SUMMARY OF PRODUCT CHARACTERISTICS (SPC)

1. NAME OF THE MEDICINAL PRODUCT

Rabeque Injection

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Each Combi-pack Contains

A. Sterilised Water for Injections BP
B. Rabeprazole sodium for Injection 20mg

Each Vial Contains

Rabeprazole Sodium 20mg

For full list of excipients, see section 6.1

3. PHARMACEUTICAL FORM

SVP lyophilized injection

4. CLINICAL PARTICULARS

4.1 Therapeutic indication

Rabeprazole for injection 20 mg is used for the treatment of

- Active duodenal ulcer with bleeding or severe erosions
- Active gastric ulcer with bleeding or severe erosions
- Short-term treatment of erosive or ulcerative gastro esophageal reflux disease (GERD)
- Prevention of acid-aspiration
- Stress-induced mucosal injury in critical care
- Pathological hypersecretory conditions, including Zollinger-Ellison syndrome

4.2 Posology and Method of Administration

The intravenous administration is recommended only in cases where the oral administration is not indicated. As soon as an oral therapy is possible the intravenous therapy should be discontinued.

Recommended dose is intravenous administration of the content of one vial (20 mg rabeprazole) once daily. Parenteral routes of administration other than intravenous are not recommended.

Injection The content of the vial needs to be reconstituted with 5 ml sterile water for injection, which should be given slowly over 5-15 min.

Infusion For intravenous infusion the reconstituted solution should be further diluted and administered as short-term infusion over 15-30 min.

4.3Contraindications

Rabeprazole is contraindicated in patients with known hypersensitivity to rabeprazole, substituted benzimidazoles or to any component of the formulation.

Rabeprazole is contraindicated in pregnancy and breastfeeding.

4.4Special Warnings and Precautions for use

Bone Fracture

Several published observational studies suggest that proton pump inhibitor (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, defined as multiple daily doses, 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 for osteoporosis-related fractures should be managed according to the established treatment guidelines.

Hypomagnesaemia

Hypomagnesaemia, symptomatic and asymptomatic, has been reported rarely in patients treated with PPIs for at least 3months, in most cases after a year of therapy. Serious adverse events include tetany, arrhythmias, and seizures. In most patients, treatment of hypomagnesaemia required magnesium replacement and 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 hypomagnesaemia (e.g. diuretics), healthcare professionals may consider monitoring magnesium levels prior to initiation of PPI treatment and periodically.

Presence of gastric malignancy

Symptomatic response to therapy with rabeprazole does not preclude the presence of gastric malignancy. Patients with healed GERD were treated for up to 40 months with rabeprazole and monitored with serial gastric

biopsies. Patients without H. pylori infection (221 of 326 patients) had no clinically important pathologic changes in the gastric mucosa. Patients with H. pylori infection at baseline (105 of 326 patients) had mild or moderate inflammation in the gastric body or mild inflammation in the gastric antrum. Patients with mild grades of infection or inflammation in the gastric body tended to change to moderate, whereas those graded moderate at baseline tended to remain stable. Patients with mild grades of infection or inflammation in the gastric antrum tended to remain stable. At baseline 8% of patients had atrophy of glands in the gastric body and 15% had atrophy in the gastric antrum. At endpoint, 15% of patients had atrophy of glands in the gastric body and 11% had atrophy in the gastric antrum. Approximately 4% of patients had intestinal metaplasia at some point during follow-up, but no consistent changes were seen.

Renal impairment
No dose adjustment is necessary in patients with renal impairment

Hepatic impairment
No dose adjustment is necessary in patients with mild to moderate hepatic impairment. Administration of rabeprazole to patients with mild to moderate liver impairment resulted in increased exposure and decreased elimination. Due to the lack of clinical data on rabeprazole in patients with severe hepatic impairment, caution should be exercised in those patients.

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

Clostridium difficile-associated Diarrhoea
Published observational studies suggest that PPI therapy such as rabeprazole sodium may be associated with an increased risk of C. difficile-associated diarrhoea (CDAD), especially in hospitalised patients. This diagnosis should be considered for diarrhoea that does not improve. Patients should use the lowest dose and shortest duration of PPI therapy appropriate to the condition being treated. CDAD has been reported with the use of nearly all antibacterial agents.

Cutaneous and Systemic Lupus Erythematosus Cutaneous lupus erythematosus (CLE) and systemic lupus erythematosus (SLE) have been reported in patients taking PPIs, including rabeprazole. These events have occurred as both new onset and an exacerbation of existing autoimmune disease. The majority of PPI-induced lupus erythematosus cases were CLE. The most common form of CLE reported in patients treated with PPIs was subacute CLE (SCLE) and

occurred within weeks to years after continuous drug therapy in patients ranging from infants to the elderly. Generally, histological findings were observed without organ involvement. Systemic lupus erythematosus (SLE) is less commonly reported than CLE in patients receiving PPIs. PPI associated SLE is usually milder than non-drug induced SLE. Onset of SLE typically occurred within days to years after initiating treatment primarily in patients ranging from young adults to the elderly. The majority of patients presented with rash; however, arthralgia and cytopaenia were also reported. Avoid administration of PPIs for longer than medically indicated. If signs or symptoms consistent with CLE or SLE are noted in patients receiving Rabeprazole sodium, discontinue the drug and refer the patient to the appropriate specialist for evaluation. Most patients improve with discontinuation of the PPI alone in 4 to 12 weeks. Serological testing (e.g. ANA) may be positive and elevated serological test results may take longer to resolve than clinical manifestations.

Cyanocobalamin (Vitamin B12) Deficiency Daily treatment with any acid-suppressing medications over a long period of time (e.g. longer than 3 years) may lead to malabsorption of cyanocobalamin (vitamin B12) 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 sodium.

Fundic Gland Polyps
PPI use is associated with an increased risk of fundic gland polyps that increases with long-term use, especially beyond one year. Most PPI users who developed fundic gland polyps were asymptomatic and fundic gland polyps were identified incidentally on endoscopy. Use the shortest duration of PPI therapy appropriate to the condition being treated.

4.5 Interaction with other medicinal products and other forms of interaction

Drugs metabolized by CYP450 Rabeprazole is metabolized by the cytochrome P450 (CYP450) drug metabolizing enzyme system. Studies in healthy subjects have shown that rabeprazole does not have clinically significant interactions with other drugs metabolized by the CYP450 system, such as warfarin and theophylline given as single oral doses, diazepam as a single intravenous dose, and phenytoin given as a single intravenous dose (with supplemental oral dosing). Steady state interactions of rabeprazole and other drugs metabolized by this enzyme system have not been studied in

patients.

Warfarin There have been reports of increased INR and prothrombin time in patients receiving proton pump inhibitors, including rabeprazole, and warfarin concomitantly. Increases in INR and prothrombin time may lead to abnormal bleeding and even death.

Cyclosporine
In vitro incubations employing human liver microsomes indicated that rabeprazole inhibited cyclosporine metabolism with an IC 50 of 62 micromolar, a concentration that is over 50 times higher than the C max in healthy volunteers following 14 days of dosing with 20 mg of rabeprazole. This degree of inhibition is similar to that by omeprazole at equivalent concentrations.

Compounds dependent on gastric pH for absorption Rabeprazole produces sustained inhibition of gastric acid secretion. An interaction with compounds which are dependent on gastric pH for absorption may occur due to the magnitude of acid suppression observed with rabeprazole. For example, in normal subjects, co-administration of rabeprazole 20 mg QD resulted in an approximately 30% decrease in the bioavailability of ketoconazole and increases in the AUC and C max for digoxin of 19% and 29%, respectively. Therefore, patients may need to be monitored when such drugs are taken concomitantly with rabeprazole. Co-administration of rabeprazole and antacids produced no clinically relevant changes in plasma rabeprazole concentrations.

Concomitant use of atazanavir and proton pump inhibitors is not recommended. Coadministration of atazanavir with proton pump inhibitors is expected to substantially decrease atazanavir plasma concentrations and thereby reduce its therapeutic effect.

Drugs metabolized by CYP2C19 In a clinical study in Japan evaluating rabeprazole in patients categorized by CYP2C19 genotype (n=6 per genotype category), gastric acid suppression was higher in poor metabolizers as compared to extensive metabolizers. This could be due to higher rabeprazole plasma levels in poor metabolizers. Whether or not interactions of rabeprazole sodium with other drugs metabolized by CYP2C19 would be different between extensive metabolizers and poor metabolizers has not been studied.

Combined administration with Clarithromycin

Combined administration consisting of rabeprazole, amoxicillin and clarithromycin resulted in increases in plasma concentrations of rabeprazole and 14-hydroxyclarithromycin.

Concomitant administration of clarithromycin with other drugs can lead to serious adverse reactions due to drug interactions. Because of these drug interactions, clarithromycin is contraindicated for co-administration with certain drugs.

Methotrexate
Case reports, published population pharmacokinetic studies, and retrospective analyses suggest that concomitant administration of PPIs and methotrexate (primarily at a high dose; see methotrexate prescribing information) may elevate and prolong serum levels of methotrexate and/or its metabolite hydroxymethotrexate. However, no formal drug interaction studies of methotrexate with PPIs have been conducted.

Clopidogrel
Concomitant administration of rabeprazole and clopidogrel in healthy subjects had no clinically meaningful effect on exposure to the active metabolite of clopidogrel. No dose adjustment of clopidogrel is necessary when administered with an approved dose of rabeprazole sodium.

Clinically Relevant Interactions Affecting Drugs Co-Administered with Rabeprazole Sodium and Interactions with Diagnostics

Antiretrovirals	
Clinical Impact	The effect of PPI on antiretroviral drugs is variable. The clinical importance and the mechanisms behind these interactions are not always known. • Decreased exposure of some antiretroviral drugs (e.g. rilpivirine, atazanavir and nelfinavir) when used concomitantly with rabeprazole may reduce the antiviral effect and promote the development of drug resistance. • Increased exposure of other antiretroviral drugs (e.g. saquinavir) when used concomitantly with rabeprazole may increase toxicity. • There are other antiretroviral drugs that do not result in clinically relevant interactions with rabeprazole.
Intervention	Rilpivirine-containing products Concomitant use with rabeprazole sodium is contraindicated [see **Contraindications**]. See the prescribing information. Atazanavir See the prescribing information for atazanavir for dosing information. Nelfinavir Avoid concomitant use with rabeprazole sodium. See prescribing information for nelfinavir. Saquinavir See the prescribing information for saquinavir and monitor for potential saquinavir toxicities. Other antiretrovirals See the prescribing information.
Warfarin	
Clinical Impact	Increased INR and prothrombin time in patients receiving PPIs, including rabeprazole and warfarin concomitantly. Increases in INR and prothrombin time may lead to abnormal bleeding and even death.
Intervention	Monitor INR and prothrombin time. Dose adjustment of warfarin may be needed to maintain target INR range. See prescribing information for warfarin.
Methotrexate	

Clinical Impact	Concomitant use of rabeprazole with methotrexate (primarily at high doses) may elevate and prolong serum levels of methotrexate and/or its metabolite hydroxymethotrexate, possibly leading to methotrexate toxicities. No formal drug interaction studies of methotrexate with PPIs have been conducted.
Intervention	A temporary withdrawal of rabeprazole sodium may be considered in some patients receiving high-dose methotrexate administration.
Digoxin	
Clinical	Potential for increased exposure of digoxin.
Impact	
Intervention	Monitor digoxin concentrations. Dose adjustment of digoxin may be needed to maintain therapeutic drug concentrations.
Drugs Dependent on Gastric pH for Absorption (e.g. iron salts, erlotinib, dasatinib, nilotinib, mycophenolate mofetil, ketoconazole, itraconazole)	
Clinical Impact	Rabeprazole can reduce the absorption of other drugs due to its effect on reducing intragastric acidity.
Intervention	Mycophenolate mofetil (MMF) Co-administration of PPIs in healthy subjects and in transplant patients receiving MMF has been reported to reduce the exposure to the active metabolite, mycophenolic acid (MPA), possibly due to a decrease in MMF solubility at an increased gastric pH. The clinical relevance of reduced MPA exposure on organ rejection has not been established in transplant patients receiving rabeprazole sodium and MMF. Use rabeprazole sodium with caution in transplant patients receiving MMF.
Combination Therapy with Clarithromycin and Amoxicillin	
Clinical Impact	Concomitant administration of clarithromycin with other drugs can lead to serious adverse reactions, including potentially fatal arrhythmias, and are contraindicated. Amoxicillin also has drug interactions.
Intervention	See **Contraindications** and **Warnings and Precautions** in the prescribing information for clarithromycin.
Tacrolimus	
Clinical Impact	Potentially increased exposure of tacrolimus, especially in transplant patients who are intermediate or poor metabolisers of CYP2C19.
Intervention	Monitor tacrolimus whole blood trough concentrations. Dose adjustment of tacrolimus may be needed to maintain therapeutic drug concentrations. See prescribing information for tacrolimus.
Interactions with Investigations of Neuroendocrine Tumours	
Clinical Impact	Serum chromogranin A (CgA) levels increase secondary to PPI-induced decreases in gastric acidity. The increased CgA level may cause false-positive results in diagnostic investigations for neuroendocrine tumours.
Intervention	Temporarily stop rabeprazole sodium treatment at least 14 days before assessing CgA levels and consider repeating the test if initial CgA levels are high. If serial tests are performed (e.g. for monitoring), the same commercial laboratory should be used for testing, as reference ranges between tests may vary.
Interaction with Secretin Stimulation Test	
Clinical Impact	Hyper-response in gastrin secretion in response to secretin stimulation test, falsely suggesting gastrinoma.
Intervention	Temporarily stop treatment with rabeprazole sodium at least 14 days before assessing to allow gastrin levels to return to baseline.
False-Positive Urine Tests for THC	

Clinical Impact	There have been reports of false-positive urine screening tests for tetrahydrocannabinol (THC) in patients receiving PPIs.
Intervention	An alternative confirmatory method should be considered to verify positive results.

4.6 Pregnancy and Lactation

Pregnancy

There are no data on the safety of rabeprazole in human pregnancy. Reproduction studies performed in rats and rabbits have revealed no evidence of impaired fertility or harm to the foetus due to Rabeprazole sodium, although low foeto-placental transfer occurs in rats. Rabeprazole is contraindicated in pregnancy

Breast-feeding

It is not known whether Rabeprazole sodium is excreted in human breasts milk, no studies in breast-feeding women have been performed. Rabeprazole sodium is however excreted in rat mammary secretions. Therefore, Rabeprazole should not be used during breast-feeding.

4.7 Effects on ability to drive and use machines

Based on the Pharmacodynamics properties and the adverse events profile, it is unlikely that Rabeprazole for injection 20 mg would cause an impairment of driving performance or compromises the ability to use machinery. If, however, alertness is impaired due to somnolence, it is recommended that driving and operating complex machinery is avoided.

4.8 Undesirable effects

The most commonly reported adverse drug reactions, during controlled clinical trials with rabeprazole were headache, diarrhea, abdominal pain, asthenia, flatulence, rash and dry mouth. The majority of adverse events experienced during clinical studies were mild or moderate in severity, and transient in nature.

The following adverse events have been reported from clinical trial and post-marketing experience. the frequencies as common ≥1/100 to <1/10; uncommon ≥1/1,000 to <1/100; rare ≥1/10,000 to <1/1,000; very rare
<1/10,000; not known = cannot be estimated from available data.

System Organ Class	Common	Uncommon	Rare	Very Rare	Not known
Infections And Infestations	Infection				
Blood and lymphatic system disorders			Neutropenia, Leucopenia, Thrombocytopenia, Leucocytosis		
Immune system disorders			Hypersensitivity[1],[2]		
Metabolism and nutrition disorders			Anorexia		Hyponatremia, Hypomagnesaemia
Psychiatric disorders	Insomnia	Nervousness	Depression		Confusion
Nervous system disorders	Headache, Dizziness	Somnolence			

Eye disorders			Visual disturbance		
Vascular disorders					Peripheral oedema
Respiratory, thoracic and mediastinal disorders	Cough, Pharyngitis, Rhinitis	Bronchitis, Sinusitis			
Gastrointestinal disorders	Diarrhoea, Vomiting, Nausea, Abdominal pain, Constipation, Flatulence,	Dyspepsia, Dry mouth, Eructation	Gastritis, Stomatitis, Taste disturbance		Microscopic colitis

	Fundic gland polyps (benign)				
Hepato biliary disorde			Hepatitis, Jaundice		

rs			, Hepatic encephal opathy[3]		
Skin and subcutaneous tissue disorders	Rash	Erythema[2]	Pruritus, Sweating, Bullous reactions[2]	Erythema multiforme, TEN, SJS	Subacut e cutaneous lupus erythem atosus
Musculoskeletal and connec tive tissue disorde rs	Non-specific pain, Back pain	Myalgia, Legcramps, Arthralgia			Fracture of hip, wrist or spine[4]
Renal and urinary disorders		Urinary tract infec tion	Tubuloin terstitial nephritis (with possible progressi on to renal failure)		

Reproductive system and breast disorders				Gynecomastia	
General disorders and administration site conditions	Asthenia, Influenza-like illness	Chest pain, Chills, Pyrexia			
Investigations		Increased hepatic enzymes[3]	Weight increased		

Reporting of suspected adverse reactions. Healthcare professionals are requested to report any suspected adverse reactions via the Pharmacy and Poisons Board pharmacovigilance Electronic Reporting system (PvERS) https//pv.pharmacyboardkenya.org.

4.9 Overdose

As Rabeque is usually given by healthcare professionals, there are least chances that your doctor/nurse will give too much of medicine. Experience to date with deliberate or accidental overdose is limited. The maximum established exposure has not exceeded 60mg twice daily, or 160mg once daily. Effects are generally minimal, representative of the known adverse event profile and reversible without further medical intervention. No specific antidote is known. Rabeprazole sodium is extensively protein bound and is, therefore, not dialysable. As in any case of overdose, treatment should be symptomatic and general supportive measures should be utilised.

5. PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic Properties

Pharmacotherapeutic group Alimentary tract and metabolism, Drugs for peptic ulcer and gastrooesophageal Reflux disease (GORD), proton pump inhibitors, ATC code A02B C04

Mechanism of Action Rabeprazole sodium belongs to the class of antisecretory compounds, the substituted benzimidazoles, that do not exhibit anticholinergic or H2 histamine antagonist properties, but suppress gastric acid secretion by the specific inhibition of the H^+/K^+ - ATPase enzyme (the acid or proton pump) The effect is dose related and leads to inhibition of both basal and stimulated acid secretion irrespective of the stimulus. Animal studies indicate that after administration, rabeprazole sodium rapidly disappears from both the plasma and gastric mucosa. As a weak base, rabeprazole is rapidly absorbed following all doses and is concentrated in the acid environment of the parietal cells. Rabeprazole is converted to the active sulphenamide form through protonation and it subsequently reacts with the available cysteines on the proton pump.

Antisecretory Activity After oral administration of a 20mg dose of rabeprazole sodium the onset of the antisecretory effect occurs within one hour, with the maximum effect occurring within two to four hours. Inhibition of basal and food stimulated acid secretion 23 hours after the first dose of rabeprazole sodium are 69% and 82% respectively and the duration of inhibition lasts up to 48 hours. The inhibitory effect of rabeprazole sodium on acid secretion increases slightly with repeated once daily dosing, achieving steady state inhibition after three days. When the drug is discontinued, secretory activity normalises over 2 to 3 days.

Decreased gastric acidity due to any means, including proton pump inhibitors such as rabeprazole, increases counts of bacteria normally present in the gastrointestinal tract. Treatment with proton pump inhibitors may possibly increase the risk of gastrointestinal infections such as Salmonella, Campylobacter and Clostridium difficile.

Serum Gastrin Effects In clinical studies patients were treated once daily with 10 or 20mg rabeprazole sodium, for up to 43 months duration. Serum gastrin levels increased during the first 2 to 8 weeks reflecting the inhibitory effects on acid secretion and remained stable while treatment was continued. Gastrin values returned to pretreatment levels, usually within 1 to 2 weeks after

discontinuation of therapy.

Human gastric biopsy specimens from the antrum and the fundus from over
500 patients receiving rabeprazole or comparator treatment for up to 8 weeks have not detected changes in ECL cell histology, degree of gastritis, incidence of atrophic gastritis, intestinal metaplasia or distribution of H. pylori infection. In over 250 patients followed for 36 months of continuous therapy, no significant change in findings present at baseline was observed.

Other Effects Systemic effects of rabeprazole sodium in the CNS, cardiovascular and respiratory systems have not been found to date. Rabeprazole sodium, given in oral doses of 20mg for 2 weeks, had no effect on thyroid function, carbohydrate metabolism, or circulating levels of parathyroid hormone, cortisol, oestrogen, testosterone, prolactin, cholecystokinin, secretin, glucagon, follicle stimulating hormone (FSH), luteinising hormone (LH), renin, aldosterone or somatotrophic hormone.

Studies in healthy subjects have shown that rabeprazole sodium does not have clinically significant interactions with amoxicillin. Rabeprazole does not adversely influence plasma concentrations of amoxicillin or clarithromycin when coadministered for the purpose of eradicating upper gastrointestinal H. pylori infection.

5.2 Pharmacokinetic Properties

Absorption

Rabeprazole is rapidly absorbed. When Rabeprazole is administered with a high fat meal, its T max is variable and may delay its absorption up to 4 hours or longer, however, the C max and the extent of Rabeprazole absorption (AUC) are not significantly altered.

Distribution

Rabeprazole is 96.3% bound to human plasma proteins.

Metabolism

Rabeprazole sodium, as is the case with other members of the proton pump inhibitor (PPI) class of compounds, is metabolised through the cytochrome P450 (CYP450) hepatic drug metabolising system. In vitro studies with human liver microsomes indicated that rabeprazole sodium is metabolized by isoenzymes of CYP450 (CYP2C19 and CYP3A4). In these studies, at expected human plasma concentrations

rabeprazole neither induces nor inhibits CYP3A4; and although in vitro studies may not always be predictive of in vivo status these findings indicate that no interaction is expected between rabeprazole and cyclosporin. In humans the thioether (M1) and carboxylic acid (M6) are the main plasma metabolites with the sulphone (M2), desmethylthioether(M4) and mercapturic acid conjugate (M5) minor metabolites observed at lower levels. Only the desmethyl metabolite (M3) has a small amount of antisecretory activity, but it is not present in plasma.

Elimination
Approximately 90% of the dose is eliminated in urine mainly as the two metabolites a mercapturic acid conjugate (M5) and a carboxylic acid (M6), plus two unknown metabolites. The remainder of the dose was recovered in faeces.

5.3 Preclinical Safety Data

Non-clinical effects were observed only at exposures sufficiently in excess of the maximum human exposure that make concerns for human safety negligible in respect of animal data. Studies on mutagenicity gave equivocal results. Tests in mouse lymphoma cell line were positive, but in vivo micronucleus and in vivo and in vitro DNA repair tests were negative. Carcinogenicity studies revealed no special hazard for humans.

6. PHARMACEUTICAL PARTICULARS

6.1 List of Excipients

Mannitol BP

Sodium hydroxide BP

Water for injection BP.

6.2 Incompatibilities

This medicinal product must not be used with other medicinal products.

6.3 Shelf Life

24 months.

Shelf-life after reconstitution

Chemical and physical in-use stability has been demonstrated for 12 hours at 30°C. From a microbiological point of view, the product

should be used immediately

6.4 Special Precautions for Storage

Store below 30^0C. Keep out of reach of children.

6.5 Nature and content of

container

Primary Container Closure System
10 ml USP Type I clear tubular glass vial stoppered with 20m slotted bromobutyl 'RFU' rubber stopper and 20 mm flip off aluminium seal cap.

Secondary Container Closure System
Such a vial is packed with diluent in a carton along with diluent and pack insert.

6.6 Special Precautions for disposal

No special requirements.

7. MARKETING AUTHORIZATION HOLDER AND

MANUFACTURING SITE

MAH: SURGILINKS LTD
Surgilinks Building Mombasa Road
P.O. Box 14461 – 00800 Nairobi Kenya

Manufacturing site
Gufic Biosciences Limited.
National Highway No. 8, Near
Grid, Kabilpore - 396424,
City
Navsari, Dist. Navsari,
Gujarat State, INDIA.

8. MARKETING AUTHORIZATION NUMBER(S)

16796

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE

AUTHORIZATION

19/11/2011

10.DATE OF REVISION OF THE TEXT

12/08/2026