechanism of action to omeprazole and both increase gastric pH, the following statement is made by analogy to omeprazole. Decreased gastric acidity due to lansoprazole increases gastric counts of bacteria normally present in the gastrointestinal tract. Treatment with lansoprazole may lead to a slightly increased risk of gastrointestinal infections such as Salmonella and Campylobacter.

Patients suffering from gastro-duodenal ulcers, the possibility of *H. pylori* infection as an aetiological factor should be considered.
If lansoprazole, in combination with antibiotics, is used for eradication therapy of *H. pylori*, then also instructions for the use of these antibiotics should be followed.

Because of limited safety data for patients on maintenance treatment for longer than 1 year, regular review of the treatment and a thorough benefit risk assessment should regularly be performed in these patients.

Very rarely cases of colitis have been reported in patients taking lansoprazole. Therefore, in the case of severe and/or persistent diarrhoea, discontinuation of therapy should be considered.

The treatment for the prevention of peptic ulceration of patients in need of continuous NSAID treatment should be restricted to high risk patients (e.g. previous gastrointestinal bleeding, perforation or ulcer, advanced age, concomitant use of medication known to increase the likelihood of upper GI adverse events [e.g. corticosteroids or anticoagulants], the presence of a serious co-morbidity factor or the prolonged use of NSAID maximum recommended doses).

If visual disturbances occur during long-term use (>1 year), an ophthalmologist should be consulted.

Proton pump inhibitors, especially if used in high doses and over long durations (>1 year), may modestly increase the risk of hip, wrist and spine fracture, predominantly in the elderly or in presence of other recognised risk factors.

Observational studies suggest that proton pump inhibitors may increase the overall risk of fracture by 10–40%. Some of this increase may be due to other risk factors. Patients at risk of osteoporosis should receive care according to current clinical guidelines and they should have an adequate intake of vitamin D and calcium.

Hypomagnesaemia
Severe hypomagnesaemia has been reported in patients treated with PPIs like lansoprazole for at least three months, and in most cases for a year. Serious manifestations of hypomagnesaemia such as fatigue, tetany, delirium, convulsions, dizziness and ventricular arrhythmia can occur but they may begin insidiously and be overlooked. In most affected patients, hypomagnesaemia improved after magnesium replacement and discontinuation of the PPI.
For patients expected to be on prolonged treatment or who take PPIs with digoxin or drugs that may cause hypomagnesaemia (e.g., diuretics), health care professionals should consider measuring magnesium levels before starting PPI treatment and periodically during treatment.

This medicinal product contains 83 mg, 166 mg of sucrose. When taken according to the dosage recommendations, the maximum daily dose supplies up to 664 mg of sucrose. Patients with rare hereditary problems of fructose intolerance, glucose-galactose malabsorption or sucrase-isomaltase insufficiency should not take this medicinal product.

4.5 Interaction with other medicinal products and other forms of interaction

Effects of lansoprazole on other drugs

Medicinal products with pH dependent absorption
Lansoprazole may interfere with the absorption of drugs where gastric pH is critical to bioavailability.

Atazanavir
A study has shown that co-administration of lansoprazole (60 mg once daily) with atazanavir 400 mg to healthy volunteers resulted in a substantial reduction in atazanavir exposure (approximately 90% decrease in AUC and C_{max}). Co-administration of atazanavir with proton pump inhibitors such as lansoprazole is not recommended (see section 4.3).

Ketoconazole and itraconazole
The absorption of ketoconazole and itraconazole from the gastrointestinal tract is enhanced by the presence of gastric acid. Administration of lansoprazole may result in subtherapeutic concentrations of ketoconazole and itraconazole and the combination should be avoided. The effect may also be present if lansoprazole is combined with other drugs with pH dependent absorption.

Digoxin
Coadministration of lansoprazole and digoxin may lead to increased digoxin plasma levels. In patients receiving digoxin, the plasma levels should therefore be monitored and the dose of digoxin adjusted if necessary when initiating and ending lansoprazole treatment.

Medicinal products metabolised by P450 enzymes
Lansoprazole may give rise to increased plasma concentrations of drugs metabolised by CYP3A4. Caution is advised when combining lansoprazole with drugs which are metabolised by this enzyme. Caution should be exercised when combining lansoprazole with drugs which have a narrow therapeutic index, as the effect of lansoprazole on the metabolism of other drugs has not been extensively investigated.

Tacrolimus
Coadministration of lansoprazole increases the plasma concentrations of tacrolimus (a CYP3A and P-gp substrate). Lansoprazole exposure increased the mean exposure of tacrolimus by up to 81%. Monitoring of tacrolimus plasma concentrations is advised when concomitant treatment with lanzoprazole is initiated or ended.

Carbamazepine
Caution is advised during cotreatment with carbamazepine (a CYP3A substrate) and lansoprazole. The drug combination may result in increased carbamazepine concentrations as well as reduced lansoprazole concentrations.

Phenytoin
Studies have shown that the dosage of phenytoin (CYP2C19 and CYP2C9 substrate) may have to be reduced when administered concomitantly with lansoprazole. Caution and monitoring of phenytoin plasma concentrations is advised when initiating and ending lansoprazole treatment in patients on phenytoin, although no clinically significant interactions have been reported between the two substances.

Warfarin
Caution and increased monitoring frequency is advised when initiating or ending lansoprazole cotreatment in patients treated with warfarin.

Theophylline
Lansoprazole gives a 14% reduction in the plasma concentrations of theophylline. Individual patients may receive a clinically relevant decrease. Caution is advised when combining the two drugs.

Medicinal products transported by P-glycoprotein
Lansoprazole has been observed to inhibit the transport protein, P-glycoprotein (P-gp) *in vitro*. It may not be excluded that lanzoprazole may affect transport via this protein giving rise to increased plasma concentrations of P-gp substrates such as digoxin.

Therapy of *Helicobacter pylori* infection
Therapy of *Helicobacter pylori* infection is intended to be combined, with concurrent administration of lansoprazole with two antibiotics. The influence of this combined administration has not yet been investigated systemically. For reasons of theoretical considerations, enhanced interactions with other medicinal products must be expected as a precaution. Monitoring of the serum levels of other medicinal products taken during the 1-week eradication therapy is therefore recommended. This concerns particularly such medicinal products also metabolized via the cytochrome P450 system.

The following interactions between lansoprazole and one/two antibiotics used in eradication therapy have been found so far:

Co-administered medicinal products	**Dosage and duration of combined administration**	**Effect***
lansoprazole + clarithromycin	30 mg + 500 mg 3 times/day for 5 days	Increased plasma levels of a clarithromycin metabolite by 16%; increased bioaivalability of lansoprazole by 19% up to 32%
lansoprazole + amoxicillin	30 mg + 1000 mg 3 times/day for 5 days	Decelerates uptake of amoxicillin
lansoprazole + metronidazole	Not yet investigated	
lansoprazole + clarithromycin + amoxicillin	30 mg + 500 mg + 1000 mg twice daily for 5 days	Increase bioavailability and half-life of lansoprazole by 30% each; increased plasma levels of a clarithromycin metabolite by 30%

*The effects of clarithromycin on the pharmacokinetics of lansoprazole are likely to be dependent on the patient's CYP2C19 genotype. A poor metaboliser would have more marked effects than an extensive metaboliser.

Effects of other drugs on lansoprazole

Drugs which inhibits CYP2C19
Fluvoxamine
A dose reduction may be considered when combining lansoprazole with the CYP2C19 inhibitor fluvoxamine. Drugs which inhibit CYP2C19 may increase the plasma concentration of lansoprazole. Fluvoxamine, an inhibitor of CYP2C19, increased the plasma concentrations of lansoprazole up to 4-fold.

Drugs which induces CYP2C19 and CYP3A4
Enzyme inducers affecting CYP2C19 and CYP3A4 such as rifampicin, and St John's wort (*Hypericum perforatum*) can markedly reduce the plasma concentrations of lansoprazole.

Others
Sucralfate/Antacids
Antacids and sucralfate may decrease the bioavailability of lansoprazole. The lansoprazole dose should therefore be taken at least an hour prior or after.

Clinically significant interactions of lansoprazole with NSAIDs or diazepam have not been demonstrated. Formal interaction studies with lansoprazole and NSAIDs have not been conducted.

4.6 Fertility, pregnancy and lactation

Pregnancy
For lansoprazole no clinical data on exposed pregnancies are available. Animal studies do not indicate direct or indirect harmful effects with respect to pregnancy, embryonal/fetal development, parturition or postnatal development.

The use of lansoprazole during pregnancy is not recommended.

Lactation
It is not known whether lansoprazole is excreted in human breast milk. Animal studies have shown excretion of lansoprazole in milk. A decision on whether to continue/discontinue breast-feeding or to continue/discontinue therapy with lansoprazole should be made taking into account the benefit of breast-feeding to the child and the benefit of lansoprazole therapy to the woman.

4.7 Effects on ability to drive and use machines

Adverse drug reactions such as dizziness, vertigo, visual disturbances, somnolence and fatigue may occur (see section 4.8). Under these conditions the ability to react may be decreased. This should be taken into account when driving or using machines.

4.8 Undesirable effects

The following undesirable effects have been observed during treatment with lansoprazole with the following frequencies: common (>1/100, <1/10), uncommon (>1/1,000, <1/100), rare (>1/10,000, <1/1,000), very rare (<1/10,000) including isolated reports, not known (cannot be estimated from the available data).

	Common (>1/100, <1/10)	**Uncommon (>1/1,000, <1/100)**	**Rare (>1/10,000, <1/1,000)**	**Very rare (<1/10,000) including isolated reports**	**Not known (cannot be estimated fr the availabl data)**
Blood and lymphatic system disorders		Thrombocytopeniaeosinophilia, leucopenia	anaemia	Agranulocytosis, pancytopenia	
Psychiatric disorders		Depression	hallucination, confusion, insomnia		
Nervous system disorders	Headache and dizziness		somnolence, drowsiness,		

			vertigo, tremor and paraesthesia, restlessness		
Eye disorders			Visual disturbances		
Cardiac disorders			Palpitation and chest pain		
Vascular disorders			Peripheral oedema		
Gastrointestinal disorders	Dry mouth or throat, vomiting, nausea,diarrhoea, stomach ache, constipation, flatulence and dyspepsia		Pancreatitis, candidiasis of oesophagus, taste disturbances and glossitis	Colitis, stomatitis and black tongue	
Hepatobiliary disorders	Increase in liver enzyme levels		Hepatitis, jaundice and icterus		
Skin and subcutaneous tissue disorders	Eczema, urticaria and itching		Erythema multiforme, petechiae, hair loss, photosensitivity and purpura	Stevens-Johnson syndrome and toxic epidermal necrolysis	
Musculoskeletal and connective tissue disorders		Muscle and joint pain, fracture of the hip, wrist or spine (see section 4.4)			
Renal and urinary disorders			Interstitial nephritis		
Reproductive system and breast disorders			Gynaecomastia	Galactorrhoea	
Metabolism and nutritional disorders					hypomagnes (See section
General disorders and administrative site conditions	Fatigue	oedema	Angioedema, bronchial constriction, impotence, hyperhidrosis, anorexia and fever	Anaphylactic shock, and general malaise	
Investigations				Increase in cholesterol and triglyceride levels	

4.9 Overdose

The effects of overdose of lansoprazole in humans are not known (although the acute toxicity is likely to be low) and, consequently, instructions for treatment cannot be given. However, daily doses of up to 180 mg of lansoprazole orally and up to 90 mg of lansoprazole intravenously have been administered in trials without significant undesirable effects.

Please refer to section 4.8. for possible symptoms of lansoprazole overdose.

In case of suspected overdose the patient should be monitored. Lansoprazole is not significantly eliminated by haemodialysis. If necessary, gastric emptying, charcoal and symptomatic therapy are recommended.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Proton pump inhibitors
ATC Code: A02B C03

Lansoprazole is a gastric proton pump inhibitor. It inhibits the final stage of gastric acid formation by inhibiting the activity of H^+/K^+ ATPase of the parietal cells in the stomach. The inhibition is dose-dependent and reversible, and the effect applies to both basal and stimulated secretion of gastric acid. Lansoprazole is concentrated in the parietal cells and becomes active in their acidic environment, whereupon it reacts with the sulphydryl group of H^+/K^+ATPase causing inhibition of the enzyme activity.

Effect on gastric acid secretion:
Lansoprazole is a specific inhibitor of the parietal cell proton pump. A single oral dose of lansoprazole 30 mg inhibits pentagastrin-stimulated gastric acid secretion by about 80%. After repeated daily administration for seven days, about 90% inhibition of gastric acid secretion is achieved. It has a corresponding effect on the basal secretion of gastric acid. A single oral dose of 30 mg reduces basal secretion by about 70%, and the patients' symptoms are consequently relieved starting from the very first dose. After eight days of repeated administration the reduction is about 85%. A rapid relief of symptoms is obtained by 30 mg daily, and most patients with duodenal ulcer recover within 2 weeks, patients with gastric ulcer and reflux oesophagitis within 4 weeks. By reducing gastric acidity, lansoprazole creates an environment in which appropriate antibiotics can be effective against *H. pylori.*

5.2 Pharmacokinetic properties

Absorption and distribution:
Lansoprazole is rapidly inactivated by gastric acid and lansoprazole is consequently administered as enteric coated granules in gelatin capsules. Absorption from the duodenum is rapid and plasma peak concentration is achieved within 1.5-2.0 hours. Bioavailability after a single dose of 30 mg and after repeated daily administration is 80-90%. Intake of food slows the absorption rate of lansoprazole and reduces its bioavailability (AUC) by about 25%. Antacids and sucralfate may reduce the bioavailability of lansoprazole. The plasma protein binding of lansoprazole is about 95%, but this has not been found to have a significant effect on other protein bound drugs.

Studies have shown that granules from opened capsules give equivalent AUC as the intact capsule if the granules are suspended in a small amount of orange juice, apple juice, or tomato juice mixed with a tablespoon of apple or pear puree or sprinkled on a tablespoon of yoghurt, pudding or cottage cheese. Equivalent AUC has also been shown for granules suspended in apple juice administered through a naso-gastric tube.

Metabolism and elimination:
The metabolism of lansoprazole is mainly catalysed by the enzyme CYP2C19. The enzyme CYP3A4 also contributes to the metabolism. CYP2C19 is subject to genetic polymorphism and 2%-6% of the population, called poor metabolisers (PMs)**,** are homozygote for a mutant CYP2C19 allele and therefore lacks a functional CYP2C19 enzyme. The exposure of lansoprazole is several-fold higher in PMs than in extensive metabolisers (EMs).

The plasma elimination half-life of lansoprazole is 1.0-2.0 hours following single or multiple doses in healthy subjects. There is no evidence of accumulation following multiple doses in healthy subjects. There is no change in half-life during treatment. A single dose of lansoprazole has an inhibitory effect on gastric acid secretion lasting more than 24 hours. Since lansoprazole is activated in the parietal cells, its plasma concentration is not related to gastric acid inhibition. Lansoprazole is mainly metabolised in the liver. Three metabolites have been identified in the plasma: the sulphone, 5-hydroxy lansoprazole and the sulphide. These metabolites have no significant effect on acid secretion. About 15-50% of the metabolites are secreted in the urine and the remainder in the faeces. Three metabolites have been identified in the urine: 5-hydroxy sulphone, 5-hydroxy sulphide and 5-hydroxy lansoprazole.

In patients with cirrhosis the AUC of lansoprazole is significantly increased and the elimination half-life is prolonged, but no signs of accumulation of lansoprazole have been detected. The bioavailability of lansoprazole is not significantly changed in renal insufficiency. Elimination of lansoprazole in the elderly is slightly delayed.

A study with ^{14}C labelled lansoprazole indicated that approximately one-third of the administered radiation was excreted in the urine and two-thirds was recovered in the faeces.

Pharmacokinetics in elderly patients
The clearance of lansoprazole is decreased in the elderly, with elimination half-life increased approximately 50% to 100%. Peak plasma levels were not increased in the elderly.

Pharmacokinetics in paediatric patients
The evaluation of the pharmacokinetics in children aged 1-17 years of age showed a similar exposure as compared to adults with doses of 15 mg for those below 30 kg of weight and 30 mg for those above. The investigation of a dose of 17 mg/m^2 body surface or 1 mg/kg body weight also resulted in comparable exposure of lansoprazole in children aged 2-3 months up to one year of age compared to adults.

Higher exposure of lansoprazole in comparison to adults has been seen in infants below the age of 2-3 months with doses of both 1.0 mg/kg and 0.5 mg/kg body weight given as a single dose.

Pharmacokinetics in hepatic insufficiency
The exposure of lansoprazole is doubled in patients with mild hepatic impairment and much more increased in patients with moderate and severe hepatic impairment.

5.3 Preclinical safety data

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

In two rat carcinogenicity studies, lansoprazole produced dose-related gastric ECL cell hyperplasia and ECL cell carcinoids associated with hypergastrinaemia due to inhibition of acid secretion. Intestinal metaplasia was also observed, as were Leydig cell hyperplasia and benign Leydig cell tumours. After 18 months of treatment retinal atrophy was observed. This was not seen in monkeys, dogs or mice.

In mouse carcinogenicity studies dose-related gastric ECL cell hyperplasia developed as well as liver tumours and adenoma of rete testis.

The clinical relevance of these findings is unknown.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Gastro-resistant granules:
Sugar spheres (sucrose, maize starch)
Hypromellose
Talc
Magnesium carbonate
Methacrylic acid ethylacrylate copolymer (1:1) dispersion 30 %
Triethyl citrate
Titanium dioxide (E171)

Capsule shells:
Black iron oxide (E172)
Titanium dioxide (E171)
Red iron oxide (E172)
Gelatin

Ink:
Shellac
Black iron oxide (E172)
Propylene glycol

6.2 Incompatibilities

Not applicable

6.3 Shelf life

Blisters: 18 months
Bottles: 24 months

6.4 Special precautions for storage

Do not store above 25°C. Store in the original package.
Bottles only: Keep the bottle tightly closed.

6.5 Nature and contents of container

Blister packs (aluminium/aluminium) with 7, 14, 15, 28, 30, 50, 56, 84, 98 and 100 gastro-resistant Capsules.
Bottles (HDPE bottles with PP closure and dessicant) with 7, 14, 15, 28, 30, 50, 56, 84, 98 and 100 gastro-resistant Capsules.

Not all pack sizes may be marketed.

6.6 Special precautions for disposal

No special requirements

7 MARKETING AUTHORISATION HOLDER

Teva Pharma B.V.
Computerweg 10
3542 DR Utrecht
The Netherlands

8 MARKETING AUTHORISATION NUMBER

PA 749/24/2

9 DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

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10 DATE OF REVISION OF THE TEXT

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