SUMMARY OF PRODUCT CHARACTERISTICS

1. NAME OF THE MEDICINAL PRODUCT

Prednisolon Omet Pharma 2.5 mg tablets
Prednisolon Omet Pharma 5 mg tablets
Prednisolon Omet Pharma 10 mg tablets
Prednisolon Omet Pharma 25 mg tablets

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Each Prednisolon Omet Pharma 2.5 mg tablet contains 2.5 mg of prednisolone.
Each Prednisolon Omet Pharma 5 mg tablet contains 5 mg of prednisolone.
Each Prednisolon Omet Pharma 10 mg tablet contains 10 mg prednisolone.
Each Prednisolon Omet Pharma 25 mg tablet contains 25 mg prednisolone.

Excipient(s) with known effect

Each Prednisolon Omet Pharma 2.5 mg tablet contains 39 mg lactose monohydrate.
Each Prednisolon Omet Pharma 5 mg tablet contains 39 mg lactose monohydrate.
Each Prednisolon Omet Pharma 10 mg tablet contains 39 mg lactose monohydrate.
Each Prednisolon Omet Pharma 25 mg tablet contains 98 mg lactose monohydrate.

For the full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

Tablets

Prednisolon Omet Pharma 2.5 mg tablets:
Slightly red, round, plain tablet

Prednisolon Omet Pharma 5 mg tablets:
Slightly yellow, round, plain tablet with score line on one side and ‘5’embosses on the other side

Prednisolon Omet Pharma 10 mg tablets:
White, round, plain tablet with score line on one side.

Prednisolon Omet Pharma 25 mg tablets:
White, round, biconvex tablet with score line on one side.

The score line is not for dividing the tablets in equal doses but only for tablets presentation.

4. CLINICAL PARTICULARS

4.1 Therapeutic indications

Prednisolone is indicated to treat conditions where the anti-inflammatory and immunosuppressive effects of prednisolone are desired. Examples include rheumatoid arthritis, systemic lupus erythematosus, certain vasculitides such as temporal arteritis and polyarteritis nodosa, sarcoidosis, bronchial asthma, ulcerative colitis, hemolytic anemia and granulocytopenia, and severe allergic conditions.

In tumor treatment, in some cases of acute leukemia, lymphoma, breast cancer and prostate cancer.

4.2 Posology and method of administration

Individual
Generally, the dosage depends on the nature and severity of the disease. The dose should begin with a dose that provides the desired control of the disease. Then the dose should be reduced to the lowest dose that provides adequate control of the disease.
Dose adjustment or discontinuation must always be considered depending on the indication and disease activity. During stress (e.g. surgery, infection, trauma) it may be necessary to increase the dose temporarily.

If the entire maintenance dose is given in the morning (8 a.m.), prednisolone acts in accordance with the natural circadian rhythm of the adrenal cortex and causes minimal adrenal inhibition. This type of dosing is generally preferable; however, in some cases, such as rheumatoid arthritis patients with pronounced morning stiffness and asthmatic who require corticosteroid protection during the night, a late evening or split dose may be preferable.

In some cases of asthma, allergic conditions, dermatoses, etc., it may be beneficial to give the double daily dose as an undivided dose once every other day in the morning.
Therapy is gradually withdrawn, especially after high doses.
Discontinuation of the dose should be done gradually because the own ACTH secretion may be reduced after longer treatment.

Adults
The daily oral dose is typically between 5 and 30 mg depending on the nature of the disease and severity. In very severe acute cases, up to 50-60 mg or more may be given over a few days. When a satisfactory effect has been achieved, the daily dose is reduced by 2.5-5 mg every two to five days (more rapidly at the highest doses) to the lowest possible maintenance dose. It is desirable that this dose does not exceed 10 mg/day.

Paediatric population
It is important that the maintenance dose in children is gradually reduced to the lowest dose which gives adequate control of the disease and a minimum of side effects. In children this is especially important due to the risk of growth inhibition (see section 4.4 and 4.8).

<u>Method of administration</u>
Prednisolon Omet Pharm should be swallowed with water. Prednisolon Omet Pharm can be taken before or after a meal.

4.3 Contraindications

- Hypersensitivity to the active substance or any of the excipients listed in section 6.1.
- Administration of live vaccines is contraindicated in patients receiving immunosuppressive doses of corticosteroids.
- Systemic fungal infections

In general, no contraindications apply in conditions where the use of glucocorticoids may be lifesaving.

4.4 Special warnings and precautions for use

Because complications of corticosteroid treatment are dependent on the dose and treatment duration, a risk/benefit assessment must be made for each patient concerning dose and treatment length, and also if daily or intermittent treatment must be used. The lowest possible dose of corticosteroid that can control the condition being treated must be used, and when dose reduction is possible, this must be undertaken gradually (see section 4.2).

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

Glucocorticoids should not be given for infections without concomitant causal treatment.

Chickenpox and measles can have a more severe or even fatal course in non-immunised children and adults treated with corticosteroids. Children, or adults who have not had these diseases, and who are taking immunosuppressive doses of corticosteroids, should be advised to avoid exposure to chickenpox and measles, and to seek medical care if exposed.
In cases of active tuberculosis, prednisolone treatment should be limited to cases of fulminant or disseminated tuberculosis in which the corticosteroid is used for treatement of the disease in combination with an appropriate tuberculosis treatment. If corticosteroids are indicated in patients with latent tuberculosis or in tuberculin-reactive patients, these patients must be monitored carefully as reactivation of the disease may occur. In the event of long-term corticosteroid treatment, patients should receive prophylactic anti-tuberculosis treatment.

High-dose corticosteroids may interfere with active immunisation.
Vaccination with live vaccines should be done under close supervision and not in patients on long-term treatment with corticosteroids in immunosuppressive doses.

Immune system
As rare cases of skin reactions and anaphylactic/anaphylactoid reactions have occurred in patients treated with corticosteroids, appropriate precautions should be taken before administration, especially if the patient has had a previous allergic reaction to any medicine.

Endocrine system
Long-term treatment with pharmacological doses corticosteroids of may lead to secondary adrenal insufficiency. The risk can be reduced by giving the treatment every other day (see Section 4.2).

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 discontinuation of treatment may lead to acute adrenal insufficiency which can be fatal. The risk of secondary adrenal insufficiency can be reduced by gradually lowering the dose. This type of relative insufficiency may persist for months after discontinuation of treatment, so hormone therapy should be re-initiated for stressful situations occurring during this period. As mineralocorticoid excretion may be impaired, salts and/or mineralocorticoids should be administered concomitantly.

A "steroid withdrawal syndrome", seemingly unrelated to adrenal insufficiency, may also occur after abrupt withdrawal of glucocorticoids. This syndrome produces symptoms such as anorexia, nausea,

vomiting, lethargy, headache, fever, joint pain, distension, myalgia, weight loss and/or hypotension. These effects are thought to be due to the sudden change in glucocorticosteroid concentration rather than to low corticosteroid levels.

Patients with hypothyroidism or liver cirrhosis have an enhanced effect of corticosteroids.

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

Psychiatric reactions
Potentially serious psychiatric disorders can occur during treatment with corticosteroids, including prednisolone. These can range from euphoria, sleep disturbances, mood swings, personality changes and severe depression to psychotic manifestations. In addition, pre-existing emotional instability and psychotic tendencies may be exacerbated by corticosteroids (see section 4.8). The onset of symptoms typically occurs within days or weeks of starting treatment.
Most reactions resolve after dose reduction or discontinuation, but specific treatment may be necessary.
Psychological effects have been reported with corticosteroid withdrawal, the frequency is unknown. Patients/carers should be advised to seek medical attention if the patient develops psychiatric symptoms, especially if depression or suicidal ideation is suspected. Patients/caregivers should be aware that psychiatric disorders may occur either during or immediately after dose reduction/discontinuation of systemic steroids.

Central and peripheral nervous system
Corticosteroids should be used with caution in patients with convulsive disorders.

Cardiovascular system
Adverse effects of glucocorticoids on the cardiovascular system, such as dyslipidaemia and hypertension, may predispose treated patients with pre-existing cardiovascular risk factors to further cardiovascular events at high doses and long duration of treatment. Therefore, corticosteroids should be started in these patients only after careful consideration, and risk-modifying measures and additional cardiac monitoring should be considered if necessary. Low dose and treatment every other day may reduce complications of corticosteroid therapy.

Blood vessels
Since corticosteroids have been reported to increase the coagulation tendency of the blood in rare cases and thus could accelerate the development of intravascular thrombosis, thromboembolism and thrombophlebitis, corticosteroids should be used with caution in patients with thromboembolic diseases.

Gastrointestinal tract
High doses of corticosteroids can cause acute pancreatitis.

There are no clear data establishing that corticosteroids cause peptic ulcers. Glucocorticoid therapy may mask peritonitis and other signs and symptoms associated with gastrointestinal conditions such as perforation, obstruction or pancreatitis. In combination with NSAIDs, the risk of gastrointestinal ulcers is increased.

Corticosteroids should therefore be used with caution in non-specific ulcerative colitis if there is a likelihood of impending perforation, abscess or other pyogenic infection, diverticulitis, newly created anastomoses, or active or latent peptic ulcer.

Liver and biliary tract
Liver and biliary tract diseases have been reported in rare cases and in the majority of these cases the condition was reversible after discontinuation of treatment. Appropriate monitoring measures are required.

Musculoskeletal system
Acute myopathy has been reported with high corticosteroid doses, most commonly in patients with neuromuscular transmission disorders (e.g. myasthenia gravis), or in patients concomitantly treated with anticholinergics, e.g. neuromuscular blocking drugs (such as pancuronium) (see section 4.5). This acute myopathy is generalised, may involve eye and respiratory muscles, and may lead to tetraparesis. Elevated creatine kinase may occur. Clinical improvement or recovery after discontinuation of corticosteroids may take weeks or years.

Corticosteroids should be used with caution in patients with osteoporosis

Kidney and urinary tract
Corticosteroids should be used with caution in patients with renal insufficiency.

Acute renal crisis (renal crisis in scleroderma)
Caution is required in patients with systemic sclerosis as an increased incidence of (potentially fatal) renal crisis in scleroderma, with hypertension and decreased urine output, has been observed with a daily prednisolone dose of 15 mg or more. Blood pressure and renal function (S-creatinine) should therefore be routinely monitored. In case of suspected renal crisis, blood pressure should be kept under close control.

Effects on electrolytes and fluid balance
Systemic corticosteroids should be used with caution in patients with heart failure or hypertension. Medium and high doses of hydrocortisone or cortisone may lead to increased blood pressure, salt and water retention and increased potassium excretion. These effects are less likely with synthetic derivatives, except when used in high doses. Dietary restrictions with lower salt intake 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.

Tumour lysis syndrome (TLS)
Tumour lysis syndrome (TLS) has been reported post-marketing in patients with malignancies, including haematological malignancies and solid tumours, following the use of systemic corticosteroids alone or in combination with other cytotoxic agents. Patients at high risk of TLS, such as those with tumours that have a high cell division rate, high tumour burden and high sensitivity to cytotoxic agents, should be closely monitored and appropriate precautions taken.

Paediatric population
Corticosteroids cause growth retardation in infants, children and adolescents and therefore long-term treatment with pharmacological doses should be avoided. If long-term treatment is required, the growth and development of the infant/child should be closely monitored (see section 4.2).
Infants and children on long-term corticosteroid therapy are at particular risk of developing elevated intracranial pressure.

Excipient(s)
Lactose
This medicinal product contains lactose. 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 Prednisolon Omet Pharma may require dose adjustment.

Phenobarbital, phenytoin, carbamazepine
Phenobarbital (which is also a metabolite of primidone), phenytoin and carbamazepine, either alone or or in combination, induce the metabolism of hydrocortisone, prednisolone and methylprednisolone (shown in children with asthma) resulting in increased dose requirements.
The interaction probably applies to the whole group of glucocorticoids.

Rifampicin
Rifampicin induces the microsomal oxidation of glucocorticoids (hydrocortisone, prednisolone, methylprednisolone). This results in an increased steroid requirement during rifampicin treatment and a reduced steroid requirement after such treatment.

NSAID
1) The incidence of gastrointestinal bleeding and ulceration may increase if corticosteroids are given together with an NSAID.

2) Corticosterioids may increase the clearance of high doses of acetylsalicylic acid. This can lead to reduced levels of salicylates and increase the risk of salicylate toxicity upon discontinuation of prednisolone.

Estrogens (including oral contraceptives containing estrogens)
Estrogens increase the concentration of transcortin. The effect of glucocorticoids that bind to transcortin may be enhanced and dose adjustments may be necessary if estrogens are added to or removed from a stable treatment regimen.

Potassium-reducing diuretics
Potassium-reducing diuretics (e.g. thiazides, furosemide, ethacrynic acid) and other potassium-reducing drugs such as amphotericin B, xanthines and beta2-agonists, may enhance the potassium-lowering effect of glucocorticoids. Serum potassium should be closely monitored in patients receiving glucocorticoids and potassium-reducing agents.

Isoniazid
Prednisolone also has a potential effect of increasing the acetylation rate and clearance of isoniazid.

Oral anticoagulants
There are reports of altered efficacy of anticoagulants given concomitantly with prednisolone. Prothrombin time (INR) should be monitored during treatment.

Anticholinergics, neuromuscular blocking agents
Corticosteroids may affect the efficacy of anticholinergics.
1) Acute myopathy has been reported with concomitant use of high doses of corticosteroids and anticholinergics, such as neuromuscular blocking agents (see section 4.4).
2) Antagonism with the neuromuscular blocking effect of pancuronium and vecuronium has been reported in patients taking glucocorticosteroids. This interaction can be expected with all competitive neuromuscular blocking agents.

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

Antidiabetics
Glucocorticoids increase the blood sugar. Patients with diabetes mellitus receiving concomitant insulin and/or oral hypoglycaemic agents may need to adjust the doses of such treatment.

CYP3A inhibitors, including drugs containing cobicistat
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.

4.6 Fertility, pregnancy and lactation

Pregnancy
In animal experiments, corticosteroids have been shown to cause malformations of various kinds (cleft palate, skeletal malformations,see section 5.3). The relevance of this for humans is not known. Reduced placental and birth weight has been confirmed in humans and animals following long-term treatment. Long-term treatment is also associated with a risk of adrenocortical suppression in the neonate. During pregnancy, corticosteroids should be given only following careful consideration.

Breast-feeding
Prednisolone passes into breast milk, however, at therapeutic doses the risk of affecting the child appears unlikely.

Fertility
Studies in animals have shown that corticosteroids reduce fertility (see section 5.3).

4.7 Effects on ability to drive and use machines

The effect of corticosteroids on the ability to drive and operate machinery has not been systematically studied. Side effects such as dizziness, visual disturbances and fatigue are possible after treatment with corticosteroids. In case of such side effects, patients should not drive or use machines.

4.8 Undesirable effects

Apart from substitution therapy, corticosteroid treatment always involves an overdose compared to the physiological state. Side effects occur mainly with long-term treatment but also depend on dose size and individual sensitivity.

The following adverse reactions 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), no known frequency (cannot be calculated from available data).

Organ / system	**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 the available data)**

Infections and infestations	Opportunistic infection Activation of infection (e.g. tuberculosis)				
Blood and lymphatic system disorders					Leukocytosis (due to redistribution of intravascular granulocytes)
Immune system disorders					Drug hypersensitivity Anaphylactic reaction Anaphylactoid reaction
Endocrine disorders	Inhibition of hypothalamic pituitary adrenal (HPA) axis, Cushing-like symptoms. Growth retardation (in children).				Steroid withdrawal syndrome (see section 4.4) Pheochromocytosis-related crisis (see section 4.4).
Metabolism and nutrition disorders	Hypokalaemia Sodium retention Increased gluconeogenesis Catabolic effects				Metabolic acidosis Fluid retention Hypokalaemic alkalosis Dyslipidaemia Impaired glucose tolerance (diabetes mellitus may worsen and latent diabetes become manifest) Lipomatosis Increased appetite (which may lead to weight gain)
Psychiatric disorders		Activation of previous mental disorders (high dose)	Depression, mania in patients without a history of mental illness		Affective disorder (includes euphoria, affective lability, drug dependence, suicidal ideation) Psychotic disorder (includes delusions, hallucinations and schizophrenia) Mental illness Personality change Confusional state Anxiety Mood swings Abnormal behavior Insomnia

					Irritability
Nervous system disorders			Benign intracranial hypertension		Epidural lipomatosis Seizures Amnesia Cognitive disturbance Dizziness Headache
Eye disorders		Cataract Glaucoma			Central serous chorioretinopathy (see section 4.4) Exophthalmos Blurred vision (see section 4.4)
Cardiac disorders					Heart failure (in susceptible patients) Bradycardia*
Vascular disorders	Oedema Hypertension				Thromboembolic events
Respiratory, thoracic and mediastinal disorders					Hiccup
Gastrointestinal disorders					Peptic ulcer (possibly with perforation and bleeding) Intestinal perforation Pancreatitis Ulcerative oesophagitis Distended abdomen Abdominal pain Diarrhoea Dyspepsia Nausea
Skin and subcutaneous tissue disorders	Skin atrophy, Impaired wound healing				Angioedema Hirsutism Petechiae Ecchymosis Erythema Hyperhidrosis Striae Pruritus Urticaria Acne

Musculoskeletal and connective tissue disorders	Muscular atrophy Osteoporosis		Aseptic bone necrosis Tendon rupture		Muscle weakness Myalgia Myopathy Pathological fracture Neuropathic arthropathy Arthralgia
Renal and urinary disorders					Acute renal crisis (scleroderma renal crisis)**
Reproductive organs and mammary gland					Irregular menstruation
General symptoms and/or symptoms at the site of administration					Fatigue Feeling sick
Investigations					Increased urinary calcium levels Elevated alanine aminotransferase Elevated aspartate aminotransferase Elevated blood alkaline phosphatase Elevated blood urea Suppression of skin test reactions[1]

[1] Not MedDRA term.
*At high doses.
**Acute renal crisis (scleroderma renal crisis)
The incidence of acute renal crisis varies between the different subpopulations. 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 via the national reporting system listed in Appendix V.

4.9 Overdose

Reports of acute toxicity and/or death following glucocorticoid overdose are rare. It is possible that acute overdose may aggravate pre-existing conditions such as ulcers, electrolyte disturbances, infections and oedema.

Treatment: Generally not required. If warranted, gastric lavage, charcoal. In case of overdose there is no specific antidote, 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-allergic effects. Per unit of weight, prednisolone has a 4-5 times higher anti-inflammatory effect than cortisone, but affects electrolyte metabolism to a lesser extent. The mechanism of action is not yet fully understood.

5.2 Pharmacokinetic properties

Absorption
Prednisolone is rapidly absorbed in the gastrointestinal tract when given orally. Maximum plasma concentration is reached after 1 to 2 hours after oral administration. The plasma half-life is normally 2 to 4 hours. Its initial absorption, but not its overall bioavailability, is affected by food.

Distribution
Prednisolone is highly bound to plasma proteins and has a high affinity for transcortin. Volume of distribution and clearance are reported to increase with transition from low to medium doses.

Metabolism
Prednisolone is metabolised mainly 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 to intravenously administered prednisolone after a single dose of 10 mg. At normal dosing, the duration of effect is estimated 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 given amount is excreted in the urine. 7-15% is excreted in unchanged form.

5.3 Preclinical safety data

In animal studies, corticosteroids have been found to give rise to deformities of various kinds (palate fissure, skeletal deformities). Reduced placental and birth weight has been confirmed in animals following long-term treatment.
Corticosteroids have been found to reduce fertility when administered to rats.

6. PHARMACEUTICAL PARTICULARS

6.1 List of excipients

2.5 mg:
Cellulose microcrystalline
Silica, colloidal anhydrous Lactose monohydrate
Starch maize
Magnesium stearate
Iron oxide red (E172)

5 mg:
Cellulose microcrystalline
Silicon dioxide colloidal
Lactose monohydrate
Starch maize
Magnesium stearate
quinoline yellow (E104)

10 mg:
Cellulose microcrystalline
Silicon dioxide colloidal
Lactose monohydrate
Starch maize
Magnesium stearate

25 mg:
Cellulose microcrystalline
Silicon dioxide colloidal
Lactose monohydrate
Starch maize
Magnesium stearate

6.2 Incompatibilities

None known

6.3 Shelf life

2 year

6.4 Special precautions for storage

This medicinal product does not require any special storage conditions

6.5 Nature and contents of container

The tablets are supplied in blisters made of polyamide/aluminium/poly(vinyl chloride) with aluminium foil lidding.

Blister packs containing 10, 25 or 100 tablets.

Not all pack sizes may be marketed

6.6 Special precautions for disposal and other handling

No special requirements for disposal.
Any unused medicinal product or waste material should be disposed of in accordance with local requirements.

7. MARKETING AUTHORISATION HOLDER

Omet Pharma AB
Tippvägen 2.
296 35 Åhus
Sweden

8. MARKETING AUTHORISATION NUMBER(S)

[To be completed nationally]

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

<Date of first authorisation: {DD month YYYY}>
<Date of latest renewal: {DD month YYYY}>

10. DATE OF REVISION OF THE TEXT

<{MM/YYYY}>
<{DD/MM/YYYY}>
<{DD month YYYY}>

17 June 2026