SUMMARY OF PRODUCT CHARACTERISTICS

1. Name of the medicinal product

Monokast Tablets 5mg

Monokast Tablets 10mg

2. Qualitative and quantitative composition

Monokast Tablets 5mg; Each Film coated tablet contains: Montelukast Sodium Equivalent to Montelukast 5 mg

Excipient(s) with known effect 50.00 mg of lactose.

Monokast Tablets 10mg;Each Film coated tablet contains: Montelukast Sodium Equivalent to Montelukast 10 mg

Excipient(s) with known effect 50.00 mg of lactose.

For a full list of excipients, see section 6.1.

3. Pharmaceutical form

Monokast Tablets 5mg

Film coated Tablet

Brown colored, Triangular, biconvex film coated tablets.

Monokast Tablets 10mg

Film Coated Tablet

Yellow colored, round, biconvex film coated tablets.

4. Clinical particulars

MONTELUKAST is indicated in the treatment of asthma as add-on therapy in those patients with mild to moderate persistent asthma who are inadequately controlled on inhaled corticosteroids and in whom “asneeded” short acting β-agonists provide inadequate clinical control of asthma. In those asthmatic patients in whom MONTELUKAST is indicated in asthma, MONTELUKAST can also provide symptomatic relief of seasonal allergic rhinitis.

MONTELUKAST is also indicated in the prophylaxis of asthma in which the predominant component is exercise-induced bronchoconstriction.

4.1. Therapeutic indications

MONTELUKAST 10 mg Film-coated Tablets are indicated in adults and children 15 years of age and older for the prophylaxis and chronic treatment of atopic asthma.

In those adult asthmatic patients, in whom MONTELUKAST is indicated in asthma, MONTELUKAST may also provide some symptomatic relief of seasonal allergic rhinitis.

MONTELUKAST 5 mg Film-coated tablet Tablets are indicated in paediatric patients over 6 years -14 years of age for the prophylaxis and chronic treatment of atopic asthma.

4.2. Posology and method of administration

MONTELUKAST should be taken once daily in the evening. MONTELUKAST can be taken with or without food.

MONTELUKAST 10 mg Film-coated Tablets

Adults and Children 15 years of age and older with atopic asthma

One 10 mg film-coated tablet daily. Clinical studies in adults 15 years of age and older did not demonstrate additional clinical benefit to montelukast doses above 10 mg once daily.

MONTELUKAST 5 mg Film Coated Tablets

Paediatric patients 6 to 14 years of age with atopic asthma

One 5 mg tablet daily.

General recommendations;

A therapeutic effect of MONTELUKAST on parameters of asthma control occurs within one day.

General recommendations:

The therapeutic effect of MONTELUKAST on parameters of asthma control occurs within one day. MONTELUKAST may be taken with or without food. Patients should be advised to continue taking MONTELUKAST even if their asthma is under control, as well as during periods of worsening asthma. MONTELUKAST should not be used concomitantly with other products containing the same active ingredient, montelukast.

No dosage adjustment is necessary for the elderly, or for patients with renal insufficiency, or mild to moderate hepatic impairment. There are no data on patients with severe hepatic impairment. Thedosage is the same for both male and female patients.

Therapy with MONTELUKAST in relation to other treatments for asthma

MONTELUKAST can be added to a patient's existing treatment regimen. Inhaled corticosteroids:

Treatment with MONTELUKAST can be used as add-on therapy in patients when inhaled corticosteroids plus "as needed" short acting βagonists provide inadequate clinical control. MONTELUKAST should notbe abruptly substituted for inhaled corticosteroids (see section 4.4). Paediatric population

Do not give MONTELUKAST 10mg to children less than 15 years of age. The safety and efficacy of MONTELUKAST 10mg in children less than 15 years has not been established.

5 mg tablets are available for paediatric patients 6 to 14 years of age

Method of administration: Oral use.

4.3. Contraindications

Hypersensitivity to the active substance or to any of the excipients listed in section 6.1

4.4. Special warnings and precautions for use

Patients should be advised never to use oral montelukast to treat acute asthma attacks and to keep their usual appropriate rescue medication for this purpose readily available. If an acute attack occurs, a shortacting inhaled β-agonist should be used. Patients should seek their doctor's advice as soon as possible if they need more inhalations of short-acting β-agonists than usual.

Montelukast should not be substituted abruptly for inhaled or oral corticosteroids.

There are no data demonstrating that oral corticosteroids can be reduced when montelukast is givenconcomitantly.

In rare cases, patients on therapy with anti-asthma agents including montelukast may present with systemic eosinophilia, sometimes presenting with clinical features of vasculitis consistent with ChurgStrauss syndrome, a condition which is often treated with systemic corticosteroid therapy. These cases have been sometimes associated with the reduction or withdrawal of oral corticosteroid therapy. Although a causal relationship with leukotriene receptor antagonism has not been established, physicians should be alert to eosinophilia, vasculitic rash, worsening pulmonary symptoms, cardiac complications, and/or neuropathy presenting in their patients. Patients who develop these symptoms should be reassessed and their treatment regimens evaluated.

Treatment with montelukast does not alter the need for patients with aspirin-sensitive asthma toavoid taking aspirin and other non-steroidal anti-inflammatory drugs.

This medicinal product contains lactose monohydrate.

Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency orglucose-galactose malabsorption should not take this medicine.

Neuropsychiatric events have been reported in adults, adolescents, and children taking MONTELUKAST (see section 4.8). Patients and physicians should be alert for neuropsychiatric events. Patients and/or caregivers should be instructed to notify their physician if these changes occur. Prescribers should carefully evaluate the risks and benefits of continuing treatment with MONTELUKAST if such events occur.

4.5. Interaction with other medicinal products and other forms of interaction

Montelukast may be administered with other therapies routinely used in the prophylaxis and chronic treatment of asthma. In druginteractions studies, the recommended clinical dose of montelukast did not have clinically important effects on the pharmacokinetics of the following medicinal products: theophylline, prednisone, prednisolone, oral contraceptives (ethinyl estradiol/ norethindrone 35/1), terfenadine, digoxin and warfarin.

The area under the plasma concentration curve (AUC) for montelukast was decreased approximately 40% in subjects with co-administration of phenobarbital. Since montelukast is metabolised by CYP 3A4, 2C8, and 2C9, caution should be exercised, particularly in children, when montelukast is co-administered with inducers of CYP 3A4, 2C8, and 2C9, such as phenytoin,phenobarbital and rifampicin.

In vitro studies have shown that montelukast is a potent inhibitor of CYP 2C8. However, data from a clinical drug-drug interaction study involving montelukast and rosiglitazone (a probe substrate representative of medicinal products primarily metabolized by CYP 2C8) demonstrated that montelukast does not inhibit CYP 2C8 in vivo. Therefore, montelukast is not anticipated to markedly alter the metabolism of medicinal products metabolised by this enzyme (e.g., paclitaxel, rosiglitazone, and repaglinide.)

In vitro studies have shown that montelukast is a substrate of CYP 2C8, and to a less significant extent, of 2C9, and 3A4. In a clinical drug-drug interaction study involving montelukast and gemfibrozil (an inhibitor of both CYP 2C8 and 2C9) gemfibrozil increased the systemic exposure of montelukast by 4.4-fold. No routine dosage adjustment of montelukast is required upon co-administration with gemfibrozil or other potent inhibitors of CYP 2C8, but the physician should be aware of the potential for an increase in adverse reactions.

Based on in vitro data, clinically important drug interactions with less potent inhibitors of CYP 2C8 (e.g., trimethoprim) are not anticipated. Co-administration of montelukast with itraconazole, a strong inhibitor of CYP 3A4, resulted in no significant increase in the systemic exposure of montelukast.

4.6 Fertility, pregnancy, and lactation

Pregnancy

Animal studies do not indicate harmful effects with respect to effects on pregnancy orembryonal/foetal development.

Available data from published prospective and retrospective cohort studies with montelukast use inpregnant women evaluating major birth defects have not established a drug-associated risk.

Available studies have methodologic limitations, including small sample size, in some cases retrospective data collection, and inconsistent comparator groups.

MONTELUKAST may be used during pregnancy only if it is considered to be clearly essential.

Breastfeeding

It is unknown whether montelukast is excreted in human milk. Studies in rats have shown thatmontelukast is excreted in milk (see section 5.3).

MONTELUKAST may be used in breast-feeding only if it is considered to be clearly essential

4.7. Effects on ability to drive and use machines.

Montelukast has no or negligible influence on the ability to drive and use machines. However, individuals have reported drowsiness or dizziness.

4.8. Undesirable effects

Montelukast has been evaluated in clinical studies as follows:

Body System Class	**Adult and Adolescent Patients 15 years and older (two 12- week studies; n = 795)**	**Paediatric Patients 6 to 14 years old (one 8-week study; n=201) (two 56-week studies; n=615)**
Nervous system disorders	headache	headache
Gastrointestin al disorders	abdominal pain	

• 10 mg film-coated tablets in approximately 4000 adult and adolescent asthmatic patients 15 years of age and older.

• 10 mg film-coated tablets in approximately 400 adult and adolescent asthmatic patients with seasonal allergic rhinitis 15 years of age and older.

• 5 mg tablets in approximately 1750 paediatric asthmatic patients 6 to 14 years of age. The following drug-related adverse reactions in clinical studies were reported commonly (1/100 to<1/10) in asthmatic patients treated with montelukast and at a greater incidence than in patients treated with placebo:

With prolonged treatment in clinical trials with a limited number of patients for up to 2 years for adults, and up to 12 months for paediatric patients and adolescents 6 to 14 years of age, the safety profile did not change.

Tabulated list of Adverse Reactions

Adverse reactions reported in post-marketing use are listed, by System Organ Class and specific Adverse Experience Term, in the table below. Frequency Categories were estimated based on relevant clinical trials.

System Organ Class	Adverse Reactions	Frequency Category*
Infections and infestations	upper respiratory infection†	Very Common
Blood and lymphatic system disorders	increased bleeding tendency	Rare
	thrombocytopenia	Very Rare
Immune system disorders	hypersensitivity reactions including anaphylaxis	Uncommon

	hepatic eosinophilic infiltration	Very Rare
Psychiatric disorders	dream abnormalities including nightmares, insomnia, somnambulism, anxiety, agitation including aggressive behaviour or hostility, depression, psychomotor hyperactivity (including irritability, restlessness, tremor§)	Uncommon
	disturbance in attention, memory impairment, tic	Rare
	hallucinations, disorientation, suicidal thinking and behaviour (suicidality), obsessive-compulsive symptoms, dysphemia	Very Rare
Nervous system disorders	dizziness, drowsiness paraesthesia/hypoesthesia, seizure	Uncommon
Cardiac disorders	palpitations	Rare
Respiratory, thoracic and mediastinal disorders	epistaxis	Uncommon
	Churg-Strauss Syndrome (CSS) (see section 4.4)	Very Rare
	pulmonary eosinophilia	Very Rare
Gastrointestinal disorders	diarrhoea‡, nausea‡, vomiting‡	Common
	dry mouth, dyspepsia	Uncommon
Hepatobiliary disorders	elevated levels of serum transaminases (ALT, AST)	Common
	hepatitis (including cholestatic, hepatocellular, and mixed-pattern liver injury).	Very Rare
Skin and subcutaneous tissue disorders	rash‡	Common
	bruising, urticaria, pruritus	Uncommon
	angiooedema	Rare
	erythema nodosum, erythema multiforme	Very Rare
Musculoskeletal and connective tissue disorders	arthralgia, myalgia including muscle cramps	Uncommon
Renal and urinary disorders	enuresis in children	Uncommon
General disorders and administration site conditions	pyrexia‡	Common
	asthenia/fatigue, malaise, oedema	Uncommon

*Frequency Category: Defined for each Adverse Reactions by the incidence reported in the clinical trials data base: Very Common (≥1/10), Common (≥1/100 to <1/10), Uncommon (≥1/1000 to

<1/100), Rare (≥1/10,000 to <1/1000), Very Rare (<1/10,000).

† This adverse experience, reported as Very Common in the patients who received montelukast, was also reported as Very Common in the patients who received placebo in clinical trials.

‡ This adverse experience, reported as Common in the patients who received montelukast, was also reported as Common in the patients who received placebo in clinical trials.

§ Frequency Category: Rare

Reporting of suspected adverse reactions: Healthcare professionals are asked to report any suspected adverse reactions via pharmacy and poisons board, Pharmacovigilance Electronic Reporting System (PvERS)

https://pv.pharmacyboardkenya.org

4.9. Overdose

In chronic asthma studies, montelukast has been administered at doses up to 200 mg/day to adult patients for 22 weeks and in shortterm studies, up to 900 mg/day to patients for approximately one week without clinically important adverse experiences.

There have been reports of acute overdose in post-marketing experience and clinical studies with montelukast. These include reports in adults and children with a dose as high as 1000 mg (approximately 61 mg/kg in a 42 month old child). The clinical and laboratory findings observed were consistent with the safety profile in adults and paediatric patients. There were no adverse experiences in the majority of overdose reports Symptoms of overdose

The most frequently occurring adverse experiences were consistent with the safety profile of montelukast and included abdominal pain, somnolence, thirst, headache, vomiting, and psychomotor hyperactivity..

Management of overdose No specific information is available on the treatment of overdose with montelukast. It is not known whether montelukast is dialysable by peritoneal- or haemo-dialysis.

5. Pharmacological properties

5.1. Pharmacodynamic properties

Pharmacotherapeutic group: Leukotriene receptor antagonist, **ATC Code:** RO3D CO3

Mechanism of action

The cysteinyl leukotrienes (LTC , LTD , LTE) are potent inflammatory 4 4 4 eicosanoids released from various cells including mast cells and eosinophils. These important pro-asthmatic mediators bind tocysteinyl leukotriene (CysLT) receptors. The CysLT type-1 (CysLT) receptor is found in the human airway (including airway smooth muscle cells and airway macrophages) and on other pro- inflammatory cells (including eosinophils and certain myeloid stem cells). CysLTs have been correlated with the pathophysiology of asthma and allergic rhinitis. In asthma, leukotriene-mediated effects include bronchoconstriction, mucous secretion, vascular permeability, and eosinophil recruitment. In allergic rhinitis, CysLTs are released from the nasal mucosa after allergen exposure during both early- and late-phase reactions and are associated with symptoms of allergic rhinitis.

Intranasal challenge with CysLTs has been shown to increase nasal airway resistance andsymptoms of nasal obstruction.

Pharmacodynamic effects

Montelukast is an orally active compound which binds with high affinity and selectivity to theCysLT receptor. In clinical studies, montelukast inhibits bronchoconstriction due to inhaled

LTD at doses as low as 5 mg. Bronchodilation was observed within 2 hours of oral administration.

The bronchodilation effect caused by a β agonist was additive to that caused by montelukast. Treatment with montelukast inhibited both early- and late phase bronchoconstriction due to antigen challenge. Montelukast, compared with placebo, decreased peripheral blood eosinophils in adult and paediatric patients. In a separate study, treatment with montelukast significantly decreased eosinophils in the airways (as measured in sputum) and in peripheral blood while improving clinical asthma control.

Clinical efficacy and safety

In studies in adults, montelukast 10 mg once daily, compared with placebo, demonstrated significant improvements in morning FEV1 (10.4% vs 2.7% change from baseline), AM peak expiratory flow rate (PEFR) (24.5 L/min vs 3.3 L/min change from baseline), and significant decrease in total β-agonist use (-26.1% vs -4.6% change from baseline). Improvement in patient- reported daytime and night-time asthma symptoms scores was significantly better than placebo. Studies in adults demonstrated the ability of montelukast to add to the clinical effect of inhaled corticosteroid (% change from baseline for inhaled beclomethasone plus montelukast vs beclomethasone, respectively for FEV1 : 5.43% vs 1.04%; β-agonist use: -8.70% vs 2.64%).

Compared with inhaled beclomethasone (200 µg twice daily with a spacer device), montelukast demonstrated a more rapid initial response, although over the 12-week study, beclomethasone provided a greater average treatment effect (% change from baseline for montelukast vs beclomethasone, respectively for

FEV1 : 7.49% vs 13.3%; β-agonist use: -28.28% vs -43.89%). However, compared with beclomethasone, a high percentage of patients treated with montelukast achieved similar clinical responses (e.g. 50% of patients treated with beclomethasone achieved an improvement in FEV1 of approximately 11% or more over baseline while approximately 42% of patients treated with montelukast achieved the same response).

In an 8-week study in paediatric patients 6 to 14 years of age, montelukast 5 mg once daily, compared with placebo, significantly improved respiratory function (FEV1 8.71% vs 4.16% change from baseline; AM PEFR 27.9 L/min vs 17.8 L/min change from baseline) and decreased 'as- needed' β-agonist use (-11.7% vs +8.2% change from baseline).

In a 12-month study comparing the efficacy of montelukast to inhaled fluticasone on asthma control in paediatric patients 6 to 14 years of age with mild persistent asthma, montelukast was non-inferior to fluticasone in increasing the percentage of asthma rescue-free days (RFDs), the primary endpoint. Averaged over the 12-month treatment period, the percentage of asthma RFDs increased from 61.6 to 84.0 in the montelukast group and from 60.9 to 86.7 in the fluticasone group. The between group difference in LS mean increase in the percentage of asthma RFDs was statistically significant (-2.8 with a 95% CI of -4.7, - 0.9), but within the limit pre-defined to be clinically not inferior. Both montelukast and fluticasone also improved asthma control on secondary variables assessed over the 12 month treatment period:

• FEV1 increased from 1.83 L to 2.09 L in the montelukast group and from 1.85 L to 2.14 L in the fluticasone group. The between-group difference in LS mean increase in FEV1 was -0.02 L with a 95% CI of -0.06, 0.02. The mean increase from baseline in % predicted FEV1was 0.6% in the montelukast treatment group, and 2.7% in the fluticasone treatment group.

• The difference in LS means for the change from baseline in the % predicted FEV1 was significant:- 2.2% with a 95% CI of -3.6, -0.7.

• The percentage of days with β-agonist use decreased from 38.0 to

15.4 in the montelukast group, and from 38.5 to 12.8 in the fluticasone group. The between group difference in LS means for the percentage of days with β-agonist use was significant:2.7 with a 95% CI of 0.9, 4.5.

• The percentage of patients with an asthma attack (an asthma attack being defined as a period of worsening asthma that required treatment with oral steroids, an unscheduled visit to the doctor's office, an emergency room visit, or hospitalisation) was 32.2 in the montelukast group and 25.6 in the fluticasone group; the odds ratio (95% CI) being significant: equal to 1.38 (1.04, 1.84).

• The percentage of patients with systemic (mainly oral) corticosteroid use during the study period was 17.8% in the montelukast group and 10.5% in the

fluticasone group. The between group difference in LS means was significant: 7.3% with a 95% CI of 2.9; 11.7.

Significant reduction of exercise-induced bronchoconstriction (EIB) was demonstrated in a 12- week study in adults (maximal fall in FEV1 22.33% for montelukast vs 32.40% for placebo; time to recovery to within 5% of baseline FEV1 44.22 min vs 60.64 min). This effect was consistent throughout the 12-week study period. Reduction in EIB was also demonstrated in a short term study in paediatric patients (maximal fall in FEV1 18.27% vs 26.11%; time to recovery to within 5% of baseline FEV1 17.76 min vs 27.98 min). The effect in both studies was demonstrated at the end of the once-daily dosing interval.

In aspirin-sensitive asthmatic patients receiving concomitant inhaled and/or oral corticosteroids, treatment with montelukast, compared with placebo, resulted in significant improvement in asthma control (FEV1 8.55% vs -1.74% change from baseline and decrease in total βagonist use -27.78% vs 2.09% change from baseline).

5.2. Pharmacokinetic properties

Absorption

Montelukast is rapidly absorbed following oral administration. For the 10 mg film-coated tablet, themean peak plasma concentration (Cmax) is achieved 3 hours (Tmax) after administration in adults in the fasted state. The mean oral bioavailability is 64%. The oral bioavailability and Cmax are not influenced by a standard meal. Safety and efficacy were demonstrated in clinical trials where the 10mg film-coated tablet was administered without regard to the timing of food ingestion.

For the 5 mg tablet, the Cmax is achieved in 2 hours after administration in adults in the fasted state. The mean oral bioavailability is 73% and is decreased to 63% by a standard meal.

Distribution

Montelukast is more than 99% bound to plasma proteins. The steadystate volume of distribution ofmontelukast averages 8-11 litres. Studies in rats with radiolabelled montelukast indicate minimal distribution across the blood-brain barrier. In addition, concentrations of radiolabelled material at 24 hours post-dose were minimal in all other tissues.

Biotransformation

Montelukast is extensively metabolised. In studies with therapeutic doses, plasma concentrations of metabolites of montelukast are undetectable at steady state in adults and children.

Cytochrome P450 2C8 is the major enzyme in the metabolism of montelukast. Additionally CYP 3A4 and 2C9 may have a minor contribution, although itraconazole, an inhibitor of CYP 3A4, wasshown not to change pharmacokinetic variables of montelukast in healthy subjects that received

10mg montelukast daily. Based on *in vitro* results in human liver microsomes, therapeutic plasma concentrations of montelukast do not inhibit cytochromes P450 3A4, 2C9, 1A2, 2A6, 2C19, or 2D6. The contribution of metabolites to the therapeutic effect of montelukast is minimal.

Elimination

The plasma clearance of montelukast averages 45 ml/min in healthy adults. Following an oral doseof radiolabelled montelukast, 86% of the radioactivity was recovered in 5-day faecal collections and <0.2% was recovered in urine. Coupled with estimates of montelukast oral bioavailability, thisindicates that montelukast and its metabolites are excreted almost exclusively via the bile.

Characteristics in patients

No dosage adjustment is necessary for the elderly or mild to moderate hepatic insufficiency. Studies in patients with renal impairment have not been undertaken. Because montelukast and its metabolites are eliminated by the biliary route, no dose adjustment is anticipated to be necessary inpatients with renal impairment. There are no data on the pharmacokinetics of montelukast in patients with severe hepatic insufficiency (Child-Pugh score>9).

With high doses of montelukast (20- and 60-fold the recommended adult dose), decrease in plasma theophylline concentration was observed. This effect was not seen at the recommended dose of 10 mg once daily.

5.3. Preclinical safety data

In animal toxicity studies, minor serum biochemical alterations in ALT, glucose, phosphorus and triglycerides were observed which were transient in nature. The signs of toxicity in animals were increased excretion of saliva, gastro-intestinal symptoms, loose stools and ion imbalance. These occurred at dosages which provided >17-fold the systemic exposure seen at the clinical dosage. In monkeys, the adverse effectsappeared at doses from 150 mg/kg/day (>232-fold the systemic exposure seen at the clinical dose). In animal studies, montelukast did not affect fertility or reproductive performance at systemic exposure exceeding the clinical systemic exposure by greater than 24-fold. A slight decrease in pup body weightwas noted in the female fertility study in rats at 200 mg/kg/day (>69-fold the clinical systemic exposure). In studies in rabbits, a higher incidence of incomplete ossification, compared with concurrent control animals, was seen at systemic exposure >24-fold the clinical systemic exposure seen at the clinical dose. No abnormalities were seen in rats. Montelukast has been shown to cross the placental barrier and is excreted in breast milk of animals.

No deaths occurred following a single oral administration of montelukast sodium at doses up to 5,000 mg/kg in mice and rats (15,000 mg/m2 and 30,000 mg/m2 in mice and rats, respectively) the maximum dose tested. This dose is equivalent to 25,000 times the recommended daily adult human dose (based on anadult patient weight of 50 kg).

Montelukast was determined not to be phototoxic in mice for UVA, UVB or visible light spectra at doses up to 500 mg/kg/day (approximately >200-fold based on systemic exposure).

Montelukast was neither mutagenic in in vitro and in vivo tests nor tumorigenic in rodent species.

6. Pharmaceutical particulars

6.1. List of excipients

Monokast Tablets 5mg

Microcrystalline Cellulose

Lactose Monohydrate 50MG

Colloidal Silicon Dioxide

Magnesium Stearate

Sodium Starch Glycolate

Opadry II Yellow (85G82757)

Monokast Tablets 10mg

Lactose Monohyrate 49.54 MG

Magnesium Stearate

Primojel

Aerosil 200

Avicel 200

Tabcoat TC Yellow (TC-117-220004)

6.2. Incompatibilities

Not Applicable

6.3. Shelf life

36 months

6.4. Special precautions for storage:

Store below 30℃

Protect from light and moisture.

Store below 30°C.

6.5. Nature and contents of container

Aluminium - Aluminium Blister Pack 14 Tablet are packed in one cartonpack.

6.6. Special precautions for disposal and other handling:

There are no special storage precautions. Any unused product or waste material should be disposed of inaccordance with local requirements.

7. MARKETING AUTHORISATION HOLDER

Global Napi Pharmaceuticals

Plot # 22-23 Industrial Triangle, Kahutta

Road, Islamabad, Pakistan.

8. MARKETING AUTHORISATION NUMBER(S)

Monokast Tablets 5mg: H2022/CTD9970/20485

Monokast Tablets 10mg: H2022/CTD9762/20486

9. DATE OF FIRST AUTHORISATION / RENEWAL OF THE AUTHORISATION

Monokast Tablets 5mg: 21.07.2023

Monokast Tablets 10mg: 21.07.2023

10. DATE OF REVISION OF THE TEXT

Monokast Tablets 5mg: 21.07.2023

Monokast Tablets 10mg: 21.07.2023