SUMMARY OF PRODUCT CHARACTERISTICSFAROGLIP S 100 TABLET (Sitagliptin Tablets, 100 mg)

1. NAME OF THE MEDICINAL PRODUCT FAROGLIP S

100 TABLET (Sitagliptin Tablets, 100 mg)

Legal Category: Prescription only medicine

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Name of Ingredient	Quantity per Tablet
Sitagliptin Phosphate Monohydrate	128.50 mg (equivalent to Sitagliptin 100.00 mg)
Microcrystalline Cellulose (PH 102)	227.50 mg
Anhydrous Dibasic Calcium Phosphate	20.0 mg
Croscarmellose Sodium	12.00 mg
Sodium Stearyl Fumarate	4.00 mg
Magnesium stearate	8.00 mg
Insta Moistshield Aqua II (White) [IC-AMS-II-1675]	10.00 mg
Opadry OY-B-37203 (Tan)	0.80 mg
Opadry II 85G52259 (Yellow)	1.20 mg

3. PHARMACEUTICAL FORM

Oral Tablet

Description: Golden yellow, round, bi-convex film coated engraved with “ACME” on one face and other side being plain.

4. CLINICAL PRECAUTION

4.1. Therapeutic Indications:

For adult patients with type 2 diabetes mellitus, FAROGLIP S is indicated to improve glycaemic control:

as monotherapy:

- in patients inadequately controlled by diet and exercise alone and for whom metformin is inappropriate due to contraindications or intolerance.

as dual oral therapy in combination with:

- metformin when diet and exercise plus metformin alone do not provide adequate glycaemic control.
- a sulphonylurea when diet and exercise plus maximal tolerated dose of a sulphonylurea alonedo not provide adequate glycaemic control and when metformin is inappropriate due to contraindications or intolerance.
- a peroxisome proliferator-activated receptor gamma (PPARγ) agonist (i.e. a thiazolidinedione) when use of a PPARγ agonist is appropriate and when diet and exercise plusthe PPARγ agonist alone do not provide adequate glycaemic control.

as triple oral therapy in combination with:

- a sulphonylurea and metformin when diet and exercise plus dual therapy with these medicinalproducts do not provide adequate glycaemic control.
- a PPARγ agonist and metformin when use of a PPARγ agonist is appropriate and when diet and exercise plus dual therapy with these medicinal products do not provide adequate glycaemic control.

FAROGLIP S is also indicated as add-on to insulin (with or without metformin) when diet and exercise plus stable dose of insulin do not provide adequate glycaemic control.

4.2. Posology and method of administrationPosology

The dose is 100 mg sitagliptin once daily. When used in combination with metformin and/or a PPARγ agonist, the dose of metformin and/or PPARγ agonist should be maintained, and FAROGLIP S administered concomitantly.

When FAROGLIP S is used in combination with a sulphonylurea or with insulin, a lower dose of the sulphonylurea or insulin may be considered to reduce the risk of hypoglycaemia.

If a dose of FAROGLIP S is missed, it should be taken as soon as the patient remembers. A double dose should not be taken on the same day.

Special populationsRenal impairment

When considering the use of sitagliptin in combination with another anti-diabetic medicinal product, its conditions for use in patients with renal impairment should be checked.

For patients with mild renal impairment (glomerular filtration rate [GFR] ≥ 60 to < 90 mL/min),no dose adjustment is required.

For patients with moderate renal impairment (GFR ≥ 45 to < 60 mL/min), no dosage adjustmentis required.

For patients with moderate renal impairment (GFR ≥ 30 to < 45 mL/min), the dose of FAROGLIP S is 50 mg once daily.

For patients with severe renal impairment (GFR ≥ 15 to <30 mL/min) or with end-stage renal disease (ESRD) (GFR < 15 mL/min), including those requiring haemodialysis or peritoneal dialysis, the dose of FAROGLIP S is 25 mg once daily. Treatment may be administered without regard to the timing of dialysis.

Because there is a dosage adjustment based upon renal function, assessment of renal function is recommended prior to initiation of FAROGLIP S and periodically thereafter.

Hepatic impairment

No dose adjustment is necessary for patients with mild to moderate hepatic impairment. FAROGLIP S has not been studied in patients with severe hepatic impairment and care shouldbe exercised.

However, because sitagliptin is primarily renally eliminated, severe hepatic impairment is not expected to affect the pharmacokinetics of sitagliptin.

Elderly

No dose adjustment is necessary based on age.

Paediatric population

Sitagliptin should not be used in children and adolescents 10 to 17 years of age because of insufficient efficacy. Currently available data are described in sections 4.8, 5.1, and 5.2. Sitagliptin has not been studied in paediatric patients under 10 years of age.

Method of administration

FAROGLIP S can be taken orally with or without food.

4.3. Contraindication

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

4.4. Special warning and precautions for useGeneral

FAROGLIP S should not be used in patients with type 1 diabetes or for the treatment of diabetic ketoacidosis.

Acute pancreatitis

Use of DPP-4 inhibitors has been associated with a risk of developing acute pancreatitis. Patients should be informed of the characteristic symptom of acute pancreatitis: persistent, severe abdominal pain. Resolution of pancreatitis has been observed after discontinuation of sitagliptin (with or without supportive treatment), but very rare cases of necrotising or haemorrhagic pancreatitis and/or death have been reported. If pancreatitis is suspected, FAROGLIP S and other potentially suspect medicinal products should be discontinued; if acute pancreatitis is confirmed, FAROGLIP S should not be restarted. Caution should be exercised in patients with a history of pancreatitis.

Hypoglycaemia when used in combination with other anti-hyperglycaemic medicinal products

In clinical trials of FAROGLIP S as monotherapy and as part of combination therapy with medicinal products not known to cause hypoglycaemia (i.e. metformin and/or a PPARγ agonist), rates of hypoglycaemia reported with sitagliptin were similar to rates in patients taking placebo. Hypoglycaemia has been observed when sitagliptin was used in combination with insulin or a sulphonylurea. Therefore, to reduce the risk of hypoglycaemia, a lower dose of sulphonylurea or insulin may be considered.

Renal impairment

Sitagliptin is renally excreted. To achieve plasma concentrations of sitagliptin similar to those in patients with normal renal function, lower dosages are recommended in patients with GFR

< 45 mL/min, as well as in ESRD patients requiring haemodialysis or peritoneal dialysis.

When considering the use of sitagliptin in combination with another anti-diabetic medicinal product, its conditions for use in patients with renal impairment should be checked.

Hypersensitivity reactions

Post-marketing reports of serious hypersensitivity reactions in patients treated with sitagliptin have been reported. These reactions include anaphylaxis, angioedema, and exfoliative skin conditions including Stevens-Johnson syndrome. Onset of these reactions occurred within the first 3 months after initiation of treatment, with some reports occurring after the first dose. If a hypersensitivity reaction is suspected, FAROGLIP S should be discontinued. Other potential causes for the event should be assessed, and alternative treatment for diabetes initiated.

Bullous pemphigoid

There have been post-marketing reports of bullous pemphigoid in patients taking DPP-4 inhibitors including sitagliptin. If bullous pemphigoid is suspected, FAROGLIP S should be discontinued.

Sodium

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

4.5. Interaction of other medicinal products and other forms of interactionEffects of other medicinal products on sitagliptin

Clinical data described below suggest that the risk for clinically meaningful interactions by co-administered medicinal products is low.

In vitro studies indicated that the primary enzyme responsible for the limited metabolism of sitagliptin is CYP3A4, with contribution from CYP2C8. In patients with normal renal function, metabolism, including via CYP3A4, plays only a small role in the clearance of sitagliptin. Metabolism may play a more significant role in the elimination of sitagliptin in the setting of severe renal impairment or end-stage renal disease (ESRD). For this reason, it is possible that potent CYP3A4 inhibitors (i.e. ketoconazole, itraconazole, ritonavir, clarithromycin) could alter the pharmacokinetics of sitagliptin in patients with severe renal impairment or ESRD. The effect of potent CYP3A4 inhibitors in the setting of renal impairment has not been assessed ina clinical study.

In vitro transport studies showed that sitagliptin is a substrate for p-glycoprotein and organic anion transporter-3 (OAT3). OAT3 mediated transport of sitagliptin was inhibited in vitro by probenecid, although the risk of clinically meaningful interactions is considered to be low. Concomitant administration of OAT3 inhibitors has not been evaluated in vivo.

Metformin: Co-administration of multiple twice-daily doses of 1,000 mg metformin with 50 mg sitagliptin did not meaningfully alter the pharmacokinetics of sitagliptin in patients with type 2 diabetes.

Ciclosporin: A study was conducted to assess the effect of ciclosporin, a potent inhibitor of p-glycoprotein, on the pharmacokinetics of sitagliptin. Co-administration of a single 100 mg oral dose of sitagliptin and a single 600 mg oral dose of ciclosporin increased the AUC and Cmax of sitagliptin by approximately 29 % and 68 %, respectively. These changes in sitagliptin pharmacokinetics were not considered to be clinically meaningful. The renal clearance of sitagliptin was not meaningfully altered. Therefore, meaningful interactions would not be expected with other p-glycoprotein inhibitors.

Effects of sitagliptin on other medicinal products

Digoxin: Sitagliptin had a small effect on plasma digoxin concentrations. Following administration of 0.25 mg digoxin concomitantly with 100 mg of sitagliptin daily for 10 days,

the plasma AUC of digoxin was increased on average by 11 %, and the plasma Cmax on average by 18 %. No dose adjustment of digoxin is recommended. However, patients at risk ofdigoxin toxicity should be monitored for this when sitagliptin and digoxin are administered concomitantly.

In vitro data suggest that sitagliptin does not inhibit nor induce CYP450 isoenzymes. In clinical studies, sitagliptin did not meaningfully alter the pharmacokinetics of metformin, glyburide, simvastatin, rosiglitazone, warfarin, or oral contraceptives, providing in vivo evidence of a low propensity for causing interactions with substrates of CYP3A4, CYP2C8, CYP2C9, and organic cationic transporter (OCT). Sitagliptin may be a mild inhibitor of p-glycoprotein in vivo.

6. Fertility, pregnancy and lactation general principlePregnancy

There are no adequate data from the use of sitagliptin in pregnant women. Studies in animals have shown reproductive toxicity at high doses. The potential risk for humans is unknown. Dueto lack of human data, FAROGLIP S should not be used during pregnancy.

Breast-feeding

It is unknown whether sitagliptin is excreted in human breast milk. Animal studies have shown excretion of sitagliptin in breast milk. FAROGLIP S should not be used during breast-feeding.

Fertility

Animal data do not suggest an effect of treatment with sitagliptin on male and female fertility. Human data are lacking.

7. Effect on ability to drive and use medicines

FAROGLIP S has no or negligible influence on the ability to drive and use machines. However, when driving or using machines, it should be taken into account that dizziness and somnolencehave been reported.

In addition, patients should be alerted to the risk of hypoglycaemia when FAROGLIP S is usedin combination with a sulphonylurea or with insulin.

8. Undesirable effect

Summary of the safety profile

SUMMARY OF PRODUCT CHARACTERISTICSFAROGLIP S 100 TABLET (Sitagliptin Tablets, 100 mg)

Serious adverse reactions including pancreatitis and hypersensitivity reactions have been reported. Hypoglycaemia has been reported in combination with sulphonylurea (4.7 %-13.8 %)and insulin (9.6 %).

Tabulated list of adverse reactions

Adverse reactions are listed below (Table 1) by system organ class and frequency.
Frequenciesare 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) and not known (cannot beestimated from the available data).

Table 1. The frequency of adverse reactions identified from placebo-controlled clinicalstudies of sitagliptin monotherapy and post-marketing experience

Adverse reaction	**Frequency of adverse reaction**
Blood and lymphatic system disorders	
thrombocytopenia	Rare
Immune system disorders	
hypersensitivity reactions including anaphylacticresponses*,†	Frequency not known
Metabolism and nutrition disorders	
hypoglycaemia†	Common
Nervous system disorders	
headache	Common
dizziness	Uncommon
Respiratory, thoracic and mediastinal disorders	
interstitial lung disease*	Frequency not known
Gastrointestinal disorders	
constipation	Uncommon
vomiting*	Frequency not known
acute pancreatitis*,†,‡	Frequency not known

fatal and non-fatal haemorrhagic and necrotizingpancreatitis*,†	Frequency not known
Skin and subcutaneous tissue disorders	
pruritus*	Uncommon
angioedema*,†	Frequency not known
rash*,†	Frequency not known
urticaria*,†	Frequency not known
cutaneous vasculitis*,†	Frequency not known
exfoliative skin conditions including Stevens-Johnson syndrome*,†	Frequency not known
bullous pemphigoid*	Frequency not known
Musculoskeletal and connective tissue disorders	
arthralgia*	Frequency not known
myalgia*	Frequency not known
back pain*	Frequency not known
arthropathy*	Frequency not known
Renal and urinary disorders	
impaired renal function*	Frequency not known
acute renal failure*	Frequency not known

*Adverse reactions were identified through post-marketing surveillance.

† **See section 4.4.**

‡ See *TECOS Cardiovascular Safety Study* below.

Description of selected adverse reactions

In addition to the drug-related adverse experiences described above, adverse experiences reported regardless of causal relationship to medication and occurring in at least 5 % and more commonly in patients treated with sitagliptin included upper respiratory tract infection and nasopharyngitis. Additional adverse experiences reported regardless of causal relationship to medication that occurred more frequently in patients treated with sitagliptin (not reaching the

5 % level, but occurring with an incidence of > 0.5 % higher with sitagliptin than that in the control group) included osteoarthritis and pain in extremity.

Some adverse reactions were observed more frequently in studies of combination use of sitagliptin with other anti-diabetic medicinal products than in studies of sitagliptin monotherapy. These included hypoglycaemia (frequency very common with the combination of sulphonylurea and metformin), influenza (common with insulin (with or without metformin)), nausea and vomiting (common with metformin), flatulence (common with metformin or pioglitazone), constipation (common with the combination of sulphonylurea and metformin), peripheral oedema (common with pioglitazone or the combination of pioglitazone and metformin), somnolence and diarrhoea (uncommon with metformin), and dry mouth (uncommon with insulin (with or without metformin)).

Paediatric population

In clinical trials with sitagliptin in paediatric patients with type 2 diabetes mellitus aged 10 to17 years, the profile of adverse reactions was comparable to that observed in adults.

TECOS Cardiovascular Safety Study

The Trial Evaluating Cardiovascular Outcomes with Sitagliptin (TECOS) included 7,332 patients treated with sitagliptin, 100 mg daily (or 50 mg daily if the baseline eGFR was ≥ 30 and < 50 mL/min/1.73 m^2), and 7,339 patients treated with placebo in the intention-to-treat population. Both treatments were added to usual care targeting regional standards for HbA_{1c} and CV risk factors. The overall incidence of serious adverse events in patients receiving sitagliptin was similar to that in patients receiving placebo.

In the intention-to-treat population, among patients who were using insulin and/or a sulfonylurea at baseline, the incidence of severe hypoglycaemia was 2.7 % in sitagliptin-treated patients and 2.5 % in placebo-treated patients; among patients who were not using insulin and/or a sulfonylurea at baseline, the incidence of severe hypoglycaemia was 1.0 % in sitagliptin-treated patients and 0.7 % in placebo-treated patients. The incidence of adjudication-confirmed pancreatitis events was 0.3 % in sitagliptin-treated patients and 0.2 % in placebo- treated patients.

9. Overdose

During controlled clinical trials in healthy subjects, single doses of up to 800 mg sitagliptin were administered. Minimal increases in QTc, not considered to be clinically relevant, were observed in one study at a dose of 800 mg sitagliptin. There is no experience with doses above

800 mg in clinical studies. In Phase I multiple-dose studies, there were no dose-related clinical adverse reactions observed with sitagliptin with doses of up to 600 mg per day for periods of up to 10 days and 400 mg per day for periods of up to 28 days.

In the event of an overdose, it is reasonable to employ the usual supportive measures, e.g., remove unabsorbed material from the gastrointestinal tract, employ clinical monitoring (including obtaining an electrocardiogram), and institute supportive therapy if required.

Sitagliptin is modestly dialysable. In clinical studies, approximately 13.5 % of the dose was removed over a 3- to 4-hour haemodialysis session. Prolonged haemodialysis may be considered if clinically appropriate. It is not known if sitagliptin is dialysable by peritoneal dialysis.

5. PHARMACOLOGICAL PROPERTIES

5.1. Pharmacodynamic properties

Pharmacotherapeutic group: [Dipeptidyl peptidase 4 (DPP-4) inhibitors]

ATC code: A10BH01

Mechanism of action:

Sitagliptin is a member of a class of oral anti-hyperglycaemic agents called dipeptidyl peptidase 4 (DPP-4) inhibitors. The improvement in glycaemic control observed with this medicinal product may be mediated by enhancing the levels of active incretin hormones. Incretin hormones, including glucagon-like peptide-1 (GLP-1) and glucose dependent insulinotropic polypeptide (GIP), are released by the intestine throughout the day, and levels are increased inresponse to a meal. The incretins are part of an endogenous system involved in the physiologicregulation of glucose homeostasis. When blood glucose concentrations are normal or elevated, GLP-1 and GIP increase insulin synthesis and release from pancreatic beta cells by intracellular signaling pathways involving cyclic AMP. Treatment with GLP-1 or with DPP-4 inhibitors inanimal models of type 2 diabetes has been demonstrated to improve beta cell responsiveness to glucose and stimulate insulin biosynthesis and release. With higher insulin levels, tissue glucose uptake is enhanced. In addition, GLP-1 lowers glucagon secretion from pancreatic alpha cells. Decreased glucagon concentrations, along with higher insulin levels, lead to reduced hepatic glucose production, resulting in a decrease in blood glucose levels. The effects of GLP-1 and GIP are glucose-dependent such that when blood glucose concentrations are low, stimulation of insulin release and suppression of glucagon secretion by GLP-1 are not observed. For both GLP-1 and GIP, stimulation of insulin release is enhanced as glucose rises

above normal concentrations. Further, GLP-1 does not impair the normal glucagon response to hypoglycaemia. The activity of GLP-1 and GIP is limited by the DPP-4 enzyme, which rapidly hydrolyzes the incretin hormones to produce inactive products. Sitagliptin prevents the hydrolysis of incretin hormones by DPP-4, thereby increasing plasma concentrations of the active forms of GLP-1 and GIP. By enhancing active incretin levels, sitagliptin increases insulin release and decreases glucagon levels in a glucose-dependent manner. In patients withtype 2 diabetes with hyperglycaemia, these changes in insulin and glucagon levels lead to lowerhaemoglobin A1c (HbA1c) and lower fasting and postprandial glucose concentrations. The glucose-dependent mechanism of sitagliptin is distinct from the mechanism of sulphonylureas,which increase insulin secretion even when glucose levels are low and can lead to hypoglycaemia in patients with type 2 diabetes and in normal subjects. Sitagliptin is a potent and highly selective inhibitor of the enzyme DPP-4 and does not inhibit the closely-related enzymes DPP-8 or DPP-9 at therapeutic concentrations.

In a two-day study in healthy subjects, sitagliptin alone increased active GLP-1 concentrations,whereas metformin alone increased active and total GLP-1 concentrations to similar extents. Co-administration of sitagliptin and metformin had an additive effect on active GLP-1 concentrations. Sitagliptin, but not metformin, increased active GIP concentrations.

5.2. P h a r m a c o k i n e t i c s

propertiesAbsorption

Following oral administration of a 100-mg dose to healthy subjects, Sitagliptin was rapidly absorbed, with peak plasma concentrations (median Tmax) occurring 1 to 4 hours post-dose, mean plasma AUC of sitagliptin was 8.52 μM•hr, Cmax was 950 nM. The absolute bioavailability of sitagliptin is approximately 87 %. Since co-administration of a high-fat meal with sitagliptin had no effect on the pharmacokinetics, Sitagliptin may be administered with or without food.

Plasma AUC of Sitagliptin increased in a dose-proportional manner. Dose proportionality was not established for Cmax and C24hr (Cmax increased in a greater than dose-proportional manner and C24hr increased in a less than dose proportional manner).

Distribution

The mean volume of distribution at steady state following a single 100-mg intravenous dose of sitagliptin to healthy subjects is approximately 198 litres. The fraction of sitagliptin reversiblybound to plasma proteins is low (38 %).

Biotransformation

Sitagliptin is primarily eliminated unchanged in urine, and metabolism is a minor pathway. Approximately 79 % of Sitagliptin is excreted unchanged in the urine. Following a [14C] Sitagliptin oral dose, approximately 16 % of the radioactivity was excreted as metabolites of sitagliptin. Six metabolites were detected at trace levels and are not expected to contribute to the plasma DPP-4 inhibitory activity of sitagliptin. *In vitro* studies indicated that the primary enzyme responsible for the limited metabolism of Sitagliptin was CYP3A4, with contribution from CYP2C8.

In vitro data showed that Sitagliptin is not an inhibitor of CYP isozymes CYP3A4, 2C8, 2C9, 2D6, 1A2, 2C19 or 2B6, and is not an inducer of CYP3A4 and CYP1A2.

Elimination

Following administration of an oral [14C] Sitagliptin dose to healthy subjects, approximately 100 % of the administered radioactivity was eliminated in faeces (13 %) or urine (87 %) within one week of dosing. The apparent terminal t1/2 following a 100- mg oral dose of Sitagliptin was approximately 12.4 hours. Sitagliptin accumulates only minimally with multiple doses. The renal clearance was approximately 350 ml/min.

Elimination of Sitagliptin occurs primarily via renal excretion and involves active tubular secretion. Sitagliptin is a substrate for human organic anion transporter-3 (hOAT-3), which may be involved in the renal elimination of sitagliptin. The clinical relevance of hOAT-3 in sitagliptin transport has not been established. Sitagliptin is also a substrate of p-glycoprotein, which may also be involved in mediating the renal elimination of Sitagliptin. However, ciclosporin, a p-glycoprotein inhibitor, did not reduce the renal clearance of Sitagliptin. Sitagliptin is not a substrate for OCT2 or OAT1 or PEPT1/2 transporters. *In vitro*, sitagliptin did not inhibit OAT3 (IC50=160 μM) or p-glycoprotein (up to 250 μM) mediated transport at therapeutically relevant plasma concentrations. In a clinical study Sitagliptin had a small effecton plasma digoxin concentrations indicating that sitagliptin may be a mild inhibitor of pglycoprotein.

5.3. Preclinical safety data:

Renal and liver toxicity were observed in rodents at systemic exposure values 58 times the human exposure level, while the no-effect level was found at 19 times the human exposure level. Incisor teeth abnormalities were observed in rats at exposure levels 67 times the clinical exposure level; the no-effect level for this finding was 58-fold based on the 14-week rat study.

The relevance of these findings for humans is unknown. Transient treatment-related physical signs, some of which suggest neural toxicity, such as open-mouth breathing, salivation, white foamy emesis, ataxia, trembling, decreased activity, and/or hunched posture were observed in dogs at exposure levels approximately 23 times the clinical exposure level. In addition, very slight to slight skeletal muscle degeneration was also observed histologically at doses resultingin systemic exposure levels of approximately 23 times the human exposure level. A noeffect level for these findings was found at an exposure 6-fold the clinical exposure level.

Sitagliptin has not been demonstrated to be genotoxic in preclinical studies. Sitagliptin was notcarcinogenic in mice. In rats, there was an increased incidence of hepatic adenomas and carcinomas at systemic exposure levels 58 times the human exposure level. Since hepatotoxicity has been shown to correlate with induction of hepatic neoplasia in rats, this increased incidence of hepatic tumours in rats was likely secondary to chronic hepatic toxicityat this high dose. Because of the high safety margin (19-fold at this no-effect level), these neoplastic changes are not considered relevant for the situation in humans.

No adverse effects upon fertility were observed in male and female rats given sitagliptin priorto and throughout mating.

In a pre-/postnatal development study performed in rats sitagliptin showed no adverse effects. Reproductive toxicity studies showed a slight treatment-related increased incidence of foetal rib malformations (absent, hypoplastic and wavy ribs) in the offspring of rats at systemic exposure levels more than 29 times the human exposure levels. Maternal toxicity was seen in rabbits at more than 29 times the human exposure levels. Because of the high safety margins, these findings do not suggest a relevant risk for human reproduction. Sitagliptin is secreted in considerable amounts into the milk of lactating rats (milk/plasma ratio: 4:1).

6. PHARMACEUTICAL PARTICULARS

6.1. List of excipientsTablet core:

Sitaglitpin Phosphate
Microcrystalline Cellulose (PH 102)
Anhydrous Dibasic Calcium
PhosphateCroscarmellose Sodium
Sodium Stearyl
FumarateMagnesium
Stearate

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Film coating:

Insta moistshield Aqua II (White) [IC-AMS-II-1675]

Opadry OY-B-37203 (Tan)

Qualitative and Quantitative Formula: **Insta moist-shield Aqua II (White) [IC-AMS-II-1675]**

Ingredients	**% w/w**
Polyvinyl Alcohol USP	35-45
Polyethylene Glycol USPNF/BP/EP/IP	10-20
Talc USP/BP/EP/IP	1-6
Triacetin USP/ EP/IP/BP	0.5-2.5
Tribasic Calcium Phosphate USP/IP/EP/BP	10-20
Titanium Dioxide USP/BP/EP/IP	15-25

Qualitative and Quantitative Formula: **Opadry OY-B-37203**

Ingredients	**% w/w**
Polyvinyl Alcohol – Part hydrolysed (USP, FCC, PhEur, JPE)	45.519
Titanium Dioxide (USP, FCC, PhEur, JP, ChP, GB)	26.750
Talc (USP, FCC, PhEur, JP, JECFA)	19.999
Iron Oxide Red (NF, JPE, JECFA, CP)	3.000
Iron Oxide Yellow (NF, JPE, JECFA, CP)	2.000
Lecithin (SOYA) (NF, JPE, FCC, JSFA)	1.999
Xanthan Gum (NF, FCC, PhEur, JPE,ChP,JECFA)	0.483
Ferrosoferric Oxide (NF)/Black Iron Oxide (JPE, JECFA, ChP)	0.250

Qualitative and Quantitative Formula: **Opadry II 85G52259**

Ingredients	**% w/w**
Polyvinyl Alcohol – Part hydrolysed (USP, FCC, PhEur, JPE)	44.000
Talc (USP, FCC, PhEur, JP, JECFA)	20.000
Quinoline Yellow Aluminium Lake (JECFA)	17.150
Macrogol/ PEG (USP, FCC, PhEur, JECFA, JP)	12.350
Lecithin (SOYA) (NF, JPE, FCC, JSFA)	3.500
Titanium Dioxide (USP, FCC, PhEur, JP, ChP, GB)	3.000

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6.2. Incompatibilities

Not applicable.

6.3. Shelf-life

24 months

6.4. Special precaution for storage

Do not store above 30°C. Protect from light.

6.5. Nature and content of container

Aluminium–Aluminium blister pack, each blister contains 10 Tablets.

The blisters are further packed in Paperboard carton (Each carton contains 1 blisters with 1package insert).

6.6. Special precaution for disposal

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

7. MARKETING AUTHORIZATION HOLDER

Name: FAROMED LIFESCIENCES LLP

Business Address: Plot No. 29-33, Ancillary Industrial Area,Deonar, Govandi, Mumbai-400 043, India.

Postal Address: Spaces, Inspire Hub, Western Heights, J.P Road,2036, 2nd floor, Andheri West, Mumbai, India 400053.

Email Address: anurag@omnicals.com

Manufacturer Details:

Name: The ACME Laboratories Ltd.

Address: Dhulivita, Dhamrai, Dhaka, BangladeshE-Mail: plant@acmeglobal.com (Plant).

8. MARKETING AUTHORIZATION NUMBER

TAN 22 HM 0258

9. DATE OF FIRST REGISTRATION / RENEWAL OF THE REGISTRATION

19th July, 2022

10. DATE OF REVISION OF THE TEXT