DATA SHEET

JORVEZA®
Budesonide

1. NAME OF THE MEDICINE

JORVEZA® contains budesonide.

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Each orally disintegrating tablet contains either 0.5 mg or 1 mg of budesonide.

Each budesonide orally disintegrating tablet also contains sucralose and sodium.

For the full list of excipients, see section 6.1 LIST OF EXCIPIENTS.

3. PHARMACEUTICAL FORM

Orally disintegrating tablet.

Jorveza® 0.5 mg tablet. White or almost white, round, biplane orally disintegrating tablet with '0.5' debossed on one side.

Jorveza® 1 mg tablet. White or almost white, round, biplane orally disintegrating tablet.

4. CLINICAL PARTICULARS

4.1 THERAPEUTIC INDICATIONS

Jorveza® is indicated for the treatment of eosinophilic oesophagitis (EoE) in adults (older than 18 years of age).

4.2 DOSE AND METHOD OF ADMINISTRATION

The treatment with this medicinal product should be initiated by a physician experienced in the diagnosis and treatment of eosinophilic oesophagitis.

Dosage

The recommended daily dose for induction treatment is 2 mg budesonide as one 1 mg tablet in the morning and one 1 mg tablet in the evening.

The usual duration of induction treatment is 6 weeks. For patients who are not appropriately responding during 6 weeks the treatment can be extended to up to 12 weeks.

The recommended daily dose for maintenance of remission is 1 mg budesonide as one 0.5 mg tablet in the morning and one 0.5 mg tablet in the evening, or 2 mg budesonide as one 1 mg tablet in the morning and one 1 mg tablet in the evening, depending on the individual clinical requirement of the patient.

A maintenance dose of 1 mg budesonide twice daily is recommended for patients with a long standing disease history and/or high extent of oesophageal inflammation in their acute disease state, see also section 5.1 PHARMACODYNAMIC PROPERTIES.

The duration of maintenance therapy is determined by the treating physician.

Method of administration

The orally disintegrating tablet should be taken after a meal.

It should be placed on the tip of the tongue and gently pressed against the top of the mouth, where it will dissolve. This will usually take between two and five minutes, but can take up to 10 minutes or longer in some patients. The effervescence process starts after the orally disintegrating tablet comes into contact with saliva and stimulates the production of further saliva. The dissolved material should be swallowed with saliva little by little while the tablet dissolves. The orally disintegrating tablet should not be taken with liquid or food.

There should be at least 30 minutes after dosing before eating or drinking or performing oral hygiene. Any oral solutions, sprays or chewable tablets should not be used for at least 30 minutes before or after administration of Jorveza®.

The orally disintegrating tablet should not be chewed or swallowed undissolved. These measures ensure optimal exposure of the oesophageal mucosa to the active substance.

The tablet should be taken immediately once removed from the blister package.

Special populations
Renal impairment
There are currently no data available for patients with renal impairment. Because budesonide is not excreted via the kidneys, patients with mild to moderate impairment may be treated with caution with the same doses as patients without renal impairment. Budesonide is not recommended for use in patients with severe renal impairment.

Hepatic impairment
During treatment of patients with hepatic impairment with other budesonide containing products, budesonide levels were increased. However, no systematic study investigating different levels of hepatic impairment is available. Patients with hepatic impairment should not be treated with Jorveza® (see sections 4.4 SPECIAL WARNINGS AND PRECAUTIONS FOR USE and 5.2 PHARMACOKINETIC PROPERTIES).

Paediatric population
The safety and efficacy of Jorveza® in children and adolescents under the age of 18 years have not been established. No data are available. (see section 4.4 SPECIAL WARNINGS AND PRECAUTIONS FOR USE).

4.3 CONTRAINDICATIONS

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

Jorveza® is contraindicated in patients with uncontrolled infections or active tuberculosis.

4.4 SPECIAL WARNINGS AND PRECAUTIONS FOR USE

Infections
Suppression of the inflammatory response and immune function increases the susceptibility to infections and their severity. Symptoms of infections can be atypical or masked.

Jorveza® should not be used in patients with uncontrolled infections or active tuberculosis.

In clinical studies conducted with Jorveza®, oral, oropharyngeal and oesophageal candida infections have been observed with a high frequency (see section 4.8 ADVERSE EFFECTS (UNDESIRABLE EFFECTS).

If indicated, symptomatic candidiasis of the mouth and throat can be treated with topical or systemic anti-fungal therapy whilst still continuing treatment with Jorveza®.

Chickenpox, herpes zoster and measles can have a more serious course in patients treated with glucocorticosteroids. In patients who have not had these diseases, the vaccination status should be checked, and particular care should be taken to avoid exposure. If patients are infected or suspected of being infected, consider reduction or discontinuation of glucocorticosteroid treatment.

Vaccines
The co-administration of live vaccines and glucocorticosteroids should be avoided as this is likely to reduce the immune response to vaccines. The antibody response to other vaccines may be diminished.

Special populations
Patients with tuberculosis, hypertension, diabetes mellitus, osteoporosis, peptic ulcer, glaucoma, cataract, family history of diabetes or family history of glaucoma may be at higher risk of experiencing systemic glucocorticosteroid adverse reactions (see below and section 4.8 ADVERSE EFFECTS (UNDESIRABLE EFFECTS)) and should therefore be monitored for the occurrence of such effects. In these patients, caution should be exercised and the benefits of an oral glucocorticosteroid must be weighed against its risk. Jorveza® should not be used in patients with active tuberculosis or uncontrolled infection.

Use in Hepatic Impairment
Reduced liver function may affect the elimination of budesonide, causing higher systemic exposure. The risk of adverse reactions (systemic glucocorticosteroid effects) will be increased. However, no systematic data are available. Patients with hepatic impairment should therefore not be treated. Patients with hepatic impairment should therefore not be treated with Jorveza®.

Renal impairment
There are currently no data available for patients with renal impairment. Because budesonide is not excreted via the kidneys, patients with mild to moderate impairment may be treated with caution with the same doses as patients without renal impairment. Jorveza® is not recommended for use in patients with severe renal impairment.

Use in the elderly
There are insufficient data concerning the use of Jorveza® in patients aged ≥65 years. Caution should be exercised in elderly patients due to the potential for decreased hepatic, renal or cardiac function, or due to concomitant disease or therapies.

Paediatric use
The safety and efficacy of Jorveza® in children and adolescents under the age of 18 years have not been established; no data are available. Jorveza® should not be used in children and adolescents under the age 18 years. Glucocorticosteroids, including Jorveza®, may reduce growth velocity in children.

Systemic effects of glucocorticosteroids
Systemic effects of glucocorticosteroids (e.g., Cushing's syndrome, adrenal suppression, growth retardation, cataract, glaucoma, decreased bone mineral density and a wide range of psychiatric effects) may occur (see also section 4.8 ADVERSE EFFECTS (UNDESIRABLE EFFECTS) particularly when prescribed at high doses and for prolonged periods. These adverse reactions also depend on concomitant and previous glucocorticosteroid treatment and the individual sensitivity.

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.

Others
Cases of angioedema and/or contact dermatitis have been reported with the use of Jorveza. Treatment with Jorveza should be stopped if a patient develops swelling of the face, particularly around the mouth (lips, tongue or throat) and/or difficulties to breathe or swallow.

Glucocorticosteroids may cause suppression of the hypothalamic–pituitary–adrenal (HPA) axis and reduce the stress response. When patients are subject to surgery or other stresses, supplementary systemic glucocorticosteroid treatment is therefore recommended.

Concomitant treatment with ketoconazole or other CYP3A4 inhibitors should be avoided (see section 4.5 INTERACTIONS WITH OTHER MEDICINES AND OTHER FORMS OF INTERACTIONS).

Effects on laboratory tests
No data available.

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

Excipient with known effect
The 0.5 mg and 1 mg orally disintegrating tablets contain 26 mg of sodium in each tablet.

Therefore, the maximum daily dose of sodium intake from either strength is 52 mg per day if taken as recommended as either 2 x 0.5 mg tablet or 2 x 1 mg tablet (see section 4.2 DOSE AND METHOD OF ADMINISTRATION). This is equivalent to 2.6% of the WHO recommended maximum daily intake of 2 g sodium for an adult.

4.5 INTERACTIONS WITH OTHER MEDICINES AND OTHER FORMS OF INTERACTIONS

CYP3A4 inhibitors
Co-treatment with potent CYP3A inhibitors such as ketoconazole, ritonavir, itraconazole, clarithromycin, cobicistat and grapefruit juice may cause a marked increase of the plasma concentration of budesonide and is expected to increase the risk of systemic adverse reactions. Therefore, concomitant use 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 once daily orally increased the plasma concentration of budesonide (3 mg single dose) approximately 6-fold during concomitant administration. When ketoconazole was administered approximately 12 hours after budesonide, the plasma concentration of budesonide increased approximately 3-fold.

Oestrogens, oral contraceptives
Elevated plasma concentrations and enhanced effects of glucocorticosteroids have been reported in women also receiving oestrogens or oral contraceptives. No such effect has been observed with budesonide and concomitant intake of low-dose combination oral contraceptives.

Cardiac glycosides
The action of glycoside can be potentiated by potassium deficiency which is a potential and known adverse reaction of glucocorticoids.

Saluretics
Concommitant use of glucocorticoids may result in enhanced potassium excretion and aggravated hypokalaemia.

Drug Food Interactions
Inhibitors of CYP3A4 such as grapefruit juice may cause a marked increase of the plasma concentration of budesonide. Therefore, concomitant use should be avoided.

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 of up to 20 µg/kg/day did not affect fertility.
Use in Pregnancy – Category B3
Administration during pregnancy should be avoided unless there are compelling reasons for therapy with Jorveza®. There are few data of pregnancy outcomes after oral administration of budesonide in humans. Although data on the use of inhaled budesonide in a large number of exposed pregnancies indicate no adverse effect, the maximal concentration of budesonide in plasma has to be expected to be higher in the treatment with Jorveza® compared to inhaled budesonide. In pregnant animals, budesonide, like other glucocorticosteroids, has been shown to cause abnormalities of fetal development (smaller litter size, intrauterine growth retardation of foetuses and skeletal and visceral abnormalities). Some glucocorticosteroids have been reported to produce cleft palate in animals. The clinical relevance of these findings to humans has not been established.

Use in Lactation
Budesonide is excreted in human milk (data on excretion after inhalative use is available). However, only minor effects on the breast-fed child are anticipated after oral use of Jorveza® within the therapeutic range. A decision must be made whether to discontinue breast-feeding or to discontinue/abstain from budesonide therapy taking into account the benefit of breast-feeding for the child and the benefit of therapy for the woman.

4.7 EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

Jorveza® has no or negligible influence on the ability to drive and use machines.

4.8 ADVERSE EFFECTS (UNDESIRABLE EFFECTS)

Summary of the safety profile
Fungal infections in the mouth, pharynx and the oesophagus were the most frequently observed adverse reactions in clinical studies with Jorveza®. In the clinical studies BUL-1/EEA and BUL-2/EER, a total of 44 out of 268 patients (16.4%) exposed to Jorveza® experienced cases of suspected fungal infections associated with clinical symptoms, which were all of mild or moderate intensity and which did not interfere with their daily activities. The total number of infections (including those without symptoms diagnosed by endoscopy and histology through proactive investigation required in the study protocol) was 92, occurring in 72 out of 268 patients (26.9%). The frequency of the fungal infections was not dose related. All patients received oral antifungal treatment or no medical intervention. None of the patients ceased Jorveza® due to a local fungal infection during the two studies.

Tabulated list of adverse reactions

Adverse reactions observed in clinical studies with Jorveza® are listed in the table below, by MedDRA system organ class and frequency. Frequencies are defined as 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) or not known (cannot be estimated from the available data).

MedDRA system organ class	**Very common**	**Common**	**Uncommon**
Infections and infestations	Oesophageal candidiasis	Oral and/or oropharyngeal candidiasis	
Immune system disorders			Hypersensitivity reactions including angioedema and contact dermatitis
Psychiatric disorders			Sleep disorder, anxiety
Nervous system disorders		Headache	Dizziness, dysgeusia
Eye disorders			Dry eye
Vascular disorders			Hypertension
Respiratory, thoracic and mediastinal disorders			Cough, dry throat, oropharyngeal pain
Gastrointestinal disorders		Gastroesophageal reflux disease, nausea, oral paraesthesia, dyspepsia	Abdominal pain, upper abdominal pain, dry mouth, dysphagia, erosive gastritis, gastric ulcer, glossodynia, lip oedema
Skin and subcutaneous tissue disorders			Rash, urticaria
General disorders and administration site conditions		Fatigue	Sensation of foreign body
Investigations		Blood cortisol decreased	

The following known adverse reactions of the therapeutic class (corticosteroids, budesonide) could also occur with Jorveza® (frequency = not known).

MedDRA system organ class	**Adverse reactions**
Immune system disorders	Immune suppression (e.g. increased risk of infection)
Endocrine disorders	Cushing's syndrome, moon-face, truncal obesity, adrenal suppression, growth retardation in children, disturbance of sex hormone secretion (e.g. amenorrhea, hirsutism, impotence)
Metabolism and nutrition disorders	Hypokalaemia, hyperglycaemia, reduced glucose tolerance, diabetes mellitus, sodium retention with oedema
Psychiatric disorders	Depression, irritability, euphoria, psychomotor hyperactivity, aggression
Nervous system disorders	Dysgeusia, pseudotumor cerebri including papilloedema in adolescents

MedDRA system organ class	Adverse reactions
Eye disorders	Glaucoma, cataract (including subcapsular cataract), blurred vision, central serous chorioretinopathy (CSCR) (see also section 4.4 Special Warnings and Precautions for Use; Visual Disturbances)
Vascular disorders	Increased risk of thrombosis, vasculitis (withdrawal syndrome after long-term therapy), hypertension
Gastrointestinal disorders	Duodenal ulcers, pancreatitis, constipation
Skin and subcutaneous tissue disorders	Allergic exanthema, petechiae, delayed wound healing, , ecchymosis, steroid acne, red striae
Musculoskeletal and connective tissue disorders	Muscle and joint pain, muscle weakness and twitching, osteoporosis, osteonecrosis
General disorders and administration site conditions	Malaise

Post-Market Adverse Reactions
Adverse reactions seen with Jorveza® during Post-Marketing Surveillance:

Nervous system disorders: Dysgeusia

Reporting of suspected adverse reactions
Reporting suspected adverse effects 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 https://nzphvc.otago.ac.nz/reporting/

4.9 OVERDOSE

For information on the management of overdose, contact the Poison Information Centre in Australia on 131126, and in New Zealand telephone 0800 764 766.

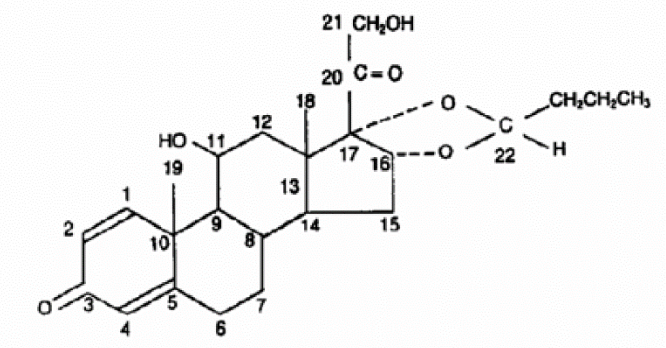
In case of short-term overdose no emergency medical treatment is required. There is no specific antidote. Subsequent treatment should be symptomatic and supportive.

5. PHARMACOLOGICAL PROPERTIES

5.1 PHARMACODYNAMIC PROPERTIES

Pharmacotherapeutic group: Antidiarrheals, intestinal anti-inflammatory/anti-infective agents, corticosteroids acting locally, ATC code: A07EA06

Chemical Structure



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

C25H34O6 = 430.5

CAS number: 51333-22-3

Physicochemical properties: Budesonide is a white or almost-white crystalline powder, with a pKa of 12.85 ± 0.10.
Budesonide is practically insoluble in water, freely soluble in methylene chloride, sparingly soluble in ethanol (96%).

Mechanism of action
Budesonide is a non-halogenated glucocorticosteroid, which acts primarily as an anti-inflammatory via binding to the glucocorticoid receptor. The exact mechanism of action in the treatment of EoE is not fully understood. In the treatment of EoE with Jorveza®, budesonide may inhibit antigen-stimulated secretion of many pro-inflammatory signal molecules, which may result in a significant reduction of the oesophageal eosinophilic inflammatory infiltrate.

Pharmacodynamics
The primary pharmacodynamic effect of budesonide is its anti-inflammatory activity. Budesonide has a high glucocorticoid effect and a weak mineralocorticoid effect, and the affinity of budesonide to GCS receptors, which reflects the intrinsic potency of the drug, is about 200-fold that of cortisol and 15-fold that of prednisolone.

Effect on hypothalamus-pituitary-adrenal and endogenous cortisol levels
Treatment with systemically active glucocorticosteroids is associated with a suppression of endogenous cortisol concentrations and impairment of the HPA axis function.

Following 6 weeks of Jorveza® 1 mg BID treatment in patients with EoE, the rate of patients with adverse events of decreased plasma cortisol levels was 5% with Jorveza® and 0% with placebo.

Clinical Trials
In a randomised, placebo-controlled, double-blind phase III clinical study (BUL-1/EEA) including 88 adult patients with active symptomatic and histological EoE (randomisation rate: 2:1), 1 mg budesonide given twice daily as an orally disintegrating tablet for 6 weeks induced clinico-pathologic remission (defined as both peak of < 16 eosinophils/mm^2 high power field (hpf); < 5 eos/hpf) in oesophageal biopsies and no or only minimal symptoms of dysphagia or pain during swallowing on each day in week 6) in 34 out of 59 patients (57.6%) *versus* 0/29 patients (0%) in the placebo-group (p=0.00000001). Open-label extension of the treatment with 1 mg budesonide orally disintegrating tablet twice daily for further 6 weeks in patients in 23 budesonide-treated patients without remission in the double-blind phase increased the rate of patients with clinico-pathologic remission to 84.7%.

In a randomised, placebo-controlled, double-blind phase III clinical study (BUL-2/EER) including 204 adult patients with EoE in clinico-pathological remission, patients were randomised to treatment with 1 mg budesonide twice daily (BID), 0.5 mg budesonide BID, or placebo (all given as orally disintegrating tablets) for 48 weeks (n=68 for all treatment groups). Primary endpoint was the rate of patients free of treatment failure, with treatment failure defined as clinical relapse (severity of dysphagia or pain during swallowing of ≥4 points on a 0-10 numerical rating scale, respectively), and/or histological relapse (peak of ≥ 48 eosinophils/mm^2 hpf; >15 eos/hpf), and/or food impaction requiring endoscopic intervention, and/or need of an endoscopic dilation, and/or premature withdrawal for any reason. Significantly more patients in the 1 mg BID (75%) group and the 0.5 mg BID (73.5%) group were free of treatment failure at week 48 compared to placebo (4.4%); p<0.0001; both treatments.

The most stringent secondary endpoint “deep disease remission”, i.e., deep clinical, deep endoscopic and histological remission showed a clinically relevant higher efficacy in the 1 mg BID group (52.9%) compared to the 0.5 mg BID group (39.7%), indicating that a higher dose of budesonide is of advantage to achieve and maintain deep disease remission.

For information about the observed adverse reactions, see section 4.8 ADVERSE EFFECTS (UNDESIRABLE EFFECTS).

5.2 PHARMACOKINETIC PROPERTIES

Absorption
Following administration of Jorveza®, budesonide is rapidly absorbed. Pharmacokinetic data following administration of single doses of 1 mg budesonide to fasted healthy subjects in two different studies show a median lag time of 0.17 hours (range 0.00 – 0.52 hours) and a median time to peak plasma concentration of 1.00 – 1.22 hour (range 0.50 – 2.00 hours). The mean peak plasma concentration (± standard deviation) was 0.44 – 0.49 ng/mL (range 0.18 – 1.05 ng/ml) and the area under the plasma-concentration–time curve ($AUC_{0-\infty}$) was 1.50 – 2.23 hr*ng/mL (range 0.81 – 5.14 hr*ng/mL).

Single dose pharmacokinetic data in fasted patients with EoE are available with 4 mg budesonide: Median lag-time was 0.00 hours (range 0.00 – 0.17), median time to peak plasma concentration was 1.00 hour (range 0.67 – 2.00 hours); peak plasma concentration was 2.56 ± 1.36 ng/mL, and AUC_{0-12} was 8.96 ± 4.21 hr*ng/mL.

Patients showed a 35% increase in peak plasma concentrations and a 60% increase in AUC_{0-12} compared to healthy subjects.

Dose proportionality of the systemic exposure (C_{max} and AUC) from 0.5 mg orally disintegrating tablets to 1 mg orally disintegrating tablets has been demonstrated.

Distribution
The apparent volume of distribution following oral administration of 1 mg budesonide to healthy subjects was 35.52 ± 14.94 L/kg and 42.46 ± 23.90 L/kg following administration of 4 mg budesonide to patients with EoE. Plasma protein binding is on average 85-90%.

Biotransformation
Metabolism of budesonide is decreased in EoE patients compared to healthy subjects resulting in increased plasma concentrations of budesonide.

Budesonide undergoes extensive biotransformation by CYP3A4 in the mucosa of the small intestine and in the liver to metabolites of low glucocorticosteroid activity. The glucocorticosteroid activity of the major metabolites, 6β-hydroxybudesonide and 16α-hydroxyprednisolone, is less than 1% of that of budesonide. CYP3A5 does not contribute significantly to the metabolism of budesonide.

Elimination
The median elimination half-life is 2 - 3 hours in healthy subjects (receiving 1 mg budesonide) and 4 - 5 hours in EoE patients (receiving 4 mg budesonide). Clearance of budesonide is about 13 – 15 L/hour/kg in healthy subjects and 6.54 ± 4.4 L/hour/kg in EoE patients. Budesonide is eliminated only in marginal if any amounts by the kidney. No budesonide, but only budesonide metabolites were detected in urine.

Hepatic impairment
A relevant proportion of budesonide is metabolised in the liver by CYP3A4. The systemic exposure of budesonide is considerably increased in patients with severely impaired hepatic function. No studies have been conducted with Jorveza® in patients with impaired liver function. Jorveza® should not be used in patients with hepatic impairment.

5.3 PRECLINICAL SAFETY DATA

Genotoxicity
Budesonide had no mutagenic effects in a number 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. As this was not confirmed in further studies in male Sprague-Dawley and Fischer rats, it was concluded that budesonide does not increase the incidence of brain tumours in 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

Jorveza® 0.5 mg and 1 mg orally disintegrating tablets:

Sodium acid citrate
Docusate sodium
Macrogol 6000
Magnesium stearate
Mannitol
Sodium dihydrogen citrate
Povidone
Sodium bicarbonate
Sucralose

6.2 INCOMPATIBILITIES

Not applicable.

6.3 SHELF LIFE

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

In New Zealand, the approved shelf-life is 24 months from date of manufacture when stored as per conditions noted in Section 6.4 Special Precautions for Storage. The expiry date can be found on the packaging.

6.4 SPECIAL PRECAUTIONS FOR STORAGE

Store below 25°C. Store in the original package in order to protect from light and moisture.

6.5 NATURE AND CONTENTS OF CONTAINER

Al/Al-blister.

Jorveza® 0.5 mg:
Pack sizes: 20, 60, 100 or 200 orally disintegrating tablets. Not all pack sizes may be marketed.

Jorveza® 1 mg:
Pack sizes: 20, 30, 60 or 90 orally disintegrating tablets. Not all pack sizes may be marketed.

6.6 SPECIAL PRECAUTIONS FOR DISPOSAL

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

7. MEDICINE SCHEDULE (POISONS STANDARD)

Schedule 4 – Prescription Only Medicine

8. SPONSOR

JORVEZA® is supplied in Australia by:
Dr Falk Pharma Australia Pty Ltd
815 Pacific Highway,
Chatswood NSW 2067
Phone: 1800 373 255

JORVEZA® is supplied in New Zealand by:
Dr Falk Pharma New Zealand Ltd
29 Northcroft Street
Takapuna
Auckland 0622
New Zealand

Phone: 0800 44 88 69

9. DATE OF FIRST APPROVAL

20 June 2022

Summary table of changes

Section changed	Summary of new information