AUSTRALIAN PRODUCT INFORMATION

BUDENOFALK® (BUDESONIDE) FOAM

1 NAME OF THE MEDICINE

Budesonide

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

BUDENOFALK® foam contains the active ingredient budesonide.

For the full list of excipients, see Section 6.1 List of excipients.

3 PHARMACEUTICAL FORM

Foam

4 CLINICAL PARTICULARS

4.1 THERAPEUTIC INDICATIONS

BUDENOFALK® foam is indicated in the treatment of active rectal and rectosigmoid disease in ulcerative colitis.

4.2 DOSE AND METHOD OF ADMINISTRATION

For adults aged >18 years of age: Apply one actuation of 2 mg budesonide daily. BUDENOFALK® 2 mg foam can be applied in the morning or evening.

BUDENOFALK® 2 mg foam should be in room temperature when applied.

The canister is first fitted with an applicator and then shaken for about 15 seconds before the applicator is inserted into the rectum as far as comfortable. Note that the dose is only sufficiently accurate when the pump dome is held downwards as vertically as possible. To administer a dose of BUDENOFALK® 2 mg foam , the pump dome is fully pushed down and very slowly released. Following the activation the applicator should be held in position for 10-15 seconds before being withdrawn from the rectum.

The best results are obtained when the intestine is evacuated prior to administration of BUDENOFALK® 2mg foam.

The attending physician determines the duration of use. An acute episode generally subsides after 6 to 8 weeks.

Treatment may be continued in patients showing progressive improvement, but it should not be persisted with if the response has been inadequate. Continuous treatment beyond 8

weeks has not been assessed. BUDENOFALK® 2 mg foam should not be used after this time.

Topical steroids including BUDENOFALK® have not been shown to be effective in the maintenance of remission of ulcerative colitis.

Do not use BUDENOFALK® foam after 4 weeks of first opening the container.

4.3 CONTRAINDICATIONS

BUDENOFALK® foam is contraindicated in patients with the following:

- hypersensitivity to budesonide or any of the ingredients
- hepatic cirrhosis

4.4 SPECIAL WARNINGS AND PRECAUTIONS FOR USE

Identified precautions

Topical steroids have not been demonstrated to maintain remission in ulcerative colitis. BUDENOFALK® should only be used for treatment of active ulcerative colitis. Treatment should not continue beyond 8 weeks.

Treatment with BUDENOFALK® foam results in lower systemic steroid levels than conventional oral steroid therapy. Particular care is needed in patients who are transferred from systemic glucocorticosteroid treatment with higher systemic effect to BUDENOFALK® foam. These patients may have adrenocortical suppression. Therefore, monitoring of adrenocortical function may be considered in these patients and their dose of systemic steroid should be reduced cautiously.

Caution is required in patients with tuberculosis, hypertension, diabetes mellitus, osteoporosis, peptic ulcer, glaucoma, cataracts, family history of diabetes, family history of glaucoma, or any other condition in which glucocorticoids may have undesirable effects.

Systemic effects of corticosteroids may occur, particularly when prescribed at high doses and for prolonged periods. Such effects may include Cushing's syndrome, adrenal suppression, growth retardation, decreased bone mineral density, cataract, glaucoma and a wide range of psychiatric/behavioural effects (see Section 4.6 Adverse Effects - Undesirable Effects).

Corticosteroids may cause suppression of the HPA axis and reduce the stress response. When patients are subject to surgery or other stresses, supplementary systemic glucocorticoid treatment is recommended.

As with all glucocorticosteroids, some degree of adrenal suppression may occur in particularly sensitive patients, therefore, monitoring of haematological and adrenal function is strongly advised.

Infection: Suppression of the inflammatory response and immune function increases the susceptibility to infections and their severity. The risk of deterioration of bacterial, fungal, amoebic and viral infections during glucocorticoid treatment should be carefully considered. The clinical presentation may often be atypical and serious infections such as septicaemia and tuberculosis may be masked, and therefore may reach an advanced stage before being recognised.

Chickenpox: Chickenpox is of particular concern since this normally minor illness may be fatal in immunosuppressed patients. Patients without a definite history of chickenpox should be advised to avoid close personal contact with chickenpox or herpes zoster and if exposed they should seek urgent medical attention. If the patient is a child, parents must be given the above advice. 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.

Measles: Patients with compromised immunity who have come into contact with measles should, wherever possible, receive normal immunoglobulin as soon as possible after exposure.

Live vaccines: Live vaccines should not be given to individuals with chronic corticosteroid use. The antibody response to other vaccines may be diminished.

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.

Concomitant treatment with ketoconazole or other CYP3A4 inhibitors should be avoided (see Section 4.4 - Interactions with Other Medicines and Other Forms of Interactions).

This medicine contains cetyl alcohol and propylene glycol, which can cause local skin reactions (e.g. contact dermatitis).

Use in hepatic impairment

Based on the experience with oral preparations of budesonide in patients suffering from late stage primary biliary cirrhosis (PBC) with hepatic cirrhosis an increased systemic availability of budesonide in all patients with severely impaired hepatic function is to be expected. However, in patients with liver disease without hepatic cirrhosis oral budesonide in daily doses of 3 mg TID was safe and well tolerated. There is no evidence that a specific dose recommendation for patients with non-cirrhotic liver diseases or only slightly impaired liver function is necessary. As the plasma levels of budesonide appear to be generally slightly higher with rectal budesonide, BUDENOFALK® foam should be used only with caution in patients with hepatic impairment.

Use in the elderly

The experience in elderly with BUDENOFALK® foam is limited.

Paediatric use

BUDENOFALK® foam is not recommended for use in children or adolescents. Long term effects, including on height and bone density have not been assessed.

Effects on laboratory tests

Because adrenal function may be suppressed by treatment with budesonide, an ACTH stimulation test for diagnosing pituitary insufficiency might show false results (low values).

4.5 INTERACTIONS WITH OTHER MEDICINES AND OTHER FORMS OF INTERACTIONS

Pharmacodynamic interactions

Cardiac glycosides:

The action of the glycoside can be potentiated by potassium deficiency.

Saluretics:

Potassium excretion can be enhanced.

Pharmacokinetic interactions

Cytochrome P450:

- *CYP3A4 inhibitors:*
  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. Ketoconazole 200 mg orally once daily increased the plasma concentrations of budesonide (3 mg single dose) approximately 6-fold during concomitant administration. When ketoconazole was administered 12 hours after budesonide, the concentrations

increased approximately 3-fold. As there are not enough data to give dose recommendations, the combination should be avoided.
Other potent inhibitors of CYP3A4 such as ritonavir, itraconazole, clarithromycin, and grapefruit juice are also likely to cause a marked increase of the plasma concentrations of budesonide. Therefore concomitant application of budesonide should be avoided.

- *CYP3A4 inducers:*
  Compounds or drugs such as carbamazepine and rifampicin, which induce CYP3A4, might reduce the systemic but also the local exposure of budesonide at the gut mucosa. An adjustment of the budesonide dose might be necessary.
- *CYP3A4 substrates:*
  Compounds or drugs which are metabolized by CYP3A4 might be in competition with budesonide. This might lead to an increased budesonide plasma concentration if the competing substance has a stronger affinity to CYP3A4, or – if budesonide binds stronger to CYP3A4 – the competing substance might be increased in plasma and a dose-adaption/reduction of this drug might be required.

Elevated plasma concentrations and enhanced effects of corticosteroids have been reported in women also receiving oestrogens or oral contraceptives, but this has not been observed with oral low dose combination contraceptives.

4.6 FERTILITY, PREGNANCY AND LACTATION

Effects on fertility

There are no data on the effect of budesonide on human fertility. Subcutaneous administration of budesonide to rats at doses up to 20 µg/kg/day did not affect fertility.

Use in pregnancy – Pregnancy Category B3

Administration during pregnancy should be avoided unless there are compelling reasons for therapy with BUDENOFALK® foam.

There are no data on pregnancy outcomes after rectal administration of budesonide in humans. Although data on the use of inhaled budesonide in a large number of exposed pregnancies indicate no adverse effects, the maximal concentration of budesonide in plasma is expected to be higher with rectal budesonide compared to inhaled budesonide.

In pregnant animals, administration of budesonide, like other glucocorticoids, has been shown to cause fetal death and abnormalities of fetal development (reductions in fetal/pup growth and litter size, skeletal and visceral abnormalities) The relevance of these findings to humans has not been established.

Use in lactation

Budesonide is excreted in human milk. However, only minor effects on the breast-fed infant are anticipated after BUDENOFALK® administration within the therapeutic range. A decision should be made whether to discontinue breastfeeding or to discontinue BUDENOFALK® taking into account the benefit of breastfeeding for the child and the benefit of therapy for the woman.

4.7 EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

The effects of this medicine on a person's ability to drive and use machines were not assessed as part of its registration.

4.8 ADVERSE EFFECTS (UNDESIRABLE EFFECTS)

Reporting suspected adverse effects

Reporting suspected adverse reactions after registration 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 at www.tga.gov.au/reporting-problems.

In clinical trial BUF-6/UCA involving a total of 120 patients receiving BUDENOFALK® foam budesonide was well tolerated. The table below shows the adverse events:

Table 1

Results of study BUF-6/UCA: Summary of number and rate (%) of treatment-emergent adverse events by system organ class

Adverse event by body system	**BUDENOFALK® foam 2 mg** **N = 120**	**Hydrocortisone acetate 100 mg** **N = 128**
Body as a whole		
any event	17 (14%)	24 (19%)
abdominal pain	7 (6%)	9 (7%)
headache	4 (3%)	6 (5%)
infection	4 (3%)	2 (2%)
asthenia	2 (2%)	2 (2%)
flu syndrome	2 (2%)	2 (2%)

back pain	1 (<1%)	2 (2%)
fever	1 (<1%)	2 (2%)
Digestive system		
any event	15 (13%)	14 (11%)
diarrhoea	5 (4%)	2 (2%)
gastrointestinal disorder	3 (3%)	3 (2%)
rectal disorder	2 (2%)	3 (2%)
rectal haemorrhage	2 (2%)	1 (<1%)
nausea	0	2 (2%)
Haemic and lymphatic system		
any event	4 (3%)	6 (5%)
sedimentation rate increased	3 (3%)	4 (3%)
leukocytosis	2 (2%)	1 (<1%)
Metabolic and nutritional		
any event	2 (2%)	5 (4%)
alkaline phosphatase increased	0	2 (2%)
peripheral oedema	0	2 (2%)
Musculoskeletal system		
any event	1 (<1%)	7 (5%)
myalgia	1 (<1%)	2 (2%)
arthralgia	0	2 (2%)
Respiratory system		
any event	3 (3%)	7 (5%)

bronchitis	0	4 (3%)
cough increased	1 (<1%)	2 (2%)
Skin and appendages		
any event	5 (4%)	4 (3%)
acne	2 (2%)	1 (<1%)
Urogenital system		
any event	2 (2%)	2 (2%)
urinary tract infection	2 (2%)	0

Undesirable effects were reported in 14% of patients in clinical trials with BUDENOFALK® foam. Burning in the rectum or pain were common, and nausea, headache and an increase in liver enzymes were uncommon.

Post-marketing adverse effects

The following suspect adverse drug reactions presented by body system have been spontaneously reported in international post marketing surveillance as well as in clinical trials of BUDENOFALK® preparations including enteric capsules and foam.

The assessment of undesirable effects is based on 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), including isolated reports.

Adverse drug reactions by frequency and system organ class (SOC):

Infections and parasitic diseases

Uncommon: urinary tract infections

Blood and lymphatic system disorders

Uncommon: anaemia, increase in erythrocyte sedimentation rate, leukocytosis

Metabolism and nutrition disorders

Uncommon: increased appetite

Psychiatric disorders

Common: Depression, irritability, euphoria

Uncommon: insomnia, psychomotor hyperactivity, anxiety

Rare: aggression

Musculoskeletal and connective tissue disorders:

Common: Muscle and joint pain, muscle weakness and twitching, osteoporosis

Rare: Osteonecrosis

Eye disorders:

Rare: Glaucoma, cataract, blurred vision

Nervous system disorders

Uncommon: headache, dizziness, disturbances of smell

Vascular disorders

Uncommon: hypertension

Gastrointestinal disorders

Common: dyspepsia

Uncommon: nausea, abdominal pain, flatulence, abdominal complaints, anal fissure, aphthous stomatitis, frequent urge to defecate, haemorrhoids, rectal bleeding, duodenal and gastric ulcer

Rare: pancreatitis

Hepatobiliary disorders

Uncommon: increase in transaminases (ALT, AST), increase in parameters of cholestasis (GGT, AP)

Skin and subcutaneous tissue disorders

Uncommon: acne, increased sweating

Rare: ecchymosis

Investigations

Uncommon: increase in amylase, change in cortisol

General disorders and administration site conditions

Common: burning in the rectum and pain

Uncommon: asthenia, increase in body weight

Systemically acting glucocorticoids

Occasionally side effects may occur which are typical for systemically acting glucocorticoids. These side effects, listed below, depend on the dosage, the period of treatment, concomitant or previous treatment with other glucocorticoids and the individual sensitivity.

Immune system disorders

Interference with the immune response (e.g. increase in risk of infections).

An exacerbation or the reappearance of extraintestinal manifestations (especially affecting skin and joints) can occur on switching a patient from the systemically acting glucocorticosteroids to the locally acting budesonide.

Metabolism and nutrition disorders

Cushing's syndrome: moon-face, truncal obesity, reduced glucose tolerance, diabetes mellitus, sodium retention with oedema formation, increased excretion of potassium, inactivity or atrophy of the adrenal cortex, growth retardation in children, disturbance of sex hormone secretion (e.g. amenorrhoea, hirsutism, impotence)

Psychiatric disorders

Depression, irritability, euphoria

In addition, a wide range of other psychiatric/behavioural effects may occur.

Eye disorders

Glaucoma, cataract

Nervous system disorders

Pseudotumor cerebri (including papilloedema) in adolescents

Vascular disorders

Hypertension, increased risk of thrombosis, vasculitis (withdrawal syndrome after long-term therapy)

Gastrointestinal disorders

Stomach complaints, duodenal ulcer, pancreatitis, constipation

Skin and subcutaneous tissue disorders

Allergic exanthema, red striae, petechiae, ecchymoses, steroid acne, delayed wound healing.
Local skin reactions such as contact dermatitis may occur.

Musculoskeletal, connective tissue and bone disorders

Aseptic bone necrosis (femur and head of the humerus), diffuse muscle pain and weakness, osteoporosis.

General disorders:

Tiredness, malaise.

Some of these undesired effects were reported after long-term use of orally administered budesonide.

There are no data on the long term use of BUDENOFALK® foam in patients with ulcerative colitis and long term use is not recommended.

Due to its local action, the risk of undesired effects of BUDENOFALK® foam is generally lower than with systemically acting glucocorticoids.

4.9 OVERDOSE

To date, no cases of overdosage with budesonide are known. In view of the properties of budesonide contained in BUDENOFALK® 2mg foam, an overdose resulting in toxic damage is extremely unlikely. For information on the management of overdose, contact the Poisons Information Centre on 13 11 26 (Australia).

5 PHARMACOLOGICAL PROPERTIES

5.1 PHARMACODYNAMIC PROPERTIES

Mechanism of action

Pharmacodynamic properties

The exact mechanism of action of budesonide in the treatment of ulcerative colitis/procto-sigmoiditis is not fully understood. Data from clinical pharmacology studies and controlled clinical trials strongly indicate that the mode of action of budesonide is predominantly based on a local action in the gut. Budesonide is a glucocorticoid with a high local anti-inflammatory effect. At a dosage of 2 mg budesonide, applied rectally, budesonide leads to practically no suppression of the hypothalamus-hypophysis-adrenal cortex axis.

BUDENOFALK® 2mg foam investigated up to the daily dosage of 4 mg budesonide showed virtually no influence on the basal plasma cortisol level.

Clinical trials

Study BUF-6/UCA was an active-controlled, multicentre, randomised, open-label,

parallel-group trial involving 251 patients with proctitis or proctosigmoiditis. A rectally applied hydrocortisone comparator (hydrocortisone acetate 100 mg foam [Colifoam®]) was compared to budesonide 2 mg (BUDENOFALK®) foam.

This study was designed to demonstrate equivalence between the two treatments with equivalence to be confirmed if the 95% CI for the between group difference in remission rate was no more than 15%. Equivalence was demonstrated only in the intent-to-treat population, not in the per-protocol population.

The primary efficacy parameter for this study was clinical remission defined as Disease Activity Index (DAI) ≤ 3 at the end of the 8-week treatment. DAI is defined as the sum of the scores of four parameters: weekly stool frequency, weekly rectal bleeding, mucosal appearance and physician's rating of disease activity.

Table 2

Clinical remission results

	Number (%) of patients with clinical remission based on DAI ≤ 3		**Difference in proportion (%) of response**	**[95% CI]**
	BUDENOFALK® Foam 2 mg o.d.	**Hydrocortisone acetate 100 mg foam o.d.**	**vs. comparator**	
Analysis				
PP	48/88 (55 %)	46/91 (51 %)	4.00*; 0.99**	(-10.6, 18.6)* (-14.5, 16.5)**
ITT	63/120 (53 %)	67/128 (52 %)	0.16*; -2.63**	(-12.3, 12.6)* (-15.8, 10.5)**

LOCF; * Response is experiencing clinical remission, with 'Not recorded' taken to be "Lack of remission"; ** Response is experiencing clinical remission, excluding data classified as

'Not recorded'

5.2 PHARMACOKINETIC PROPERTIES

Absorption

After oral administration, the systemic availability of budesonide is about 10%. After rectal administration the AUC is about 1.5-fold higher than in historical controls considering the identical oral budesonide dose. Peak levels are obtained after an average of 2-3 hours after administering BUDENOFALK® 2mg foam .

Distribution

Budesonide has a high volume of distribution (about 3 L/kg). Plasma protein binding averages 85 -90%.

Metabolism

Budesonide undergoes extensive biotransformation in the liver (approximately 90 %) to metabolites of low glucocorticoid activity. The glucocorticoid activity of the major metabolites, 6β-hydroxybudesonide and 16α-hydroxyprednisolone, is less than 1 % of that of budesonide.

Excretion

The average elimination half-life is about 3 - 4 hours. The mean clearance rate is about 10 - 15 L/min for budesonide, determined by HPLC-based methods.

Specific patient populations (liver disease):

Compromised hepatic function has an influence on the pharmacokinetics of budesonide with a reduced elimination rate and increased oral systemic availability.

BUDENOFALK® foam

The systemic bioavailability was calculated to be 15.3 % and 13.8 % after single and multiple dosing, respectively. Comparison of data after single and multiple dosing reveals no indication for a potential accumulation of budesonide in serum.

Pharmacokinetic data are summarised in the following table for BUDENOFALK® 2 mg foam after single dose and steady state dosing in 18 healthy subjects:

Table 3

Pharmacokinetic Parameters	**BUDENOFALK® foam (2 mg budesonide)**	
	Day 1* **Mean ± S.D.** **(n=18)**	**Day 5**** **Mean ± S.D.** **(n=18)**
t_{max} (h)	2.14 ± 1.28	1.81 ± 0.88
C_{max} (ng/ml)	0.84 ± 0.55	0.90 ± 0.49
C_{max}/D (ng/(ml x mg))	0.42 ± 0.28	0.45 ± 0.24
$C_{average}$ (ng/ml)	-	0.36 ± 0.21
AUC_{0-12} h or AUC_{ss} (ng x h/ml)	4.59 ± 2.94	4.30 ± 2.58
AUC∞ or AUC_{ss} (ng x h/ml)	5.36 ± 3.60	4.30 ± 2.58
AUC∞/D or AUC_{ss}/D (ng x h/(ml x mg))	2.68 ± 1.80	2.15 ± 1.29
k_e (h-1)	0.19 ± 0.07	-
$t_{½}$ (h)	4.05 ± 1.28	-
MRT (h)	6.36 ± 1.73	-
Cl/f (l/min)	9.33 ± 8.36	10.10 ± 5.14

* single dose on day 1; ** b.i.d. on days 2 to 5

Healthy male volunteers (n=18)

Scintigraphic study of a single rectal dose of 99mTc-labelled budesonide 2 mg (BUDENOFALK®) foam in 12 patients showed that the spread of the budesonide foam ranged between 11 and 40 cm (mean of 25.4 ± 10.3 cm) depending on the individual patient (this range includes from reaching the distal half of the sigmoid, to reaching the proximal third of the descending colon). The maximal spread was reached between 2 and 6 hours (mean of 4 hours) depending on the individual patient and remained relatively stable between 4 hours and 6 hours. The distal half sigmoid was reached in all patients on average after 2 hours and accounted for 27.4 % of the radiolabelled budesonide foam at 2 hours.

This study maximised conditions for spread of budesonide within the colon by having study subjects undergo a pre-study colonoscopy which would have emptied the bowel of faecal matter and by having them lie down for 4 hours after administration. It is anticipated that in clinical practise the spread would be somewhat less than was demonstrated in this study.

5.3 PRECLINICAL SAFETY DATA

Genotoxicity

Budesonide had no genotoxic effects in a battery of *in vitro* and *in vivo* tests.

Carcinogenicity

The carcinogenic potential of budesonide has been assessed in mice and rats at respective oral doses up to 200 and 50 µg/kg/day. No oncogenic effect was noted in mice. One study showed an increased incidence of malignant gliomas in male Sprague-Dawley rats given budesonide 50 µg/kg/day; however this was not confirmed in further studies in male Sprague-Dawley and Fischer rats. In male rats dosed with 10, 25 and 50 µg/kg/day, those receiving 25 and 50 µg/kg/day showed an increased incidence of primary hepatocellular tumours; however this was also observed in rats treated with prednisolone and triamcinolone acetonide, thus indicating a class effect of corticosteroids in rats.

6 PHARMACEUTICAL PARTICULARS

6.1 LIST OF EXCIPIENTS

BUDENOFALK foam contains the following excipients: cetyl alcohol, emulsifying wax, purified water, disodium edetate, steareth-10, propylene glycol, citric acid monohydrate and butane, isobutane and propane as propellants.

6.2 INCOMPATIBILITIES

Incompatibilities were either not assessed or not identified as part of the registration of this medicine.

6.3 SHELF LIFE

In Australia, information on the shelf life can be found on the public summary of the Australian Register of Therapeutic Goods (ARTG). The expiry date can be found on the

packaging.

6.4 SPECIAL PRECAUTIONS FOR STORAGE

Store below 25˚C. Do not refrigerate or freeze.

This is a pressurised container, containing flammable propellant.

Do not expose to temperature higher than 50°C, protect from direct sunlight.

Do not pierce or burn even when empty.

6.5 NATURE AND CONTENTS OF CONTAINER

BUDENOFALK® foam is supplied in aluminium pressurised container with metering valve together with 14 PVC applicators coated with white soft paraffin and liquid paraffin for administration of the foam and 14 plastic bags for hygienic disposal of the applicators.

Pack sizes: Original pack with 1 pressurised container, contains at least 14 doses of 1.2 g foam each.*

Original pack with 2 pressurised containers, contain at least 2 x 14 doses of 1.2 g foam each.

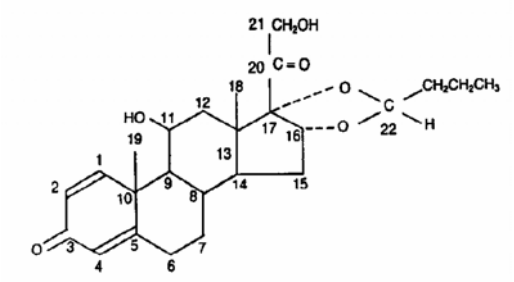
(*currently not marketed)

6.6 SPECIAL PRECAUTIONS FOR DISPOSAL

In Australia, any unused medicine or waste material should be disposed of by taking to your local pharmacy.

6.7 PHYSICOCHEMICAL PROPERTIES

Chemical structure



Chemical name: 16α,17α-butylidene dioxy-11ß, 21-dihydroxy-1,4-pregnadiene-3 ,20-dione

$C_{25}H_{34}O_6$ = 430.5

Budesonide is a white or almost white, crystalline powder. It is practically insoluble in water, freely soluble in methylene chloride, sparingly soluble in alcohol.

CAS number: 51333-22-3

7 MEDICINE SCHEDULE (POISONS STANDARD)

S4 – Prescription Only Medicine

8 SPONSOR

Dr Falk Pharma Australia Pty Ltd
815 Pacific Highway,
Chatswood, NSW 2067

9 DATE OF FIRST APPROVAL

12 June 2012

10 DATE OF REVISION

23 November 2020

BUDENOFALK® ® is a registered trademark of Dr. Falk Pharma GmbH, Germany.

SUMMARY TABLE OF CHANGES

Section Changed	Summary of new information
All	Change to product name throughout PI to remove incorrect dosage form enema.