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Cliente *Customer*
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Colores *Colors print*
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Prospecto *Leaflet*
PANTOSALA 40 mg
RJ PAN MALAYAN

Código *Code*
B1XXXX-01

Sustituye *Replaces*
-

Medidas *Format*
140 x 600 mm

Plegado *Fold*
*35 x 100 mm

Material *Material*
Specialprint 40 g

Plano *Layout leaflet*
-

Barcode *Barcode*
-

Tipografía *Font*
Myriad Pro Light Condensed 9 pt
Interl. 9 pt

Observaciones *Observations*
*** Lomo en 100 mm.**

Aprobado *Approved*
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PACKAGE INSERT

PANTOSALA 40 mg powder for solution for injection

1. NAME OF THE MEDICINAL PRODUCT
PANTOSALA 40 mg powder for solution for injection

2. QUALITATIVE AND QUANTITATIVE COMPOSITION
Each vial contains 45.2 mg pantoprazole sodium sesquihydrate equivalent to 40 mg pantoprazole.

Excipient with known effect
Each vial contains 1 mg edetate tetrasodium.
For the full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM
Powder for solution for injection.
White to off-white powder.

4. CLINICAL PARTICULARS
4.1 Therapeutic indications
PANTOSALA 40 mg is indicated in adults for treatment of short-term use for symptomatic improvement and healing of gastrointestinal diseases which require a reduction in acid secretion:
- Moderate to severe reflux oesophagitis.
- Gastric and duodenal ulcer.

4.2 Posology and method of administration
This medicine should be administered by a healthcare professional and under appropriate medical supervision.
Intravenous administration of pantoprazole is recommended only if oral administration is not appropriate.

Posology
Gastric and duodenal ulcer, moderate and severe reflux oesophagitis
The recommended intravenous dose is one vial of PANTOSALA 40 mg (40 mg pantoprazole) per day.
As soon as oral therapy is possible, treatment with pantoprazole i.v. should be discontinued and pantoprazole p. o. should be administered instead.

Special populations
Paediatric population
The safety and efficacy of pantoprazole 40 mg powder for solution for injection in children aged under 18 years have not been established. Therefore, pantoprazole 40 mg powder for solution for injection is not recommended for use in patients below 18 years of age until further data become available.

Hepatic impairment
In patients with severe liver impairment the daily dose has to be reduced to 20 mg pantoprazole. (See section 4.4).

Renal impairment
The daily dose of 40 mg pantoprazole should not be exceeded in patients with impaired kidney function (see section 5.2).

Elderly patients
The daily dose of 40 mg pantoprazole should not be exceeded in elderly patients.

Method of administration
A ready-to-use solution is prepared in 10 ml of sodium chloride 9 mg/ml (0.9%) solution for injection. The prepared solution may be administered directly or may be administered after mixing it with 100 ml sodium chloride 9 mg/ml (0.9%) solution for injection or glucose 50 mg/ml (5%) solution for injection.
After preparation the solution must be used within 12 hours.
The medicinal product should be administered intravenously over 2-15 minutes.
For instructions on reconstitution and dilution of the medicinal product before administration, see section 6.6.

4.3 Contraindications
Pantoprazole i.v. should generally not be used in cases of known hypersensitivity to one of the constituents of pantoprazole i.v.
Pantoprazole, like other PPIs, should not be co-administered with atazanavir (See section 4.5).

4.4 Special warnings and precautions for use
The intravenous administration of pantoprazole i.v. is recommended only if oral application is not appropriate.
Pantoprazole is not indicated for mild gastro-intestinal complaints such as nervous dyspepsia.

Gastric malignancy
In the presence of any alarm symptom (e.g. significant unintentional weight loss, recurrent vomiting, dysphagia, haematemesis, anaemia or melaena) and when gastric ulcer is suspected or present, malignancy should be excluded, as symptomatic response to pantoprazole may mask the symptoms of gastric malignancy and delay diagnosis.
Further investigation is to be considered if symptoms persist despite adequate treatment.

Hepatic impairment
In patients with severe liver impairment, the liver enzymes should be monitored during therapy. In the case of a rise of the liver enzymes, the treatment should be discontinued (See section 4.2).

Renal Impairment
The daily dose of 40 mg pantoprazole should not be exceeded in elderly patients or in patients with impaired kidney function

Co-administration with HIV protease inhibitors
Co-administration of pantoprazole is not recommended with HIV protease inhibitors for which absorption is dependent on acidic intragastric pH such as atazanavir, due to significant reduction in their bioavailability (see section 4.5).
Co-administration of atazanavir with proton pump inhibitors is not recommended (See section 4.5).

Gastrointestinal infections caused by bacteria
Treatment with pantoprazole may lead to a slightly increased risk of gastrointestinal infections caused by bacteria such as Salmonella and Campylobacter or C. difficile.
Pantoprazole, like all proton pump inhibitors (PPIs), might be expected to increase the counts of bacteria normally present in the upper gastrointestinal tract. Treatment with pantoprazole may lead to a slightly increased risk of gastrointestinal infections caused by bacteria such as *Salmonella* and *Campylobacter* and in hospitalized patients, possibly also *Clostridium difficile.*

Concomitant use with Methotrexate
Literature suggests that concomitant use of PPIs with methotrexate (primarily at high dose; see methotrexate prescribing information) may elevate and prolong serum levels of methotrexate and/or its metabolite, possibly leading to methotrexate toxicities. In high-dose methotrexate administration a temporary withdrawal of the PPI may be considered in some patients (See section 4.8).

Excipients
This medicinal product contains less than 1 mmol sodium (23 mg) per vial, i.e. is essentially 'sodium-free'.

Hypomagnesaemia
Hypomagnesaemia, symptomatic and asymptomatic, has been reported rarely in patients treated with PPIs for at least three months, 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), health care professionals may consider monitoring magnesium levels prior to initiation of PPI treatment and periodically. (See section 4.8).

Bone fractures
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.
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 the 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.
To date there has been no experience with treatment in children.

Cutaneous lupus erythematosus (CLE) and systemic lupus erythematosus (SLE)
Cutaneous lupus erythematosus (CLE) and systemic lupus erythematosus (SLE) have been reported in patients taking PPIs. 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 cytopenia 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 PANTOSALA 40 mg, 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., Antinuclear antibody) may be positive and elevated serological test results may take longer to resolve than clinical manifestations.

Subacute cutaneous lupus erythematosus (SCLE)
Proton pump inhibitors 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 the healthcare professional should consider stopping PANTOSALA 40 mg. SCLE after previous treatment with a proton pump inhibitor may increase the risk of SCLE with other proton pump inhibitors.

Interference with laboratory tests
Increased Chromogranin A (CgA) level may interfere with investigations for neuroendocrine tumours. To avoid this interference, proton pump inhibitor treatment should be stopped 14 days before CgA measurements.

4.5 Interaction with other medicinal products and other forms of interaction
Medicinal products with pH dependent absorption pharmacokinetics
Because of profound and long-lasting inhibition of gastric acid secretion, pantoprazole may interfere with the absorption of other medicinal products where a gastric pH is an important determinant of oral bioavailability, e.g. some azole antifungals such as ketoconazole, itraconazole, posaconazole and other medicine such as erlotinib.

HIV protease inhibitors
Co-administration of pantoprazole is not recommended with atazanavir and other HIV protease inhibitors whose absorption is pH-dependent due to substantial reduction in their bioavailability that might impact the efficacy of these medicines. Therefore, the co-administration of proton pump inhibitors with atazanavir is not recommended (See section 4.4).

Coumarin anticoagulants (phenprocoumon or warfarin)
Although no interaction during concomitant administration of pantoprazole with phenprocoumon or warfarin has been observed in clinical pharmacokinetic studies, a few isolated cases of increased International Normalised Ratio (INR) and prothrombin time have been reported during concomitant treatment in the post-marketing period. Increases in INR and prothrombin time may lead to abnormal bleeding, and even death. Therefore, in patients treated with coumarin anticoagulants (e.g. phenprocoumon or warfarin), monitoring of prothrombin time/INR is recommended after initiation, termination or during irregular use of pantoprazole.

Methotrexate
Case reports, published population pharmacokinetic studies, and retrospective analyses suggest that concomitant administration of PPIs and methotrexate (primarily at 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 (See section 4.4).

Other interactions studies
Pantoprazole is extensively metabolized in the liver via the cytochrome P450 enzyme system. The main metabolic pathway is demethylation by CYP2C19 and other metabolic pathways include oxidation by CYP3A4.
An interaction of pantoprazole with other drugs or compounds which are metabolized using the same enzyme system cannot be ruled out. However, in targeted studies involving a range of such drugs and substances no clinically significant interactions were observed; studies have been carried out on carbamazepine, caffeine, diazepam, diclofenac, digoxin, ethanol, glibenclamide, metoprolol, naproxen, nifedipine, phenytoin, piroxicam, theophylline, and an oral contraceptive.
Interaction studies with drugs also metabolized with these pathways, like carbamazepine, diazepam, glibenclamide, nifedipine, and an oral contraceptive containing levonorgestrel and ethinyl oestradiol, did not reveal clinically significant interactions.
Results from a range of interaction studies demonstrate that pantoprazole does not affect the metabolism of active substances metabolised by CYP1A2 (such as caffeine, theophylline), CYP2C9 (such as piroxicam, diclofenac, naproxen), CYP2D6 (such as metoprolol), CYP2E1 (such as ethanol), or does not interfere with p-glycoprotein related absorption of digoxin.
There were no interactions with concomitantly administered antacids.
Interaction studies have also been performed by concomitantly administering pantoprazole with the respective antibiotics (clarithromycin, metronidazole, amoxicillin). No clinically relevant interactions were found.

Medicinal products that inhibit or induce CYP2C19
Inhibitors of CYP2C19 such as fluvoxamine could increase the systemic exposure of pantoprazole. A dose reduction may be considered for patients treated long-term with high doses of pantoprazole, or those with hepatic impairment.
Enzyme inducers affecting CYP2C19 and CYP3A4 such as rifampicin and St John´s wort (Hypericum perforatum) may reduce the plasma concentrations of PPIs that are metabolized through these enzyme systems.
Concomitant administration of pantoprazole and tacrolimus may increase whole blood levels of tacrolimus, especially in transplant patients who are intermediate or poor metabolizers of CYP2C19.

4.6 Fertility, pregnancy and lactation
Pregnancy
Limited data from the use of pantoprazole in pregnant women (between 300-1000 pregnancy outcome) indicate no malformative or feto/neonatal toxicity of pantoprazole. Studies in animals have shown reproductive toxicity (See section 5.3). The potential risk for humans is unknown.
As a precautionary measure, PANTOSALA 40 mg should not be used during pregnancy, unless clearly necessary.

Breastfeeding
Animal studies have shown excretion of pantoprazole in breast milk. There is insufficient information on the excretion of pantoprazole in human milk but excretion into human milk has been reported. A risk to the newborns/infants cannot be excluded. Therefore, a decision on whether to continue/discontinue breast-feeding or to continue/discontinue/abstain therapy with pantoprazole should be made taking into account the benefit of breast-feeding for the child, and the benefit of PANTOSALA 40 mg therapy for the women.

Fertility
There was no evidence of impaired fertility following the administration of pantoprazole in animal studies (see section 5.3).

4.7 Effects on ability to drive and use machines
Pantoprazole has no or negligible influence on the ability to drive and use machines.
Adverse drug reactions, such as dizziness and visual disturbances may occur (See section 4.8). If affected, patients should not drive or operate machines.

4.8 Undesirable effects
Approximately 5% of patients can be expected to experience adverse drug reactions (ADRs). The most commonly reported ADR is injection site thrombophlebitis. Diarrhoea and headache occurred in approximately 1% of patients.
The table below lists adverse reactions reported with pantoprazole, ranked under the following frequency classification:
Very common (≥ 1/10),
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 the available data).
For all adverse reactions reported from post-marketing experience, it is not possible to apply any adverse reaction frequency and therefore they are mentioned with a "not known" frequency.
Within each frequency grouping, adverse reactions are presented in order of decreasing seriousness.

Table 1
Adverse reactions with pantoprazole in clinical trials and post-marketing experience

MedDRA SOC	***Frequency***	***Undesirable effect***
Blood and lymphatic system disorders	Rare	Agranulocytosis
	Very rare	Thrombocytopenia, leukopenia, pancytopenia
Immune system disorders	Rare	Hypersensitivity (including anaphylactic reactions and anaphylactic shock)
	Postmarketing experience"	Systemic lupus erythematosus
Metabolism and nutrition disorders	Rare	Hyperlipidaemias and lipid increases (triglycerides, cholesterol), weight changes
	Not known	Hyponatraemia, hypomagnesaemia (See section 4.4)
Psychiatric disorders	Uncommon	Sleep disorders
	Rare	Depression (and all aggravations)
	Very rare	Disorientation (and all aggravations)
	Not known	Hallucination, confusion (especially in pre-disposed patients, as well as the aggravation of these symptoms in case of pre-existence)
Nervous system disorders	Uncommon	Headache, dizziness
	Rare	Taste disorders
Eye disorders	Rare	Disturbances in vision/blurred vision
Gastrointestinal disorders	Common	Fundic gland polyps (benign)
	Uncommon	Diarrhoea, nausea/vomiting, abdominal distension and bloating, constipation, dry mouth, abdominal pain and discomfort
Hepatobiliary disorders	Uncommon	Liver enzymes increased (transaminases, γ-GT)
	Rare	Bilirubin increased
	Not known	Hepatocellular injury, jaundice, hepatocellular failure
Skin and subcutaneous tissue disorders	Uncommon	Rash/exanthema/eruption, pruritus
	Rare	Urticaria, angioedema
	Not known	Stevens-Johnson syndrome, Lyell syndrome, erythema multiforme, photosensitivity, subacute cutaneous lupus erythematosus (see section 4.4)
	Postmarketing experience	Cutaneous lupus erythematosus
Musculoskeletal and connective tissue disorders	Uncommon	Fracture of the hip, wrist or spine (See section 4.4)
	Rare	Arthralgia, myalgia
Renal and urinary disorders	Not known	Interstitial nephritis
Reproductive system and breast disorders	Rare	Gynaecomastia
General disorders and administration site conditions	Common	Injection site thrombophlebitis
	Uncommon	Asthenia, fatigue and malaise
	Rare	Body temperature increased, oedema peripheral

4.9 Overdose
There are no known symptoms of overdosage in man. In the case of overdosage with clinical signs of intoxication, the usual rules of intoxication therapy apply.

5. PHARMACOLOGICAL PROPERTIES
5.1 Pharmacodynamic properties
Pharmacotherapeutic group: Proton pump inhibitors, ATC code: A02BC02

Mechanism of action
Pantoprazole is a substituted benzimidazole which inhibits the secretion of hydrochloric acid in the stomach by specific blockade of the proton pumps of the parietal cells.
Pantoprazole is converted to its active form in the acidic environment in the parietal cells where it inhibits the H+, K+-ATPase enzyme, i. e. the final stage in the production of hydrochloric acid in the stomach. The inhibition is dose-dependent and affects both basal and stimulated acid secretion. In most patients, freedom from symptoms is achieved within 2 weeks. As with other proton pump inhibitors and H_2 receptor inhibitors, treatment with pantoprazole reduces acidity in the stomach and thereby increases gastrin in proportion to the reduction in acidity. The increase in gastrin is reversible. Since pantoprazole binds to the enzyme distal to the cell receptor level, it can inhibit hydrochloric acid secretion independently of stimulation by other substances (acetylcholine, histamine, gastrin). The effect is the same whether pantoprazole is given orally or intravenously.
The fasting gastrin values increase under pantoprazole in response to the decreased acid secretion. Also, CgA increases due to decreased gastric acidity. On short-term use, in most cases they do not exceed the upper limit of normal. During long-term treatment, gastrin levels double in most cases. An excessive increase, however, occurs only in isolated cases. As a result, a mild to moderate increase in the number of specific endocrine (ECL) cells in the stomach is observed in a minority of cases during long-term treatment (simple to adenomatoid hyperplasia). However, according to the studies conducted so far, the formation of carcinoid precursors (atypical hyperplasia) or gastric carcinoids as were found in animal experiments (See section 5.3) have not been observed in humans.
The increased CgA level may interfere with investigations for neuroendocrine tumours.
Available published evidence suggests that proton pump inhibitors should be discontinued 14 days prior to CgA measurements. This is to allow CgA levels that might be spuriously elevated following PPI treatment to return to reference range.
An influence of a long-term treatment with pantoprazole exceeding one year cannot be completely ruled out on endocrine parameters of the thyroid according to results in animal studies.

5.2 Pharmacokinetic properties
General pharmacokinetics
Pharmacokinetics does not vary after single or repeated administration. In the dose range of 10 to 80 mg, the plasma kinetics of pantoprazole are linear after both oral and intravenous administration.

Distribution
Pantoprazole's serum protein binding is about 98%. Volume of distribution is about 0.15 l/kg.

Elimination
The substance is almost exclusively metabolized in the liver. The main metabolic pathway is demethylation by CYP2C19 with subsequent sulphate conjugation, other metabolic pathway includes oxidation by CYP3A4. Terminal half-life is about 1 hour and clearance is about 0.1 l/h/kg. There were a few cases of subjects with delayed elimination. Because of the specific binding of pantoprazole to the proton pumps of the parietal cell the elimination half-life does not correlate with the much longer duration of action (inhibition of acid secretion). Renal elimination represents the major route of excretion (about 80%) for the metabolites of pantoprazole, the rest is excreted with the faeces. The main metabolite in both the serum and urine is desmethylpantoprazole which is conjugated with sulphate. The half-life of the main metabolite (about 1.5 hours) is not much longer than that of pantoprazole.

Characteristics in patients/special groups of subjects
Poor metabolisers lack a functional CYP2C19 enzyme. In these individuals the metabolism of pantoprazole is probably mainly catalysed by CYP3A4. After a single-dose administration of 40 mg pantoprazole, the mean area under the plasma concentration-time curve was approximately 6 times higher in poor metabolisers than in subjects having a functional CYP2C19 enzyme (extensive metabolisers). Mean peak plasma concentrations were increased by about 60%. These findings have no implications for the posology of pantoprazole.
No dose reduction is recommended when pantoprazole is administered to patients with impaired renal function (incl. dialysis patients). As with healthy subjects, pantoprazole's half-life is short. Only very small amounts of pantoprazole are dialyzed. Although the main metabolite has a moderately delayed half-life (2-3 h), excretion is still rapid and thus accumulation does not occur.
Although for patients with liver cirrhosis (classes A and B according to Child) the half-life values increased to between 7 and 9 h and the AUC values increased by a factor of 5-7, the maximum serum concentration only increased slightly by a factor of 1.5 compared with healthy subjects.
A slight increase in AUC and C_{max} in elderly volunteers compared with younger counterparts is also not clinically relevant.

5.3 Preclinical safety data
Preclinical data reveal no special hazard to humans based on conventional studies of safety pharmacology, repeated dose toxicity and genotoxicity. In the two-year carcinogenicity studies in rats neuroendocrine neoplasms were found. In addition, squamous cell papillomas were found in the forestomach of rats. The mechanism leading to the formation of gastric carcinoids by substituted benzimidazoles has been carefully investigated and allows the conclusion that it is a secondary reaction to the massively elevated serum gastrin levels occurring in the rat during chronic high-dose treatment. In the two-year rodent studies, an increased number of liver tumors was observed in rats and in female mice and was interpreted as being due to pantoprazole's high metabolic rate in the liver.
A slight increase of neoplastic changes of the thyroid was observed in the group of rats receiving the highest dose (200 mg/kg). The occurrence of these neoplasms is associated with the pantoprazole-induced changes in the breakdown of thyroxine in the rat liver. As the therapeutic dose in man is low, no harmful effects on the thyroid glands are expected.
In animal reproduction studies, signs of slight fetotoxicity were observed at doses above 5 mg/kg.
Investigations revealed no evidence of impaired fertility or teratogenic effects.
Penetration of the placenta was investigated in the rat and was found to increase with advanced gestation. As a result, concentration of pantoprazole in the foetus is increased shortly before birth.

6. PHARMACEUTICAL PARTICULARS
6.1 List of excipients
Edetate tetrasodium
Mannitol
Tromethamine (for pH adjustment)

6.2 Incompatibilities
This medicinal product must not be mixed with other medicinal products except those mentioned in section 6.6.

6.3 Shelf life
Unopened vial
Please refer to outer carton.

After reconstitution and dilution
Chemical and physical in use stability for PANTOSALA 40 mg after reconstitution with 0.9% sodium chloride and dilution with sodium chloride (0.9%) or Glucose (5%) has been demonstrated for 12 hours at 25 ºC and 24 hours at 5± 3ºC.
From a microbiological point of view, the product should be used immediately.
If not used immediately, in-use storage times and conditions are the responsibility of the user.

6.4 Special precautions for storage
Do not store above 25ºC.
Store in the original package in order to protect from light.
For storage conditions after reconstitution and dilution of the medicinal product, see section 6.3.

6.5 Nature and contents of container
Type I 10 ml amber glass vial with a chlorobutyl stopper, and a flip-off cap.

Pack size
Carton box containing 1 vial.
Carton box containing 5vials and 10 vials.
Not all pack sizes are marketed locally.

6.6 Special precautions for disposal and other handling
A ready-to-use solution is prepared by injecting 10 ml of sodium chloride 9 mg/ml (0.9%) solution for injection into the vial containing the powder. This solution may be administered directly or may be administered after mixing it with 100 ml sodium chloride 9 mg/ml (0.9%) solution for injection or glucose 50 mg/ml (5%) solution for injection. Glass or plastic containers should be used for dilution.
After reconstitution, or reconstitution and dilution, chemical and physical in use stability has been demonstrated for 12 hours at 25 ºC and 24 hours at 5± 3 ºC.
From a microbiological point of view, the product should be used immediately.
PANTOSALA 40 mg should not be prepared or mixed with solvents other than those stated.
The medicine should be administered intravenously over 2-15 minutes.
The contents of the vial are for single use only. Any product that has remained in the container or the visual appearance of which has changed (e.g. if cloudiness or precipitation is observed) should be disposed of in accordance with local requirements.

7. PRODUCT OWNER
Laboratorio Reig Jofre, S.A
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08970 Sant Joan Despí-Barcelona
Spain.

8. DATE OF REVISION OF THE TEXT
08/2021

REIG JOFRE