DELTACORTRIL® Tablets
(Prednisolone)

1. NAME OF THE MEDICINAL PRODUCT

DELTACORTRIL®

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Active ingredient: prednisolone.

Tablets containing 5 mg of prednisolone.
Enteric coated tablets containing 5 mg of prednisolone.

3. PHARMACEUTICAL FORM

Tablets
Enteric Coated Tablets

4. CLINICAL PARTICULARS

4.1. THERAPEUTIC INDICATIONS

Unspecified therapy in conditions where the anti-inflammatory and immunosuppressive effects of prednisolone are desired. Examples include rheumatoid arthritis, SLE, certain kinds of vasculitis, such as temporal arteritis and periarteritis nodosa, sarcoidosis, bronchial asthma, ulcerative colitis, haemolytic anaemia and granulocytopenia, as well as serious allergic conditions.

Treatment of tumours, in certain cases of acute leukaemia, lymphoma, breast cancer and prostate cancer.

4.2. POSOLOGY AND METHOD OF ADMINISTRATION

Posology
Individual. Generally, 10-30 mg daily. In very serious, acute cases up to 50-60 mg or more can be given for several days. When a satisfactory effect has been reached, the daily dose should be reduced by 2.5-5 mg every second to every fifth day (more rapidly for the highest doses) to the lowest possible maintenance dose. It is desirable that this does not exceed 10 mg/24 hours.

If the whole maintenance dose is given in the morning (at 8 a.m.), prednisolone acts concurrently with the natural daily adrenocortical rhythm and results in minimal adrenocortical inhibition. This type of dosing may generally be tried initially; however, in some cases, e.g., in rheumatic patients with pronounced morning stiffness and patients with asthma in need of corticosteroid therapy during the night, a late evening dose or a divided dose may be preferred.

In certain cases of asthma, allergic conditions, dermatoses, etc., a double daily dose, as an undivided dose once every second day in the morning, would be preferred.

The therapy should be discontinued gradually, especially after high doses.

The dose should be discontinued gradually, since the patient's own ACTH secretion may be reduced after long-term treatment.

An increased dose may be given before, during and after stress situations.

An increased dose in case of fever and stress.

An increased dose of insulin should be given to diabetic patients during treatment with cortisone.

4.3. CONTRAINDICATIONS

Hypersensitivity to the active substance or to any of the excipients listed in section 6.1. LIST OF EXCIPIENTS.

Systemic fungal infections.

Administration of live vaccines is contraindicated in patients receiving immunosuppressive doses of corticosteroids.

In general, none of contraindications apply in conditions when treatment with prednisolone may be life-saving.

4.4. SPECIAL WARNINGS AND PRECAUTIONS FOR USE

As the complications of glucocorticoid treatment depend on the dose and length of treatment, a risk/benefit assessment must be made in each individual case concerning dose and treatment length, and also if daily or intermittent treatment must be used.

The lowest possible corticosteroid dose that is needed to control the disease being treatment must be used. When dose reduction is possible, this must occur gradually.

Immunosuppressive effects/increased susceptibility to infection
Glucocorticoids, including prednisolone, may increase susceptibility to infection, mask some symptoms of infection and new infections may appear during treatment. Infections caused by virus, bacteria, fungi, protozoa or helminths may be associated with the use of corticosteroids alone or in combination with other immunosuppressive agents that affect cellular immunity, humoral immunity, or neutrophil function. These infections may be mild, but can be severe and at times fatal. The risk of infectious complications increases with increased dose.

Glucocorticoids should not be given in case of infections without concomitant causal treatment.

Chicken pox and measles can have a more serious or even fatal course in non-immunised children and adults who are treated with corticosteroids. Children, or adults who have not had these diseases, and who take immunosuppressant doses of corticosteroids should be warned to avoid exposure to chicken pox and measles and, if exposed, to obtain medical advice.

The use of prednisolone in active tuberculosis should be restricted to those cases of fulminating or disseminated tuberculosis in which the corticosteroid is used for treatment of the disease in combination with an appropriate tuberculosis treatment. If corticosteroids are indicated in patients with latent

tuberculosis or tuberculin reactivity, close observation is necessary as reactivation of the disease may occur. During prolonged corticosteroid therapy, these patients should receive tuberculosis prophylaxis.

High doses of corticosteroids may interfere with active immunisation. Vaccination with live vaccine should be performed under close monitoring and not to patients who are on long-term treatment with immunosuppressive doses of corticosteroids.

Immune system disorders
Because rare instances of skin reactions and anaphylactic/anaphylactoid reactions have occurred in patients being treated with corticosteroids, appropriate precautionary measures should be taken prior to administration, especially when the patient has previously had an allergic reaction to any drug.

Endocrine disorders
Long-term treatment with pharmacological doses of corticosteroids may lead to secondary adrenocortical insufficiency. The risk can be reduced by administering the treatment every second day (see section 4.2. POSOLOGY AND METHOD OF ADMINISTRATION).

Patients on corticosteroid maintenance therapy who are subjected to unusual stress (e.g. infection, surgery, trauma) require increased corticosteroid dosage before, during, and after the stressful situation.

Abrupt withdrawal of the treatment may lead to acute adrenal insufficiency, which can be fatal. The risk of secondary adrenocortical insufficiency can be minimised by gradual reduction of dosage. This type of relative insufficiency may persist for months after discontinuation of therapy; therefore, in any situation of stress occurring during that period, hormone therapy should be reinstituted. Since mineralocorticoid secretion may be impaired, salt and/or a mineralocorticoid should be administered concurrently.

A “steroid withdrawal syndrome,” seemingly unrelated to adrenocortical insufficiency, may also occur following abrupt discontinuation of glucocorticoids. This syndrome includes symptoms such as anorexia, nausea, vomiting, lethargy, headache, fever, joint pain, desquamation, myalgia, 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.

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

Pheochromocytoma-related crisis, which can be fatal, has been reported after administration of systemic corticosteroids. Corticosteroids should only be administered to patients with suspected or identified pheochromocytoma after consideration of the individual risk/benefit.

Metabolism and nutrition disorders
Corticosteroids, including prednisolone, can increase blood glucose, worsen pre-existing diabetes, and increase the risk of developing diabetes in patients who are on long-term treatment with corticosteroids.

Psychiatric disorders
Potentially serious psychiatric disorders may occur during treatment with corticosteroids, including prednisolone. This can range from euphoria, sleep disorders, mood swings, personality changes, and severe depression, to psychotic manifestations. Also, existing emotional instability or psychotic tendencies may be aggravated by corticosteroids (see section 4.8. UNDESIRABLE EFFECTS). Symptoms typically emerge within a few days or weeks of starting treatment. Most reactions resolve after either dose reduction or withdrawal, although specific treatment may be necessary.

Psychological effects have been reported upon withdrawal of corticosteroids; the frequency is unknown. Patients/caregivers should be encouraged to seek medical attention if psychological symptoms develop in the patient, especially if depressed mood or suicidal ideation is suspected. Patients/caregivers should be alert to possible psychiatric disturbances that may occur either during or immediately after dose tapering/withdrawal of systemic steroids.

Nervous system disorders
Corticosteroids should be used with caution in patients with seizure disorders.

Cardiac disorders
Adverse effects of glucocorticoids on the cardiovascular system, such as dyslipidaemia and hypertension, may predispose treated patients with existing cardiovascular risk factors to additional cardiovascular events at high doses and prolonged treatment time. Accordingly, corticosteroids should be employed judiciously in such patients, and attention should be paid to risk modification and additional cardiac monitoring if needed. Low dose and alternate day therapy may reduce the incidence of complications in corticosteroid therapy.

Vascular disorders
Because cortisone has been reported in rare cases to increase blood coagulability and thus could precipitate intravascular thrombosis, thromboembolism, and thrombophlebitis, corticosteroids should be used with caution in patients with thromboembolic disorders.

Gastrointestinal disorders
High doses of corticosteroids may cause acute pancreatitis.

There are no universal data that establishes whether corticosteroids cause stomach ulcers. Glucocorticoid therapy may mask peritonitis or other signs or symptoms associated with gastrointestinal disorders such as perforation, obstruction or pancreatitis. In combination with NSAIDs, the risk of developing gastrointestinal ulcers is increased.

Corticosteroids should be used with caution in nonspecific ulcerative colitis if there is a probability of impending perforation, abscess or other pyogenic infection, diverticulitis, fresh intestinal anastomoses, or active or latent peptic ulcer.

Hepatobiliary disorders
Hepatobiliary disorders have been reported in rare cases, and in the majority of these cases the condition was reversible after discontinuation of therapy. Therefore appropriate monitoring is required.

Musculoskeletal and connective tissue disorders
Acute myopathy has been reported with the use of high doses of corticosteroids, most often occurring in patients with disorders of neuromuscular transmission (e.g., myasthenia gravis) or in patients receiving concomitant therapy with anticholinergics, such as neuromuscular blocking drugs (e.g., pancuronium) (see section 4.5. INTERACTION WITH OTHER MEDICINAL PRODUCTS AND OTHER FORMS OF INTERACTION). This acute myopathy is generalised, may involve ocular and respiratory muscles, and may result in quadriparesis. Elevations of creatine kinase may occur. Clinical improvement or recovery after stopping corticosteroids may require weeks to years.

Corticosteroids should be used with caution in patients with osteoporosis.

Renal and urinary disorders
Corticosteroids should be used with caution in patients with renal insufficiency.

Acute renal crisis (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.

Effect on electrolytes and fluid balance
Systemic corticosteroids should be used with caution in patients with heart failure or hypertension. Average and large doses of hydrocortisone or cortisone can cause elevation of blood pressure, salt and water retention, and increased excretion of potassium. These effects are less likely to occur with the synthetic derivatives except when used in large doses. Dietary salt restriction and potassium supplementation may be necessary.

All corticosteroids increase calcium excretion.

Eye disorders
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.

Paediatric patients
Corticosteroids cause growth retardation in infants, children and adolescents and, therefore, long-term administration of pharmacological doses should be avoided. If prolonged therapy is necessary, the growth and development of infants and children should be closely monitored (see section 4.2. POSOLOGY AND METHOD OF ADMINISTRATION).

Infants and children on prolonged corticosteroid therapy are at special risk from elevated intracranial pressure.

Tumor lysis syndrome (TLS)
In post marketing experience tumor lysis syndrome (TLS) has been reported in patients with malignancies, including hematological malignancies and solid tumors, following the use of systemic corticosteroids alone or in combination with other chemotherapeutic agents. Patients at high risk of TLS, such as patients with tumors that have a high proliferative rate, high tumor burden and high sensitivity to cytotoxic agents, should be monitored closely and appropriate precautions should be taken.

Excipients
Patients with rare hereditary conditions such as 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

The following combinations with Prednisolone Pfizer may require dose adjustment.

Phenobarbital, phenytoin and carbamazepine: Phenobarbital (which is also a metabolite of primidone), phenytoin and carbamazepine, either as monotherapy or in combination, induce the metabolism of

hydrocortisone, prednisolone and methyl prednisolone (shown in children with asthma), resulting in a need for a dose increase. This interaction probably concerns the whole group of glucocorticoids.

Non-steroidal anti-inflammatory drugs:
1) The incidence of gastrointestinal bleeding and ulceration may increase if corticosteroids are given together with an NSAID.
2) Corticosteroids may increase clearance of high doses of acetylsalicylic acid, which can lead to lower salicylate levels in serum. When withdrawing corticosteroid treatment, the salicylate levels in serum can increase, which will probably lead to increased risk of toxic effects from salicylate.

Diabetic medicine: Glucocorticoids increase blood sugar content. Patients with diabetes mellitus who simultaneously receive insulin and/or oral hypoglycaemic products may need the doses of such treatment adjusted.

Oestrogens (including oral contraceptives containing oestrogens): Oestrogens increase the concentration of transcortin. The effect of glucocorticoids that bind to transcortin may be potentiated and dose adjustment may be needed if oestrogens are added to or removed from a stable treatment regimen.

Potassium-reducing products: Potassium-reducing diuretics (e.g. thiazides, furosemide, ethacrynic acid) and other medicinal products that reduce the quantity of potassium, such as amphotericin B, xanthines and beta-2 agonists, may potentiate the potassium-reducing effect of glucocorticoids. Serum-potassium should be monitored closely in patients who receive glucocorticoids and potassium-reducing products.

Rifampicin: Rifampicin induces the microsomal oxidation of glucocorticoids (hydrocortisone, prednisolone, methyl prednisolone). This leads to increased steroid requirements during treatment with rifampicin and reduced steroid requirements after such treatment.

Isoniazid: Prednisolone also has a potential effect that leads to increased acetylation rate and clearance of isoniazid.

Oral anticoagulants: Changed effect from anticoagulants has been reported when given in combination with prednisolone. The prothrombin time (INR) should be monitored during the treatment.

CYP3A inhibitors, including cobicistat-containing products: These are 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.

Anticholinergic, neuromuscular blockers: Corticosteroids can affect the effect of anticholinergics.
1) Acute myopathy has been reported in concomitant use of high doses of corticosteroids and anticholinergics, such as neuromuscular blockers (see section 4.4. SPECIAL WARNINGS AND PRECAUTIONS FOR USE).
2) Antagonism with the neuromuscular blocking effect of pancuronium and vecuronium has been reported in patients who take glucocorticosteroids. This interaction may be expected with all competitive neuromuscular blockers.

Anticholinesterases: Interaction between glucocorticoids and anticholinesterases such as ambenonium, neostigmine and pyridostigmine may lead to significant weakness in persons with myasthenia gravis. If possible, treatment with anticholinesterase should be discontinued at least 24 hours before administering glucocorticoid treatment.

4.6. FERTILITY, PREGNANCY AND LACTATION

Fertility
Animal studies have shown that corticosteroids impair fertility (see section 5.3. PRECLINICAL SAFETY DATA).

Pregnancy
Some animal studies have shown that corticosteroids may cause malformations of various types (cleft palate, skeletal malformation, see section 5.3. PRECLINICAL SAFETY DATA). The relevance to humans is unknown. Reduced placental and birth weight have been established following long-term treatment in humans and animals. There is also a risk in long-term treatment of adrenocortical failure in the newborn child. During pregnancy, corticosteroids should therefore be given after special consideration.

Breast-feeding
Prednisolone passes into breast milk but the risk of effect on the child is considered unlikely at therapeutic doses.

4.7. EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

The effect of corticosteroids on the ability to drive or use machinery has not been systematically evaluated. Undesirable effects, such as dizziness, visual disturbances and fatigue are possible after treatment with corticosteroids. If such side effects occur, patients should not drive or operate machinery.

4.8. UNDESIRABLE EFFECTS

Apart from substitution therapy, treatment with corticosteroids always constitutes an overdose, compared with the physiological state. Side effects occur predominantly during long-term treatment, but they also depend on dose size and individual sensitivity.

The following undesirable effects have been observed and reported during treatment with prednisolone 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); not known (cannot be estimated from available data).

Organ system	**Very Common ≥ 1/10**	**Common ≥ 1/100, < 1/10**	**Uncommon ≥ 1/1,000, < 1/100**	**Rare ≥ 1/10,000, < 1/1,000**	**Very Rare < 1/10,000**	**Frequency not known (cannot be estimated from the available data)**
Infections and infestations		Opportunistic infection Activation of infection (e.g. tuberculosis)				
Blood and lymphatic system disorders						Leukocytosis (due to a redistribution of intravascular granulocytes)
Immune system disorders						Drug hypersensitivity Anaphylactic reaction

Organ system	**Very Common ≥ 1/10**	**Common ≥ 1/100, < 1/10**	**Uncommon ≥ 1/1,000, < 1/100**	**Rare ≥ 1/10,000, < 1/1,000**	**Very Rare < 1/10,000**	**Frequency not known (cannot be estimated from the available data)**
						Anaphylactoid reaction
Endocrine disorders		Hypothalamic pituitary adrenal axis suppression, Cushingoid. Growth inhibition (in children).				Steroid withdrawal syndrome (see section 4.4. SPECIAL WARNINGS AND PRECAUTIONS FOR USE) Pheochromocytosis-related crisis (see section 4.4. SPECIAL WARNINGS AND PRECAUTIONS FOR USE).
Metabolism and nutrition disorders		Hypokalaemia Sodium retention Increased gluconeogenesis Catabolic effects Osteoporosis				Metabolic acidosis Fluid retention Alkalosis hypokalaemic Dyslipidaemia Glucose tolerance impaired (diabetes mellitus may be exacerbated and latent diabetes may manifest) Lipomatosis Increased appetite (which may result in weight increase)
Psychiatric disorders			Activation of previous psychological disturbances (high dose)	Depression, mania in patients with no prior history of mental illness		Affective disorder (including euphoric mood, affect lability, drug dependence, suicidal ideation) Psychotic disorder (including delusion, hallucination, and schizophrenia) Mental disorder Personality change Confusional state Anxiety Mood swings Abnormal behaviour Insomnia Irritability
Nervous system disorders				Benign intracranial		Epidural lipomatosis

Organ system	**Very Common ≥ 1/10**	**Common ≥ 1/100, < 1/10**	**Uncommon ≥ 1/1,000, < 1/100**	**Rare ≥ 1/10,000, < 1/1,000**	**Very Rare < 1/10,000**	**Frequency not known (cannot be estimated from the available data)**
				hypertension		Seizure Amnesia Cognitive disorder Dizziness Headache
Eye disorders			Cataract Glaucoma			Central serous chorioretinopathy (see section 4.4. SPECIAL WARNINGS AND PRECAUTIONS FOR USE) Exophthalmos Blurred vision (see section 4.4. SPECIAL WARNINGS AND PRECAUTIONS FOR USE)
Cardiac disorders						Cardiac failure (in susceptible patients) Bradycardia*
Vascular disorders		Oedema Hypertension				Thromboembolic events
Respiratory, thoracic and mediastinal disorders						Hiccups
Gastrointestinal disorders						Peptic ulcer (with possible perforation and haemorrhage) Intestinal perforation Pancreatitis Oesophagitis ulcerative Abdominal distention Abdominal pain Diarrhoea Dyspepsia Nausea
Skin and subcutaneous tissue disorders		Skin atrophy Poor wound healing				Angioedema Hirsutism Petechiae Ecchymosis Erythema Hyperhidrosis Skin striae Pruritus Urticaria

Organ system	Very Common ≥ 1/10	Common ≥ 1/100, < 1/10	Uncommon ≥ 1/1,000, < 1/100	Rare ≥ 1/10,000, < 1/1,000	Very Rare < 1/10,000	Frequency not known (cannot be estimated from the available data)
						Acne
Musculoskeletal and connective tissue disorders		Muscle atrophy		Aseptic bone necrosis Tendon rupture		Muscular weakness Myalgia Myopathy Pathological fracture Neuropathic arthropathy Arthralgia
Renal and urinary disorders						Acute renal crisis (renal crisis in scleroderma)**
Reproductive system and breast disorders						Menstruation irregular
General disorders and administration site conditions						Fatigue Malaise
Investigations						Urine calcium increased Alanine aminotransferase increased Aspartate aminotransferase increased Blood alkaline phosphatase increased Blood urea increased Suppression of reactions to skin tests[1]

[1] Not a MedDRA term.
*Following high doses
**Acute renal crisis (scleroderma renal crisis)
Amongst the different subpopulations, 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%)

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.

4.9. OVERDOSE

Reports of acute toxicity and/or death following overdose of glucocorticoids are rare. Acute overdose may possibly aggravate pre-existing disease states, e.g. ulcer, electrolyte imbalance, infections and oedema.

Treatment: Generally not required. If justified, gastric lavage, charcoal. In the event of overdose, no specific antidote is available; treatment is supportive and symptomatic.

5. PHARMACOLOGICAL PROPERTIES

5.1. PHARMACODYNAMIC PROPERTIES

Pharmacotherapeutic group: Glucocorticoids
ATC code: H02AB06

Synthetic glucocorticoid with anti-inflammatory, immunosuppressive and anti-allergenic effects. Prednisolone has a per unit of weight anti-inflammatory effect 4-5 times higher than cortisone, but affects electrolyte metabolism to a lesser extent. The mechanism of action has not yet been fully verified.

5.2. PHARMACOKINETIC PROPERTIES

Absorption
Prednisolone is rapidly absorbed from the gastrointestinal tract when administered orally. Peak plasma concentrations are reached 1 to 2 hours after oral administration. Usual plasma half-life is 2 to 4 hours. Its initial absorption, but not its overall bioavailability, is affected by food.

Distribution
Prednisolone is extensively bound to plasma proteins and has a high affinity to transcortin. The volume of distribution and clearance are reported to increase with an increase from low to moderate doses.

Metabolism
Prednisolone is metabolised primarily in the liver to a biologically inactive compound. Prednisolone can be reversibly converted to prednisone by 11β-hydroxysteroid dehydrogenase.

The absolute bioavailability of prednisolone is, on average, 82% compared with intravenously administered prednisolone after a single dose of 10 mg. With normal dosage, the effect duration is calculated to be 12-36 hours.

Elimination
Prednisolone is excreted in the urine as free and conjugated metabolites, together with small amounts of unchanged prednisolone. More than 90% of the administered amount is excreted in the urine. 7-15% is excreted in unchanged form.

5.3. PRECLINICAL SAFETY DATA

In animal trials, corticosteroids have been shown to cause various types of malformation (cleft palate, skeletal malformations). After long-term treatment in animals, reduced placenta and birth weights have been observed.

Corticosteroids have been shown to reduce fertility when administered to rats.

6. PHARMACEUTICAL PARTICULARS

6.1. LIST OF EXCIPIENTS

Magnesium stearate
Gelatine
Microcrystalline cellulose
Talc
Potato starch
Lactose monohydrate

6.2. INCOMPATIBILITIES

Not applicable.

6.3. SHELF LIFE

Please see pack for expiry of product.

6.4. SPECIAL PRECAUTIONS FOR STORAGE

No special precautions for storage.

6.5. NATURE AND CONTENTS OF CONTAINER

- DELTACORTRIL® (prednisolone) 5 mg Tablets: pack of 1000s in HDPE bottle.
- DELTACORTRIL® (prednisolone) 5 mg Enteric Coated Tablets: pack of 100s in HDPE bottle.

Not all pack size will be marketed.

6.6. SPECIAL PRECAUTION FOR DISPOSAL AND HANDLING

No special requirement

6.7 DRUG PRODUCT SPECIFICATION

DELTACORTRIL® (prednisolone) 5 mg Tablets:
BP Specs.

DELTACORTRIL® (prednisolone) 5 mg Enteric Coated Tablets:
Pfizer Specs.

7. REGISTRATION HOLDER / MARKETING AUTHORISATION HOLDER

Pfizer Pakistan Limited
B-2, S.I.T.E., Karachi.

7.1. MANUFACTURER

Name of Manufacturing site	Address of site	Manufacturing step (if applicable)
Pfizer Pakistan Limited	B-2, S.I.T.E., Karachi, Pakistan	Manufacturing, Packaging & Batch Release

8. REGISTRATION / MARKETING AUTHORISATION NUMBER

DELTACORTRIL® (prednisolone) 5 mg Tablets: 000448
DELTACORTRIL® (prednisolone) 5 mg Enteric Coated Tablets: 053218

9. DATE FROM WHICH MARKETING IS AUTHORIZED

DELTACORTRIL® (prednisolone) 5 mg Tablets:
17-Apr-1976
DELTACORTRIL® (prednisolone) 5 mg Enteric Coated Tablets:
26-Nov-2008

10. DATE OF REVISION OF THE TEXT

Deltacortril/LPD/PK-02
According to Sweden Approved SmPC dated: 27 October 2023 & approved information in Pakistan

Marketed by:

Pfizer Pakistan Limited.

*Please visit our website **www.pfizerpro.com.pk** for latest version of Product leaflet.*