Summary of Product Characteristics for Pharmaceutical Products

1. Name of the medicinal product:

Paradym (Diclofenac Potassium 50 mg and Acetaminophen 325 mg) Tablet

2. Qualitative and quantitative composition:

Each uncoated tablet contains:
Acetaminophen 325 mg
Diclofenac Potassium 50 mg.

For full list of excipients, see section 6.1.

3. Pharmaceutical form:

Uncoated tablet

Pink colour, elongated, biconvex, scored on one side uncoated tablet.

4. Clinical particulars

4.1. Therapeutic indications:

Musculoskeletal system inflammatory and degenerative affections such as: rheumatoid arthritis ankylosing spondylitis, arthrosis (including spondylarthrosis, painful vertebral syndrome); abarticular inflammatory affections; acute gout; post-traumatic pains or post-operative inflammation; inflammation and oedema. Paradym is indicated for symptomatic or add-on therapy.

4.2 Posology and method of administration

Posology:

Undesirable effects may be minimized by using the lowest effective dose for the shortest duration necessary to control symptoms (see section 4.4 Special warnings and precautions for use).

Adults: 1 tablet 2-3 times daily. If the symptoms are more important by night or in the morning, the tablets should be taken in the evening.

Children: the pharmaceutical form and dosage are not indicated for children.

Method of administration: Paradym tablets should be swallowed with a drink, preferably while eating. Elderly patients: the dosage should be reduced and monitoring of biological parameters is recommended.

The daily dose of paracetamol cannot exceed 2 g in the following situations: Liver failure, Gilbert's syndrome, Chronic alcoholism.

Impaired renal function: The dose of paracetamol should be reduced in terms of the creatinine clearance: (Glomerular filtration rate) GFR of 10 - 50 mL/min Dose of paracetamol is 500 mg every 6 hours, GFR of less than 10 mL/min Dose of paracetamol is 500 mg every 8 hours

4.3 Contraindications:

- Established congestive heart failure (NYHA II-IV)
- Ischemic heart disease
- Peripheral arterial disease and/or cerebrovascular disease.
- Gastrointestinal ulcer
- Known hypersensitivity to the active ingredients or any of the excipients
- Severe hepatic failure
- Moderate to severe renal failure
- Due to cross-allergy, diclofenac should not be given to patients, especially asthmatics, who have experienced symptoms of asthma, urticaria or acute rhinitis after taking aspirin or other non-steroidal anti-inflammatory drugs (prostaglandin-synthetase inhibitors).
- Severe heart failure
- Third term of pregnancy

4.4 Special warnings and precautions for use:

Undesirable effects may be minimised by using the lowest effective dose for the shortest duration necessary to control the symptoms (see Section 4.2 and Gastrointestinal and cardiovascular risks, hereunder).

Cardiovascular and cerebrovascular effects :

As fluid retention and oedema have been reported in association with non-steroidal anti-inflammatory drugs (NSAIDs) therapy, caution is required in patients with history of high blood pressure and/or heart failure.

Clinical studies and epidemiologic data suggest that diclofenac use, especially at high dose (150 mg daily) and prolonged use can be associated to a slight increase in arterial thrombotic events (such as cardiac infarction or stroke).

Patients with significant risk factors for cardiovascular events (e.g. hypertension, hyperlipidaemia, diabetes mellitus, smoking) should only be treated with diclofenac after careful consideration.

As the cardiovascular risks of diclofenac may increase with dose and duration of exposure, the shortest duration possible and the lowest effective daily dose should be used. The patient's need for symptomatic relief and response to therapy should be reevaluated periodically.

Gastrointestinal effects.

Gastrointestinal (GI) bleeding, ulceration or perforation have been reported with all NSAIDs at any time during treatment, with or without warning symptoms or in patients without any history of GI events. In elderly patients these events are usually more severe.

Should GI bleeding or ulceration occur in patients receiving diclofenac, treatment should be discontinued.

As with others anti-inflammatory drugs, allergic reactions including anaphylactic reactions can occur, even without prior exposure to the drug.
NSAIDs can mask the signs or symptoms of infection (due to antalgic and antipyretic effects) resulting in delayed diagnosis and treatment.
Patients with GI disorders or with a history ulcer as well as patients with ulcerous colitis, Crohn's disease, impaired hepatic function should be closely monitored.
During diclofenac treatment, increase in several hepatic enzymes levels can occur.
Hepatic function monitoring is recommended, as a preventive measure, during long term treatment. Paradym treatment should be discontinued if hepatic function tests remain abnormal or worsen, if clinical symptoms of hepatic affections occur, or in case of other signs (such as eosinophilia, rash, …). Hepatitis can appear without prodromal symptoms. Risk of hepatic toxicity significantly increases in case of chronic alcoholism. Dosage reduction is required in alcoholic patients. Special care is recommended in case of observed hepatic failure. The same recommendation is applicable for patients treated by hepatic enzymes inductors (alcohol, barbiturates and anti-epileptics). In those cases, paracetamol toxic metabolites accumulation may lead to or worse hepatic lesions.
In patient with hepatic porphyria, special care is recommended as diclofenac can induce an attack.
Due to the role of prostaglandins in maintaining renal blood flow, particular monitoring is required when diclofenac is used in patients with impaired heart, hepatic or renal function, in elderly patients, in patients treated by diuretics and in patients who have lost large extracellular volumes (for example during the peri-operative or post-operative phase of major surgical procedures). The effect is reversible upon discontinuation of the treatment.
In case of prolonged use, blood analysis, including haematocrit, transaminase levels, total proteins and serum albumin, should be performed regularly.
As with others NSAIDs, Paradym may temporarily inhibit platelets aggregation.
Special care is required in patients with haemostasis disorders.
Special attention should be paid in elderly patients, especially regarding gastro-intestinal and renal undesirable effects. It is recommended to administer the lowest effective dose, particularly in debilitated patients.
Administration of Paradym to patients with bronchial asthma should be carefully considered because of the risk of worsening symptoms.
As with others NSAIDs, increase in uraemia and creatininaemia can occur.

Prostaglandins synthesis inhibitors can modify the renal function especially if this function is already affected for example in case of sodium depletion, cardiac decompensation or severe liver affection.
As with others prostaglandins synthesis inhibitors, the following renal abnormalities can occur: glomerulonephritis, interstitial nephritis, papillary necrosis, nephrotic syndrome, acute renal failure. Paracetamol.
A frequent or time extended use is unadvised. A time extended use, unless controlled by a medical professional, can harm the health.
The maximal dose should not be exceeded. In order to prevent the risk of overdose, no other medical product containing paracetamol should be taken simultaneously.
Taking at once a dose corresponding to several times the daily dose can seriously damage the liver; there might not be any conscious loss. Despite, it is recommended to call a doctor in regard to the risk of irreversible liver damage.
Caution should be given if the following risk factors, lowering the liver toxicity threshold, are present: liver failure (including Gilbert's syndrome), acute hepatitis, kidney failure, chronic alcoholism and very meagre adults (< 50 kg). In those cases, the posology should be adapted (see 4.2).
A concomitant treatment with drugs influencing the liver function, dehydration, chronic malnutrition (low glutathione liver stock) are as well regarded as risk factors for the emergence of liver toxicity and that can lower the liver toxicity threshold. The maximal daily dose should certainly not be exceeded in these patients.
Caution should be given in case of paracetamol administration to patients with glucose6-phosphate dehydrogenase deficiency and with haemolytic anaemia.
In case of acute fever, signs of secondary infection or persistency of the complaints, the patients should be referred to the doctor.
Paracetamol administration in patients with moderate to severe renal failure may lead to accumulation of conjugated derivatives.

4.5 Interactions with other medicinal products and other interactions.

Lithium:
Diclofenac may increase serum lithium levels. In case of NSAID and lithium concomitant use, special attention to signs of lithium intoxication should be paid and serum lithium levels should be closely monitored.

Digoxin:
Diclofenac may increase plasma levels of digoxin. Plasma digoxin levels should be monitored when initiating Paradym (diclofenac) and when discontinuing treatment, since adjustment of the dose may be necessary.

Diuretics:
Diclofenac can lead to sodium retention, oedema and to a decrease in antihypertensive and diuretic treatment. Concomitant treatment with

potassium-sparing diuretic can lead to hyperkalaemia. Kalaemia should therefore be closely monitored.

Nsaids:
Concomitant administration of several nsaids (including salicylates and pyrazole compounds) should in general be avoided due to potential effect on bioavailabilty of these drugs, hence reduced activities, and to the increased risk of undesirable effects.
Anticoagulants: during clinical trials, diclofenac did not interfere with anticoagulants but isolated reports showed an increased risk of haemorrhage when diclofenac and anticoagulants are concomitantly administered. Close monitoring is therefore required. As with other nsaids, elevated doses of diclofenac can temporarily inhibit platelets aggregation.
As paracetamol is poorly linked to plasmatic proteins, the concomitant use of paracetamol and oral anticoagulants is allowed. However, concomitant use of paracetamol (at more than 2 g daily during a long period) with oral anticoagulants may lead to slight variations in INR values. In this case a regular monitoring of INR values is recommended.

Oral antidiabetics:
Clinical trials have shown that diclofenac does not influence the effect of antidiabetic agents, although there have been isolated reports of hypoglycaemia and hyperglycaemia that have required dose adjustment.
Methotrexate: caution should be exercised when nsaids are administered less than 24 hours before or after a methotrexate treatment because its plasma level and toxicity can be increased.

Cyclosporin:
Nsaids effects on renal prostaglandins may increase cyclosporin cytotoxicity.

Quinolones:
Isolated cases of convulsions have been reported during concomitant use of quinolones and nsaids.

Corticosteroids:
Concomitant use of diclofenac and corticosteroids can increase the risk of gastrointestinal adverse reactions.
Antihypertensive agents: as with diuretics, sodium retention may occur during treatment with nsaids, hence a decrease in the antihypertensive effect. The same interaction also exists with ACE inhibitors. Paracetamol is fully metabolized in the liver. Some of its metabolites are toxic to the liver, a concomitant administration of potent enzymes inducers (rifampicin, certain anti-convulsants) can lead to liver-toxic reactions, especially with high doses of paracetamol.

Metoclopramide:
Paracetamol absorption can be increased when associated with metoclopramide.

Chloramphenicol:
Paracetamol increases chloramphenicol clearance.

Colestyramine:
Colestyramine may decrease the intestinal absorption of paracetamol. While using concomitantly paracetamol and colestyramine, paracetamol should be administered 1 hour prior or 4 hours after the administration of colestyramine.

Probenecid:
Probenecid can decrease by almost half the clearance of paracetamol by the inhibition the conjugation with glucuronic acid. A reduction in the dose of paracetamol should therefore be considered if concomitant treatment with probenecid.

Zidovudine:
Concomitant administration of paracetamol and zidovudine can lead to neutropenia and liver toxicity. The chronic/frequent use of paracetamol in patients treated with zidovudine should be avoided. If required, white blood cells and liver function should be monitored, especially in undernourished patients.

Vitamin K antagonists:
A stronger effect of the vitamin K antagonists can arise, especially if paracetamol is taken often and in high doses. In this case, a frequent monitoring of the International Normalised Ratio (INR) is recommended.
Lamotrigine: a decreased bioavailability of lamotrigine, with possible reduced therapeutic effect can appear because of likely induction in the metabolism of lamotrigine by paracetamol.

Metoclopramide and domperidone:
Accelerated intestinal resorption of paracetamol can arise due to the accelerated stomach emptying.
Diagnosis tests: paracetamol can interfere with the determination of blood uric acid by the phosphotungstic acid method and with the determination of blood glucose by the glucose oxydase-peroxydase method.

4.6 Fertility,Pregnancy and lactation:

Fertility

As with other NSAIDs, the use of diclofenac may impair female fertility and is not recommended in women attempting to conceive. In women

who have difficulties conceiving or who are undergoing investigation of infertility, withdrawal of diclofenac should be considered.

Pregnancy

During pregnancy, Paradym should not be given unless clearly necessary and only at the lowest effective doses. As with others prostaglandins synthesis inhibitors, this rule should particularly be followed during the third trimester of pregnancy due to inhibition of uterine contractions resulting in delayed or prolonged labour and/or premature closure of the ductus arteriosus. Hemorrhagic risk is also increased in case of NSAIDs use at the end of pregnancy.

Lactation

At 150 mg daily dose (50 mg every 8 hours), diclofenac is excreted in mother's milk at very low level. It is unlikely that children be affected by therapeutic doses used by their mothers. Paracetamol can be administered during breastfeeding. Therefore, Paradym can be prescribed to breastfeeding mothers.

4.7 Effects on ability to drive and use machines

Vertigo or central nervous system effects can occur during treatment with Paradym. If these effects appear while using Paradym, driving a car and using machines are not recommended.

4.8 Undesirable effects

The following frequencies are used for the description of the occurrence of adverse reactions:
very common (>1/10)
common (>1/100, 1/1000, 1/10,000, < 50 kg).

Diclofenac Potassium

Blood and lymphatic system disorders	
Very rare	Thrombocytopenia, leucopoenia, anaemia (including haemolytic and aplastic anaemia), agranulocytosis.
Immune system disorders	
Rare Very rare	Hypersensitivity, anaphylactic and anaphylactoid reactions (including hypotension and shock). Angioneurotic oedema (including face oedema).
Psychiatric disorders	
Very rare	Disorientation, depression, insomnia, nightmare, irritability, psychotic disorder.
Nervous system disorders	
Common Rare Very rare Unknown	Headache, dizziness. Somnolence, tiredness. Paraesthesia, memory impairment, convulsion, anxiety, tremor, aseptic meningitis, taste disturbances, cerebrovascular accident. Confusion, hallucinations, disturbances of sensation,

	malaise.
Eye disorders	
Very rare Unknown	Visual disturbance, vision blurred, diplopia. Optic neuritis.
Ear and labyrinth disorders	
Common Very rare	Vertigo. Tinnitus, hearing impaired.
Cardiac disorders	
Uncommon*	Myocardial infarction, cardiac failure, palpitations, chest pain.
Unknown	Kounis syndrome
Vascular disorders	
Very rare	Hypertension, hypotension, vasculitis.
Respiratory, thoracic and mediastinal disorders	
Rare Very rare	Asthma (including dyspnoea). Pneumonitis.
Gastrointestinal disorders	
Common Rare Very rare Unknown	Nausea, vomiting, diarrhoea, dyspepsia, abdominal pain, flatulence, anorexia. Gastritis, gastrointestinal haemorrhage, haematemesis, diarrhoea haemorrhagic, melaena, gastrointestinal ulcer with or without bleeding or perforation (sometimes fatal particularly in the elderly). Colitis (including haemorrhagic colitis and exacerbation of ulcerative colitis or Crohn's disease), constipation, stomatitis (including ulcerative stomatitis), glossitis, oesophageal disorder, diaphragm-like intestinal strictures, pancreatitis. Ischaemic colitis
Hepatobiliary disorders	
Common Rare Very rare	Transaminases increased. Hepatitis, jaundice, liver disorder. Fulminant hepatitis, hepatic necrosis, hepatic failure.
Skin and subcutaneous tissue disorders	
Common Rare Very rare	Rash. Urticaria. Bullous eruptions, eczema, erythema, erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis (Lyell's syndrome), dermatitis exfoliative, loss of hair, photosensitivity reaction, purpura , allergic purpura, pruritus.
Renal and urinary disorders	
Very rare	Acute renal failure, haematuria, proteinuria, nephrotic syndrome, interstitial nephritis, renal papillary necrosis.
Reproductive system and breast disorders	
Very rare	Impotence
General disorders and administration site conditions	

Rare	Oedema

Reporting of suspected adverse reactions: Healthcare professionals are asked to report any suspected adverse reactions ND PQMPs to https://pv.pharmacyboardkenya.org

4.9 Overdose

Symptoms Diclofenac overdose

There is no typical clinical picture resulting from diclofenac over dosage. Over dosage can cause symptoms such as headache, nausea, vomiting, epigastric pain, gastrointestinal haemorrhage, diarrhoea, dizziness, disorientation, excitation, coma, drowsiness, tinnitus, fainting or convulsions. In the case of significant poisoning acute renal failure and liver damage are possible.

Therapeutic measures: Management of acute poisoning with NSAIDs, including diclofenac, essentially consists of supportive measures and symptomatic treatment. Supportive measures and symptomatic treatment should be given for complications such as hypotension, renal failure, convulsions, gastrointestinal disorder, and respiratory depression.

Special measures such as forced diuresis, dialysis or haemo-perfusion are probably of no help in eliminating NSAIDs, including diclofenac, due to the high protein binding and extensive metabolism.

Activated charcoal may be considered after ingestion of a potentially toxic overdose, and gastric decontamination (e.g. vomiting, gastric lavage) after ingestion of a potentially life threatening overdose

Paracetamol Overdose

It has to be kept in mind that a massive overdose with a glutathione depletion exceeding 70% (which theoretically requires that an adult absorb 15 g paracetamol and a child a dose equal or higher than 150 mg/kg body weight) leads to an increased quantity of reactive metabolite which, as it cannot be detoxified, causes hepatic cytolysis potentially leading to a complete and irreversible necrosis. Paracetamol accumulation due to metabolism impairment has not been observed at therapeutic doses. Glutathione depletion, which could increase the toxicity risk, does not usually occur.

Symptoms: Early symptoms, that can occur only 12 hours after ingesting a potentially toxic dose, might include: nausea, vomiting, anorexia, abdominal pain and sweating. Clinical and biological proofs of liver disorder can appear later (48 to 72 hours). As a consequence, in case of any suspicion of paracetamol overdose, the patient should be

immediately hospitalized and serum levels should be determined at the earliest from the 4th hour post-ingestion on. Values exceeding 200 µg/ml at the 4th hour or 50 µg/ml at the 12th hour let suspect a high risk of hepatic necrosis. The usual liver function tests should be performed early and regularly repeated (every 24 hours). Treatment: The overdose treatment in a specialized environment includes administering at the earliest the N-acetylcysteine antidote. Early treatment can result in a total functional recovery. N-acetylcysteine proposed posology: initial dose 150 mg/kg in 30 minutes, then 50 mg/kg in 4 hours and 100 mg/kg during the following 16 hours. A close monitoring of hepatic function is recommended (every 24 hours).

5. Pharmacological properties

In postoperative pain treatment, the doses of morphinic analgesics can be significantly reduced when Paradym is associated to the treatment.

5.1. Pharmacodynamic properties

Paradym contains the following active substances: diclofenac and paracetamol.

Diclofenac is a non-steroidal anti-inflammatory drug (NSAID) with analgesic and antipyretic properties. Inhibition of prostaglandin synthesis has been shown experimentally to be an important component of the mechanism of action. Prostaglandins synthesis reduction induces the following effects: -

Inflammatory symptoms reduction by partial suppression of one of the main mediators of inflammation.

Pain attenuation (by decrease of prostaglandins production, involved in sensitization of nociceptors to inflammatory mediators such as bradykinin).

Dysmenorrhoea pain relief (dysmenorrhoea is due to increase in uterine activity and high prostaglandins levels in menstrual blood).

Decrease in fever (prostaglandins acts on hypothalamic centre and thermoregulation)

Diclofenac exhibits anti-inflammatory and analgesic properties in clinical signs of rheumatic diseases, as relief of symptoms such as pain at rest or in motion, early morning stiffness and swollen joints. These properties are also manifested as improvement in function. Diclofenac has been shown in clinical trials to significantly relieve non rheumatic moderate to severe pain. Diclofenac relieves pain and reduces blood volumes in primary dysmenorrhoea.

In posttraumatic and postoperative pain, diclofenac relieves spontaneous pain, movement induced pain and reduces oedema.

Paracetamol is an antalgic and antipyretic. It might exercise its peripheral analgesic activity by elevating the pain sensation thresholds.

Its antipyretic activity might be due to an action on the hypothalamic centres.

5.2 Pharmacokinetic properties

Absorption

Diclofenac is rapidly and completely absorbed from diclofenac tablets. Absorption started immediately following the administration. Food intake does not affect the quantity absorbed but can delay and reduce the absorption.

Both oral and rectal paracetamol are rapidly and totally absorbed; however rectal resorption may change according to exposure time with rectal membrane.

Peak plasma concentration from diclofenac tablets is reached within 2 hours.

Distribution:

Diclofenac plasma concentrations are dose proportional. Almost half of diclofenac is metabolized by the liver (first –pass metabolism), in comparaison to parenteral administration, 50% of diclofenac reaches the systemic circulation after oral administration. The pharmacokinetic is not affected by repeated doses. Repeated diclofenac oral administration, at recommended interval dose, does not lead to accumulation of diclofenac in plasma.

Diclofenac is highly bound to plasma proteins (99.7%), mainly to albumin (99.4%). This should be taken into consideration when others plasma proteins highly bounded drugs are coadministered.

Distribution volume is about 0.12 to 0.17 l/kg.

Diclofenac penetrates synovial fluids where the concentrations are maximal 2 – 4 hours after peak plasma concentration. The concentration in synovial fluid remains higher than in plasma for a 12 hours period. Half life in synovial fluid is about 3 – 6 hours.

Paracetamol is weakly bound to plasmatic proteins (20 to 50%) and its diffusion is rapid. Metabolism and elimination:

Metabolism

The biotransformation of diclofenac is partly performed by glucuronoconjugation of the intact molecule but mainly by single and multiple hydroxylation and methoxylation which lead to different phenol metabolites eliminated by glucuronoconjugation. Two of those phenol metabolites are active but significantly less active than diclofenac. The total systemic clearance of diclofenac in plasma is 263 ± 56 ml/min (mean ± SD). The terminal half-life in plasma is 1-2 hours. Four metabolites, two of which are active, have also a brief terminal half-life in plasma (1-3 hours). Another metabolite, inactive, has a long terminal

half-life in plasma. Approximately 60% of the dose administered is excreted in the urine in the form of glucuronoconjugates of unchanged diclofenac or of its metabolites, and less than 1% as unchanged substance. The remainder of the dose is eliminated as metabolites through the bile in faeces.
Paracetamol is metabolised in the liver and follows two major metabolic routes. It is excreted via the urine under glucuronoconjugated (60 to 80 %) and sulfoconjugated (20 to 40%) forms. A small fraction (less than 4%) is transformed with the intervention of cytochrome P450 into a metabolite formed by oxidative process and which would have been involved in the hepatotoxicity of paracetamol at high doses; indeed, at therapeutic doses, this metabolite is eliminated by conjugation with glutathione. The conjugation ability is not changed in elderly patients and the kinetics is linear for doses until 7 g. In case of massive intoxication, the conjugation ability is exceeded, and the hepatotoxic metabolite quantity is increased. At therapeutic doses, the paracetamol half-life is about 3 hours.

5.3 Preclinical safety data

Diclofenac

Preclinical data from acute and repeated dose toxicity studies, as well as from genotoxicity, mutagenicity, and carcinogenicity studies with diclofenac revealed no specific hazard for humans at the intended therapeutic doses. In standard preclinical animal studies, there was no evidence that diclofenac had a teratogenic potential in mice, rats or rabbits.

Diclofenac had no influence on the fertility of parent animals in rats. Except for minimal fetal effects at maternally toxic doses the prenatal, perinatal, and postnatal development of the offspring was not affected.

Administration of NSAIDs (including diclofenac) inhibited ovulation in the rabbit and implantation and placentation in the rat, and led to premature closure of the ductus arteriosus in the pregnant rat. Maternally toxic doses of diclofenac were associated with dystocia, prolonged gestation, decreased fetal survival, and intrauterine growth retardation in rats. The slight effects of diclofenac on reproduction parameters and delivery as well as constriction of the ductus arteriosus in utero are pharmacologic consequences of this class of prostaglandin synthesis inhibitors.

Paracetamol

Conventional studies using the currently accepted standards for the evaluation of toxicity to reproduction and development are not available.

6. Pharmaceutical particulars

6.1 List of excipients

Microcrystalline cellulose
Dibasic Calcium Phosphate
Maize starch
Colour Erythrosine Supra
Methyl Hydroxybenzoate
Propyl Hydroxybenzoate
Povidone (PVP K-30)
Sodium starch glycolate
Hydrophobic Colloidal Anhydrous Silica
Purified Talcum
Magnesium Stearate
Purified water

6.2 Incompatibilities:

Not known

6.3 Shelf life

36 months

6.4 Special precautions for storage:

Store below 30 ^{o}C.
Store in the original packaging away from heat, light and moisture.

6.5 Nature and contents of container:

10 tablets packed in a blister strip.10 such blisters are packed in a unit carton with a packaging insert. (10 X 10 Tablets)

6.6 Special precautions for disposal and other handling:

Any unused product or waste material should be disposed of in accordance with local requirements.

7. Marketing authorization holder and manufacturing site addresses:

Marketing authorization holder:
Next Wave (India),
SCO No. 313, Second Floor, Sector 29,
Gurugram, 122009, Haryana,
India.

Manufacturing site address:
Next wave (India)
Rampur Ghat Road, Paonta Sahib Distt.
Sirmour, H.P.-173025

India.

8. Marketing authorization number
CTD9438

9. Date of first registration
02/08/2023

10. Date of revision of the text:
16/09/2023

11. Dosimetry:
Not Applicable

12. Instructions for Preparation of Radiopharmaceuticals:
Not Applicable