SUMMARY OF PRODUCT CHARACTERISTICS

1. NAME OF THE MEDICINAL PRODUCT

Efmody 5 mg modified-release hard capsules
Efmody 10 mg modified-release hard capsules
Efmody 20 mg modified-release hard capsules

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Efmody 5 mg modified-release hard capsules.

Each modified-release hard capsule contains 5 mg hydrocortisone.

Efmody 10 mg modified-release hard capsules.

Each modified-release hard capsule contains 10 mg hydrocortisone.

Efmody 20 mg modified-release hard capsules.

Each modified-release hard capsule contains 20 mg hydrocortisone.

For the full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

Modified-release hard capsules.

Efmody 5 mg modified-release hard capsules.

A capsule (approx.19 mm long) with an opaque blue cap and opaque white body printed with "CHC 5 mg" containing white to off white granules.

Efmody 10 mg modified-release hard capsules.

A capsule (approx.19 mm long) with an opaque green cap and opaque white body printed with "CHC 10 mg" containing white to off white granules.

Efmody 20 mg modified-release hard capsules.

A capsule (approx. 22 mm long) with an opaque orange cap and opaque white body printed with "CHC 20 mg" containing white to off white granules.

4. CLINICAL PARTICULARS

4.1 Therapeutic indications

Treatment of congenital adrenal hyperplasia (CAH) in adolescents aged 12 years and over and adults.

4.2 Posology and method of administration

Posology

Treatment should be initiated by physicians experienced in the management of CAH.

As maintenance therapy the dose must be individualised according to the response of the individual patient. The lowest possible dose should be used.

Monitoring of the clinical response is necessary and patients should be observed closely for signs that might require dose adjustment, including changes in clinical status resulting from remissions or exacerbations of the disease, changes in electrolytes particularly hypokalaemia, individual responsiveness to the medicinal product, and the effect of stress (e.g. surgery, infection, trauma). As the treatment has a modified-release profile, blood tests are used to monitor clinical response, assessment of the evening dose should be done with a morning blood test and assessment of the morning dose should be done with an early afternoon blood test.

During excessive physical and/or mental stress it may be necessary to increase the dose of Efmody, and/or add additional immediate release hydrocortisone especially in the afternoon or evening.

Dose adjustments should be considered in case of concomitant use of potent CYP3A4 inducers or inhibitors (see section 4.5).

Treatment in CAH

Recommended replacement doses of hydrocortisone are 10-15 mg/m^2/day in adolescents aged 12 years and over who have not completed growth, and 15-25 mg/day in adolescents who have completed growth and adult patients with CAH. In patients with some remaining endogenous cortisol production a lower dose may be sufficient.

At initiation the total daily dose should be split into two doses with two thirds to three quarters of the dose given in the evening at bedtime and the rest given in the morning. Patients should then be titrated based on their individual response.

The morning dose should be taken on an empty stomach at least 1 hour before a meal and the evening dose taken at bedtime at least 2 hours after the last meal of the day.

Changing from conventional oral glucocorticoid treatment to Efmody

When changing patients from conventional oral hydrocortisone replacement therapy to Efmody, the identical total daily dose should be given, but the dose should be given in two doses with two thirds to three quarters of the dose given in the evening at bedtime and the rest given in the morning.

When changing patients from other glucocorticoids to Efmody an appropriate conversion factor should be used, and the patient monitored for response carefully.

Conversion to Efmody might elicit symptoms of adrenal insufficiency or overreplacement during dose optimisation.

A starting dose exceeding 40 mg per day of hydrocortisone is not recommended.

During serious trauma, intercurrent illness or periods of stress

In severe situations, an increase in dose is immediately required and oral administration of hydrocortisone must be replaced with parenteral treatment (see section 4.4).

In less severe situations when parenteral administration of hydrocortisone is not required, during periods of physical and/or mental stress, additional immediate release hydrocortisone at the same total daily dose as Efmody should be given in three divided doses; Efmody should be continued as well with the usual regimen (i.e. a doubled daily dose of hydrocortisone) to allow for easy return to the normal replacement dose of Efmody once additional hydrocortisone is no longer required.

In case of long-term increases in hydrocortisone daily dose due to prolonged periods of stress or illness, the additional hydrocortisone should be carefully weaned off.

Missed doses

If a dose of Efmody is missed it is recommended that it be taken as soon as possible.

Special populations

Elderly

No clinical data on the safety and efficacy of Efmody are available in elderly patients over the age of 65 years.

Renal impairment

There is no need for dose adjustment in patients with mild to moderate renal impairment. In patients with severe renal impairment monitoring of the clinical response is recommended and adjustment of dose may be necessary (see section 4.4).

Hepatic impairment

There is no need for dose adjustment in patients with mild to moderate hepatic impairment. In patients with severe hepatic impairment monitoring of the clinical response is recommended and adjustment of dose may be necessary (see section 4.4).

Paediatric population

No clinical data on the safety and efficacy of Efmody are available in children aged below 12 years. Other hydrocortisone containing medicinal products are available for children below 12 years.

Adolescents

No clinical data on the safety and efficacy of Efmody are available in adolescents aged 12 to 18 years.

Method of administration

The capsules must be given orally.

Patients should be advised to swallow the capsules with water to wash the capsules down.

The capsules should not be chewed as chewing the capsule could affect the release profile.

4.3 Contraindications

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

4.4 Special warnings and precautions for use

Adrenal crisis

Acute adrenal insufficiency may develop in patients with known adrenal insufficiency who are on inadequate daily doses or in situations with increased cortisol need. Therefore, patients should be advised of the signs and symptoms of acute adrenal insufficiency and of adrenal crisis and the need to seek immediate medical attention. Sudden discontinuation of therapy with hydrocortisone risks triggering an adrenal crisis and death.

During adrenal crisis parenteral, preferably intravenous administration of hydrocortisone in high doses, together with sodium chloride 9 mg/ml (0.9%) solution for infusion, should be administered according to current treatment guidelines.

Pre-operatively, during serious trauma or during intercurrent illness

Pre-operatively, anaesthetists must be informed if the patient is taking corticosteroids or has previously taken corticosteroids.

Parenteral administration of hydrocortisone is warranted during transient illness episodes such as severe infections, in particular gastroenteritis associated with vomiting and/or diarrhoea, high fever of any aetiology or extensive physical stress, such as for instance serious accidents and surgery under general anaesthesia. Where parenteral hydrocortisone is required, the patient should be treated in a facility with resuscitation facilities in case of evolving adrenal crisis.

In less severe situations when parenteral administration of hydrocortisone is not required, for instance low grade infections, moderate fever of any aetiology and stressful situations such as minor surgical procedures, there should be high awareness of the risk of developing acute adrenal insufficiency.

Infections

Infection should not be more likely at a replacement dose of hydrocortisone, but all infections should be taken seriously, and an increase in steroid dose be initiated early (see section 4.2). Patients with CAH are at risk of life-threatening adrenal crisis during infection so clinical suspicion of infection should be high and specialist advice should be sought early.

Immunisation

Treatment schedules of corticosteroids for people with CAH do not cause immunosuppression and are not, therefore, contraindications for administration of live vaccines.

Undesirable effects of corticosteroid replacement therapy

Most undesirable effects of corticosteroids are dose and duration of exposure related. Undesirable effects are therefore less likely when using corticosteroids as replacement therapy.

Impaired glucose tolerance and diabetes are associated with treatment with glucocorticoids. Patients should be warned of the signs of diabetes and the need to seek medical advice if they occur.
All glucocorticoids increase calcium excretion and reduce the bone-remodelling rate. Long-term glucocorticoid replacement therapy may therefore reduce bone mineral density (see section 4.8).

Patients should be warned that potentially severe psychiatric adverse reactions; euphoria, mania, psychosis with hallucinations and delirium have been seen in adult patients at replacement doses of hydrocortisone (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 should be encouraged to seek medical advice if worrying psychological symptoms develop, especially if depressed mood or suicidal ideation is suspected. Patients 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.

Rare instances of anaphylactoid reactions have occurred in patients receiving corticosteroids, medical advice should be sought immediately in the case of anaphylactoid symptoms (see section 4.8).

Gastric emptying and motility disorders

Modified-release formulations, such as Efmody are not recommended in patients with increased gastrointestinal motility, i.e. chronic diarrhoea, due to the risk of impaired cortisol exposure. There are no data in patients with confirmed slow gastric emptying or decreased motility disease/disorder. The clinical response should be monitored in patients with these conditions.

Growth retardation

Corticosteroids may cause growth retardation in childhood and adolescence; this may be irreversible. Treatment should be limited to the minimum dose required to achieve desired clinical response and when reduction in dose is possible, the reduction should be gradual. Excessive weight gain with decreased height velocity or other symptoms or signs of Cushing syndrome indicate excessive glucocorticoid replacement. Children require frequent assessment to assess growth, blood pressure, and general well-being.

Accelerated sexual maturation

Adolescents with CAH may show accelerated sexual maturation. Patients should be closely monitored; and if signs of early puberty or accelerated sexual maturation are present, an increase in dose should be considered. Careful and regular monitoring of adolescent patients with dose adjustment according to the response of the individual patient is recommended.

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 which have been reported after use of systemic and topical corticosteroids.

Treatment of CAH often warrants additional treatment with mineralocorticosteroids.

Precaution

In both men and women who have lower fertility due to CAH, fertility may be restored shortly after beginning treatment with Efmody, which can lead to unexpected pregnancies. Patients should be informed of the potential for restored fertility when starting treatment with Efmody, to be able to consider if a contraceptive measure is needed (see section 4.6).

4.5 Interaction with other medicinal products and other forms of interaction

Hydrocortisone is metabolised by cytochrome P450 3A4 (CYP3A4). Concomitant administration of medicinal products that are inhibitors or inducers of CYP3A4 may therefore lead to unwanted alterations in serum concentrations of hydrocortisone with the risk of adverse reactions, particularly adrenal crisis. The need for dose adjustment when such medicinal products are used can be anticipated and patients should be closely monitored.

Medicinal products inducing CYP3A4, requiring a potential increase in Efmody dosing, include but are not limited to:

- Anticonvulsants: phenytoin, carbamazepine and oxcarbazepine
- Antibiotics: rifampicin and rifabutin
- Barbiturates including phenobarbital and primidone
- Antiretroviral medicinal products: efavirenz and nevirapine
- Herbal medicinal products such as St. John's Wort

Medicinal products/substances inhibiting CYP3A4, requiring a potential decrease in hydrocortisone dosing, include but are not limited to:

- Anti-fungals: itraconazole, posaconazole, voriconazole
- Antibiotics: erythromycin and clarithromycin
- Antiretroviral medicinal products: ritonavir
- Grapefruit juice
- Liquorice

The desired actions of hypoglycaemic medicinal products including insulin are antagonised by corticosteroids.

4.6 Fertility, pregnancy and lactation

Pregnancy

Hydrocortisone crosses the placenta. Hydrocortisone is preferentially metabolised by placental 11βHSD2 to inactive cortisone reducing the fetal exposure. There are no indications that replacement therapy with hydrocortisone in pregnant women is associated with adverse consequences for the fetus. Hydrocortisone for replacement therapy can be used during pregnancy.
Studies in animals have shown reproductive toxicity of corticosteroids (see section 5.3).

Breast-feeding

Hydrocortisone is excreted in breast milk. However, the doses of hydrocortisone used for replacement therapy probably do not clinically significantly affect the child. Hydrocortisone for replacement therapy can be used during breast-feeding.

Fertility

In both men and women who have lower fertility due to CAH, fertility may be restored shortly after beginning treatment with Efmody. In women, a reduction of 17-OH progesterone and androstenedione will lead to a corresponding fall in progesterone and testosterone which may restore menses/fertility. (see section 4.4).

4.7 Effects on ability to drive and use machines

Efmody has minor influence on the ability to drive and use machines. Fatigue and dizziness have been reported. Untreated and poorly replaced adrenal insufficiency may affect the ability to drive and use machines.

4.8 Undesirable effects

Summary of safety profile

In the clinical trial programme the overall most common serious adverse events were acute adrenal insufficiency (4.2% of patients treated with Efmody), another common reaction, in relation to Efmody was fatigue (11.7% of patients), headache (7.5%), increased appetite (5.8%), dizziness (5.8%) and increased weight (5.8%).

Tabulated list of adverse reactions

The commonest reactions reported to Efmody in the pooled population in the clinical trial programme, are tabulated below. Frequencies are defined as very common (≥ 1/10), common (≥ 1/100 to < 1/10), uncommon (≥ 1/1,000 to < 1/100).

Table 1. Tabulated summary of adverse reactions seen in clinical trial programme

MedDRA system organ classification	**Event**	**Frequency**
Endocrine disorders	Adrenal insufficiency including acute events	Common
Metabolism and nutrition disorders	Increased appetite	Common
	Decreased appetite	Common
	Impaired fasting glucose	Common
Psychiatric disorders	Insomnia	Common
	Abnormal dreams	Common
	Depressed mood	Common
	Sleep disorder	Common
Nervous System Disorders	Headache	Common
	Dizziness	Common
	Carpal tunnel syndrome	Common
	Paraesthesia	Common
Gastrointestinal disorders	Nausea	Common
	Abdominal pain upper	Common
Skin and subcutaneous tissue disorders	Acne	Common
	Hair growth abnormal	Common
Musculoskeletal and connective tissue disorders	Arthralgia	Common
	Muscle fatigue*	Common
	Myalgia	Common
	Pain in extremity	Common
General disorders and administration site conditions	Asthenia	Common
	Fatigue	Very Common
Investigations	Weight increased	Common
	Renin increased	Common

*Includes muscular weakness

Description of selected adverse reactions

Adrenal insufficiency (including acute events).

Events of acute adrenal insufficiency were reported during the clinical trial programme but none were considered related to Efmody. Acute adrenal insufficiency should be monitored for and treated promptly in patients with adrenal insufficiency (see section 4.2 and 4.4).

Rare instances of anaphylactoid reactions have occurred in patients receiving corticosteroids especially when a patient has a history of allergies to medicinal products.

Historical cohorts of adults treated from childhood for CAH have been found to have reduced bone mineral density and increased fracture rates (see section 4.4) - it is unclear if these relate to hydrocortisone therapy using current replacement regimens.

Historical cohorts of adults treated from childhood for CAH have been found to have raised cardiovascular risk factors and a higher risk of cerebrovascular disease than the general population - it is unclear if these relate to hydrocortisone therapy using current replacement regimens.

Paediatric population

No paediatric patients were included in the clinical development programme for Efmody. Hydrocortisone has been used for more than 60 years in paediatrics with a safety profile similar to that in adults. Growth retardation has been seen in children treated with hydrocortisone for CAH and can

be caused by both the disorder and hydrocortisone. Accelerated sexual maturation has been seen in hydrocortisone-treated paediatric CAH patients and is associated with excess adrenal androgen production (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. Healthcare professionals are asked to report any suspected adverse reactions via Yellow Card Scheme. Website: www.mhra.gov.uk/yellowcard or search for MHRA Yellow Card in the Google Play or Apple App Store.

4.9 Overdose

Reports of acute toxicity and/or deaths following hydrocortisone overdose 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/her unusually susceptible to ill effects from hydrocortisone. In which case, symptomatic treatment should be instituted as necessary.

5. PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Corticosteroids for systemic use; glucocorticoids. ATC code: H02AB09.

Mechanism of action

Hydrocortisone is a glucocorticoid. Glucocorticoids have multiple effects in multiple tissues through actions on the intracellular steroid receptors.

Pharmacodynamic effects

Hydrocortisone is a glucocorticoid and the synthetic form of endogenously produced cortisol. Glucocorticoids are adrenocortical steroids, both naturally occurring and synthetic, which are readily absorbed from the gastro-intestinal tract. Cortisol is the principal corticosteroid secreted by the adrenal cortex. Naturally occurring glucocorticoids (hydrocortisone and cortisone), which also have salt-retaining properties, are used as replacement therapy in adrenocortical deficiency states. They are also used for their potent anti-inflammatory effects in disorders of many organ systems. Glucocorticoids cause profound and varied metabolic effects. In addition, they modify the body's immune responses to diverse stimuli.

Clinical efficacy and safety

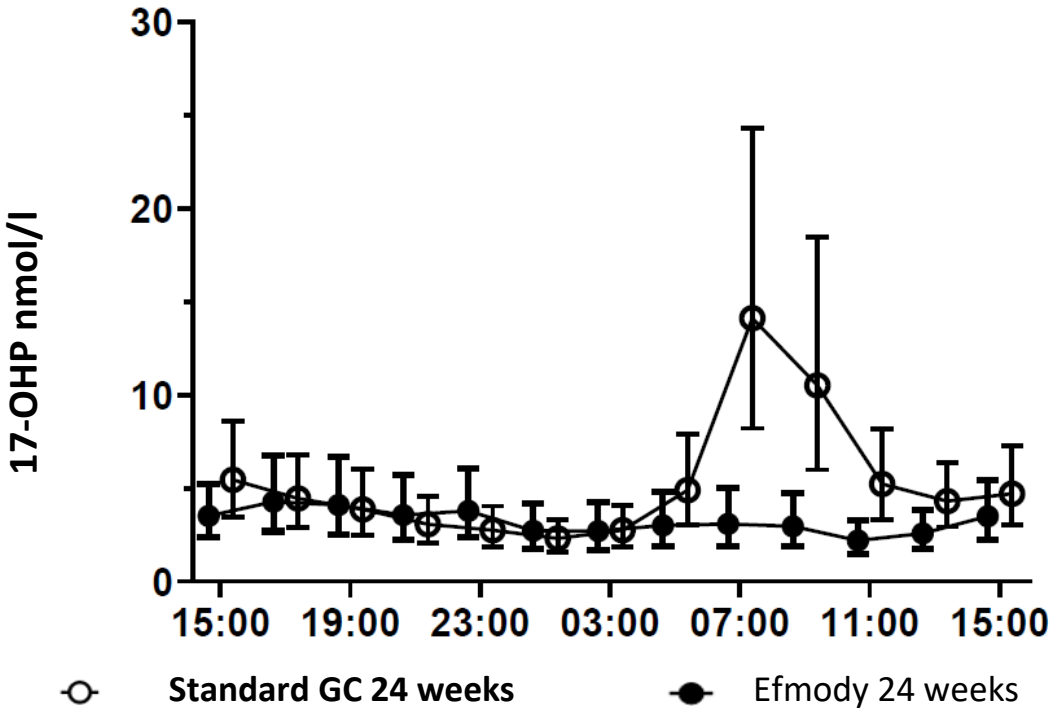
A study in 122 participants with genetically diagnosed 21-hydroxylase deficiency randomised to Efmody or continuation of standard care with blinded titration of dose and regular overnight profiles failed to meet its primary endpoint of superiority in change from baseline to 24 weeks of the mean of the 24-hour standard deviation score (SDS) profile for 17-hydroxyprogesterone 17-OHP. The 17-OHP SDS was lower in the Efmody cohort than standard therapy at 4 and 12 weeks. At 24 weeks the 17-OHP SDS was lower in the morning period (07:00 hrs to 15:00 hrs) but not in the evening or overnight (see also Figure 1 for the geometric mean 24-hour profile of 17-OHP after 24 weeks intensive treatment). A reduction in the 17-OHP area under the curve occurred in both groups, with greater reduction in the Efmody cohort. The percentage of patients with controlled 09:00 hrs 17-OHP (<36nmol/l) was 50% at baseline and at 24 weeks was 91% in the Efmody cohort and 71% in the standard therapy cohort. Efmody patients suffered no adrenal crises (compared to 3 in the control arm) and had fewer sick day episodes where increased dosing due to stress was required was required (26 vs 36 in the control arm) despite reporting more episodes of intercurrent infective or gastro-intestinal

illness. Glucocorticoid daily dose, measured as a hydrocortisone equivalent dose, increased in most subjects during the study (see Table 2).

Table 2. Glucocorticoid daily dose changes during the phase 3 study DIUR-005

Dose	**Hydrocortisone modified-release hard capsules group**		**Standard glucocorticoid group**	
	Baseline	**24 weeks**	**Baseline**	**24 weeks**
All (hydrocortisone dose equivalents)*				
Median daily dose (mg)	25.0	30.0	25.0	31.3
On hydrocortisone at baseline				
Median daily dose (mg)	20.0	25.0	23.75	25.0
On prednis(ol)one at baseline				
Median daily dose (mg)	30	27.5	26.6	32.8
On dexamethasone at baseline				
Median daily dose (mg)	30	30	40	40

Figure 1. End of study geometric mean 24-hour profile of 17-OHP after 24 weeks intensive treatment with either Efmody (closed circles) or standard therapy (open circles).



A safety extension study of 91 patients with titration by investigators was characterised by dose reductions with median daily dose of Efmody at 18 months interim analysis (n=50) being 20 mg (from a median baseline daily dose of 30 mg) with 17-OHP levels remaining in the clinically determined optimal range and androstenedione at or below the reference range for normal individual.

In the safety assessment of clinical studies, differences between the treatment arms in treatment related AE's were reported (by preferred term [PT]). The most notable differences between the Efmody and standard glucocorticoid therapy pools, respectively, were observed for headache (7.5% vs 1.6%), increased appetite (5.8% vs 3.3%), weight increase (including abnormal weight gain) (9.2% vs1.6%), decreased appetite (5.0% vs 0%) and nausea (4.2% vs 1.6%)."

5.2 Pharmacokinetic properties

Absorption

Following a single oral administration in fasted dexamethasone-suppressed healthy adults, the rate of absorption of hydrocortisone from Efmody 20 mg was delayed and reduced compared to immediate release hydrocortisone tablets 20 mg, as reflected by a lower C_{max} and a significantly longer T_{max} for Efmody (median T_{max} for serum cortisol of 4.5 hours and 0.88 hours for Efmody and hydrocortisone tablets respectively). Efmody appeared to be more bioavailable relative to immediate release hydrocortisone tablets, with overall exposure to serum cortisol and derived free cortisol approximately 19% and 13% higher respectively for Efmody.

In the same population, food (high fat meal started 30 minutes before dosing) was found to delay and reduce the rate of absorption of hydrocortisone from Efmody 20 mg, as reflected by a longer T_{max} (median T_{max} for serum cortisol of 6.75 hours and 4.5 hours for fed and fasted subjects respectively) and lower C_{max} (reduced by approximately 20% in fed subjects). Overall exposure appeared similar in fed and fasted subjects (90% confidence intervals for the fed/fasted ratio of the geometric least square mean of AUC_{0-t} and AUC_{0-inf} were within 80-125%). This effect is therefore not considered clinically significant.

Distribution

90% or more of circulating hydrocortisone is reversibly bound to protein.

The binding is accounted for by two protein fractions. One, corticosteroid-binding globulin is a glycoprotein; the other is albumin.

Biotransformation

Hydrocortisone is metabolised in the liver and most body tissues to hydrogenated and degraded forms such as tetrahydrocortisone and tetrahydrocortisol which are excreted in the urine, mainly conjugated as glucuronides, together with a very small proportion of unchanged hydrocortisone. Hydrocortisone is both metabolised by and a regulator of CYP3A4.

Elimination

In the fasted dexamethasone-suppressed healthy adult population described above, terminal elimination half-life values were similar for Efmody and hydrocortisone tablets (geometric mean $t_{1/2}$ for serum cortisol of 1.38 hours and 1.40 hours respectively). Clearance appeared higher for hydrocortisone tablets relative to Efmody (geometric mean CL/F for serum cortisol of 22.24 L/h and 18.48 L/h respectively).

Paediatrics

The pharmacokinetics of Efmody have not been studied in the paediatric population.

Other populations

No studies have been conducted in patients with hepatic or renal impairment.

5.3 Preclinical safety data

Administration of corticosteroids to pregnant animals can cause abnormalities of fetal development including cleft palate, intrauterine growth retardation and effects on brain growth and development.

6. PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Granules

Microcrystalline cellulose
Povidone
Methacrylic acid-methyl methacrylate copolymer (1:2)
Methacrylic acid-methyl methacrylate copolymer (1:1)
Talc
Dibutyl sebacate

Capsule

Gelatin

Efmody 5 mg modified-release hard capsules (white/blue)

Titanium dioxide (E171)
Indigotine (E132)

Efmody 10 mg modified-release hard capsules (white/green)

Titanium dioxide (E171)
Indigotine (E132)
Yellow iron oxide (E172)

Efmody 20 mg modified-release hard capsules (white/orange)

Titanium dioxide (E171)
Yellow iron oxide (E172)
Red iron oxide (E172)

Printing ink

Shellac
Black iron oxide (E172)
Propylene glycol
Potassium hydroxide

6.2 Incompatibilities

Not applicable

6.3 Shelf life

3 years

6.4 Special precautions for storage

Store in the original package.

Keep the bottle tightly closed in order to protect from moisture.

This medicinal product does not require any special temperature storage conditions.

6.5 Nature and contents of container

The capsules are provided in high-density polyethylene bottles with child resistant, tamper-evident polypropylene screw cap with integrated desiccant. Each bottle contains 50 modified-release hard capsules.

Pack sizes:
Carton containing 1 bottle of 50 modified-release hard capsules.
Carton containing 2 bottles of 50 modified-release hard capsules (100 capsules).

Not all pack sizes may be marketed.

6.6 Special precautions for disposal and other handling

Any unused medicinal product or waste material should be disposed of in accordance with local requirements.

7. MARKETING AUTHORISATION HOLDER

Neurocrine Netherlands B.V.
Van Heuven Goedhartlaan 935 A,
1181LD Amstelveen,
The Netherlands
diurnalinfo@neurocrine.com

8. MARKETING AUTHORISATION NUMBER(S)

Efmody 5 mg modified-release hard capsules	PLGB 50616/0011 (50 and 100 (2x50) capsules)
Efmody 10 mg modified-release hard capsules	PLGB 50616/0012 (50 and 100 (2x50) capsules)
Efmody 20 mg modified-release hard capsules	PLGB 50616/0013 (50 capsules)

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

Date of first authorisation (UK Northern Ireland): 27 May 2021
Date of first authorisation (UK Great Britain): 01 July 2021

10. DATE OF REVISION OF THE TEXT

28/10/2024

Detailed information on this medicine is available on the European Medicines Agency web site: http://www.ema.europa.eu.