SUMMARY OF PRODUCT CHARACTERISTICS

1. Name of the Medical Product
1.1 Product Name :
VILDARIL 50 (Vildagliptin Tablets 50 mg)

1.2 Strength :
Each uncoated tablet contains:
Vildagliptin 50 mg

1.3 Pharmaceutical Dosage Form :
Tablet

2. Qualitative & Quantitative Composition:
Each uncoated tablet contains:
Vildagliptin 50 mg
Excipients q.s
For a full list of excipients, see section 6.1 of SmPC

3. Pharmaceutical Form:
Tablet
White to off white colored, circular flat beveled edged uncoated tablets, plain on both sides.

4. Clinical Particulars :
4.1 Therapeutic Indications:
Vildagliptin is indicated as an adjunct to diet and exercise to improve glycaemic control in adults with type 2 diabetes mellitus:

- as monotherapy in patients in whom metformin is inappropriate due to contraindications or intolerance.
- in combination with other medicinal products for the treatment of diabetes, including insulin, when these do not provide adequate glycaemic control.

4.2 Posology and Method of administration:
Posology
Adults
When used as monotherapy, in combination with metformin, in combination with thiazolidinedione, in combination with metformin and a sulphonylurea, or in combination with insulin (with or without metformin), the recommended daily dose of vildagliptin is 100 mg, administered as one dose of 50 mg in the morning and one dose of 50 mg in the evening.
When used in dual combination with a sulphonylurea, the recommended dose of vildagliptin is 50 mg once daily administered in the morning. In this patient population, vildagliptin 100 mg daily was no more effective than vildagliptin 50mg once daily.
When used in combination with a sulphonylurea, a lower dose of the sulphonylurea may be considered to reduce the risk of hypoglycaemia.
Doses higher than 100 mg are not recommended.
If a dose of Vildaril is missed, it should be taken as soon as the patient remembers. A double dose should not be taken on the same day.
The safety and efficacy of vildagliptin as triple oral therapy in combination with metformin and a thiazolidinedione have not been established.

Additional information on special populations
Elderly (≥ 65 years)
No dose adjustments are necessary in elderly patients

Renal impairment
No dose adjustment is required in patients with mild renal impairment (creatinine clearance ≥ 50 ml/min). In patients with moderate or severe renal impairment or with end-stage renal disease (ESRD), the recommended dose of Vildaril is 50 mg once daily.

Hepatic impairment
Vildaril should not be used in patients with hepatic impairment, including patients with pre-treatment alanine aminotransferase (ALT) or aspartate aminotransferase (AST) > 3x the upper limit of normal (ULN).

Pediatric population
Vildaril is not recommended for use in children and adolescents (< 18 years). The safety and efficacy of Vildagliptin in children and adolescents (< 18 years) have not been established. No data are available.

Method of administration
Oral use
Vildaril can be administered with or without a meal

4.3 Contraindications:
Hypersensitivity to the active substance or to any of the excipients.

4.4 Special warning and precautions for use:
General
Vildagliptin is not a substitute for insulin in insulin-requiring patients. Vildagliptin should not be used in patients with type 1 diabetes or for the treatment of diabetic ketoacidosis.
Renal impairment
There is limited experience in patients with ESRD on haemodialysis. Therefore, Vildagliptin should be used with caution in these patients.
Hepatic impairment
Vildagliptin should not be used in patients with hepatic impairment, including patients with pre-treatment ALT or AST > 3x ULN.

Liver enzyme monitoring
Rare cases of hepatic dysfunction (including hepatitis) have been reported. In these cases, the patients were generally asymptomatic without clinical sequelae and liver function test results returned to normal after discontinuation of treatment. Liver function tests should be performed prior to the initiation of treatment with Vildagliptin in order to know the patient's baseline value. Liver function should be monitored during treatment with Vildagliptin at three-month intervals during the first year and periodically thereafter. Patients who develop increased transaminase levels should be monitored with a second liver function evaluation to confirm the finding and be followed thereafter with frequent liver function tests until the abnormality(ies) return(s) to normal. Should an increase in AST or ALT of 3x ULN or greater persist, withdrawal of Vildagliptin therapy is recommended.
Patients who develop jaundice or other signs suggestive of liver dysfunction should discontinue Vildagliptin.
Following withdrawal of treatment with Vildagliptin and LFT normalisation, treatment with Vildagliptin should not be reinitiated.

Cardiac failure
A published clinical trial of Vildagliptin in patients with New York Heart Association (NYHA) functional class I-III showed that treatment with Vildagliptin was not associated with a change in left-ventricular function or worsening of pre-existing congestive heart failure (CHF) versus placebo. Clinical experience in patients with NYHA functional class III treated with Vildagliptin is still limited and results are inconclusive.

There is no experience of Vildagliptin use in clinical trials (published) in patients with NYHA functional class IV and therefore use is not recommended in these patients.

Skin disorders
Skin lesions, including blistering and ulceration have been reported in extremities of monkeys in published non-clinical toxicology studies. Although skin lesions were not observed at an increased incidence in published clinical trials, there was limited experience in patients with diabetic skin complications. Furthermore, there have been post-marketing reports of bullous and exfoliative skin lesions. Therefore, in keeping with routine care of the diabetic patient, monitoring for skin disorders, such as blistering or ulceration, is recommended.

Acute pancreatitis
Use of Vildagliptin has been associated with a risk of developing acute pancreatitis. Patients should be informed of the characteristic symptom of acute pancreatitis.
If pancreatitis is suspected, Vildagliptin should be discontinued; if acute pancreatitis is confirmed, Vildagliptin should not be restarted. Caution should be exercised in patients with a history of acute pancreatitis.

Hypoglycemia
Sulphonylureas are known to cause hypoglycemia. Patients receiving Vildagliptin in combination with a sulphonylurea may be at risk for hypoglycemia. Therefore, a lower dose of sulphonylurea may be considered to reduce the risk of hypoglycemia.

Lactose
Patients with rare hereditary problems of galactose intolerance, total lactase deficiency or glucose-galactose malabsorption should not take this medicinal product.

Sodium

This medicinal product contains less than 1 mmol sodium (23 mg) per tablet, that is to say essentially 'sodium-free'.

4.5 Interactions with other medicinal products and other forms of Interactions :
Vildagliptin has a low potential for interactions with co-administered medicinal products. Since Vildagliptin is not a cytochrome P (CYP) 450 enzyme substrate and does not inhibit or induce CYP 450 enzymes, it is not likely to interact with active substances that are substrates, inhibitors or inducers of these enzymes.

Combination with pioglitazone, metformin and glyburide
Results from published studies conducted with these oral antidiabetics have shown no clinically relevant pharmacokinetic interactions.

Digoxin (Pgp substrate), warfarin (CYP2C9 substrate)
Published clinical studies performed with healthy subjects have shown no clinically relevant pharmacokinetic interactions. However, this has not been established in the target population.

Combination with amlodipine, ramipril, valsartan or simvastatin
Published drug-drug interaction studies in healthy subjects were conducted with amlodipine, ramipril, valsartan and simvastatin. In these published studies, no clinically relevant pharmacokinetic interactions were observed after co-administration with Vildagliptin.

Combination with ACE-inhibitors
There may be an increased risk of angioedema in patients concomitantly taking ACE-inhibitors.
As with other oral antidiabetic medicinal products the hypoglycemic effect of Vildagliptin may be reduced by certain active substances, including thiazides, corticosteroids, thyroid products and sympathomimetic.

4.6 Pregnancy and Lactation:
Pregnancy
There are no adequate data from the use of Vildagliptin in pregnant women. Published studies in animals have shown reproductive toxicity at high doses. The potential risk for humans is unknown. Due to lack of human data, Vildagliptin should not be used during pregnancy.

Breast-feeding
It is unknown whether Vildagliptin is excreted in human milk. Published animal studies have shown excretion of Vildagliptin in milk. Vildagliptin should not be used during breast-feeding.

Fertility
No studies on the effect on human fertility have been conducted for Vildagliptin.

4.7 Effects on ability to drive and use machine:
No studies on the effects on the ability to drive and use machines have been performed. Patients who experience dizziness as an adverse reaction should avoid driving vehicles or using machines.

4.8 Undesirable Effects:
Summary of the safety profile
Safety data were obtained from a total of 5451 patients exposed to vildagliptin at a daily dose of 100 mg (50 mg twice daily) in randomised double-blind placebo-controlled published trials of at least 12 weeks duration. Of these patients, 4622 patients received vildagliptin as monotherapy and 829 patients received placebo.
The majority of adverse reactions in these published trials were mild and transient, not requiring treatment discontinuations. No association was found between adverse reactions and age, ethnicity, duration of exposure or daily dose. Hypoglycaemia has been reported in patients receiving vildagliptin concomitantly with sulphonylurea and insulin. The risk of acute pancreatitis has been reported with the use of vildagliptin.

Tabulated list of adverse reactions
Adverse reactions reported in patients who received Vildagliptin in published double-blind studies as monotherapy and add-on therapies are listed below for each indication by system organ class and absolute 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), not known (cannot be estimated from the available data). Within each frequency grouping, adverse reactions are presented in order of decreasing seriousness.

Combination with metformin
Table 1 Adverse reactions reported in patients who received Vildagliptin 100 mg daily in combination with metformin in published double-blind studies (N=208)

System Organ Class-Adverse Reaction	Frequency
Infections and infestations	
Nasopharyngitis	Very common
Upper respiratory tract infection	Common
Metabolism and nutrition disorders	
Hypoglycaemia	Uncommon

Nervous system disorders	
Dizziness	Common
Headache	Common
Tremor	Common
Eye disorders	
Vision blurred	Common
Gastrointestinal disorders	
Constipation	Common
Nausea	Common
Gastro-oesophageal reflux disease	Common
Diarrhoea	Common
Abdominal pain, including upper	Common
Vomiting	Common
Flatulence	Uncommon
Pancreatitis	Rare
Hepatobiliary disorders	
Hepatitis	Not known*
Skin and subcutaneous tissue disorders	
Hyperhidrosis	Common
Rash	Common
Pruritis	Common
Dermatitis	Common
Urticaria	Uncommon
Exfoliative and bullous skin lesions, including bullous pemphigoid	Not known*
Infections and infestations	
Nasopharyngitis	Very common
Upper respiratory tract infection	Common
Metabolism and nutrition disorders	
Hypoglycaemia	Uncommon
Nervous system disorders	
Dizziness	Common
Headache	Common
Tremor	Common
Eye disorders	
Vision blurred	Common
Gastrointestinal disorders	
Constipation	Common
Nausea	Common
Gastro-oesophageal reflux disease	Common
Diarrhoea	Common
Abdominal pain, including upper	Common
Vomiting	Common
Flatulence	Uncommon
Pancreatitis	Rare
Hepatobiliary disorders	
Hepatitis	Not known*
Skin and subcutaneous tissue disorders	
Hyperhidrosis	Common
Rash	Common
Pruritis	Common
Dermatitis	Common

Urticaria	Uncommon
Exfoliative and bullous skin lesions, including bullous pemphigoid	Not known*
Cutaneous vasculitis	Not known*
Musculoskeletal and connective tissue disorders	
Arthralgia	Common
Myalgia	Common
Reproductive system and breast disorders	
Erectile dysfunction	Uncommon
General disorders and administration site conditions	
Asthenia	Common
Oedema peripheral	Common
Fatigue	Uncommon
Chills	Uncommon
Investigations	
Abnormal liver function tests	Uncommon
Weight increase	Uncommon
* Based on post-marketing experience.	

Description of selected adverse reactions

Hepatic impairment

Rare cases of hepatic dysfunction (including hepatitis) have been reported. In these cases, the patients were generally asymptomatic without clinical sequelae and liver function returned to normal after discontinuation of treatment. In data from published controlled monotherapy and add-on therapy trials of up to 24 weeks in duration, the incidence of ALT or AST elevations ≥ 3xULN (classified as present on at least 2 consecutive measurements or at the final on-treatment visit) was 0.2%, 0.3% and0.2% for vildagliptin 50 mg once daily, vildagliptin 50 mg twice daily and all comparators, respectively. These elevations in transaminases were generally asymptomatic, non-progressive in nature and not associated with cholestasis or jaundice.

Angioedema

Rare cases of angioedema have been reported on vildagliptin at a similar rate to controls. A greater proportion of cases were reported when vildagliptin was administered in combination with an angiotensin converting enzyme inhibitor (ACE-Inhibitor). The majority of events were mild in severity and resolved with ongoing vildagliptin treatment.

Hypoglycaemia

Hypoglycaemia was uncommon when vildagliptin (0.4%) was used as monotherapy in comparative controlled monotherapy published studies with an active comparator or placebo (0.2%). No severe or serious events of hypoglycaemia were reported. When used as add-on to metformin, hypoglycaemia occurred in 1% of vildagliptin-treated patients and in 0.4% of placebo-treated patients. When pioglitazone was added, hypoglycaemia occurred in 0.6% of vildagliptin-treated patients and in 1.9% of placebo-treated patients. When sulphonylurea was added, hypoglycaemia occurred in 1.2% of vildagliptin treated patients and in 0.6% of placebo-treated patients. When sulphonylurea and metformin were added, hypoglycaemia occurred in 5.1%of vildagliptin treated patients and in 1.9% of placebo treated patients. In patients taking vildagliptin in combination with insulin, the incidence of hypoglycaemia was 14% for vildagliptin and 16% for placebo.

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 Overdosage:
Information regarding overdose with Vildagliptin is limited.
Symptoms
Information on the likely symptoms of overdose was taken from a rising dose tolerability published study in healthy subjects given Vildagliptin for 10 days. At 400 mg, there were three cases of muscle pain, and individual cases of mild and transient paraesthesia, fever, edema and a transient increase in lipase levels. At 600 mg, one subject experienced edema of the feet and hands, and increases in creatinine phosphokinase (CPK), aspartate aminotransferase (AST), and C - reactive protein (CRP) and myoglobin levels. Three other subjects experienced edema of the feet, with paraesthesia in two cases. All symptoms and laboratory abnormalities resolved without treatment after discontinuation of the study medicinal product.

Management
In the event of an overdose, supportive management is recommended. Vildagliptin cannot be removed by hemodialysis. However, the major hydrolysis metabolite (LAY 151) can be removed by hemodialysis.

5. Pharmacological properties:
5.1 Pharmacodynamic Properties:
Pharmacotherapeutic group: Alimentary tract and metabolism, Drugs used in diabetes, Blood glucose lowering drugs, Excl. Insulins, Dipeptidyl peptidase 4 (DPP-4) inhibitors
ATC Code: A10BH02

Vildagliptin, a member of the islet enhancer class, is a potent and selective dipeptidyl peptidase 4 (DPP-4) inhibitors.

Mechanism of action
The administration of Vildagliptin results in a rapid and complete inhibition of DPP-4 activity, resulting in increased fasting and postprandial endogenous levels of the incretin hormones GLP-1 (glucagon-like peptide 1) and GIP (glucose-dependent insulinotropic polypeptide).

Pharmacodynamic effects
By increasing the endogenous levels of these incretin hormones, Vildagliptin enhances the sensitivity of beta cells to glucose, resulting in improved glucose-dependent insulin secretion. Treatment with Vildagliptin 50-100 mg daily in patients with type 2 diabetes significantly improved markers of beta cell function including HOMA-β (Homeostasis Model Assessment–β), proinsulin to insulin ratio and measures of beta cell responsiveness from the frequently-sampled meal tolerance test. In non-diabetic (normal glycemic) individuals, Vildagliptin does not stimulate insulin secretion or reduce glucose levels. By increasing endogenous GLP-1 levels, Vildagliptin also enhances the sensitivity of alpha cells to glucose, resulting in more glucose-appropriate glucagon secretion.
The enhanced increase in the insulin/glucagon ratio during hyperglycemia due to increased incretin hormone levels results in a decrease in fasting and postprandial hepatic glucose production, leading to reduced glycaemia.
The known effect of increased GLP-1 levels delaying gastric emptying is not observed with Vildagliptin treatment.

5.2 Pharmacokinetics Properties:

Absorption

Following oral administration in the fasting state, Vildagliptin is rapidly absorbed, with peak plasma concentrations observed at 1.7 hours. Food slightly delays the time to peak plasma concentration to 2.5 hours, but does not alter the overall exposure (AUC). Administration of Vildagliptin with food resulted in a decreased C_{max} (19%). However, the magnitude of change is not clinically significant, so that Vildagliptin can be given with or without food. The absolute bioavailability is 85%.

Distribution

The plasma protein binding of Vildagliptin is low (9.3%) and Vildagliptin distributes equally between plasma and red blood cells. The mean volume of distribution of Vildagliptin at steady-state after intravenous administration (V_{ss}) is 71 litres, suggesting extravascular distribution.

Biotransformation

Metabolism is the major elimination pathway for Vildagliptin in humans, accounting for 69% of the dose. The major metabolite (LAY 151) is pharmacologically inactive and is the hydrolysis product of the cyano moiety, accounting for 57% of the dose, followed by the glucuronide (BQS867) and the amide hydrolysis products (4% of dose). In vitro data in human kidney microsomes suggest that the kidney may be one of the major organs contributing to the hydrolysis of Vildagliptin to its major inactive metabolite, LAY151. DPP-4 contributes partially to the hydrolysis of Vildagliptin based on an *in vivo* study using DPP-4 deficient rats. Vildagliptin is not metabolised by CYP 450 enzymes to any quantifiable extent. Accordingly, the metabolic clearance of Vildagliptin is not anticipated to be affected by co-medications that are CYP 450 inhibitors and/or inducers. *In vitro* studies demonstrated that Vildagliptin does not inhibit/induce CYP 450 enzymes. Therefore, Vildagliptin is not likely to affect metabolic clearance of co-medications metabolised by CYP 1A2, CYP 2C8, CYP 2C9, CYP 2C19, CYP 2D6, CYP 2E1 or CYP 3A4/5.

Elimination

Following oral administration of [^{14}C] Vildagliptin, approximately 85% of the dose was excreted into the urine and 15% of the dose is recovered in the faeces. Renal excretion of the unchanged Vildagliptin accounted for 23% of the dose after oral administration. After intravenous administration to healthy subjects, the total plasma and renal clearances of Vildagliptin are 41 and 13 l/h, respectively. The mean elimination half-life after intravenous administration is approximately 2 hours. The elimination half-life after oral administration is approximately 3 hours.

Linearity/non-linearity

The Cmax for vildagliptin and the area under the plasma concentrations versus time curves (AUC) increased in an approximately dose proportional manner over the therapeutic dose range.

Characteristics in specific groups of patients

Gender

No clinically relevant differences in the pharmacokinetics of vildagliptin were observed between male and female healthy subjects within a wide range of age and body mass index (BMI). DPP-4 inhibition by vildagliptin is not affected by gender.

Elderly

In healthy elderly subjects (≥ 70 years), the overall exposure of vildagliptin (100 mg once daily) was increased by 32%, with an 18% increase in peak plasma concentration as compared to young healthy subjects (18-40 years). These changes are, however, not considered to be clinically relevant. DPP-4 inhibition by vildagliptin is not affected by age.

Hepatic impairment
The effect of impaired hepatic function on the pharmacokinetics of vildagliptin was studied in patients with mild, moderate and severe hepatic impairment based on the Child-Pugh scores (ranging from 6 for mild to 12 for severe) in comparison with healthy subjects. The exposure to vildagliptin after a single dose in patients with mild and moderate hepatic impairment was decreased (20% and 8%, respectively), while the exposure to vildagliptin for patients with severe impairment was increased by 22%. The maximum change (increase or decrease) in the exposure to vildagliptin is ~30%, which is not considered to be clinically relevant. There was no correlation between the severity of the hepatic disease and changes in the exposure to vildagliptin.

Renal impairment
A multiple-dose, open-label trial was conducted to evaluate the pharmacokinetics of the lower therapeutic dose of vildagliptin (50 mg once daily) in patients with varying degrees of chronic renal impairment defined by creatinine clearance (mild: 50 to < 80 ml/min, moderate: 30 to < 50 ml/min and severe: < 30 ml/min) compared to normal healthy control subjects.

Vildagliptin AUC increased on average 1.4, 1.7 and 2-fold in patients with mild, moderate and severe renal impairment, respectively, compared to normal healthy subjects. AUC of the metabolites LAY151 and BQS867 increased on average about 1.5, 3 and 7-fold in patients with mild, moderate and severe renal impairment, respectively. Limited data from patients with end stage renal disease (ESRD) indicate that vildagliptin exposure is similar to that in patients with severe renal impairment. LAY151 concentrations were approximately 2- 3-fold higher than in patients with severe renal impairment. Vildagliptin was removed by haemodialysis to a limited extent (3% over a 3- 4 hour haemodialysis session starting 4 hours post dose).

Ethnic group
Limited data suggest that race does not have any major influence on vildagliptin pharmacokinetics

5.3 Preclinical Safety data:
Intra-cardiac impulse conduction delays were observed in dogs with a no-effect dose of 15 mg/kg (7-fold human exposure based on C_{max}).

Accumulation of foamy alveolar macrophages in the lung was observed in rats and mice. The no-effect dose in rats was 25 mg/kg (5-fold human exposure based on AUC) and in mice 750 mg/kg (142-fold human exposure).

Gastrointestinal symptoms, particularly soft faeces, mucoid faeces, diarrhea and, at higher doses, faecal blood were observed in dogs. A no-effect level was not established.

Vildagliptin was not mutagenic in conventional *in vitro* and *in vivo* tests for genotoxicity.

A fertility and early embryonic development study in rats revealed no evidence of impaired fertility, reproductive performance or early embryonic development due to Vildagliptin. Embryo-foetal toxicity was evaluated in rats and rabbits. An increased incidence of wavy ribs was observed in rats in association with reduced maternal body weight parameters, with a no-effect dose of 75 mg/kg (10-fold human exposure). In rabbits, decreased foetal weight and skeletal variations indicative of developmental delays were noted only in the presence of severe maternal toxicity, with a no-effect dose of 50 mg/kg (9-fold human exposure). A pre- and postnatal development study was performed in rats. Findings were only observed in association with maternal toxicity at ≥ 150 mg/kg and included a transient decrease in body weight and reduced motor activity in the F1 generation.

A published two-year carcinogenicity study was conducted in rats at oral doses up to 900 mg/kg (approximately 200 times human exposure at the maximum recommended dose). No increases in tumour

incidence attributable to Vildagliptin were observed. Another published two-year carcinogenicity study was conducted in mice at oral doses up to 1,000 mg/kg. An increased incidence of mammary adenocarcinomas and haemangiosarcomas was observed with a no-effect dose of 500 mg/kg (59-fold human exposure) and 100 mg/kg (16-fold human exposure), respectively. The increased incidence of these tumours in mice is considered not to represent a significant risk to humans based on the lack of genotoxicity of Vildagliptin and its principal metabolite, the occurrence of tumours only in one species and the high systemic exposure ratios at which tumours were observed.

In a 13-week toxicology published study in cynomolgus monkeys, skin lesions have been recorded at doses ≥ 5 mg/kg/day. These were consistently located on the extremities (hands, feet, ears and tail). At 5 mg/kg/day (approximately equivalent to human AUC exposure at the 100 mg dose), only blisters were observed. They were reversible despite continued treatment and were not associated with histopathological abnormalities. Flaking skin, peeling skin, scabs and tail sores with correlating histopathological changes were noted at doses ≥ 20 mg/kg/day (approximately 3 times human AUC exposure at the 100 mg dose). Necrotic lesions of the tail were observed at ≥ 80 mg/kg/day. Skin lesions were not reversible in the monkeys treated at 160 mg/kg/day during a 4-week recovery period.

6. Pharmaceutical particulars:

6.1 List of Excipients:

Microcrystalline Cellulose, Sodium Starch Glycolate Type B, Hydrophobic Colloidal Silica and Magnesium Stearate.

6.2 Incompatibilities: Not applicable

6.3 Shelf life: 24 months

6.4 Special Precautions for storage: Store below 30°C. Protect from Moisture.

6.5 Nature and contents of container:

10 tablets in Alu-Alu blister pack, 3 such blisters in a printed carton along with Patient Information Leaflet.

6.6 Special precautions for disposal: Not applicable

7. Marketing Authorization Holder:

Ajanta Pharma Limited
Ajanta Tower,
54A, M Vasanji Road, Chakala, Andheri (E),
Mumbai - 400 093, India
Telephone: 91 - 69132111/2112
E-Mail: rnd.info@ajantapharma.com
shailesh.singh@ajantapharma.com

Manufacturing Site Address:

Ajanta Pharma Ltd.
Plot No. B-4/5/6, MIDC, Paithan,
Chhatrapati Sambhajinagar 431148
Maharashtra State, India.

8. Marketing Authorization Number:

H2021/CTD8151/16557

9. Date of first registration /renewal of the registration:
2026 December 01

10. Date of revision of text:
2026 December 01