For the use of only a Registered Medical Practitioner or a Hospital or a Laboratory

Not to be sold by retail without the prescription of a Registered Medical Practitioner

Prescribing Information

1. Generic Name

Rabeprazole Gastro-resistant Tablets IP
(Brand Name: R-PPi® 20 Tablets)

2. Qualitative and Quantitative Composition

Each Gastro-resistant Tablet Contains:
Rabeprazole Sodium IP …… 20 mg
Excipients …… q.s.
Colours: Red Oxide of Iron & Titanium Dioxide IP

3. Dosage Form and Strength

Dosage Form: Tablets.
Dosage Strength: Rabeprazole 20 mg (in a gastro-resistant form) per tablet.

4. Clinical Particulars

4.1 Therapeutic Indication

R-PPi Tablets are indicated for the treatment of following:

- Duodenal ulcer.
- Gastric ulcer.
- Symptomatic (i.e., heartburn and regurgitation) treatment of gastro-esophageal reflux disease (GERD).
- Erosive/ulcerative esophagitis.
- Maintenance therapy for healing of erosive esophagitis.
- *Helicobacter pylori*-associated peptic ulcers (in combination with appropriate antibiotic therapy for eradication of *H. pylori*).
- Pathological hypersecretory conditions including Zollinger-Ellison syndrome.

4.2Posology and Method of Administration

For oral administration.

Adults

1) **Duodenal Ulcer:** 20 mg once daily up to 4 weeks. Most patients with duodenal ulcer heal within 4 weeks. However, a few patients may require an additional 4 weeks of therapy to achieve healing. For the treatment of duodenal ulcers, R-PPi Tablets should be administered after a meal.
2) **Gastric Ulcer:** 20 mg once daily up to 6 weeks. However, some patients may require an additional 6 weeks of therapy to achieve healing.

3) **Symptomatic Treatment of GERD:** 20 mg once daily up to 4 weeks. If symptoms do not resolve completely after 4 weeks, an additional course of treatment may be considered.
4) **Erosive/Ulcerative Esophagitis:** 20 mg once daily for 4 to 8 weeks. For those patients who have not healed after 8 weeks of treatment, an additional 8-week course may be considered.
5) **Maintenance Therapy for Healing of Erosive Esophagitis:** 20 mg once daily. In controlled studies, duration of rabeprazole therapy has not extended beyond 12 months.
6) ***Helicobacter Pylori*-Associated Peptic Ulcers:** Patients should be treated with following combination therapy for eradication of *H. pylori* bacteria*:* Rabeprazole 20 mg + Amoxicillin 1000 mg + Clarithromycin 500 mg, twice daily for 7 days with morning and evening meals.
7) **Zollinger-Ellison Syndrome:** The recommended starting dose is 60 mg once daily. The dose may be titrated upwards to 120 mg/day based on individual patient needs. Single daily doses up to 100 mg/day may be given. Dose of 120 mg daily should be divided and given as 60 mg twice daily. Treatment should continue for as long as clinically indicated. Some patients with Zollinger-Ellison syndrome have been treated continuously for up to one year.

Adolescents (Age Above 12 Years)

- **Symptomatic GERD:** 20 mg once daily up to 8 weeks.

Neither the time of day nor food intake has shown any effect on the rabeprazole efficacy. For the treatment of duodenal ulcers and *Helicobacter pylori* eradication, R-PPi Tablets should be administered after a meal. For all other indications, R-PPi Tablets may be taken with or without food. R-PPi Tablets should be swallowed whole with water and not to be cut, crush or chew.
Or, as prescribed by the physician.

4.3Contraindications

R-PPi Tablets are contraindicated in the following:

- Patients with known hypersensitivity to rabeprazole or to any substituted benzimidazole derivative or to any component of the formulation. Hypersensitivity reactions may include anaphylaxis, anaphylactic shock, angioedema, bronchospasm, acute interstitial nephritis, and urticaria.
- In patients receiving rilpivirine-containing products.

4.4Special Warnings and Precautions for Use

Gastric Malignancy: In adults, symptomatic response to rabeprazole therapy does not preclude the presence of gastric malignancy; therefore the possibility of malignancy should be excluded prior to commencing treatment with rabeprazole. Consider additional follow-up

and diagnostic testing in adult patients who have a suboptimal response or an early symptomatic relapse after completing treatment with a proton pump inhibitor (PPI).

Acute Interstitial Nephritis: Acute interstitial nephritis has been observed in patients taking PPIs, including rabeprazole. Acute interstitial nephritis may occur at any point during PPI therapy and is generally attributed to an idiopathic hypersensitivity reaction. Discontinue rabeprazole if acute interstitial nephritis develops.

Cyanocobalamin (Vitamin B_{12}) Deficiency: Daily treatment with acid-suppressing drugs over a long period of time (e.g., longer than 3 years) may lead to malabsorption of cyanocobalamin caused by hypo- or achlorhydria. Rare reports of cyanocobalamin deficiency occurring with acid-suppressing therapy have been reported in the literature. This diagnosis should be considered if clinical symptoms consistent with cyanocobalamin deficiency are observed in patients treated with rabeprazole.

Hypomagnesemia: Hypomagnesemia, symptomatic and asymptomatic, has been reported rarely in patients treated with PPIs. Serious adverse events include tetany, arrhythmias, and seizures. In most patients, treatment of hypomagnesemia required magnesium replacement as well as discontinuation of the PPI. For patients expected to be on prolonged treatment or who take PPIs with medications such as digoxin or drugs that may cause hypomagnesemia (e.g., diuretics), monitoring of serum magnesium levels prior to initiation of PPI treatment and periodically thereafter should be considered.

Risk of Bone Fractures: Observational studies suggest that PPI therapy may be associated with an increased risk for osteoporosis-related fractures of the hip, wrist, or spine. The risk of fracture was increased in patients who received high-dose and long-term PPI therapy (a year or longer). Patients should use the lowest dose and shortest duration of PPI therapy appropriate to the condition being treated. 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.

***Clostridium Difficile*-Associated Diarrhea (CDAD):** Published observational studies suggest that PPI therapy like rabeprazole may be associated with an increased risk of *Clostridium difficile*-associated diarrhea, especially in hospitalized patients. This diagnosis should be considered for diarrhea that does not improve. Patients should use the lowest dose and shortest duration of PPI therapy appropriate to the condition being treated.

Subacute Cutaneous Lupus Erythematosus (SCLE): PPIs are associated with very infrequent cases of SCLE. If lesions occur, especially in sun-exposed areas of the skin, and if accompanied by arthralgia, the patient should seek medical help promptly and rabeprazole therapy should be stopped immediately. The occurrence of SCLE with previous PPI treatment may increase the risk of SCLE with other PPIs.

Thrombocytopenia and Neutropenia: There have been post-marketing reports of blood dyscrasias (thrombocytopenia and neutropenia). In the majority of cases where an alternative etiology cannot be identified, the events were uncomplicated and resolved on discontinuation of rabeprazole therapy.

Hepatic Effects: Hepatic enzyme abnormalities have been seen in clinical trials and have also been reported in post-marketing studies. In the majority of cases where an alternative etiology cannot be identified, the events were uncomplicated and resolved on discontinuation of rabeprazole.

4.5Drug Interactions

Antiretroviral Drugs: The effect of PPI on antiretroviral drugs is variable. The clinical importance and the mechanisms behind these interactions are not always known.

1) Decreased exposure of some antiretroviral drugs (e.g., rilpivirine, atazanavir, and nelfinavir) when used concomitantly with rabeprazole may reduce antiviral effect and promote the development of drug resistance.
   - **Rilpivirine-Containing Products:** Concomitant use with rabeprazole is contraindicated.
   - **Atazanavir:** Co-administration of atazanavir 300 mg/ritonavir 10 mg with omeprazole (40 mg once daily) or atazanavir 400 mg with lansoprazole (60 mg once daily) to healthy volunteers resulted in a substantial reduction in atazanavir exposure. The absorption of atazanavir is pH dependent. Although not studied, similar results are expected with other PPIs. Therefore PPIs, including rabeprazole, should not be co-administered with atazanavir.
   - **Nelfinavir:** Avoid concomitant use with rabeprazole.
2) Increased exposure of other antiretroviral drugs (e.g., saquinavir) when used concomitantly with rabeprazole may increase toxicity. It is recommended to monitor for potential saquinavir toxicities.
3) There are other antiretroviral drugs which do not result in clinically relevant interactions with rabeprazole.

Drugs Dependent on Gastric pH for Absorption (e.g., iron salts, erlotinib, dasatinib, nilotinib, mycophenolate mofetil, ketoconazole, itraconazole): Rabeprazole produces a profound and long lasting inhibition of gastric acid secretion. An interaction with drugs whose absorption is pH dependent may occur. Rabeprazole can reduce the absorption/bioavailability of such drugs due to its effect on reduction of intragastric acidity.

- **Ketoconazole/Itraconazole:** Co-administration of rabeprazole with ketoconazole or itraconazole may result in a significant decrease in antifungal plasma levels. In healthy adult subjects, co-administration of rabeprazole 20 mg at steady state with a single 400 mg of ketoconazole resulted in approximately an average of 31% reduction in both C_{max} and AUC of ketoconazole. Therefore, individual patients may need to be monitored to determine if a dosage adjustment is necessary when ketoconazole or itraconazole are taken concomitantly with rabeprazole.
- **Mycophenolate Mofetil:** Co-administration of PPIs with mycophenolate mofetil in healthy and transplant patients has been reported to reduce exposure to the active metabolite, mycophenolic acid. This is possibly due to a decrease in mycophenolate mofetil solubility at an increased gastric pH. The clinical relevance of reduced mycophenolic acid exposure on organ rejection has not been established in transplant patients receiving PPIs and mycophenolate mofetil. Use rabeprazole with caution in transplant patients receiving mycophenolate mofetil.

Antacids: In clinical trials, antacids were used concomitantly with the administration of rabeprazole and, in a specific drug-drug interaction study, no interaction with liquid antacids was observed.

Methotrexate: Literature suggests that concomitant use of PPIs with methotrexate may elevate and prolong serum levels of methotrexate and/or its metabolite, possibly leading to methotrexate toxicity. A temporary withdrawal of rabeprazole therapy may be considered in some patients receiving high-dose of methotrexate.

Warfarin: Steady state interactions of rabeprazole and warfarin have not been adequately evaluated in patients. There have been reports of increased international normalized ratio (INR) and prothrombin time in patients receiving a PPI and warfarin concomitantly. Increases in INR and prothrombin time may lead to abnormal bleeding and even death. Thus, patients treated with rabeprazole and warfarin concomitantly may need to be monitored for increases in INR and prothrombin time.

Digoxin: When rabeprazole and digoxin are administered concomitantly, there is potential for increased exposure of digoxin. Monitor digoxin concentrations. Dose adjustment of digoxin may be needed to maintain therapeutic drug concentrations.

Clopidogrel: Clopidogrel is metabolised to its active metabolite by CYP2C19. Inhibition of CYP2C19 by rabeprazole would be expected to result in reduced drug levels of the active metabolite of clopidogrel and a reduction in its antiplatelet activity and therefore, its clinical efficacy. Concomitant use of rabeprazole with clopidogrel should be discouraged or while using rabeprazole, consider an alternative antiplatelet therapy.

Tacrolimus: Potentially increased exposure of tacrolimus has been reported, especially in transplant patients who are intermediate or poor metabolizers of CYP2C19; thus, monitoring of plasma levels of tacrolimus is advised. Dose adjustment of tacrolimus may be needed to maintain therapeutic drug concentration.

Drug/Laboratory Tests Interactions

Increased Chromogranin A (CgA) Levels: Increased CgA level may interfere with investigations for neuroendocrine tumors. To avoid this interference, rabeprazole treatment should be stopped for at least 5 days before CgA measurements. If CgA and gastrin levels have not returned to reference range after initial measurement, measurements should be repeated 14 days after cessation of PPI treatment.

Secretin Stimulation Test: Hyper-response in gastrin secretion in response to secretin stimulation test, falsely suggesting gastrinoma. Temporarily stop treatment with rabeprazole at least 14 days before assessing to allow gastrin levels to return to baseline.

False Positive Urine Tests for Tetrahydrocannabinol (THC): There have been reports of false positive urine screening tests for THC in patients receiving PPIs. An alternative confirmatory method should be considered to verify positive results.

4.6Use in Special Populations

Pregnant Women

Pregnancy Category C. Reproduction studies performed in rats and rabbits have revealed no evidence of impaired fertility or harm to the fetus due to rabeprazole, although low feto-placental transfer occurs in rats. No evidence of adverse developmental effects were seen in animal reproduction studies with rabeprazole administered during organogenesis at 13 and 8-times the human area under the plasma concentration-time curve (AUC) at the recommended

dose for GERD, in rats and rabbits, respectively. There are no available human data on rabeprazole use in pregnant women. Thus, caution should be exercised when prescribing rabeprazole to pregnant women.

Lactating Women
It is not known whether rabeprazole is excreted in human breast milk. No studies in lactating women have been performed. Rabeprazole is however excreted in rat mammary secretions. Therefore, rabeprazole should not be used during breast feeding. Accordingly, a decision should be made whether to discontinue nursing or to discontinue/abstain from rabeprazole therapy, taking into account the benefit of the drug to the mother.

Paediatric Patients
Rabeprazole is not recommended for use in children due to a lack of data on safety and efficacy. In adolescents above 12 years of age, the safety and effectiveness of rabeprazole has been established in the treatment of symptomatic GERD. Thus, in the treatment of symptomatic GERD, R-PPi Tablets can be administered in children above 12 years of age (20 mg once daily). However, for other indications, R-PPi Tablets are not recommended for use in paediatric patients below 18 years of age.

Geriatric Patients
No overall differences in safety or effectiveness were observed between elderly and younger subjects, but greater sensitivity of some older individuals cannot be ruled out. No dosage adjustment is generally necessary in the elderly patients.

Renal Impairment Patients
No dosage adjustment is necessary for patients with renal impairment.

Hepatic Impairment Patients
Administration of rabeprazole to patients with mild to moderate hepatic impairment resulted in increased exposure and decreased elimination. No dosage adjustment is necessary in patients with mild to moderate hepatic impairment. There is no information in patients with severe hepatic impairment. Thus, R-PPi Tablets should be avoided in patients with severe hepatic impairment; however, if treatment is necessary, monitor patients for adverse reactions.

4.7Effect on Ability to Drive and Use Machines
Based on the pharmacodynamic properties and the adverse events profile, it is unlikely that rabeprazole would cause an impairment of driving performance or compromise the ability to use machinery. However, somnolence may occur in some individuals which impair patient's mental alertness. If affected by somnolence/dizziness, patients should avoid driving a vehicle or operating machinery.

4.8Undesirable Effects

Clinical Trials Experience

Because clinical trials are conducted under varying conditions, adverse reaction rates observed in the clinical trials of a drug may not reflect the rates observed in practice.

In controlled clinical trials, the most commonly reported adverse reactions (≥ 2%) in patients treated with rabeprazole versus placebo include the following: Pain (3% vs. 1%), pharyngitis (3% vs. 2%), flatulence (3% vs. 1%), infection (2% vs. 1%), and constipation (2% vs. 1%).

Less common adverse reactions seen in controlled clinical trials (< 2% of patients treated with rabeprazole and greater than placebo) and for which there is a possibility of a causal relationship to rabeprazole, include the following: Headache, abdominal pain, diarrhoea, dry mouth, dizziness, peripheral edema, elevated hepatic enzymes, hepatitis, hepatic encephalopathy, myalgia, and arthralgia.

Post-Marketing Experience

Acute kidney injury as an adverse drug reaction reported with the use of proton pump inhibitors. The following adverse reactions have been identified during post-approval use of rabeprazole. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate their frequency or establish a causal relationship to drug exposure:

Blood and Lymphatic System Disorders: Agranulocytosis, hemolytic anemia, leukopenia, pancytopenia, thrombocytopenia.

Ear and Labyrinth Disorders: Vertigo.

Eye Disorders: Blurred vision.

Hepatobiliary Disorders: Jaundice.

Immune System Disorders: Anaphylaxis, angioedema, systemic lupus erythematosus, Stevens-Johnson syndrome, toxic epidermal necrolysis (sometime fatal).

Infections and Infestations: *Clostridium difficile*-associated diarrhea.

Investigations: Increases in prothrombin time/INR (in patients treated with concomitant warfarin), elevations in thyroid stimulating hormone (TSH).

Metabolism and Nutrition Disorders: Hyperammonemia, hypomagnesemia.

Musculoskeletal System Disorders: Bone fracture, rhabdomyolysis.

Nervous System Disorders: Coma.

Psychiatric Disorders: Delirium, disorientation.

Renal and Urinary Disorders: Interstitial nephritis.

Respiratory, Thoracic and Mediastinal Disorders: Interstitial pneumonia.

Skin and Subcutaneous Tissue Disorders: Severe dermatologic reactions including bullous and other drug eruptions of the skin, cutaneous lupus erythematosus, erythema multiforme.

4.9Overdose

Symptoms: Experience with deliberate or accidental overdose with rabeprazole is limited. The maximum established exposure has not exceeded 60 mg twice daily, or 160 mg once daily. Effects are generally minimal, representative of the known adverse event profile and reversible without further medical intervention.

Treatment: No specific antidote is known. Rabeprazole is extensively protein bound and is, therefore, not readily dialyzable. As in any case of overdose, treatment should be symptomatic and general supportive measures should be utilized.

5. Pharmacological Properties

5.1 Mechanism of Action

Rabeprazole belongs to a class of antisecretory compounds (substituted benzimidazole, proton-pump inhibitors - PPIs). As a weak base, rabeprazole is rapidly absorbed following oral dose and is concentrated in the acidic environment of the parietal cells. In gastric parietal cells (at pH 1.2), rabeprazole is converted to the active 'sulphenamide' form through 'protonation' and it subsequently reacts with the available cysteines on the proton pump.

Rabeprazole suppress gastric acid (hydrochloric acid – HCl) secretion by inhibiting the gastric H+/K+-ATPase enzyme at the secretory surface of the gastric parietal cell. Because this enzyme is regarded as the acid (proton) pump within the parietal cell, rabeprazole has been characterized as a gastric proton-pump inhibitor. Rabeprazole blocks the final step of gastric acid secretion. The effect is dose-related and leads to inhibition of both basal and stimulated acid secretion irrespective of the stimulus.

5.2Pharmacodynamic Properties

Oral administration of a 20 mg dose of rabeprazole provides rapid and effective reduction of gastric acid secretion. The onset of the antisecretory effect occurs within one hour, with the maximum effect occurring within 2 to 4 hours. Inhibition of basal and food-stimulated acid secretion 23 hours after the first dose of rabeprazole is 69% and 82% respectively, and the duration of inhibition lasts up to 48 hours. The duration of pharmacodynamic action of rabeprazole is much longer than its pharmacokinetic half-life (approximately one hour). This effect is probably due to the prolonged binding of rabeprazole to the parietal H+/K+-ATPase enzyme. The inhibitory effect of rabeprazole on acid secretion increases slightly with repeated once daily dosing, achieving steady state inhibition after 3 days. When the drug is discontinued, secretory activity normalizes over 2 to 3 days.

With once daily doses of rabeprazole 10 or 20 mg, serum gastrin levels increases in 2 to 8 weeks reflecting the inhibitory effects on acid secretion. Gastrin values returned to pretreatment levels, usually within 1 to 2 weeks after discontinuation of therapy.

5.3Pharmacokinetic Properties

The pharmacokinetics (C_{max} and AUC) of rabeprazole are linear over the range of 10 mg to 40 mg. There is no significant accumulation when doses of 10 mg to 40 mg are administered every 24 hours; the pharmacokinetics of rabeprazole is not altered by multiple dosing.

Absorption: R-PPi Tablets contains rabeprazole sodium as a gastro-resistant tablet. This is necessary because rabeprazole is acid-labile. Absorption of rabeprazole therefore, begins only after the tablet leaves the stomach. After oral administration of rabeprazole 20 mg, peak plasma concentrations (C_{max}) occur over a range of 2 to 5 hours (T_{max}). Absolute bioavailability for rabeprazole 20 mg (compared to intravenous administration) is

approximately 52% (because of extensive pre-systemic metabolism). Additionally, the bioavailability does not appear to increase with repeat dose administration of rabeprazole.

Effect of Food: With rabeprazole, there was no clinically relevant interaction with food. Neither food nor the time of day of administration affects the absorption of rabeprazole. Thus, rabeprazole may be administered without regard to timing of meals.

Distribution: Rabeprazole is 96.3% bound to human plasma proteins.

Metabolism: Rabeprazole is extensively metabolized. Rabeprazole is metabolized in the liver primarily by cytochromes P450 3A (CYP3A) to a sulphone metabolite and cytochrome P450 2C19 (CYP2C19) to desmethyl rabeprazole. The thioether and sulphone are the primary metabolites measured in human plasma. These metabolites were not observed to have significant antisecretory activity. A significant portion of rabeprazole is also metabolized via systemic non-enzymatic reduction to a thioether compound.

Excretion: Following a single 20 mg oral dose of rabeprazole, approximately 90% of the drug was eliminated in the urine, primarily as thioether carboxylic acid, glucuronide, and mercapturic acid metabolites. The remainder of the dose was recovered in the feces. No unchanged rabeprazole was recovered in the urine or feces. In healthy subjects, the plasma half-life is approximately one hour (range 0.7 to 1.5 hours) and the total body clearance is estimated to be 283 ± 98 ml/min.

6. Nonclinical Properties

6.1 Animal Toxicology

Carcinogenesis: In an 88/104-week carcinogenicity study in CD-1 mice, rabeprazole at oral doses up to 100 mg/kg/day did not produce any increased tumor occurrence. The highest tested dose produced a systemic exposure to rabeprazole (AUC) of 1.40 μg•hr/ml which is 1.6 times the human exposure (plasma $AUC_{0-\infty}$ = 0.88 μg•hr/ml) at the recommended dose for GERD (20 mg/day).

In a 28-week carcinogenicity study in p53+/- transgenic mice, rabeprazole at oral doses of 20, 60, and 200 mg/kg/day did not cause an increase in the incidence rates of tumors but produced gastric mucosal hyperplasia at all doses. The systemic exposure to rabeprazole at 200 mg/kg/day is about 17 to 24 times the human exposure at the recommended dose for GERD.

In a 104-week carcinogenicity study in Sprague-Dawley rats, males were treated with oral doses of 5, 15, 30 and 60 mg/kg/day and females with 5, 15, 30, 60, and 120 mg/kg/day. Rabeprazole produced gastric enterochromaffin-like (ECL) cell hyperplasia in male and female rats and ECL cell carcinoid tumors in female rats at all doses including the lowest tested dose. The lowest dose (5 mg/kg/day) produced a systemic exposure to rabeprazole (AUC) of about 0.1 μg•hr/ml which is about 0.1 times the human exposure at the recommended dose for GERD. In male rats, no treatment related tumors were observed at doses up to 60 mg/kg/day producing a rabeprazole plasma exposure (AUC) of about 0.2 μg•hr/ml (0.2 times the human exposure at the recommended dose for GERD).

Mutagenesis: Rabeprazole was positive in the Ames test, the Chinese hamster ovary cell (CHO/HGPRT) forward gene mutation test, and the mouse lymphoma cell forward gene mutation test. Its demethylated-metabolite was also positive in the Ames test. Rabeprazole

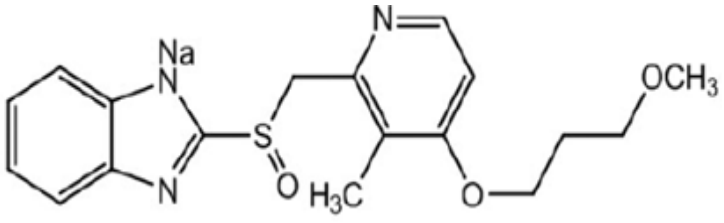
was negative in the *in vitro* Chinese hamster lung cell chromosome aberration test, the *in vivo* mouse micronucleus test, and the *in vivo* and *ex vivo* rat hepatocyte unscheduled DNA synthesis (UDS) tests.
Impairment of Fertility: Rabeprazole at intravenous doses up to 30 mg/kg/day (plasma AUC of 8.8 μg•hr/ml, about 10 times the human exposure at the recommended dose for GERD) was found to have no effect on fertility and reproductive performance of male and female rats.
Developmental Toxicity: Embryo-fetal developmental studies have been performed in rats during organogenesis at intravenous doses of rabeprazole up to 50 mg/kg/day (plasma AUC of 11.8 μg•hr/ml, about 13 times the human exposure at the recommended oral dose for GERD) and rabbits at intravenous doses up to 30 mg/kg/day (plasma AUC of 7.3 μg•hr/ml, about 8 times the human exposure at the recommended oral dose for GERD) and have revealed no evidence of harm to the fetus due to rabeprazole.

7. Description

R-PPi Tablets are Light brown coloured, round, biconvex, plain on both sides & Gastro resistant tablets.

Each tablet of R-PPi contains 20 mg of rabeprazole (in gastro-resistant form) for oral administration.

Rabeprazole sodium is a substituted benzimidazole, proton pump inhibitor (PPI) class of antisecretory agents. Rabeprazole sodium is a white to slightly yellowish-white solid. It is very soluble in water and methanol, freely soluble in ethanol, chloroform, and ethyl acetate and insoluble in ether and n-hexane.
Molecular Weight: 381.42 g/mol.
Molecular Formula: C18H20N3NaO3S
Chemical Name: 2-[[[4-(3methoxypropoxy)-3-methyl-2-pyridinyl]-methyl]sulfinyl]-1H–benzimidazole sodium salt..
Structural Formula:



Inactive ingredients (excipients) of R-PPi Tablets contain Mannitol, Light Magnesium Oxide, Crospovidone, Hydroxy Propyl Cellulose, Isopropyl Alcohol, Croscarmellose Sodium, Talcum, Magnesium Stearate, Methylene Chloride, Instacoat Sol & Instacoat En.

8. Pharmaceutical Particulars

8.1 Incompatibilities

None known.

8.2Shelf-life

24 Months

8.3Packaging Information

15 tablets per alu-alu blister.

8.4Storage and Handling Instructions

Store protected from light and moisture, at a temperature not exceeding 30°C.
Keep out of reach of children.

9. Patient Counseling Information

Instructions to Patients

- Take R-PPi Tablets exactly as prescribed by your doctor. Do not change the dose or stop therapy without consulting your doctor.
- Swallow R-PPi Tablets whole with water, especially in the morning. Do not split, chew, or crush tablets.
- If you miss a dose of R-PPi Tablets, take it as soon as possible. If it is almost time for your next dose, do not take the missed dose. Take the next dose at your regular time. Do not take 2 doses/tablets at the same time to make up for the missed dose.
- This medicine should not be used in children under 12 years of age.
- Instruct patients not to take this medicine during pregnancy and lactation unless advised by their healthcare professionals.
- Instruct patients not to share this medication with other people even though symptoms are similar. It may harm them.
- Tell your doctor about all the medicines you take, including prescription and over-the-counter medicines, vitamins and herbal supplements. R-PPi Tablets and certain other medicines can interact with each other causing serious side effects.

10. Details of Manufacturer

Pure & Cure Healthcare Pvt. Ltd.
(A subsidiary of Akums Drugs & Pharmaceuticals Ltd.)
Plot No. 26A, 27-30, Sector-8A, I.I.E., SIDCUL, Ranipur,
Haridwar – 249 403, Uttarakhand.

11. Details of Permission or License Number with Date

Mfg. Lic. No. : 31/UA/2013, Date of Product Permission: 03/11/2014

12. Date of Revision

February 2023.

Marketed by:

EXCEL BLUE CROSS || EXCEL

Division of

BLUE CROSS LABORATORIES PVT LTD.

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