SUMMARY OF PRODUCT CHARACTERISTICS

1 NAME OF THE MEDICINAL PRODUCT

Furosemide 40 mg/5 ml Oral Solution

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Each 5ml contains Furosemide 40 mg.

Excipients with known effect
Ethanol
Liquid maltitol

For the full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

Oral Solution
Clear, cherry flavoured, oral solution

4. CLINICAL PARTICULARS

4.1 Therapeutic Indications

Furosemide oral solution is indicated in all conditions requiring prompt diuresis in patients who are unable to take solid dose forms. Indications, include cardiac, pulmonary, hepatic and renal oedema, peripheral oedema due to mechanical obstruction or venous insufficiency and hypertension.

4.2 Posology and method of administration

Posology
Furosemide 40mg/5ml has an exceptionally wide therapeutic range, the effect being proportional to the dosage. Furosemide 40mg/5ml is best given as a single dose either daily or on alternate days.

The recommended initial daily dose is 40mg. This may require adjustment until the effective dose is achieved as a maintenance dose. In mild cases, 20mg

daily or 40mg on alternate days may be sufficient, whereas in cases of resistant oedema, daily doses of 80mg and above may be used as one or two dose daily, or intermittently. Severe cases may require gradual titration of the furosemide dosage up to 600mg daily. The recommended maximum daily dose of furosemide administration is 1500mg.

Elderly: The dosage recommendations for adults apply, but in the elderly, furosemide is generally eliminated more slowly. Dosage should be titrated until the required response is achieved.

Children: Oral doses for children range from 1 to 3 mg/Kg body weight daily up to a maximum total dose of 40 mg/day.

4.3 Contraindications

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

Hypersensitivity to sulphonamides or sulphonamide derivatives.

Hypovolaemia and dehydration (with or without accompanying hypotension) (see section 4.4).

Severe hypokalaemia: severe hyponatraemia (see section 4.4).

Comatose or pre-comatose states associated with hepatic cirrhosis (see section 4.4).

Anuria or renal failure with anuria not responding to furosemide, renal failure as a result of poisoning by nephrotoxic or hepatotoxic agents, renal failure associated with hepatic coma

Impaired renal function with a creatinine clearance below 30ml/min per 1.73 m^2 body surface area (see section 4.4).

Addison's disease (see section 4.4). Digitalis intoxication (see section 4.5).

Porphyria.

Breast-feeding women (see section 4.6).

4.4 Special warnings and precautions for use

Conditions requiring correction before furosemide is started (see also section 4.3)

Hypotension.

Hypovolaemia.

Severe electrolyte disturbances – particularly hypokalaemia, hyponatraemia and acid-base disturbances.

Furosemide is not recommended

In patients at high risk for radiocontrast nephropathy - it should not be used for diuresis as part of the preventative measures against radiocontrast-induced nephropathy.

Particular caution and/or dose reduction required:

Symptomatic hypotension leading to dizziness, fainting or loss of consciousness can occur in patients treated with furosemide, particularly in the elderly, patients on other medications which can cause hypotension and patients with other medical conditions that are risks for hypotension.

Older people (lower initial dose as particularly susceptible to side-effects - see section 4.2).

difficulty with micturition including prostatic hypertrophy (increased risk of urinary retention: consider lower dose). Closely monitor patients with partial occlusion of the urinary tract

diabetes mellitus (latent diabetes may become overt: insulin requirements in established diabetes may increase: stop furosemide before a glucose tolerance test) pregnancy (see section 4.6)

gout (furosemide may raise uric acid levels/precipitate gout)

patients with hepatorenal syndrome

impaired hepatic function (see section 4.3 and below – monitoring required)

impaired renal function (see section 4.3 and below – monitoring required)

adrenal disease (see section 4.3 – contraindication in Addison's disease)

hypoproteinaemia e.g. nephritic syndrome (effect of furosemide may be impaired and its ototoxicity potentiated - cautious dose titration required).

acute hypercalcaemia (dehydration results from vomiting and diuresis - correct before giving furosemide). Treatment of hypercalcaemia with a high dose of furosemide results in fluid and electrolyte depletion - meticulous fluid replacement and correction of electrolyte required.

Patients who are at risk from a pronounced fall in blood pressure premature infants (possible development nephrocalcinosis/nephrolithiasis; renal function must be monitored and renal ultrasonography performed).

Avoidance with other medicines (see also section 4.5 for other interactions)

concurrent NSAIDs should be avoided – if not possible diuretic effect of furosemide may be attenuated

ACE-inhibitors & Angiotensin II receptor antagonists – severe hypotension may occur – dose of furosemide should be reduced/stopped (3 days) before starting or increasing the dose of these

Laboratory monitoring requirements:

Serum sodium

Particularly in the older people or in patients liable to electrolyte deficiency

Serum potassium

The possibility of hypokalaemia should be taken into account, in particular in patients with cirrhosis of the liver, those receiving concomitant treatment with corticosteroids, those with an unbalanced diet and those who abuse laxatives. Regular monitoring of the potassium, and if necessary treatment with a potassium supplement, is recommended in all cases, but is essential at higher doses and in patients with impaired renal function. It is especially important in the event of concomitant treatment with digoxin, as potassium deficiency can trigger or exacerbate the symptoms of digitalis intoxication (see section 4.5). A potassium-rich diet is recommended during long-term use.

Frequent checks of the serum potassium are necessary in patients with impaired renal function and creatinine clearance below 60ml/min per 1.73m2 body surface area as well as in cases where furosemide is taken in combination with certain other drugs which may lead to an increase in potassium levels (see section 4.5 & refer to section 4.8 for details of electrolyte and metabolic abnormalities)

Renal function

Frequent BUN in first few months of treatment, periodically thereafter. Long-term/high-dose BUN should regularly be measured. Marked diuresis can cause reversible impairment of kidney function in patients with renal dysfunction. Adequate fluid intake is necessary in such patients. Serum creatinine and urea levels tend to rise during treatment

Glucose

Adverse effect on carbohydrate metabolism - exacerbation of existing carbohydrate intolerance or diabetes mellitus. Regular monitoring of blood glucose levels is desirable.

Other electrolytes

Patients with hepatic failure/alcoholic cirrhosis are particularly at risk of hypomagnesia (as well as hypokalaemia). During long-term therapy (especially at high doses) magnesium, calcium, chloride, bicarbonate and uric acid should be regularly measured.

Clinical monitoring requirements (see also section 4.8):

Regular monitoring for blood dyscrasias. If these occur, stop furosemide immediately

liver damage

idiosyncratic reactions

Other alterations in lab values

Serum cholesterol and triglycerides may rise but usually return to normal within 6 months of starting furosemide

Concomitant use with risperidone

In risperidone placebo-controlled trials in older people with dementia, a higher incidence of mortality was observed in patients treated with furosemide plus risperidone (7.3%; mean age 89 years, range 75-97 years) when compared to patients treated with risperidone alone (3.1%; mean age 84 years, range 70-96 years) or furosemide alone (4.1%; mean age 80 years, range 67-90 years). Concomitant use of risperidone with other diuretics (mainly thiazide diuretics used in low dose) was not associated with similar findings.

No pathophysiological mechanism has been identified to explain this finding, and no consistent pattern for cause of death observed. Nevertheless, caution should be exercised and the risks and benefits of this combination or co-treatment with other potent diuretics should be considered prior to the decision to use. There was no increased incidence of mortality among patients taking other diuretics as concomitant treatment with risperidone. Irrespective of treatment, dehydration was an overall risk factor for mortality and should therefore be avoided in older patients with dementia (see section 4.3 Contraindications).

This product contains liquid maltitol. Patients with a rare hereditary problem of fructose intolerance should not take this medicine.

4.5 Interaction with other medicinal products and other forms of interaction

General- The dosage of concurrently administered cardiac glycosides, diuretics, anti-hypertensive agents, or other drugs with blood-pressure-lowering potential may require adjustment as a more pronounced fall in blood pressure must be anticipated if given concomitantly with furosemide.

The toxic effects of nephrotoxic drugs may be increased by concomitant administration of potent diuretics such as furosemide.

Some electrolyte disturbances (e.g. hypokalaemia, hypomagnesaemia) may increase the toxicity of certain other drugs (e.g. digitalis preparations and drugs inducing QT interval prolongation syndrome).

Antihypertensives – enhanced hypotensive effect possible with all types. Concurrent use with ACE inhibitors or Angiotensin II receptor antagonists can result in marked falls in blood pressure. Furosemide should be stopped or the dose reduced before starting an ACE-inhibitor or Angiotensin II receptor antagonists (see section 4.4). There is a risk of a first-dose effect with post-synaptic alpha blockers eg prazosin.

Antipsychotics – furosemide-induced hypokalaemia increases the risk of cardiac toxicity. Avoid concurrent use with pimozide. Increased risk of ventricular arrhythmias with amisulpride or sertindole. Enhanced hypotensive effect with phenothiazines.

When administering risperidone, caution should be exercised and the risks and benefits of the combination or co-treatment with furosemide or with other potent diuretics should be considered prior to the decision to use. See section 4.4 Special warnings and precautions for use regarding increased mortality in elderly patients with dementia concomitantly receiving risperidone.

Anti-arrhythmics (including amiodarone, disopyramide, flecanaide and sotalol) - risk of cardiac toxicity (because of furosemide-induced hypokalaemia). The effects of lidocaine, tocainide or mexiletine may be antagonised by furosemide.

Drugs that prolong Q-T interval – increased risk of toxicity with furosemide-induced electrolyte disturbances.

Cardiac glycosides – hypokalaemia and electrolyte disturbances (including magnesium) increases the risk of cardiac toxicity.

Vasodilators – enhanced hypotensive effect with moxisylyte (thymoxamine) or hydralazine.

Other diuretics – profound diuresis possible when furosemide given with metolazone.
Increased risk of hypokalaemia with thiazides.

Renin inhibitors – aliskiren reduces plasma concentrations of furosemide.

Nitrates – enhanced hypotensive effect.

Lithium - In common with other diuretics, serum lithium levels may be increased when lithium is given concomitantly with furosemide, resulting in increased lithium toxicity, including increased risk of cardiotoxic and

neurotoxic effects of lithium. Therefore, it is recommended that lithium levels are carefully monitored and where necessary the lithium dosage is adjusted in patients receiving this combination.

Chelating agents – sucralfate may decrease the gastro-intestinal absorption of furosemide – the 2 drugs should be taken at least 2 hours apart.

NSAIDs – increased risk of nephrotoxicity. Indometacin and ketorolac may antagonise the effects of furosemide (avoid if possible see section 4.4). NSAIDs may attenuate the action of furosemide and may cause acute renal failure in cases of pre-existing hypovolaemia or dehydration.

Salicylates – effects may be potentiated by furosemide. Salycylic toxicity may be increased by furosemide.

Antibiotics – increased risk of ototoxicity with aminoglycosides, polymixins or vancomycin - only use concurrently if compelling reasons. Increased risk of nephrotoxicity with aminoglycosides or cefaloridine. Furosemide can decrease vancomycin serum levels after cardiac surgery. Increased risk of hyponatraemia with trimethoprim. Impairment of renal function may develop in patients receiving concurrent treatment with furosemide and high doses of certain cephalosporins.

Antidepressants – enhanced hypotensive effect with MAOIs. Increased risk of postural hypotension with TCAs (tricyclic antidepressants). Increased risk of hypokalaemia with reboxetine.

Antidiabetics – hypoglycaemic effects antagonised by furosemide.

Antiepileptics – increased risk of hyponatraemia with carbamazepine. Diuretic effect reduced by phenytoin.

Antihistamines – hypokalaemia with increased risk of cardiac toxicity.

Antifungals – increased risk of hypokalaemia and nephrotoxicitiy with amphotericin.

Antivirals – plasma concentrations of diuretics may be increased by nelfinavir, ritonavir or saquinavir.

Anxiolytics and hypnotics – enhanced hypotensive effect. Chloral or triclorfos may displace thyroid hormone from binding site.

CNS stimulants (drugs used for ADHD) – hypokalaemia increases the risk of ventricular arrhythmias.

Corticosteroids – diuretic effect anatgonised (sodium retention) and increased risk of hypokalaemia.

Glychyrrizin -(contained in liquorice) may increase the risk of developing hypokalaemia.

Cytotoxics – increased risk of nephrotoxicity and ototoxicity with platinum compounds/cisplatin. Nephrotoxicity of cisplatin may be enhanced if furosemide is not given in low doses (e.g. 40 mg in patients with normal renal function) and with positive fluid balance when used to achieve forced diuresis during cisplatin treatment.

Anti-metabolites – effects of furosemide may be reduced by methotrexate and furosemide may reduce renal clearance of methotrexate.

Dopaminergics – enhanced hypotensive effect with levodopa.

Immunomodulators – enhanced hypotensive effect with aldesleukin. Increased risk of hyperkalaemia with ciclosoprin and tacrolimus. Increased risk of gouty arthritis with ciclosporin.

Muscle relaxants – enhanced hypotensive effect with baclofen or tizanidine. Increased effect of curare-like muscle relaxants.

Oestrogens – diuretic effect antagonised.

Progestogens (drosperidone) – increased risk of hyperkalaemia.

Prostaglandins – enhanced hypotensive effect with alprostadil.

Sympathomimetics – increased risk of hypokalaemia with high doses of beta2 sympathomimetics.

Theophylline – enhanced hypotensive effect.

Probenecid – effects of furosemide may be reduced by probenecid and furosemide may reduce renal clearance of probenecid.

Anaesthetic agents – general anaesthetic agents may enhance the hypotensive effects of furosemide. The effects of curare may be enhanced by furosemide

Alcohol – enhanced hypotensive effect

Laxative abuse - increases the risk of potassium loss

Others**:** Concomitant administration of aminoglutethimide may increase the risk of hyponatraemia.

4.6 Fertility, pregnancy and lactation

Pregnancy

Furosemide crosses the placental barrier and should not be given during pregnancy unless there are compelling medical reasons. It should only be used for the pathological causes of oedema which are not directly or indirectly linked to the pregnancy. The treatment with diuretics of oedema and hypertension caused by pregnancy is undesirable because placental perfusion can be reduced, so, if used, monitoring of fetal growth is required. However, furosemide has been given after the first trimester of pregnancy for oedema, hypertension and toxaemia of pregnancy without causing fetal or newborn adverse effects.

Breast-feeding (see section 4.3)

Furosemide is contraindicated as it passes into breast milk and may inhibit lactation.

4.7 Effects on ability to drive and use machines

Reduced mental alertness, dizziness and blurred vision have been reported, particularly at the start of treatment, with dose changes and in combination with alcohol. Patients should be advised that if affected, they should not drive, operate machinery or take part in activities where these effects could put themselves or others at risk.

4.8 Undesirable effects

Undesirable effects can occur with the following frequencies: 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, including isolated reports), not known (cannot be estimated from the available data)

Blood and lymphatic system disorders:

Uncommon:
Thrombocytopenia

Rare:
Eosinophilia
Leukopenia
Bone marrow depression (necessitates withdrawal of treatment). The haemopoietic status should be therefore be regularly monitored.

Very Rare:
aplastic anaemia or haemolytic anaemia
agranulocytosis

Nervous system disorders

Rare:
paraesthesia
hyperosmolar coma

Not known:
Dizziness, fainting and loss of consciousness (caused by symptomatic hypotension).

Endocrine disorder

Glucose tolerance may decrease with furosemide. In patients with diabetes mellitus this may lead to a deterioration of metabolic control; latent diabetes mellitus may become manifest. Insulin requirements of diabetic patients may increase.

Eye disorders

Uncommon: visual disturbance

Ear and labyrinth disorders

Hearing disorders and tinnitus, although usually transitory, may occur in rare cases, particularly in patients with renal failure, hypoproteinaemia (e.g. in nephritic syndrome) and/or when intravenous furosemide has been given too rapidly.

Uncommon:
Deafness (sometimes irreversible)

Cardiac disorders

Uncommon: Cardiac arrhythmias

Furosemide may cause a reduction in blood pressure which, if pronounced may cause signs and symptoms such as impairment of concentration and reactions, light headedness, sensations of pressure in the head, headache, dizziness, drowsiness, weakness, disorders of vision, dry mouth, orthostatic intolerance. The diuretic effect of furosemide can result in hypovolaemia and dehydration, especially in the elderly. There is an increased risk of thrombosis.

Hepatobiliary disorders

In isolated cases, intrahepatic cholestasis, an increase in liver transaminases or acute pancreatitis may develop.

Hepatic encephalopathy in patients with hepatocellular insufficiency may occur (see Section 4.3).

Vascular Disorder:

Rare:
vasculitis

Skin and subcutaneous tissue disorders

Uncommon:
Photosensitivity

Rare:
Skin and mucous membrane reactions may occasionally occur, e.g. itching, urticaria, other rashes or bullous lesions, fever, hypersensitivity to light, exsudative erythema multiforme (Lyell's syndrome and Stevens-Johnson syndrome), bullous exanthema, exfoliative dermatitis, purpura, AGEP (acute generalized exanthematous pustulosis) and DRESS (Drug rash with eosinophilia and systemic symptoms).

Metabolism and nutrition disorders

As with other diuretics, electrolytes and water balance may be disturbed as a result of diuresis after prolonged therapy. Furosemide leads to increased excretion of sodium and chloride and consequently increase excretion of water. In addition, excretion of other electrolytes (in particular potassium, calcium and magnesium) is increased.

Metabolic acidosis can also occur. The risk of this abnormality increases at higher dosages and is influenced by the underlying disorder (e.g. cirrhosis of the liver, heart failure), concomitant medication (see section 4.5) and diet.

Symptomatic electrolyte disturbances and metabolic alkalosis may develop in the form of a gradually increasing electrolyte deficit or e.g. where higher furosemide doses are administered to patients with normal renal function, acute severe electrolyte losses

Symptoms of electrolyte imbalance depend on the type of disturbance:

Sodium deficiency can occur; this can manifest itself in the form of confusion, muscle cramps, muscle weakness, loss of appetite, dizziness, drowsiness and vomiting.

Potassium deficiency manifests itself in neuromuscular symptoms (muscular weakness, paralysis), intestinal symptoms (vomiting, constipation, meterorism), renal symptoms (polyuria) or cardiac symptoms. Severe potassium depletion can result in paralytic ileus or confusion, which can result in coma.

Magnesium and calcium deficiency result very rarely in tetany and heart rhythm disturbances.

Serum calcium levels may be reduced; in very rare cases tetany has been observed.

Nephrocalcinosis/Nephrolithiasis has been reported in premature infants.

Serum cholesterol (reduction of serum HDL-cholesterol, elevation of serum LDL-cholesterol) and triglyceride levels may rise during furosemide treatment. During long term therapy they will usually return to normal within six months

As with other diuretics, treatment with furosemide may lead to transitory increase in blood creatinine and urea levels. Serum levels of uric acid may increase and attacks of gout may occur.

The diuretic action of furosemide may lead to or contribute to hypovolaemia and dehydration, especially in elderly patients. Severe fluid depletion may lead to haemoconcentration with a tendency for thromboses to develop.

Increased production of urine may provoke or aggravate complaints in patients with an obstruction of urinary outflow. Thus, acute retention of urine with possible secondary complications may occur. For example, in patients with bladder-emptying disorders, prostatic hyperplasia or narrowing of the urethra.

Congenital, familial and genetic disorders

If furosemide is administered to premature infants during the first weeks of life, it may increase the risk of persistence of patent ductus arteriosus.

General disorders and administration site conditions

Uncommon: Fatigue

Rare:
Severe anaphylactic or anaphylactoid reactions (e.g. with shock) occurs rarely.
fever
Malaise

Gastrointestinal disorders

Uncommon: dry mouth, thirst, nausea, bowel motility disturbances, vomiting, diarrhoea, constipation.

Rare:
Acute Pancreatitis
Gastro-intestinal disorders such as nausea, malaise or gastric upset (vomiting or diarrhoea) and constipation may occur but not usually severe enough to necessitate withdrawal of treatment.

Renal and urinary disorders

Uncommon:
serum creatinine and urea levels can be temporarily elevated during treatment with furosemide.

Rare:

interstitial nephritis, acute renal failure.
Increased urine production, urinary incontinence, can be caused or symptoms can be exacerbated in patients with urinary tract obstruction. Acute urine retention, possibly accompanied by complications, can occur for example in patients with bladder disorders, prostatic hyperplasia or narrowing of the urethra.

Pregnancy, puerperium and perinatal conditions

In premature infants with respiratory distress syndrome, administration of Furosemide in the initial weeks after birth entails an increased risk of a persistent patent ductus arteriosus.

In premature infants, furosemide can be precipitated as nephrocalcinosis/kidney stones.

Rare complications may include minor psychiatric disturbances.

Special population:
Patients with hepatic impairment

Pre-existing metabolic alkalosis (e.g. in decompensated cirrhosis of the liver) may be aggravated by furosemide treatment.

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 at www.mhra.gov.uk/yellowcard.

4.9 Overdose

Features
Overdose can cause massive diuresis resulting in dehydration, volume depletion and electrolyte disturbances with consequent hypotension and cardiac toxicity. High doses have the potential to cause transient deafness and may precipitate gout (disturbed uric acid secretion).

Management
Benefits of gastric decontamination are uncertain. In patients presenting within 1 hour of ingestion, consider activated charcoal (50g for adults: 1g/kg for children).

Observe for a minimum of 4 hours - monitor pulse and blood pressure.

Treat hypotension and dehydration with appropriate IV fluids.

Monitor urinary output and serum electrolytes (including chloride and bicarbonate). Correct electrolyte imbalances. Monitor 12 lead ECG in patients with significant electrolyte disturbances

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: High ceiling Diuretic Sulfonamide, ATC code: CO3C 1 01

Mechanism of action
The principle renal action of furosemide is to inhibit active chloride transport in the thick ascending limb. Re-absorption of sodium, chloride from the nephron is reduced and a hypotonic or isotonic urine produced.

Pharmacodynamic effects
The evidence from many experimental studies suggests that furosemide acts along the entire nephron with the exception of the distal exchange site. The main effect is on the ascending limb of the loop of Henle with a complex effect on renal circulation. Blood-flow is diverted from the juxta-medullary region to the outer cortex.

It has been established that prostaglandin (PG) biosynthesis and the renin-angiotensin system are affected by furosemide administration and that furosemide alters the renal permeability of the glomerulus to serum proteins.

5.2 Pharmacokinetic properties

Absorption
Approximately 65% of the dose is absorbed after oral administration. The plasma half-life is biphasic with a terminal elimination phase of about 1½ hours.

Furosemide is a weak carboxylic acid which exists mainly in the dissociated form in the gastrointestinal tract. Furosemide is rapidly but incompletely absorbed (60-70%) on oral administration and its effect is largely over within 4 hours. The optimal absorption site is the upper duodenum at pH 5.0.

Distribution
Furosemide is up to 99% bound to plasma proteins.

Biotransformation
Furosemide is bound to plasma albumin and little biotransformation takes place

Elimination
Regardless of route of administration 69-97% of activity from a radio-labelled dose is excreted in the first 4 hours after the drug is given. Furosemide is mainly eliminated via the kidneys (80-90%) mainly excreted in the urine, largely unchanged; but also excreted in the bile, non-renal elimination being

considerably increased in renal failure. Furosemide crosses the placental barrier and is excreted in the milk.

A small fraction of the dose undergoes biliary elimination and 10-15% of the activity can be recovered from the faeces.

In renal/ hepatic impairment
Where liver disease is present, biliary elimination is reduced up to 50%. Renal impairment has little effect on the elimination rate of furosemide, but less than 20% residual renal function increases the elimination time.

The elderly
The elimination of furosemide is delayed in the elderly where a certain degree of renal impairment is present.

New born
A sustained diuretic effect is seen in the newborn, possibly due to immature tubular function.

5.3 Preclinical safety data

Furosemide is a widely used diuretic which has been available for over thirty years and its safety profile in man is well established.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Ethanol, sodium hydroxide, cherry flavour (containing propylene glycol), liquid maltitol, disodium hydrogen phosphate, citric acid monohydrate and purified water.

6.2 Incompatibilities

None known

6.3 Shelf-life

18 months

3 months once opened

6.4 Special precautions for storage

Do not store above 25^{o}C.

6.5 Nature and contents of container

Bottles: Amber (Type III) glass

Closures:

Polypropylene Child Resistant Closures (CRCs) with LDPE liners

Capacity: 150 ml

6.6 Instructions for use and handling

Not applicable.

7. MARKETING AUTHORISATION HOLDER

Pinewood Laboratories Limited
Ballymacarbry
Clonmel
Co. Tipperary
Ireland

8. MARKETING AUTHORISATION NUMBER(S)

PL 04917/0073

9 DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

25/11/2024

10 DATE OF REVISION OF THE TEXT

25/11/2024