1. NAME OF THE MEDICINAL PRODUCT

DEXAMETHASONE TABLET BP 0.5 MG

2. QUALITATIVE & QUANTITATIVE COMPOSITION

Qualitative

Declaration Label

Claim:

Each uncoated tablets contains:
Dexamethasone BP 0.5 mg
Excipients Q.S

Excipients:

Di Calcium Phosphate
Maize Starch
Micro crystalline cellulose
Sod. Methyl Paraben
Sod. Propyl Paraben
P.V. P- K 30
Purified Water
Purified Talc
Magnesium Stearate
Sod. Starch Glycolate
Colloidal Anhydrous Silica

3. PHARMACEUTICAL FORM

Tablet

4. CLINICAL PARTICULARS

4.1 Therapeutic indications

Dexamethasone is indicated as a treatment for certain endocrine and non-endocrine disorders, in certain cases of cerebral oedema, and for diagnostic testing of adrenocortical hyperfunction. Endocrine disorders: Primary or secondary adrenocortical insufficiency, congenital adrenal hyperplasia. Non-endocrine disorders: Dexamethasone may be used in the treatment of non-endocrine corticosteroid responsive conditions, including:

Allergy and anaphylaxis: Angioneurotic oedema, anaphylaxis.
Arteritis collagenosis: Polymyalgia rheumatica, polyarteritis nodosa.

Blood disorders: Haemolytic anaemia, leukaemia, myeloma.
Cardiovascular disorders: Post-myocardial infarction syndrome.
Gastro-intestinal: Crohn's disease, ulcerative colitis.
Hypercalcaemia: Sarcoidosis.
Infections (with appropriate chemotherapy): Miliary tuberculosis.
Muscular disorders: Polymyositis.
Neurological disorders: Raised intra-cranial pressure secondary to cerebral tumours.
Ocular disorders: Anterior and posterior uveitis, optic neuritis.
Renal disorders: Lupus nephritis.
Respiratory disease: Bronchial asthma, aspiration pneumonitis.
Rheumatic disorders: Rheumatoid arthritis.
Skin disorders: Pemphigus vulgaris.

4.2 Posology and method of administration

DEXAMETHASONE TABLET 0.5 MG treatment should be initiated by physicians with experience in the diagnosis and treatment of atopic dermatitis.

Posology
General considerations: Dosage must be individualised on the basis of the disease and the response of the patient. In order to minimise side effects, the lowest possible dosage adequate to control the disease process should be used (see sec. 4.8).
The initial dosage varies from 0.5 mg to 9 mg a day depending on the disease being treated. In more severe diseases, doses higher than 9 mg may be required. The initial dosage should be maintained or adjusted until the patient's response is satisfactory. Both the dose in the evening, which is useful in alleviating morning stiffness, and the divided dosage regimen are associated with greater suppression of the hypothalamo-pituitary-adrenal axis. If satisfactory clinical response does not occur after a reasonable period of time, discontinue dexamethasone tablets and transfer the patient to other therapy.
After a favourable initial response, the proper maintenance dosage should be determined by decreasing the initial dosage in small amounts to the lowest dosage that maintains an adequate clinical response. Chronic dosage should preferably not exceed 1.5 mg dexamethasone daily.
Patients should be monitored for signs that might require dosage adjustment, including changes in clinical status resulting from remissions or exacerbations of the disease, individual drug responsiveness, and the effect of stress (e.g. surgery, infection, trauma). During stress it may be necessary to increase dosage temporarily.
To avoid hypoadrenalism and/or a relapse of the underlying disease, it may be necessary to withdraw the drug gradually
The following equivalents facilitate changing to dexamethasone from other glucocorticoids: Milligram for milligram, dexamethasone is approximately equivalent to betamethasone, 4 to

6 times more potent than methylprednisolone and triamcinolone, 6 to 8 times more potent than prednisone and prednisolone, 25 to 30 times more potent than hydrocortisone, and about 35 times more potent than cortisone.
In acute, self-limiting allergic disorders or acute exacerbations of chronic allergic disorders, the following dosage schedule combining parenteral and oral therapy is suggested

First day: Dexamethasone injection, 4 mg or 8 mg (1 ml or 2 ml) intramuscularly
Second day: Two 500 microgram dexamethasone tablets twice a day
Third day:Two 500 microgram dexamethasone tablets twice a day
Fourth day:One 500 microgram dexamethasone tablet twice a day
Fifth day:One 500 microgram dexamethasone tablet twice a day
Sixth day:One 500 microgram dexamethasone tablet
Seventh day:One 500 microgram dexamethasone tablet
Eighth day: Reassessment day
This schedule is designed to ensure adequate therapy during acute episodes while minimising the risk of overdosage in chronic cases.
Dexamethasone suppression tests:
1. Tests for Cushing's syndrome: 2 milligram dexamethasone is given orally at 11 p.m., then blood is drawn for plasma cortisol determination at 8 a.m. the following morning.
For greater accuracy, 500 microgram dexamethasone is given orally every 6 hours for 48 hours. Plasma cortisol is measured at 8 a.m. on the third morning. Twenty-four-hour urine collections are made for determination of 17-hydroxycorticosteroid excretion.
2. Test to distinguish Cushing's syndrome caused by pituitary ACTH excess from the syndrome induced by other causes: 2 milligram dexamethasone is given orally every 6 hours for 48 hours. Plasma cortisol is measured at 8 a.m. on the morning following the last dose.Twenty-four-hour urine collections are made for determination of 17 hydroxycorticosteroid excretion.
Use in children: Dosage should be limited to a single dose on alternate days to lessen retardation of growth and minimise suppression of hypothalamo-pituitary-adrenal axis.
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

4.3 Contraindications

Systemic fungal infections; systemic infection unless specific anti-infective therapy is employed; hypersensitivity to any component of the drug. Administration of live virus vaccines

4.4 Special warnings and precautions for use

There is an increased risk of systemic side-effects with CYP3A inhibitors (refer to section 4.5 drug interactions).
In post marketing experience tumour lysis syndrome (TLS) has been reported in patients with haematological malignancies following the use of dexamethasone alone or in combination with other chemotherapeutic agents. Patient at high risk of TLS, such as patients with high proliferative rate, high tumour burden, and high sensitivity to cytotoxic agents, should be monitored closely and appropriate precaution taken.

Undesirable effects may be minimised by using the lowest effective dose for the minimum period and when appropriate by administering the daily requirement as a single morning dose or whenever possible as a single morning dose on alternative days. Frequent patient review is required to appropriately titrate the dose against disease activity. When reduction in dosage is possible, the reduction should be gradual (see sec. 4.2).
Corticosteroids may exacerbate systemic fungal infections and should not be used in the presence of such infections unless they are needed to control life-threatening drug reactions due to amphotericin. Moreover, there have been cases reported in which concomitant use of amphotericin and hydrocortisone was followed by cardiac enlargement and heart failure.
Reports in the literature suggest an apparent association between use of corticosteroids and left-ventricular free-wall rupture after a recent myocardial infarction; therefore, corticosteroids should be used with great caution in these patients.

A report shows that the use of corticosteroids in cerebral malaria is associated with a prolonged coma and an increased incidence of pneumonia and gastro-intestinal bleeding.
Average and large doses of hydrocortisone or cortisone can cause elevation of blood pressure, retention of salt and water, and increased excretion of potassium, but these effects are less likely to occur with synthetic derivatives, except when used in large doses. Dietary salt restriction and potassium supplementation may be necessary. All corticosteroids increase calcium excretion.

In patients on corticosteroid therapy subjected to unusual stress (e.g. intercurrent illness, trauma, or surgical procedure), dosage should be increased before, during and after the stressful situation. Drug-induced secondary adrenocortical insufficiency may result from too rapid withdrawal of corticosteroids and may be minimised by gradual dosage reduction, being tapered off over weeks and months, depending on the dose and duration of treatment, but may persist for up to a year after discontinuation of therapy. In any stressful situation during that period, therefore, corticosteroid therapy should be reinstated. If the patient is already receiving corticosteroids, the current dosage may have to be temporarily increased.
Salt and/or a mineralocorticoid should be given concurrently, since mineralocorticoid secretion may be impaired.
Stopping corticosteroids after prolonged therapy may cause withdrawal symptoms including fever, myalgia, arthralgia, and malaise. This may occur in patients even without evidence of adrenal insufficiency.

In patients who have received more than physiological doses of systemic corticosteroids (approximately 1 mg dexamethasone) for greater than three 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 systemic corticosteroids may be reduced rapidly to physiological doses. Once a daily dose of 1 mg dexamethasone 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 three

weeks is appropriate if it is considered that the disease is unlikely to relapse. Abrupt withdrawal of doses of up to 6 mg daily of dexamethasone for three 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 three weeks or less:

• Patients who have had repeated courses of systemic corticosteroids, particularly if taken for greater than three 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 6 mg daily of dexamethasone.
• Patients repeatedly taking doses in the evening.

Patients should carry 'steroid treatment' cards, which give clear guidance on the precautions to be taken to minimise risk, and which provide details of prescriber, drug, dosage, and the duration of treatment.

Administration of live virus vaccines is contra-indicated in individuals receiving immunosuppressive doses of corticosteroids. If inactivated viral or bacterial vaccines are administered to individuals receiving immunosuppressive doses of corticosteroids, the expected serum antibody response may not be obtained. However, immunisation procedures may be undertaken in patients who are receiving corticosteroids as replacement therapy, e.g. for

Addison's disease.

Special precautions: Particular care is required when considering the use of systemic corticosteroids in patients with the following conditions, and frequent patient monitoring is necessary: renal insufficiency, hypertension, diabetes or in those with a family history of diabetes, congestive heart failure, osteoporosis, previous steroid myopathy, glaucoma (or family history of glaucoma),myasthenia gravis, non-specific ulcerative colitis, diverticulitis fresh intestinal anastomosis, active or latent peptic ulcer, existing or previous history of severe affective disorders (especially previous steroid psychosis), liver failure, and epilepsy.Signs of peritoneal irritation following gastro-intestinal perforation in patients receiving large doses of corticosteroids may be minimal or absent. Fat embolism has been reported as a possible complication of hypercortisonism.

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 three months; this should be given within ten days of exposure to chickenpox. If a diagnosis of chickenpox is confirmed, the illness warrants specialist care and urgent treatment. Corticosteroids should not be stopped and the dose may need to be increased.

The use of dexamethasone in active tuberculosis should be restricted to those cases of fulminating or disseminated tuberculosis in which the corticosteroid is used for the management of the disease in conjunction with an appropriate antituberculous regimen. If corticosteroids are indicated in patients with latent tuberculosis or tuberculin reactivity, close observation of the disease is necessary as reactivation may occur. During prolonged corticosteroid therapy, these patients should receive prophylactic chemotherapy.

There is an enhanced effect of corticosteroids in patients with hypothyroidism and in those

with cirrhosis.

Corticosteroids may mask some signs of infection, and new infections may appear during their use. 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 reach an advanced stage before being recognised. There may be decreased resistance and inability to localise infection in patients on corticosteroids.
Measles can have a more serious or even fatal course in immunosuppressed patients. In such children or adults particular care should be taken to avoid exposure to measles. If exposed ,prophylaxis with intramuscular pooled immunoglobulin (IG) may be indicated. Exposed patients should be advised to seek medical advice without delay.
Corticosteroids may activate latent amoebiasis or strongyloidiasis or exacerbate active disease.Therefore, it is recommended that latent or active amoebiasis and strongyloidiasis be ruled out before initiating corticosteroid therapy in any patient at risk of or with symptoms suggestive of either condition.
Prolonged use of corticosteroids may produce subcapsular cataracts, glaucoma with possible damage to the optic nerves, and may enhance the establishment of secondary ocular infections due to fungi or viruses. Steroids may increase or decrease the motility and number of spermatozoa.
Corticosteroids should be used cautiously in patients with ocular herpes simplex, because of possible corneal perforation. Patients/and or carers should be warned that potentially severe psychiatric adverse reactions may occur with systemic steroids. Symptoms typically
emerge within a few days or weeks of starting the treatment. Risks may be higher with high doses/ systemic exposure, 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 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. Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.
Preterm neonates: Available evidence suggests long-term neurodevelopmental adverse events after early treatment (< 96 hours) of premature infants with chronic lung disease at starting doses of 0.25 mg/kg twice daily.

4.5 Interaction with other medicinal products and other forms of interaction
Dexamethasone should be used with caution with thalidomide, as toxic epidermal necrolysis has been reported with concomitant administration of these two drugs.
Aspirin should be used cautiously in conjunction with corticosteroids in hypoprothrombinaemia.
The renal clearance of salicylates is increased by corticosteroids and, therefore, salicylate dosage should be reduced along with steroid withdrawal.
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.

Dexamethasone is metabolised by cytochrome P450 3A4 (CYP 3A4).
Concomitant administration of dexamethasone with cytochrome P450 3A4 enzyme inducers(e.g. phenytoin, barbiturates, rifabutin, carbamazepine, and rifampicin), may enhance the metabolic clearance of corticosteroids, resulting in decreased blood levels and reduced physiological activity. This may necessitate adjustment of the dosage of Dexamethasone. In addition, the concomitant administration of dexamethasone with known inhibitors of CYP 3A4 (e.g. ketoconazole, macrolide antibiotics such as erythromycin) has the potential to result in increased plasma concentrations of dexamethasone. Effects of other drugs on the metabolism of dexamethasone may interfere with dexamethasone suppression tests, which should be interpreted with caution during administration of such drugs.
Dexamethasone is a moderate inducer of CYP 3A4. Co-administration with other drugs that are metabolised by CYP 3A4 (e.g. erythromycin and anti-HIV drugs such as indinavir, ritonavir, lopinavir, saquinavir) may increase their clearance, resulting in decreased plasma concentrations. In post-marketing experience, there have been reports of both increases and decreases in phenytoin levels with dexamethasone co-administration, leading to alterations in seizure control. Although ketoconazole may increase dexamethasone plasma concentrations through inhibition of CYP 3A4, ketoconazole alone can inhibit adrenal corticosteroid synthesis and may cause adrenal insufficiency during corticosteroid withdrawal.
Aminoglutethimide and ephedrine may enhance metabolic clearance of corticosteroids and an increase in corticosteroid dosage may be necessary. False-negative results in the dexamethasone suppression test in patients being treated with indomethacin have been reported.
The prothrombin time should be checked frequently in patients who are receiving corticosteroids and coumarin anticoagulants at the same time as there have been reports that corticosteroids have altered the response to these anticoagulants. Studies have shown that the usual effect produced by adding corticosteroids is inhibition of response to coumarins,although there have been some conflicting reports of potentiation not substantiated by studies.
The desired effects of hypoglycaemic agents (including insulin) are antagonised by corticosteroids.
When corticosteroids are administered concomitantly with potassium-depleting diuretics, patients should be observed closely for development of hypokalaemia. Corticosteroids may affect the nitro blue tetrazolium test for bacterial infection and produce false-negative results.

4.6 Fertility, pregnancy and

lactation <u>Pregnancy</u>

The ability of corticosteroids to cross the placenta varies between individual drugs, however, dexamethasone readily crosses the placenta.
Administration of corticosteroids to pregnant animals can cause abnormalities of foetal development including cleft palate, intrauterine 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 (see also sec. 5.3).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 maybe treated as

though they were in the non-gravid state.
Corticosteroids may pass into breast milk, although no data are available for dexamethasone. Infants

of mothers taking high doses of systemic corticosteroids for prolonged periods may have a degree of adrenal suppression.

4.7 Effects on ability to drive and use machines

No studies on the effects on the ability to drive and use machines have been conducted.

4.8 Undesirable effects

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 sec. 4.4).
Fluid and electrolyte disturbances: Sodium retention, fluid retention, congestive heart failure in susceptible patients, potassium loss, hypokalaemic alkalosis, hypertension, increased calcium excretion (see sec. 4.4).
Musculoskeletal effects: Muscle weakness, steroid myopathy, loss of muscle mass, osteoporosis (especially in post-menopausal females), vertebral compression fractures, aseptic necrosis of femoral and humeral heads, pathological fracture of long bones, tendon rupture.
Gastro-intestinal: Peptic ulcer with possible perforation and haemorrhage, perforation of the small and large bowel particularly in patients with inflammatory bowel disease, pancreatitis, abdominal distension, ulcerative oesophagitis, dyspepsia, oesophageal candidiasis.
Dermatological: Impaired wound healing, thin fragile skin, petechiae and ecchymoses, erythema, striae, telangiectasia, acne, increased sweating, suppressed reaction to skin tests, other cutaneous reactions such as allergic dermatitis, urticaria, angioneurotic oedema.
Neurological: Convulsions, vertigo, headache. Increased intracranial pressure with papilloedema (pseudotumour cerebri) may occur usually after treatment.
Psychiatric: 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 have been estimated to be 5-6%. Psychological effects have been reported on withdrawal of corticosteroids; the frequency is unknown.
Endocrine: Menstrual irregularities, amenorrhoea, development of Cushingoid state, suppression of growth in children and adolescents, secondary adrenocortical and pituitary unresponsiveness (particularly in times of stress as in trauma, surgery or illness), decreased carbohydrate tolerance, manifestations of latent diabetes mellitus, hyperglycaemia, increased requirements for insulin or oral hypoglycaemic agents in diabetics, hirsutism.
Anti-inflammatory and immunosuppressive effects: Increased susceptibility and severity of infections with suppression of clinical symptoms and signs. Opportunistic infections, recurrence of dormant tuberculosis (see sec. 4.4).
Eye disorders: Posterior subcapsular cataracts, increased intra-ocular pressure, papilloedema,

corneal or scleral thinning, exacerbation of ophthalmic viral disease, glaucoma,exophthalmos, chorioretinopathy.

Metabolic: Negative nitrogen balance due to protein catabolism. Negative calcium balance.
Cardiovascular: Myocardial rupture following recent myocardial infarction (see sec. 4.4).
Other: Hypersensitivity, including anaphylaxis has been reported, leucocytosis, thromboembolism, weight gain, increased appetite, nausea, malaise, hiccups. Withdrawal symptoms and signs
Too rapid a reduction of corticosteroid dosage following prolonged treatment can lead to acute adrenal insufficiency, hypotension, and death (see sec. 4.4).
In some instances, withdrawal symptoms may simulate a clinical relapse of the disease for which the patient has been undergoing treatment.

4.9 Overdose

Reports of acute toxicity and/or deaths following overdosage with glucocorticoids are rare. No antidote is available. Treatment is probably not indicated for reactions due to chronic poisoning unless the patient has a condition that would render him unusually susceptible to ill effects from corticosteroids. In this case, the stomach should be emptied and symptomatic treatment should be instituted as necessary.Anaphylactic and hypersensitivity reactions may be treated with epinephrine (adrenaline), positive-pressure artificial respiration and aminophylline. The patient should be kept warm and quiet.
The biological half-life of dexamethasone in plasma is about 190 minutes.

5. Pharmacological properties

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Glucocorticoids

ATC code: H02AB02

Dexamethasone is a glucocorticoid. It possesses the actions and effects of other basic glucocorticoids, and is among the most active members. Glucocorticoids are adrenocortical steroids, both naturally occurring and synthetic, which are readily absorbed from the gastrointestinal tract. They cause profound and varied metabolic effects and in addition they modify the body's immune responses to diverse stimuli.

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

5.2 Pharmacokinetic properties

Dexamethasone is readily absorbed from the gastro-intestinal tract.

Its biological half-life in plasma is about 190 minutes.

Binding of dexamethasone to plasma proteins is less than for most other corticosteroids and is estimated to be about 77%.

Up to 65% of a dose is excreted in the urine in 24 hours, the rate of excretion being increased following concomitant administration of phenytoin.

The more potent halogenated corticosteroids such as dexamethasone, appear to cross the placental barrier with minimal inactivation.

Dexamethasone has predominant glucocorticoid activity with little propensity to promote renal retention of sodium and water. Therefore, it does not offer complete replacement therapy, and must be supplemented with salt and/or deoxycorticosterone. Cortisone and hydrocortisone also act predominately as glucocorticoids, although their mineralocorticoid action is greater than that of dexamethasone. Their use in patients with total adrenocortical insufficiency also may require supplemental salt, deoxycortisone, or both.

5.3 Preclinical safety data

In animal studies, cleft palate was observed in rats, mice, hamsters, rabbits, dogs and primates; not in horses and sheep. In some cases these divergences were combined with defects of the central nervous system and of the heart. In primates, effects in the brain were seen after exposure. Moreover, intrauterine growth can be delayed. All these effects were seen at high dosages..

6. Pharmaceutical particulars

6.1 List of excipients

Di Calcium Phosphate
Maize Starch
Micro crystalline cellulose
Sod. Methyl Paraben
Sod. Propyl Paraben
P.V. P- K 30
Purified Water
Purified Talc
Magnesium Stearate
Sod. Starch Glycolate
Colloidal Anhydrous Silica

6.2 Incompatibilities: Not Applicable.

6.3 Shelf life: 36 months.

6.4 Special precautions for storage Store at temperature not exceeding 30^{O} C in dry place.

6.5 Nature and contents of container

100 Tablets packed in Amber glass bottle packed in carton with pack insert.

6.6 Special precautions for disposal and other handling

No special requirements.

7 Manufacturer:
Name & address:
Dupen Laboratories PVT. LTD.
C1-49 & 36, Degam Road,
Industrial Township,
Vapi-396195 (Gujarat

8. **Marketing Authorisation Holder**:
Mercury Healthcare Pvt. Ltd,
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Loyalka Estate,
Chowpatty Band Stand,
Mumbai – 400 006, INDIA