SUMMARY OF PRODUCT CHARACTERISTICS

1 NAME OF THE MEDICINAL PRODUCT

Defal 6 mg Tablets

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Active substance: Deflazacort 6 mg
Excipient(s) with known effect:
Lactose monohydrate 153 mg

For the full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Tablet

Deflazacort 6 mg tablets: White, round, uncoated tablets, double-scored on one side and marked with the number 6 on the other.

4 CLINICAL PARTICULARS

4.1 Therapeutic indications

A wide range of conditions may sometimes need treatment with glucocorticoids. The indications include:
Anaphylaxis, asthma, severe hypersensitivity reactions
Rheumatoid arthritis, juvenile chronic arthritis, polymyalgia rheumatica Systemic lupus erythematosus, dermatomyositis, mixed connective tissue disease (other than systemic sclerosis), polyarteritis nodosa, sarcoidosis Pemphigus, bullous pemphigoid, pyoderma gangrenosum , Minimal change nephrotic syndrome, acute interstitial nephritis Rheumatic carditis
Ulcerative colitis, Crohn's disease Uveitis, optic neuritis
Autoimmune haemolytic anaemia, idiopathic thrombocytopenic purpura Acute and lymphatic leukaemia, malignant lymphoma, multiple myeloma Immune suppression in transplantation

4.2 Posology and method of administration

Deflazacort is a glucocorticoid derived from prednisolone and 6 mg of deflazacort has approximately the same anti-inflammatory potency as 5 mg prednisolone or prednisone.

Doses vary widely in different diseases and different patients. In more serious and life-threatening conditions, high doses of deflazacort may need to be given. When deflazacort is used long term in relatively benign chronic diseases, the maintenance dose should be kept as low as possible. Dosage may need to be increased during periods of stress or in exacerbation of illness.

The dosage should be individually titrated according to diagnosis, severity of disease and patient response and tolerance. The lowest dose that will produce an acceptable response should be used (see section 4.4).

Adults
For acute disorders, up to 120 mg/day deflazacort may need to be given initially. Maintenance doses in most conditions are within the range 3 – 18 mg/day. The following regimens are for guidance only.

Rheumatoid arthritis: The maintenance dose is usually within the range 3 – 18 mg/day. The smallest effective dose should be used and increased if necessary.

Bronchial asthma: In the treatment of an acute attack, high doses of 48 – 72 mg/day may be needed depending on severity and gradually reduced once the attack has been controlled. For maintenance in chronic asthma, doses should be titrated to the lowest dose that controls symptoms.

Other conditions: The dose of deflazacort depends on clinical need titrated to the lowest effective dose for maintenance. Starting doses may be estimated on the basis of ratio of 5 mg prednisone or prednisolone to 6 mg deflazacort.

Hepatic impairment
In patients with hepatic impairment, blood levels of deflazacort may be increased. Therefore, the dose of deflazacort should be carefully monitored and adjusted to the minimum effective dose.

Renal impairment
In renally impaired patients, no special precautions other than those usually adopted in patients receiving glucocorticoid therapy are necessary.

Elderly
In elderly patients, no special precautions other than those usually adopted in patients receiving glucocorticoid therapy are necessary. The common adverse effects of systemic corticosteroids may be associated with more serious consequences in old age (see section 4.4).

Pediatric population

There has been limited exposure of children to deflazacort in clinical trials.

In children, the indications for glucocorticoids are the same as for adults, but it is important that the lowest effective dosage is used. Alternate day administration may be appropriate (see section 4.4).

Doses of deflazacort usually lie in the range 0.25 – 1.5 mg/kg/day. The following ranges provide general guidance:

Juvenile chronic arthritis: The usual maintenance dose is between 0.25 – 1.0 mg/kg/day.

Nephrotic syndrome: Initial dose of usually 1.5 mg/kg/day followed by down titration according to clinical need.

Bronchial asthma: On the basis of the potency ratio, the initial dose should be between 0.25 – 1.0 mg/kg deflazacort on alternate days.

Deflazacort withdrawal

In patients who have received more than physiological doses of systemic corticosteroids (approximately 9 mg per day or equivalent) for greater than 3 weeks, withdrawal should not be abrupt. How dose reduction should be carried out depends largely on whether the disease is likely to relapse as the dose of systemic corticosteroids is reduced. Clinical assessment of disease activity may be needed during withdrawal. If the disease is unlikely to relapse on withdrawal of systemic corticosteroids but there is uncertainty about HPA suppression, the dose of systemic corticosteroids may be reduced rapidly to physiological doses. Once a daily dose equivalent to 9 mg deflazacort is reached, dose reduction should be slower to allow the HPA-axis to recover.

Abrupt withdrawal of systemic corticosteroid treatment, which has continued up to 3 weeks is appropriate if it is considered that the disease is unlikely to relapse. Abrupt withdrawal of doses up to 48 mg daily of deflazacort, or equivalent for 3 weeks is unlikely to lead to clinically relevant HPA-axis suppression, in the majority of patients. In the following patient groups, gradual withdrawal of systemic corticosteroid therapy should be *considered* even after courses lasting 3 weeks or less:

- Patients who have had repeated courses of systemic corticosteroids, particularly if taken for greater than 3 weeks.
- When a short course has been prescribed within one year of cessation of long-term therapy (months or years).
- Patients who may have reasons for adrenocortical insufficiency other than exogenous corticosteroid therapy.
- Patients receiving doses of systemic corticosteroid greater than 48 mg daily of deflazacort (or equivalent).
- Patients repeatedly taking doses in the evening.

4.3 Contraindications

- Hypersensitivity to the active substance, deflazacort or any of the excipients listed in section 6.1.
- Systemic infection unless specific anti-infective therapy is employed.
- Patients receiving live virus immunisation (see section 4.4 and 4.5).

4.4 Special warnings and precautions for use

A patient information leaflet should be supplied with this product.

Since complications of glucocorticoid therapy are dependent on dose and duration of therapy, the lowest possible dose must be given, and a risk/benefit decision must be made as to whether intermittent therapy should be used.

Undesirable effects may be minimised by using the lowest effective dose for the minimum period, and by administering the daily requirement as a single morning dose or whenever possible as a single morning dose on alternate days. Frequent patient review is required to appropriately titrate the dose against disease activity (see section 4.2).

Adrenal suppression

Glucocorticoid induced suppression of hypothalamic-pituitary-adrenal function is dependent on dose and duration of treatment. Recovery occurs gradually as the steroid dose is reduced and withdrawn. Adrenal cortical atrophy develops during prolonged therapy and may persist for years after stopping treatment. During prolonged therapy, any intercurrent illness, trauma or surgical procedure will require a temporary increase in dosage. However, relative insufficiency may persist for months after discontinuing therapy; therefore, in any situation of stress, corticosteroids may need to be temporarily re-introduced.

When discontinuing long-term administration of corticosteroids, it should be done gradually. The risks associated with sudden discontinuation are exacerbation or recurrence of the underlying disease, adrenocortical insufficiency (which could be fatal) or steroid withdrawal syndrome. Steroid withdrawal syndrome may present with a wide range of signs and symptoms. However, typical symptoms include fever, anorexia, nausea, lethargy, malaise, myalgia, arthralgias, rhinitis, conjunctivitis, desquamation of the skin and painful itchy skin nodules, weakness, hypotension and weight loss. This may occur in patients even without evidence of adrenal insufficiency.

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.

Anti-inflammatory/immunosuppressive effects and infection
Suppression of the inflammatory response and immune function increases the susceptibility to infections and their severity. The clinical presentation may often be atypical and serious infections such as septicaemia and tuberculosis may be masked and may reach an advanced stage before being recognised.

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 chicken pox should be advised to avoid close personal contact with chickenpox or herpes zoster and, if exposed, they should seek urgent medical attention. Passive immunisation with varicella zoster immunoglobulin (VZIG) is needed by exposed non-immune patients who are receiving systemic corticosteroids or who have used them within the previous 3 months; this should be given within 10 days of exposure to chickenpox. If a diagnosis of chickenpox is confirmed, the illness warrants specialist care and urgent treatment. Corticosteroids should not be stopped, and the dose may need to be increased.

Patients should be advised to take particular care to avoid exposure to measles and to seek immediate medical advice if exposure occurs. Prophylaxis with intramuscular normal immunoglobulin may be needed.

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

Use in active tuberculosis should be restricted to those cases of fulminating and disseminated tuberculosis in which deflazacort is used for management with appropriate antituberculosis regimen. If glucocorticoids are indicated in patients with latent tuberculosis or tuberculin reactivity, close observation is necessary as reactivation of the disease may occur. During prolonged glucocorticoid therapy, these patients should receive chemoprophylaxis.

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

Prolonged use of glucocorticoids may produce posterior subcapsular cataracts, glaucoma with possible damage to the optic nerves and may

enhance the establishment of secondary ocular infections due to fungi or viruses.

Tendonitis
Tendonitis and tendon rupture are known class effect of glucocorticoids. The risk of such reactions may be increased by co-administration of quinolones (see section 4.8).

Phaeochromocytoma
Phaeochromocytoma crisis, which can be fatal, has been reported after administration of systemic corticosteroids. Corticosteroids should only be administered to patients with suspected or identified phaeochromocytoma after an appropriate risk/benefit evaluation (see section 4.8).

Anticoagulants
The effect of corticosteroids on anticoagulants is variable. There are reports of enhanced as well as diminished effects of anticoagulants when given concurrently with corticosteroids. Therefore, coagulation indices should be monitored to maintain the desired anticoagulant effects (see section 4.5).

Gastrointestinal ulcers
Care should be taken when deflazacort is prescribed in combination with non-steroidal anti-inflammatory drugs (NSAIDs) due to increased risk of gastrointestinal ulcers (see section 4.5).

Severe cutaneous adverse reactions
Deflazacort may cause severe cutaneous adverse reactions such as toxic epidermal necrolysis (TEN) and Stevens-Johnson syndrome (SJS) (see section 4.8). If severe cutaneous adverse reactions occur, deflazacort should be discontinued and appropriate therapy and/or measures should be taken.

Special precautions
The following clinical conditions require special caution and frequent patient monitoring is necessary:

- Cardiac disease or congestive heart failure (except in the presence of active rheumatic carditis), hypertension, thromboembolic disorders. Glucocorticoids can cause salt and water retention and increased excretion of potassium. Dietary salt restriction and potassium supplementation may be necessary.
- Gastritis or oesophagitis, diverticulitis, ulcerative colitis if there is probability of impending perforation, abscess or pyogenic infections, fresh intestinal anastomosis, active or latent peptic ulcer.

- Diabetes mellitus or a family history, osteoporosis, myasthenia gravis, renal insufficiency.
- Emotional instability or psychotic tendency, epilepsy.
- Previous corticosteroid-induced myopathy.
- Liver failure.
- Hypothyroidism and cirrhosis, which may increase glucocorticoid effect.
- Ocular herpes simplex because of possible corneal perforation.

Glucocorticoids are known to cause irregular menstruation and leukocytosis; care should be taken with deflazacort.

Severe psychiatric adverse reactions

Patients and/or carers should be warned that potentially severe psychiatric adverse reactions may occur with systemic steroids (see section 4.8). Symptoms typically emerge within a few days or weeks of starting the treatment. Risks may be higher with high doses/systemic exposure (see also section 4.5) although dose levels do not allow prediction of the onset, type, severity or duration of reactions. Most reactions recover after either dose reduction or withdrawal, although specific treatment may be necessary. Patients/carers should be encouraged to seek medical advice if worrying psychological symptoms develop, especially if depressed mood or suicidal ideation is suspected. Patients/carers should also be alert to possible psychiatric disturbances that may occur either during or immediately after dose tapering/withdrawal of systemic steroids, although such reactions have been reported infrequently.

Particular care is required when considering the use of systemic corticosteroids in patients with existing or previous history of severe affective disorders in themselves or in their first-degree relatives. These would include depressive or manic-depressive illness and previous steroid psychosis.

Paediatric population

Corticosteroids cause dose-related growth retardation in infancy, childhood and adolescence which may be irreversible.

Hypertrophic cardiomyopathy has been reported after systemic administration of glucocorticosteroids in preterm infants. In infants receiving administration of systemic glucocorticosteroids, echocardiograms should be performed to monitor myocardial structure and function (see section 4.8).

Elderly

The common adverse effects of systemic corticosteroids may be associated with more serious consequences in old age, especially osteoporosis, hypertension, hypokalaemia, diabetes, susceptibility to infection and thinning of the skin. Close clinical supervision is required to avoid life-threatening reactions.

Tumour Lysis Syndrome

In post-marketing experience, tumour lysis syndrome (TLS) has been reported in patients with haematological malignancies following the use of Deflazacort alone or in combination with other chemotherapeutic agents. Patients 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 precautions should be taken (see section 4.8).

Excipient with known effect

Lactose: Patients with rare hereditary problems of galactose intolerance, the Lapp lactose deficiency or glucose-galactose malabsorption should not take this medicine.

4.5 Interaction with other medicinal products and other forms of interaction

Contraindicated combinations:

- live attenuated vaccines due to increased risk of generalised potentially fatal vaccine induced disease (see section 4.3 and 4.4). Glucocorticoids may potentiate the replication of germs from live attenuated vaccines.

The same precautions should be exercised as for other glucocorticoids. Deflazacort is metabolised in the liver. It is recommended to increase the maintenance dose of deflazacort if drugs which are liver enzyme inducers are co-administered, e.g. rifampicin, rifabutin, carbamazepine, phenobarbitone, phenytoin, primidone and aminoglutethimide. For drugs which inhibit liver enzymes, e.g. ketoconazole it may be possible to reduce the maintenance dose of deflazacort.

In patients taking estrogens, corticosteroid requirements may be reduced.

The desired effects of hypoglycaemic agents (including insulin), anti-hypertensives and diuretics are antagonised by corticosteroids and the hypokalaemic effects of acetazolamide, loop diuretics, thiazide diuretics, beta 2-agonists, xanthines and carbenoxolone are enhanced.

Corticosteroids can increase or decrease the effect of anticoagulants (see section 4.4).

The efficacy of coumarin anticoagulants may be enhanced by

concurrent corticosteroid therapy and close monitoring of the INR or prothrombin time is required to avoid spontaneous bleeding.
Concomitant administration with non-steroidal anti-inflammatory drugs can increase the risk of gastrointestinal ulcers (see section 4.4).
In patients treated with systemic corticosteroids, use of non-depolarising muscle relaxants can result in prolonged relaxation and acute myopathy. Risk factors for this include prolonged and high dose corticosteroid treatment, and prolonged duration of muscle paralysis. This interaction is more likely following prolonged ventilation (such as in the ITU setting).

The renal clearance of salicylates is increased by corticosteroids and steroid withdrawal may result in salicylate intoxication.

As glucocorticoids can suppress the normal responses of the body to attack by micro-organisms, it is important to ensure that any anti-infective therapy is effective and it is recommended to monitor patients closely. Concurrent use of glucocorticoids and oral contraceptives should be closely monitored as plasma levels of glucocorticoids may be increased. This effect may be due to a change in metabolism or binding to serum proteins. Antacids may reduce bioavailability; leave at least 2 hours between administration of deflazacort and antacids.

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
The ability of corticosteroids to cross the placenta varies between individual drugs, however, deflazacort does cross the placenta.

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

state.

Breast-feeding
Corticosteroids are excreted in breast milk, although no data are available for deflazacort. Doses of up to 50 mg daily of deflazacort are unlikely to cause systemic effects in the infant. Infants of mothers taking higher doses than this may have a degree of adrenal suppression but the benefits of breast feeding are likely to outweigh any theoretical risk.

Fertility
No data is available on Deflazacort and its effects on fertility.

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. Vertigo is a possible undesirable effect after treatment with deflazacort. If affected, patients should not drive or operate machinery.

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 section 4.4).

The following CIOMS frequency rating is used: *Very common (≥1/10); common (≥1/100 to <1/10); uncommon (≥1/1000 to <1/100); rare (≥1/10 000 to <1/1000); very rare (<1/10 000), not known (cannot be estimated from the available data).*

Endocrine disorders

Uncommon: suppression of the hypothalamic-pituitary-adrenal axis, amenorrhoea, Cushingoid facies
Not known: growth suppression in infancy, childhood and adolescence, steroid withdrawal syndrome (see section 4.4)

Metabolism and nutrition disorders *Common*: Weight gain
Uncommon: impaired carbohydrate tolerance with increased requirement for anti-diabetic therapy, sodium and water retention with hypertension, potassium loss and hypokalaemic alkalosis when co-administered with beta 2-agonist and xanthines
Not known: Negative protein and calcium balance, increased appetite. Cases of tumour lysis syndrome have been reported in association with

Deflazacort when used in patients with haematological malignancies (see section 4.4)

Infections and infestations
Uncommon: Increased susceptibility and severity of infections with suppression of clinical symptoms and signs, opportunistic infections, recurrence of dormant tuberculosis (see section 4.4).
Not known: candidiasis.

Musculoskeletal and connective tissue disorders *Uncommon*: osteoporosis, vertebral and long bone fractures *Rare*: muscle wasting
Not known: avascular osteonecrosis, tendonitis and tendon rupture when co-administered with quinolones (see section 4.4), myopathy (acute myopathy may be precipitated by non-depolarising muscle relaxants – see section 4.5), negative nitrogen balance

Reproductive system and breast disorders *Not known*: menstrual irregularity

Cardiac disorders
Not known: heart failure, hypertrophic cardiomyopathy in preterm infants

Nervous system disorders
Uncommon: headache, vertigo
Not known: restlessness, Increased intra-cranial pressure with papilloedema in children (pseudotumour cerebri), usually after treatment withdrawal, aggravation of epilepsy

Psychiatric disorders
Uncommon: depressed and labile mood, behavioural disturbances
Not known: irritable, euphoric, suicidal thoughts, psychotic reactions (including mania, delusions, hallucinations, aggravation of schizophrenia), anxiety, sleep disturbances, and cognitive dysfunction including confusion and amnesia

Reactions are common and may occur in both adults and children. In adults, the frequency of severe reactions has been estimated to be 5 – 6%. Psychological effects have been reported on withdrawal of corticosteroids; the frequency is unknown.

Eye disorders
Not known: vision blurred (see section 4.4), increased intra-ocular pressure, glaucoma, papilloedema, posterior subcapsular cataracts especially in children, chorioretinopathy (see section 4.4), corneal or

scleral thinning, exacerbation of ophthalmic viral or fungal diseases.

Gastrointestinal disorders
Uncommon: dyspepsia, peptic ulceration, haemorrhage, nausea
Not known: perforation of peptic ulcer, acute pancreatitis (especially in children)

Skin and subcutaneous tissue disorders
Uncommon: hirsutism, striae, acne *Rare*: bruising
Not known: severe cutaneous adverse reactions (SCARs) such as Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN), skin atrophy, telangiectasia

General disorders and administration site conditions *Uncommon*: oedema
Not known: impaired healing

Immune system disorders
Uncommon: hypersensitivity including anaphylaxis has been reported

Blood and lymphatic system disorders *Not known*:
leukocytosis

Vascular disorders
Not known: thromboembolism in particular in patients with underlying conditions associated with increased thrombotic tendency, rare incidence of benign intracranial hypertension

Class effect
Pheochromocytoma crisis has been reported with other systemic corticosteroids and is a known class effect (see section 4.4).

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. Reporting of suspected adverse reactions: Healthcare professionals are requested to report any suspected adverse reactions via pharmacy and poisons board, Pharmacovigilance Electronic Reporting System (PvERS) https://pv.pharmacyboardkenya.org'

4.9 Overdose

It is unlikely that treatment is needed in cases of acute

overdosage. The LD_{50} for the oral dose is greater than 4000 mg/kg in laboratory animals.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: corticosteroids for systemic use; Glucocorticoids. ATC code: H02AB13.

Mechanism of action

Deflazacort is a glucocorticoid. Its anti-inflammatory and immunosuppressive effects are used in treating a variety of diseases and are comparable to other anti-inflammatory steroids. Clinical studies have indicated that the average potency ratio of deflazacort to prednisolone is 0.69 – 0.89.

5.2 Pharmacokinetic properties

Absorption
Orally administered deflazacort appears to be well absorbed.

Distribution
The active metabolite D 21-OH achieves peak plasma concentrations in 1.5 – 2 hours. It is 40% protein-bound and has no affinity for corticosteroid-binding-globulin (transcortin).

Biotransformation
Orally administered deflazacort is immediately converted by plasma esterases to the pharmacologically active metabolite (D 21-OH). Metabolism of D 21-OH is extensive. The metabolite of D 21-OH is deflazacort 6-beta-OH.

Elimination
Its elimination plasma half-life is 1.1 – 1.9 hours. Elimination takes place primarily through the kidneys; 70% of the administered dose is excreted in the urine. The remaining 30% is eliminated in the faeces. Metabolism of D 21-OH is extensive; only 18% of urinary excretion represents D 21-OH. The metabolite of D 21-OH, deflazacort 6-beta-OH, represents one third of the urinary elimination.

5.3 Preclinical safety data

Safety studies have been carried out in the rat, dog, mouse and monkey. The findings are consistent with other glucocorticoids at comparable doses.
Teratogenic effects demonstrated in rodents and rabbits are typical of those caused by other glucocorticoids. Deflazacort was not found to be carcinogenic in the mouse, but studies in the rat produced carcinogenic findings consistent with the findings with other glucocorticoids.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Lactose monohydrate,
Corn starch,
Microcrystalline cellulose,
Magnesium stearate.

6.2 Incompatibilities

Not applicable.

6.3 Shelf life

36 months

6.4 Special precautions for storage

This medicinal product does not require any special storage conditions.

6.5 Nature and contents of container

Deflazacort is packed in blister packs of polyvinylchloride and Aluminium foil presented in cardboard cartons.

6.6 Special precautions for disposal

No special requirements for disposal or handling are required.

7 MARKETING AUTHORISATION HOLDER

FAES FARMA, S.A.
Maximum Aguirre, 14 48940 Leioa

8 MARKETING AUTHORISATION NUMBER(S)

H2025/CTD9809/20180

9 DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

2025 August 27

10 DATE OF REVISION OF THE TEXT

2025 August 27