SUMMARY OF PRODUCT CHARACTERISTICS

1 NAME OF THE MEDICINAL PRODUCT

Pevanti 20mg Tablets

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Each tablet contains 20 mg of prednisolone

Excipient with known effect
Contains lactose monohydrate 98.72mg

For the full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Tablet

8mm, white, round, biconvex tablet, scored on one side. The tablet can be divided into equal doses.

4 CLINICAL PARTICULARS

4.1 Therapeutic indications

Prednisolone is indicated for the treatment and/or suppression of inflammatory and allergic disorders.

4.2 Posology and method of administration

Posology

In adults and the elderly: The lowest effective dose should be used for the minimum period in order to minimise side effects.

Paediatric population: Prednisolone should be used only when specifically indicated, in a minimum dosage and for the shortest possible time.

The initial dosage of Prednisolone Tablets may vary from 5mg to 60mg or more depending on the disorder being treated. Divided daily dosage is usually used.

The following therapeutic guidelines should be kept in mind for all therapy with corticosteroids:

Corticosteroids are palliative symptomatic treatment by virtue of their anti-inflammatory effects; they are never curative.

The appropriate individual dose must be determined by trial and error and must be re-evaluated regularly according to activity of the disease.

As corticosteroid therapy becomes prolonged and as the dose is increased, the incidence of disabling side-effects increases.

In general, initial dosage shall be maintained or adjusted until the anticipated response is observed. The dose should be gradually reduced until the lowest dose which will maintain an adequate clinical response is reached. Use of the lowest effective dose may also minimise side-effects (see section 4.4).

In patients who have received more than physiological dose for systemic corticosteroids (approximately 7.5mg prednisolone or equivalent) for greater than 3 weeks, withdrawal should not be abrupt. How dose reduction should be carried out depends largely on whether the disease is likely to relapse as the dose of systemic corticosteroids is reduced. Clinical assessment of disease activity may be needed during withdrawal. If the disease is unlikely to relapse on withdrawal of systemic corticosteroids but there is uncertainty about hypothalamic-pituitary-adrenal (HPA) suppression, the dose of corticosteroid may be reduced rapidly to physiological doses. Once a daily dose equivalent to 7.5mg of prednisolone is reached, dose reduction should be slower to allow the HPA-axis to recover.

Abrupt withdrawal of systemic corticosteroid treatment, which has continued up to 3 weeks is appropriate if it is considered that the disease is unlikely to relapse. Abrupt withdrawal of doses of up to 40mg daily of prednisolone, or equivalent for 3 weeks is unlikely to lead to clinically relevant HPA-axis suppression, in the majority of patients. In the following patient groups, gradual withdrawal of systemic corticosteroid therapy should be considered even after courses lasting 3 weeks or less:

• patients who have had repeated courses of systemic corticosteroids, particularly if taken for greater than 3 weeks.

• when a short course has been prescribed within one year of cessation of long-term therapy (months or years).

• patients who may have reasons for adrenocortical insufficiency other than exogenous corticosteroid therapy.

• patients receiving doses of systemic corticosteroid greater than 40mg daily of prednisolone (or equivalent).

• patients repeatedly taking doses in the evening.

(See section 4.4 and 4.8)

During prolonged therapy, dosage may need to be temporarily increased during periods of stress or during exacerbations of the disease (see section 4.4)

If there is lack of a satisfactory clinical response to Prednisolone Tablets, the drug should be gradually discontinued and the patient transferred to alternative therapy.

Intermittent dosage regimen A single dose of Prednisolone Tablets in the morning on alternate days or at longer intervals is acceptable therapy for some patients. When this regimen is practical, the degree of pituitary-adrenal suppression can be minimised.

Specific dosage guidelines The following recommendations for some corticosteroid-responsive disorders are for guidance only. Acute or severe disease may require initial high dose therapy with reduction to the lowest effective maintenance dose as soon as possible. Dosage reductions should not exceed 5-7.5mg daily during chronic treatment.

Allergic and skin disorders Initial doses of 5-15mg daily are commonly adequate.

Collagenosis Initial doses of 20-30mg daily are frequently effective. Those with more severe symptoms may require higher doses.

Rheumatoid arthritis The usual initial dose is 10-15mg daily. The lowest daily maintenance dose compatible with tolerable symptomatic relief is recommended.

Blood disorders and lymphoma An initial daily dose of 15-60mg is often necessary with reduction after an adequate clinical or haematological response. Higher doses may be necessary to induce remission in acute leukaemia.

Use in elderly Treatment of elderly patients, particularly if long-term, should be planned bearing in mind the more serious consequences of the common side-effects of corticosteroids in old age (see also section 4.4).

Paediatric population Although appropriate fractions of the actual dose may be used, dosage will usually be determined by clinical response as in adults (see also section 4.4). Alternate day dosage is preferable where possible.

Method of administration**:** Oral

The daily dose should be taken in the morning after breakfast. For further information with reference to dosage see section 4.4 Special warnings and precautions for use.

4.3 Contraindications

Hypersensitivity to the active substance or to any of the excipients listed in section 6.1.
Systemic infections unless specific anti-infective therapy is employed.
Patients with ocular herpes simplex due to the possibility of perforation.

4.4 Special warnings and precautions for use

A patient information leaflet should be supplied with this product. Patients should carry “steroid treatment” cards which give clear guidance on the precautions to be taken to minimise risk and provide details of prescriber, drug, dosage and duration of treatment.

Patients/and or carers should be warned that potentially severe psychiatric adverse reactions may occur with systemic steroids (see section 4.8). Symptoms typically emerge within a few days or weeks of starting the treatment. Risks may be higher with high doses/systemic exposure (see also section 4.5 pharmacokinetic interactions that can increase the risk of side effects), although dose levels do not allow prediction of the onset, type, severity or duration of reactions. Most reactions recover after either dose reduction or withdrawal, although specific treatment may be necessary.
Patients/carers should be encouraged to seek medical advice if worrying psychological symptoms develop, especially if depressed mood or suicidal ideation is suspected. Patients/carers should also be alert to possible psychiatric disturbances that may occur either during or immediately after dose tapering/withdrawal of systemic steroids, although such reactions have been reported infrequently.
Particular care is required when considering the use of systemic corticosteroids in patients with existing or previous history of severe affective disorders in themselves or in their first degree relatives. These would include depressive or manic-depressive illness and previous steroid psychosis.

Caution is necessary when corticosteroids, including prednisolone, are prescribed to patients with the following conditions and frequent patient monitoring is necessary:

- Diabetes mellitus or in those with a family history of diabetes.
- Glaucoma or in those with a family history of glaucoma.
- Hypertension or congestive heart failure.
- Liver failure.
- Epilepsy.
- Osteoporosis: This is of special importance in post-menopausal females who are at particular risk.
- Patients with a history of severe affective disorders and particularly those with a previous history of corticosteroid induced psychoses.
- Peptic ulceration.
- Previous steroid myopathy.
- Glucocorticoids should be used cautiously in patients with myasthenia gravis receiving anticholinesterase therapy.
- Because cortisone has been reported rarely to increase blood coagulability and to precipitate intravascular thrombosis, thromboembolism, and thrombophlebitis, corticosteroids should be used with caution in patients with thromboembolic disorders.
- Renal insufficiency.
- Tuberculosis: Those with a history of, or X-ray changes characteristic of tuberculosis. The emergence of active tuberculosis can, however, be prevented by the prophylactic use of antituberculous therapy.

- Recent myocardial infarction (rupture).
- Chickenpox: Chickenpox is of particular concern since this normally minor illness may be fatal in immunosuppressed patients. Patients (or parents of children) without a definite history of chickenpox should be advised to avoid close personal contact with chickenpox or herpes zoster and if exposed they should seek urgent medical attention. Passive immunisation with varicella/zoster immunoglobulin (VZIG) is needed by exposed non-immune patients who are receiving systemic corticosteroids or who have used them within the previous 3 months; this should be given within 10 days of exposure to chickenpox. If a diagnosis of chickenpox is confirmed, the illness warrants special care and urgent treatment. Corticosteroids should not be stopped and the dose may need to be increased.
- Measles: Patients are advised to avoid exposure to measles, medical advice should be sought if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.
- Suppression of the inflammatory response and immune function increases the susceptibility to infections and their severity. The clinical presentation may often be atypical and serious infections such as septicaemia and tuberculosis may be masked and may reach an advanced stage before being recognised.
- The effect of corticosteroids may be enhanced in patients with hypothyroidism in those with chronic liver disease with impaired hepatic function.
- Live vaccines should not be given to individuals with impaired immune responsiveness. The antibody response to other vaccines may be diminished.
- Adrenal cortical atrophy develops during prolonged therapy and may persist for years after stopping treatment.

Visual disturbance

Visual disturbance may be reported with systemic and topical corticosteroid use. If a patient presents with symptoms such as blurred vision or other visual disturbances, the patient should be considered for referral to an ophthalmologist for evaluation of possible causes which may include cataract, glaucoma or rare diseases such as central serous chorioretinopathy (CSCR) which have been reported after use of systemic and topical corticosteroids.

Scleroderma renal crisis

Caution is required in patients with systemic sclerosis because of an increased incidence of (possibly fatal) scleroderma renal crisis with hypertension and decreased urinary output observed with a daily dose of 15 mg or more prednisolone. Blood pressure and renal function (s-creatinine) should therefore be routinely checked. When renal crisis is suspected, blood pressure should be carefully controlled.

Withdrawal

In patients who have received more than physiological doses of systemic corticosteroids (approximately 7.5mg prednisolone or equivalent) for greater than 3 weeks, withdrawal should not be abrupt. How dose reduction should be carried out depends largely on whether the disease is likely to relapse as the dose of systemic corticosteroids is reduced. Clinical assessment of disease

activity may be needed during withdrawal. If the disease is unlikely to relapse on withdrawal of systemic corticosteroids but there is uncertainty about HPA suppression, the dose of systemic corticosteroid may be reduced rapidly to physiological doses. Once a daily dose equivalent to 7.5mg of prednisolone is reached, dose reduction should be slower to allow the HPA-axis to recover.
Abrupt withdrawal of systemic corticosteroid treatment, which has continued up to 3 weeks is appropriate if it is considered that the disease is unlikely to relapse. Abrupt withdrawal of doses of up to 40mg daily of prednisolone, or equivalent for 3 weeks is unlikely to lead to clinically relevant HPA-axis suppression, in the majority of patients.
In the following patient groups, gradual withdrawal of systemic corticosteroid therapy should be considered even after courses lasting 3 weeks or less:
• Patients who have had repeated courses of systemic corticosteroids, particularly if taken for greater than 3 weeks,
• When a short course has been prescribed within one year of cessation of long-term therapy (months or years),
• Patients who may have reasons for adrenocortical insufficiency other than exogenous corticosteroid therapy,
• Patients receiving doses of systemic corticosteroid greater than 40mg daily of prednisolone,
• Patients repeatedly taking doses in the evening.

During prolonged therapy any intercurrent illness, trauma or surgical procedure will require a temporary increase in dosage; if corticosteroids have been stopped following prolonged therapy they may need to be temporarily reintroduced.

Use in the elderly: Treatment of elderly patients, particularly if long term, should be planned bearing in mind the more serious consequences of the common side-effects of corticosteroids in old age, especially osteoporosis, diabetes, hypertension, hypokalaemia, susceptibility to infection and thinning of the skin. Close clinical supervision is required to avoid life threatening reactions.

Paediatric population:
Corticosteroids cause growth retardation in infancy, childhood and adolescence, which may be irreversible and therefore long-term administration of pharmacological doses should be avoided. If prolonged therapy is necessary, treatment should be limited to the minimum suppression of the hypothalamo-pituitary adrenal axis and growth retardation. The growth and development of infants and children should be closely monitored. Treatment should be administered where possible as a single dose on alternate days.

Patients with rare hereditary problems of galactose intolerance, total lactase deficiency or glucose galactose malabsorption should not take this medicine.

4.5 Interaction with other medicinal products and other forms of interaction

Co-treatment with CYP3A inhibitors, including cobicistat-containing products, is expected to increase the risk of systemic side-effects. The combination should be avoided unless the benefit outweighs the increased risk

of systemic corticosteroid side-effects, in which case patients should be monitored for systemic corticosteroid side-effects.

Hepatic microsomal enzyme inducers Drugs that induce hepatic enzyme cytochrome P-450 (CYP) isoenzyme 3A4 such as phenobarbital, phenytoin, rifampicin, rifabutin, carbamazepine, primidone and aminoglutethimide may reduce the therapeutic efficacy of corticosteroids by increasing the rate of metabolism. Lack of expected response may be observed and dosage of Prednisolone Tablets may need to be increased.

Hepatic microsomal enzyme inhibitors Drugs that inhibit hepatic enzyme cytochrome P-450 (CYP) isoenzyme 3A4 (e.g. ketoconazole, troleandomycin) may decrease glucocorticoid clearance. Dosages of glucocorticoids given in combination with such drugs may need to be decreased to avoid potential adverse effects.

Antidiabetic agents Glucocorticoids may increase blood glucose levels. Patients with diabetes mellitus receiving concurrent insulin and/or oral hypoglycemic agents may require dosage adjustments of such therapy.

Non-steroidal anti-inflammatory drugs Concomitant administration of ulcerogenic drugs such as indomethacin during corticosteroid therapy may increase the risk of GI ulceration. Aspirin should be used cautiously in conjunction with glucocorticoids in patients with hypoprothrombinaemia. Although concomitant therapy with salicylate and corticosteroids does not appear to increase the incidence or severity of GI ulceration, the possibility of this effect should be considered.

Serum salicylate concentrations may decrease when corticosteroids are administered concomitantly. The renal clearance of salicylates is increased by corticosteroids and steroid withdrawal may result in salicylate intoxication. Salicylates and corticosteroids should be used concurrently with caution. Patients receiving both drugs should be observed closely for adverse effects of either drug.

Antibacterials Rifampicins accelerate metabolism of corticosteroids and thus may reduce their effect. Erythromycin inhibits metabolism of methyl prednisolone and possibly other corticosteroids.

Anticoagulants Response to anticoagulants may be reduced or, less often, enhanced by corticosteroids. Close monitoring of the INR or prothrombin time is required to avoid spontaneous bleeding.

Antiepileptics Carbamazepine, phenobarbital, phenytoin, and primidone accelerate metabolism of corticosteroids and may reduce their effect.

Antifungals Risk of hypokalaemia may be increased with amphotericin, therefore concomitant use with corticosteroids should be avoided unless corticosteroids are required to control reactions; ketoconazole inhibits metabolism of methyl prednisolone and possibly other corticosteroids.

Antivirals Ritonavir possibly increases plasma concentrations of prednisolone and other corticosteroids.

Cardiac Glycosides Increased toxicity if hypokalaemia occurs with corticosteroids.

Ciclosporin Concomitant administration of prednisolone and ciclosporin may result in decreased plasma clearance of prednisolone (i.e. increased plasma concentration of prednisolone). The need for appropriate dosage adjustment should be considered when these drugs are administered concomitantly.

Cytotoxics Increased risk of haematological toxicity with methotrexate.

Mifepristone Effect of corticosteroids may be reduced for 3-4 days after mifepristone.

Vaccines Live vaccines should not be given to individuals with impaired immune responsiveness. The antibody response to other vaccines may be diminished.

Oestrogens Oestrogens may potentiate the effects of glucocorticoids and dosage adjustments may be required if oestrogens are added to or withdrawn from a stable dosage regimen.

Somatropin Growth promoting effect may be inhibited.

Sympathomimetics Increased risk of hypokalaemia if high doses of corticosteroids given with high doses of bambuterol, fenoteral, formoteral, ritodrine, salbutamol, salmeterol and terbutaline.

Other The desired effects of hypoglycaemic agents (including insulin), antihypertensives and diuretics are antagonised by corticosteroids; and the hypokalaemic effect of acetazolamide, loop diuretics, thiazide diuretics, carbenoxolone and theophylline are enhanced.

4.6 Fertility, pregnancy and lactation

Pregnancy

The ability of corticosteroids to cross the placenta varies between individual drugs, however, 88% of prednisolone is inactivated as it crosses the placenta. Administration of corticosteroids to pregnant animals can cause abnormalities of fetal development including cleft palate, intra-uterine growth retardation and effects on brain growth and development. There is no evidence that corticosteroids result in an increased incidence of congenital abnormalities, such as cleft palate/lip in man. However, when administered for prolonged periods or repeatedly during pregnancy, corticosteroids may increase the risk of intra-uterine growth retardation. Hypoadrenalism may, in theory, occur in the neonate following prenatal exposure to corticosteroids but usually resolves spontaneously following birth and is rarely clinically important. As with all drugs, corticosteroids should only be prescribed when the benefits to the mother and child outweigh the risks. When corticosteroids are essential however, patients with normal pregnancies may be treated as though they were in the non-gravid state.

Patients with pre-eclampsia or fluid retention require close monitoring.

Breast-feeding

Corticosteroids are excreted in small amounts in breast milk. However, doses of up to 40mg daily of prednisolone are unlikely to cause systemic effects in the infant. Infants of mothers receiving 40mg or more daily should be monitored for signs of adrenal suppression but the benefits of breast-feeding are likely to outweigh any theoretical risk**.**

Fertility

No data available

4.7 Effects on ability to drive and use machines

There is no evidence to suggest that prednisolone has any affect on the ability to drive or use machines.

4.8 Undesirable effects

A wide range of psychiatric reactions including affective disorders (such as irritable, euphoric, depressed and labile mood, and suicidal thoughts), psychotic reactions (including mania, delusions, hallucinations, and aggravation of schizophrenia), behavioural disturbances, irritability, anxiety, sleep disturbances, and cognitive dysfunction including confusion and amnesia have been reported. Reactions are common and may occur in both adults and children. In adults, the frequency of severe reactions has been estimated to be 5-6%. Psychological effects have been reported on withdrawal of corticosteroids; the frequency is Not known.

The incidence of predictable undesirable effects, including hypothalamic pituitary adrenal suppression correlates with the relative potency of the drug, dosage, timing of administration and the duration of treatment (see section 4.4).

Adverse reactions are listed as per System Organ Class. The following adverse reactions have been observed at the frequencies defined using the following convention:
Not known: cannot be estimated from the available data.

System organ class	Undesirable effects
Infections and infestations	Increases susceptibility to and severity of infections with suppression of clinical symptoms and signs, opportunistic infections, recurrence of dormant tuberculosis (see section 4.4).
Blood and lymphatic system disorders	Leucocytosis
Immune system disorders	Hypersensitivity including anaphylaxis
Endocrine disorders	Cushingoid , impaired carbohydrate tolerance with increased requirement for antidiabetic therapy, manifestation of latent diabetes mellitus,

Metabolism and nutrition disorders	Sodium and water retention, increased appetite which may result in weight gain, alkalosis hypokalaemic, negative nitrogen and calcium balance
Psychiatric disorders	Euphoric mood, drug dependence, depression, insomnia, schizophrenia,
Nervous system disorders	Dizziness, headache, epilepsy
Eye disorders	Glaucoma, papilloedema, cataract subcapsular , exophthalmos, corneal or scleral thinning, exacerbation of ophthalmic viral or fungal disease and vision, blurred (see also section 4.4)
Ear and labyrinth disorders	Vertigo
Cardiac disorders	Congestive heart failure in susceptible patients Bradycardia***
Vascular disorders	Thromboembolism, hypertension
Gastrointestinal disorders	Dyspepsia, nausea, peptic ulcer with perforation and haemorrhage, abdominal distension, abdominal pain, diarrhoea, oesophageal ulceration, oesophageal candidiasis, pancreatitis acute
Skin and subcutaneous tissue disorders	Hirsutism, skin atrophy, bruising, skin striae, telangiectasia, acne, hyperhidrosis, may suppress reactions to skin tests, pruritis, rash, urticaria
Musculoskeletal and connective tissue disorders	Myopathy, osteoporosis, vertebral and long bone fractures, avascular osteonecrosis, myalgia
Renal and urinary disorders	Scleroderma renal crisis*
Reproductive system and breast disorders	Menstrual irregular and amenorrhoea
General disorders and administration site conditions	Impaired healing, withdrawal symptoms**, fatigue, malaise.
Investigations	Weight increased ,increased intra-ocular pressure
Injury, poisoning and procedural complications	Tendon rupture

*Scleroderma renal crisis

Amongst the different sub populations the occurrence of scleroderma renal crisis varies. The highest risk has been reported in patients with diffuse systemic sclerosis. The lowest risk has been reported in patients with limited systemic sclerosis (2%) and juvenile onset systemic sclerosis (1%)

***Withdrawal symptoms:** Too rapid a reduction of corticosteroid dosage following prolonged treatment can lead to acute adrenal insufficiency, hypotension and death (see section 4.4 and 4.2). A steroid "withdrawal syndrome" seemingly unrelated to adrenocortical insufficiency may also occur following abrupt discontinuance of glucocorticoids. This syndrome includes symptoms such as: anorexia, nausea, vomiting, lethargy, headache, fever, joint pain, desquamation, myalgia, arthralgia, rhinitis, conjunctivitis, painful itchy skin nodules weight loss, and/or hypotension. These effects are thought to be due to the sudden change in glucocorticoid concentration rather than to low corticosteroid levels.

***Following high doses

Additional side effects in children and adolescents

Suppression of the hypothalamo-pituitary adrenal axis particularly in times of stress, as in trauma, surgery or illness, growth suppression in infancy, childhood and adolescence.

Raised intracranial pressure with papilloedema (pseudotumor cerebri) in children, usually after treatment withdrawal.

Reporting of suspected adverse reactions
Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions via the Yellow Card Scheme Website: www.mhra.gov.uk/yellowcard or search for MHRA Yellow Card in the Google Play or Apple App Store.

4.9 Overdose

Symptoms
Reports of acute toxicity and/or death following overdosage of glucocorticoids are rare.

Management
No specific antidote is available; treatment is supportive and symptomatic. Serum electrolytes should be monitored.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic Group: Corticosteroids for systematic use, plain

ATC Code: H02A

Naturally occurring glucocorticoids (hydrocortisone and cortisone), which also have salt- retaining properties, are used as replacement therapy in adrenocortical deficiency states. Their synthetic analogs are primarily used for their potent anti-inflammatory effects in disorders of many organ systems.

Glucocorticoids cause profound and varied metabolic effects. In addition, they modify the body's immune responses to diverse stimuli.

5.2 Pharmacokinetic properties

Absorption
Prednisolone is rapidly and apparently almost completely absorbed after oral administration; it reaches peak plasma concentrations after 1-3 hours. There is however wide inter-subject variation suggesting impaired absorption in some individuals. Plasma half-life is about 3 hours in adults and somewhat less in children. Its initial absorption, but not its overall bioavailability, is affected by food. Prednisolone has a biological half-life lasting several hours, making it suitable for alternate-day administration regimens.

Distribution
Prednisolone shows dose dependent pharmacokinetics, with an increase in dose leading to an increase in volume of distribution and plasma clearance. The degree of plasma protein binding determines the distribution and clearance of free, pharmacologically active drug. Reduced doses are necessary in patients with hypoalbuminaemia.

Biotransformation
Prednisolone is metabolised primarily in the liver to a biologically inactive compound. Liver disease prolongs the half-life of prednisolone and, if the patient has hypoalbuminaemia, also increases the proportion of unbound drug and may thereby increase adverse effects.

Elimination
Prednisolone is excreted in the urine as free and conjugated metabolites, together with small amounts of unchanged prednisolone.

5.3 Preclinical safety data

There are no non-clinical data of relevance to the prescriber that are not already covered in other sections of the SmPC.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Potato starch

Lactose monohydrate

Talc

Gelatine

Magnesium stearate

6.2 Incompatibilities

Not applicable.

6.3 Shelf life

HDPE bottles: 36 months

Once opened: Use within 6 months

Blisters: 20 months

6.4 Special precautions for storage

HDPE bottles: This medicinal product does not require any special storage precautions.

Blisters: Do not store above 25ºC.

6.5 Nature and contents of container

PVC/PVDC/Aluminium blister pack or HDPE container and LDPE/HDPE cap without desiccant

Pack size: 25, 30, 100 tablets

Not all pack sizes may be marketed.

6.6 Special precautions for disposal

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

7 MARKETING AUTHORISATION HOLDER

Mercury Pharmaceuticals Ltd
Dashwood House,
69 Old Broad Street,
London, EC2M 1QS,
United Kingdom

8 MARKETING AUTHORISATION NUMBER(S)

PL 12762/0483

9 DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

Date of first authorisation: 01 December 2014
Date of latest renewal: 28 August 2019

10 DATE OF REVISION OF THE TEXT

23/11/2023