For the use of a Registered Medical Practitioner Only

PRESCRIBING INFORMATION

INSTADIP 25 / 50 / 100

(Sitagliptin Film Coated Tablets USP 25mg, 50mg, 100mg)

COMPOSITION

INSTADIP 25
Sitagliptin tablet USP 25mg
Each film coated tablet contains:
Sitagliptin Phosphate USP is equivalent to Sitagliptin .....…25 mg

INSTADIP 50
Sitagliptin tablet USP 50mg
Each film coated tablet contains:
Sitagliptin Phosphate USP is equivalent to Sitagliptin.....…50 mg

INSTADIP 100
Sitagliptin tablet USP 100mg
Each film coated tablet contains:
Sitagliptin Phosphate USP is equivalent to Sitagliptin .....…100 mg

Excipients:
Microcrystalline cellulose, dibasic calcium phosphate (anhydrous), croscarmellose sodium, sodium stearyl fumarate, magnesium stearate, Opadry Orange 03F530010 (25mg), Opadry Beige 03F570000 (50mg), Opadry Beige 03F570001(100mg)

DESCRIPTION

INSTADIP 25
Orange, round, film-coated tablets plain on both sides

INSTADIP 50
Light beige, round, film coated tablets plain on both sides

INSTADIP 100
Beige, round, film coated tablets plain on both sides

PHARMACODYNAMIC AND PHARMACOKINETIC PROPERTIES

Pharmacodynamic properties

Sitagliptin is a member of a class of oral anti-hyperglycemic agents called dipeptidyl peptidase 4 (DPP-4) inhibitors, which improve glycemic control in patients with type 2 diabetes 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 in response to a meal. The incretins are part of an endogenous system involved in the physiologic regulation 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 in animal models of type 2 diabetes has been reported 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 hypoglycemia. 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 with type 2 diabetes with hyperglycemia, these changes in insulin and glucagon levels lead to lower hemoglobin A1c (HbA1c) and lower fasting and postprandial glucose concentrations. The glucose-dependent mechanism of sitagliptin is distinct from the mechanism of sulfonylureas, which increase insulin secretion even when glucose levels are low and can lead to hypoglycemia 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.

Pharmacokinetics

After oral administration of a 100-mg dose to healthy subjects, sitagliptin has been reported to be rapidly absorbed, with peak plasma concentrations (median T_{max}) occurring 1 to 4 hours post-dose. Plasma AUC of sitagliptin has been reported to increase in a dose-proportional manner. Following a single oral 100-mg dose to healthy volunteers, mean plasma AUC of sitagliptin has been reported to be 8.52 μM•hr, C_{max} was 950 nM, and apparent terminal half-life ($t_{1/2}$) was 12.4 hours. Plasma AUC of sitagliptin has been reported to increase approximately 14% following 100-mg doses at steady-state compared to the first dose. The intra-subject and inter-subject coefficients of variation for sitagliptin AUC has been reported to be small (5.8% and 15.1%). The pharmacokinetics of sitagliptin has been reported to be generally similar in healthy subjects and in patients with type 2 diabetes.

Absorption

The absolute bioavailability of sitagliptin has been reported to be approximately 87%. Since co-administration of a high-fat meal with sitagliptin had been reported to have no effect on the pharmacokinetics, sitagliptin may be administered with or without food.

Distribution
The mean volume of distribution at steady state following a single 100-mg intravenous dose of sitagliptin to healthy subjects has been reported to be approximately 198 liters. The fraction of sitagliptin reversibly bound to plasma proteins has been reported to be low (38%).

Metabolism
Sitagliptin has been reported to be primarily eliminated unchanged in urine, and metabolism has been reported to be a minor pathway. Approximately 79% of sitagliptin has been reported to be excreted unchanged in the urine.

Following a [^{14}C]sitagliptin oral dose, approximately 16% of the radioactivity has been reported to be excreted as metabolites of sitagliptin. Six metabolites have been reported to be detected at trace levels and are not expected to contribute to the plasma DPP-4 inhibitory activity of sitagliptin. The primary enzyme responsible for the limited metabolism of sitagliptin has been reported to be CYP3A4, with contribution from CYP2C8.

Elimination
Following administration of an oral [^{14}C] sitagliptin dose to healthy subjects, approximately 100% of the administered radioactivity has been reported to be eliminated in feces (13%) or urine (87%) within one week of dosing. The apparent terminal $t_{1/2}$ following a 100-mg oral dose of sitagliptin has been reported to be approximately 12.4 hours and renal clearance was reported to be 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 reported. Sitagliptin is also a substrate of p-glycoprotein, which may also be involved in mediating the renal elimination of sitagliptin. However, cyclosporine, a p-glycoprotein inhibitor, has not beed reported to reduce the renal clearance of sitagliptin.

Characteristics in Patients

Renal Impairment
Compared to normal healthy subjects, plasma AUC of sitagliptin has been reported to increase by approximately 1.2-fold and 1.6-fold in patients with mild renal impairment (eGFR ≥ 60 mL/min/1.73 m^2 to < 90 mL/min/1.73 m^2) and patients with moderate renal impairment (eGFR ≥ 45 mL/min/1.73 m^2 to < 60 mL/min/1.73 m^2), respectively. Because increases of this magnitude are not clinically relevant, dosage adjustment in these patients is not necessary.

Plasma AUC of sitagliptin has been reported to increase approximately 2-fold in patients with moderate renal impairment (eGFR ≥ 30 mL/min/1.73 m^2 to < 45 mL/min/1.73 m^2), and approximately 4-fold in patients with severe renal impairment (eGFR < 30 mL/min/1.73 m^2), including patients with ESRD on hemodialysis. Sitagliptin has been reported to be modestly removed by hemodialysis (13.5% over a 3- to 4-hour hemodialysis session starting 4 hours post-dose). To achieve plasma concentrations of sitagliptin similar to those in patients with normal renal function, lower dosages are recommended in patients with eGFR <45 mL/min/1.73 m^2 (see DOSE AND METHOD OF ADMINISTRATION, Patients with Renal Impairment).

Hepatic Impairment:
In patients with moderate hepatic impairment (Child-Pugh score 7 to 9), mean AUC and C_{max} of sitagliptin has been reported to increase approximately 21% and 13%, respectively, compared to healthy subjects following administration of a single 100-mg dose of sitagliptin. These differences are not considered to be clinically meaningful. No dosage adjustment for sitagliptin is necessary for patients with mild or moderate hepatic impairment.

There is no clinical experience in patients reported with severe hepatic impairment (Child-Pugh score >9). However, because sitagliptin is primarily renally eliminated, severe hepatic impairment is not expected to affect the pharmacokinetics of sitagliptin.

Elderly:
No dosage adjustment is required based on age. Age did not have a clinically meaningful impact on the pharmacokinetics of sitagliptin. Elderly subjects (65 to 80 years) had been reported to have approximately 19% higher plasma concentrations of sitagliptin compared to younger subjects.

Pediatric:
No data with sitagliptin have been reported in pediatric patients.

Gender:
No dosage adjustment is necessary based on gender. Gender had been reported to have no clinically meaningful effect on the pharmacokinetics of sitagliptin.

Race:
No dosage adjustment is necessary based on race. Race had been reported to have no clinically meaningful effect on the pharmacokinetics of sitagliptin.

Body Mass Index (BMI):
No dosage adjustment is necessary based on BMI. Body mass index had been reported to have no clinically meaningful effect on the pharmacokinetics of sitagliptin.

Type 2 Diabetes:
The pharmacokinetics of sitagliptin in patients with type 2 diabetes have been reported to be generally similar to those in healthy subjects.

INDICATIONS

Monotherapy
Sitagliptin is indicated as an adjunct to diet and exercise to improve glycemic control in patients with type 2 diabetes mellitus.

Combination with metformin
Sitagliptin is indicated in patients with type 2 diabetes mellitus to improve glycemic control in combination with metformin as initial therapy or when the single agent alone, with diet and exercise, does not provide

adequate glycemic control. Initial combination therapy or maintenance of combination therapy may not be appropriate for all patients. These management options are left to the discretion of the health care provider.

Combination with a sulphonylurea
Sitagliptin is indicated in patients with type 2 diabetes mellitus to improve glycemic control in combination with a sulphonylureas when treatment with maximal tolerated dose of sulphonylurea alone, with diet and exercise, does not provide adequate glycemic control and when metformin is inappropriate due to contraindications or intolerance.

Combination with metformin and a sulphonylurea
Sitagliptin is indicated in patients with type 2 diabetes mellitus to improve glycemic control in combination with metformin and a sulphonylurea when dual therapy with these two agents and with diet and exercise does not provide adequate glycemic control.

Combination with a peroxisome proliferator-activated receptor gamma (PPARγ) agonist
Sitagliptin is indicated in patients with type 2 diabetes mellitus to improve glycemic control in combination with a PPARγ agonist (i.e. thiazolidinediones) when diet and exercise, plus the single agent do not provide adequate glycemic control.

Combination with metformin and a PPARγ agonist
Sitagliptin is indicated in patients with type 2 diabetes mellitus to improve glycemic control in combination with metformin and a PPARγ agonist (i.e., thiazolidinediones) when dual therapy with these agents, with diet and exercise, does not provide adequate glycemic control.

Combination with Insulin
Sitagliptin is indicated in patients with type 2 diabetes mellitus as an adjunct to diet and exercise to improve glycemic control in combination with insulin (with or without metformin).

DOSE AND METHOD OF ADMINISTRATION

The recommended dose of sitagliptin is 100 mg once daily as monotherapy or as combination therapy with metformin, a sulfonylurea, insulin (with or without metformin), a PPARγ agonist (i.e., thiazolidinediones), metformin plus a sulfonylurea, or metformin plus a PPARγ agonist. Sitagliptin can be taken with or without food.

When sitagliptin is used in combination with a sulfonylurea or with insulin, a lower dose of sulfonylurea or insulin may be considered to reduce the risk of sulfonylurea- or insulin-induced hypoglycaemia. (See WARNINGS AND PRECAUTIONS)

Patients with renal impairment
Because there is a dosage adjustment based upon renal function, assessment of renal function is recommended prior to initiation of sitagliptin and periodically thereafter.

For patients with mild renal impairment (estimated glomerular filtration rate [eGFR] ≥60 mL/min/1.73 m^2 to < 90 mL/min/1.73 m^2), no dosage adjustment for sitagliptin is required.

For patients with moderate renal impairment (eGFR ≥45 mL/min.1.73 m^2 to < 60 mL/min.1.73 m^2), no dosage adjustment for sitagliptin is required.

For patients with moderate renal impairment (eGFR ≥30 mL/min/1.73 m^2 to 45 mL/min/1.73 m^2), the dose of sitagliptin is 50 mg once daily.

For patients with severe renal impairment (eGFR ≥15 mL/min/1.73 m^2 to < 30 mL/min/1.73 m^2) or with end-stage renal disease (ESRD) (eGFR < 15 mL/min/1.73 m^2), including those requiring hemodialysis or peritoneal dialysis, the dose of sitagliptin is 25 mg once daily. Sitagliptin may be administered without regard to the timing of dialysis.

Pediatric population
INSTADIP should not be used in children and adolescents 10 to 17 years of age because of insufficient efficacy. INSTADIP has not been studied in paediatric patients under 10 years of age.

CONTRAINDICATIONS

Sitagliptin is contraindicated in patients who are hypersensitive to any components of this product (See WARNINGS AND PRECAUTIONS, Hypersensitivity Reactions and UNDESIRABLE EFFECTS).

WARNINGS AND PRECAUTIONS

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

Pancreatitis:
There have been reports of acute pancreatitis, including fatal and non-fatal hemorrhagic or necrotizing pancreatitis (see UNDESIRABLE EFFECTS), in patients taking sitagliptin. Patients should be informed of the characteristic symptom of acute pancreatitis: persistent, severe abdominal pain. Resolution of pancreatitis has been reported after discontinuation of sitagliptin. If pancreatitis is suspected, sitagliptin and other potentially suspect medicinal products should be discontinued.

Use in Patients with 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 eGFR < 45 mL/min/1.73 m^2), as well as in ESRD patients requiring hemodialysis or peritoneal dialysis (See DOSE AND METHOD OF ADMINISTRATION, Patients with Renal Impairment).

Hypoglycemia in Combination with a Sulfonylurea or with Insulin:
Rates of hypoglycemia with sitagliptin have been reported to be similar to rates in patients taking placebo when given as monotherapy or as part of combination therapy with agents not known to cause hypoglycemia [i.e. metformin or a PPARγ agonist (thiazolidinedione)]. As is typical with other antihyperglycemic agents, hypoglycemia has been reported when sitagliptin was used in combination with insulin or a sulfonylurea (see

UNDESIRABLE EFFECTS). Therefore, to reduce the risk of sulfonylurea- or insulin-induced hypoglycemia, a lower dose of sulfonylurea or insulin may be considered (see DOSE AND METHOD OF ADMINISTRATION).

Hypersensitivity Reactions:
There have been post-marketing reports of serious hypersensitivity reactions in patients treated with sitagliptin. These reactions include anaphylaxis, angioedema, and exfoliative skin conditions including Stevens-Johnson syndrome. Because these reactions are reported voluntarily from a population of uncertain size, it is generally not possible to reliably estimate their frequency or establish a causal relationship to drug exposure. Onset of these reactions have reported to be within the first 3 months after initiation of treatment with sitagliptin, with some reports reported after the first dose. If a hypersensitivity reaction is suspected, discontinue sitagliptin, assess for other potential causes for the event, and institute alternative treatment for diabetes (See CONTRAINDICATIONS and UNDESIRABLE EFFECTS)

Severe and Disabling Arthralgia:
There have been post-marketing reports of severe and disabling arthralgia in patients taking DPP-4 inhibitors. The time to onset of symptoms following initiation of drug therapy have been reported to vary from one day to years. Patients have been reported to experience relief of symptoms upon discontinuation of the medication. A subset of patients have been reported to experience a recurrence of symptoms when restarting the same drug or a different DPP-4 inhibitor. Consider DPP-4 inhibitors as a possible cause for severe joint pain and discontinue drug if appropriate.

Bullous Pemphigoid:
Post-marketing cases of bullous pemphigoid requiring hospitalization have been reported with DPP-4 inhibitor use. In reported cases, patients typically recovered with topical or systemic immunosuppressive treatment and discontinuation of the DPP-4 inhibitor. Tell patients to report development of blisters or erosions while receiving sitagliptin. If bullous pemphigoid is suspected, sitagliptin should be discontinued and referral to a dermatologist should be considered for diagnosis and appropriate treatment.

Effects on ability to drive and use machines
Sitagliptin has reported to have 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 somnolence have been reported.

In addition, patients should be alerted to the risk of hypoglycaemia when sitagliptin is used in combination with a sulphonylurea or with insulin.

DRUG INTERACTIONS

Sitagliptin did not reported to have clinically meaningful effects on the pharmacokinetics of the following: metformin, rosiglitazone, glyburide, simvastatin, warfarin, and oral contraceptives. Sitagliptin does not inhibit CYP isozymes CYP3A4, 2C8, or 2C9. Sitagliptin is also not expected to inhibit CYP2D6, 1A2, 2C19 or 2B6 or to induce CYP3A4.

Co-administration of multiple twice-daily doses of metformin with sitagliptin did not reported to meaningfully alter the pharmacokinetics of sitagliptin in patients with type 2 diabetes.

Concomitant medications that are commonly administered to patients with type 2 diabetes, including cholesterol-lowering agents (e.g., statins, fibrates, ezetimibe), anti-platelet agents (e.g., clopidogrel), antihypertensives (e.g., ACE inhibitors, angiotensin receptor blockers, beta-blockers, calcium channel blockers, hydrochlorothiazide), analgesics and non-steroidal anti-inflammatory agents (e.g., naproxen, diclofenac, celecoxib), anti-depressants (e.g., bupropion, fluoxetine, sertraline), antihistamines (e.g., cetirizine), proton-pump inhibitors (e.g., omeprazole, lansoprazole), and medications for erectile dysfunction (e.g., sildenafil) did not reported to have a clinically meaningful effect on the pharmacokinetics of sitagliptin.

A slight increase in the area under the curve (AUC, 11%) and mean peak drug concentration (C_{max}, 18%) of digoxin with the co-administration of sitagliptin has been reported. These increases are not considered to be clinically meaningful. Patients receiving digoxin should be monitored appropriately. No dosage adjustment of digoxin or sitagliptin is recommended.

The AUC and C_{max} of sitagliptin have been reported to increase approximately 29% and 68%, respectively, in subjects with co-administration of a single 100-mg oral dose of sitagliptin and a single 600-mg oral dose of cyclosporine, a potent probe inhibitor of p-glycoprotein. The reported changes in sitagliptin pharmacokinetics are not considered to be clinically meaningful. No dosage adjustment for sitagliptin is recommended when co-administered with cyclosporine or other p-glycoprotein inhibitors (e.g., ketoconazole).

UNDESIRABLE EFFECTS

Sitagliptin has been reported to be generally well tolerated as both monotherapy and combination therapy, with discontinuation of therapy due to clinical adverse experiences similar to placebo.

Table: ADRs associated with sitagliptin therapy

Adverse reaction	Frequency of adverse reaction
Blood and lymphatic system disorders	
thrombocytopenia†	Rare
Immune system disorders	
hypersensitivity reactions including anaphylactic responses*,†	Frequency not known
Metabolism and nutrition disorders	
hypoglycaemia†	Common
Nervous system disorders	
headache	Common
dizziness	Uncommon
Respiratory, thoracic and mediastinal disorders	
cough	Common
interstitial lung disease*	Frequency not known
upper respiratory tract infection†	Frequency not known
nasopharyngitis†	Frequency not known
Gastrointestinal disorders	
nausea	Common

abdominal pain	Common
diarrhea	Common
dyspepsia	Common
flatulence	Common
constipation	Uncommon
vomiting*	Frequency not known
acute pancreatitis*,†,‡	Frequency not known
fatal and non-fatal haemorrhagic and necrotizing pancreatitis*,†	Frequency not known
Skin and subcutaneous tissue disorders	
fungal skin infection	Common
peripheral edema	Common
pruritus*	Uncommon
angioedema*,†	Frequency not known
anaphylaxis	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	
severe and disabling arthralgia*	Frequency not known
myalgia*	Frequency not known
back pain*	Frequency not known
arthropathy*	Frequency not known
pain in extremity†	Frequency not known
Renal and urinary disorders	
impaired renal function*	Frequency not known
acute renal failure* (sometimes requiring dialysis)	Frequency not known

*Adverse reactions were identified through post-marketing surveillance.
† See WARNINGS AND PRECAUTIONS.

Paediatric population
In paediatric patients with type 2 diabetes mellitus aged 10 to17 years, the profile of adverse reactions has been reported to be comparable to that reported in adults.

USE IN SPECIAL POPULATIONS

Pregnancy

Sitagliptin was not been reported to be teratogenic in rats at oral doses up to 250 mg/kg or in rabbits given up to 125 mg/kg during organogenesis (up to 32 and 22 times, respectively, the human exposure based on the recommended daily adult human dose of 100 mg/day). In rats, a slight increase in the incidence of fetal rib malformations (absent, hypoplastic and wavy ribs) has been reported at oral doses of 1000 mg/kg/day

(approximately 100 times the human exposure based on the recommended daily adult human dose of 100 mg/day). Slight decreases in mean preweaning body weights of both sexes and postweaning body weight gains of males has been reported in the offspring of rats given oral dose of 1000 mg/kg/day. However, animal reproduction studies are not always predictive of the human response.

There are no adequate and well-controlled data reported in pregnant women; therefore, the safety of sitagliptin in pregnant women is not known. Sitagliptin, like other oral antihyperglycemic agents, is not recommended for use in pregnancy.

Lactation
Sitagliptin has been reported to be secreted in the milk of lactating rats. It is not known whether sitagliptin is secreted in human milk. Therefore, sitagliptin should not be used by a woman who is nursing.

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

Pediatric use
Safety and effectiveness of sitagliptin in pediatric patients under 18 years have not been established.

Use in the elderly
The safety and effectiveness of sitagliptin in the elderly (≥65 years) has been reported to be comparable to those seen in younger patients (<65 years). No dosage adjustment is required based on age. Elderly patients are more likely to have renal impairment; as with other patients, dosage adjustment may be required in the presence of significant renal impairment (see DOSE AND METHOD OF ADMINISTRATION, Patients with Renal Impairment).

OVERDOSE

Single doses of up to 800 mg sitagliptin has been reported to be generally well tolerated in healthy subjects. Minimal increases in QTc, not considered to be clinically relevant, have been reported at a dose of 800 mg sitagliptin. There is no data reported with doses above 800 mg. No dose-related clinical adverse reactions have been reported 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 dialyzable. Approximately 13.5% of the dose has been reported to be removed over a 3- to 4-hour hemodialysis session. Prolonged hemodialysis may be considered if clinically appropriate. It is not known if sitagliptin is dialyzable by peritoneal dialysis.

Pack Style:
INSTADIP TABLETS 25 mg, 50 mg and 100 mg is available in Aluminium cold form blister packs of 3 x 10 tablets with Show box along with pack insert.

STORAGE
Store up to 30°C

Manufacturer:
Sun Pharmaceutical Industries Ltd.
Village Ganguwala, Paonta Sahib – 173 025,
District Sirmour, Himachal Pradesh. INDIA.

Product Registration Holder:
RANBAXY (MALAYSIA) SDN. BHD.
(A Sun Pharma Company)
Lot 23, Bakar Arang Industrial Estate,
08000 Sungai Petani, Kedah,
Malaysia.

Date of Revision:
January 2023

Note for artwork:
INSTADIP TABS. 25/50/100mg PI
Pack Insert size: 140 x 400 mm
Market: Malaysia
Fonts/ point size: Arial Narrow Font - 7 pt