AUSTRALIAN PI – APO-GLICLAZIDE TABLETS (GLICLAZIDE)

1 NAME OF THE MEDICINE

Gliclazide.

2 AND 3 QUALITATIVE AND QUANTITATIVE COMPOSITION AND PHARMACEUTICAL FORM

Gliclazide MR 30 mg tablets are intended for oral administration. Each tablet contains gliclazide 30 mg, as the active ingredient.

Gliclazide is a white or almost white powder which is practically insoluble in water. Freely soluble in dichloromethane, sparingly soluble in acetone and slightly soluble in ethanol 96%. The melting point of gliclazide is approximately 168 °C.

In addition to gliclazide, each tablet contains the following inactive ingredients: hypromellose, stearic acid and colloidal anhydrous silica.

30 mg Tablets:

White to off-white, flat faced, radial edge, capsule shaped tablets, engraved “APO 30” on one side and plain on the other side.

4 CLINICAL PARTICULARS

4.1 THERAPEUTIC INDICATIONS

Type II diabetes in association with dietary measures when dietary measures alone are inadequate to control blood glucose.

During controlled clinical trials in patients with type II diabetes, modified release formulation of gliclazide (30 mg - 120 mg), taken as a single daily dose, was shown to be effective long term in controlling blood glucose levels, based on monitoring of HbA1c.

4.2 DOSE AND METHOD OF ADMINISTRATION

Gliclazide MR 30 mg Tablets are for adult use only.

The daily dose may vary from 30 mg to 120 mg taken orally, once daily. Gliclazide MR 30 mg Tablets should be taken with food because there is an increased risk of hypoglycemia if a meal is taken late, if an inadequate amount of food is consumed or if the food is low in carbohydrate. It is recommended that the medication be taken at breakfast time. If a dose is forgotten, the dose taken on the next day should not be increased.

Gliclazide MR 30 mg Tablets are modified release tablets and therefore should be neither broken nor chewed.

As with all hypoglycaemic agents, the dose should be titrated according to the individual patient's response.

The initial recommended dose is 30 mg daily, even in elderly patients (≥ 65 years).

Dose titration should be carried out in steps of 30 mg, according to the fasting blood glucose response. Each step should last for at least two weeks. A single daily dose provides an effective blood glucose control. The single daily dose may be between one and three, or even four, tablets. The daily dose should not exceed 120 mg.

Previously untreated patients should commence with a dose of 30 mg and will benefit from dose titration until the appropriate dose is reached.

Gliclazide MR 30 mg Tablets, can replace gliclazide 80 mg tablets, tablet for tablet, for doses of 1 to 4 tablets per day.

Gliclazide MR 30 mg Tablets, may be used to replace other antidiabetic treatments without any transitional period. If a patient is switched from a hypoglycaemic sulfonylurea with a prolonged half-life he/she should be carefully monitored (for 1 to 2 weeks) in order to avoid hypoglycaemia due to possible residual effects of the previous therapy.

Gliclazide MR 30 mg Tablets, may be given in combination with biguanides, alpha glucosidase inhibitors or insulin.

Elderly patients: The efficacy and tolerance of Gliclazide MR 30 mg Tablets has been confirmed in clinical trials in patients over 65 years who were given the same dosage regimen as the general population. The dosage is therefore identical to that recommended for adults under the age of 65 years.

Renal impairment: The efficacy and tolerance of Gliclazide MR 30 mg Tablets has been confirmed in clinical trials of subjects with mild to moderate renal failure (creatinine clearance of between 15 - 80 mL/min) who were given the same dosage regimen as the general population. No dosage adjustment is therefore required in subjects patients with mild to moderate renal impairment. Use of Gliclazide MR 30 mg in patients with severe renal impairment is contraindicated (see **4.3 Contraindications**).

4.3 CONTRAINDICATIONS

This medication is contraindicated in the following cases:

- Hypersensitivity to gliclazide, other sulphonylureas, sulfonamides, or to any of the excipients
- Type I diabetes, diabetic keto-acidosis, diabetic pre-coma and coma
- Severe renal or hepatic impairment.
- Treatment with miconazole (See **4.5 Interactions with other medicines and other forms of interactions**),
- Pregnancy and lactation (See **4.6 Fertility, Pregnancy and Lactation, Use in Pregnancy and Use in Lactation**).

It is generally not recommended to use this agent in combination with phenylbutazone or danazol (See **4.5 Interactions with other medicines and other forms of interactions**).

4.4 SPECIAL WARNINGS AND PRECAUTIONS FOR USE

Acute Complications such as severe trauma, fever, infection or surgery

These acute complications provoke additional metabolic stress, which accentuate the predisposition to hyperglycaemia and ketosis. Patients presenting with such conditions may require insulin to maintain control. It is not appropriate to increase the dosage of gliclazide.

Hypoglycaemia

The risks of hypoglycaemia, together with its symptoms, treatment and conditions that predispose to its development, should be explained to the patient and to family members. The patient should be informed of the importance of following dietary advice, of taking regular exercise, and of regular monitoring of blood glucose levels.

Hypoglycaemia may occur following administration of sulphonylureas. Rarely cases may be severe and prolonged. This may involve hospitalisation and glucose infusion may need to be continued for several days.

Careful selection of patients and of the dose used, as well as provision of adequate information to the patient are necessary to avoid hypoglycaemic episodes.

The following factors may increase the risk of hypoglycaemia:

- patient does not follow the doctor's treatment advice (particularly elderly subjects)
- malnutrition
- irregular mealtimes, skipping meals, periods of fasting or dietary changes
- imbalance between physical exercise and carbohydrate intake
- renal impairment
- severe hepatic impairment
- overdose of anti-diabetic agents
- certain endocrine disorders: thyroid disorders, hypopituitarism and adrenal impairment
- concomitant administration of certain other medicines (See **4.5 Interactions with other medicines and other forms of interactions**).

Experience with sulfonylureas shows that hypoglycaemia can recur even when measures such as the intake of carbohydrate such as sugar are initially effective. If a hypoglycaemic episode is severe or prolonged, and even if it is temporarily controlled by intake of sugar, immediate medical treatment or even hospitalisation is required.

Patients must be warned that artificial sweeteners are not recommended in the treatment of hypoglycaemia as they have negligible effect.

Hypoglycaemia may be difficult to recognise in elderly patients and those receiving beta-blockers.

This treatment should only be prescribed if the patient is likely to have a regular food intake (including breakfast). It is important to have a regular carbohydrate intake due to the increased risk of hypoglycaemia if a meal is delayed, an inadequate amount of food is consumed or the food is low in carbohydrate. Hypoglycaemia is more likely to occur during periods of low-calorie diet, following prolonged or strenuous exercise, following alcohol intake or during treatment with a combination of hypoglycaemic agents.

Patient awareness

Comprehensive instructions must be given to the patient about the nature of the disease and what must be done to detect and prevent complications.

Poor Blood Glucose Control

Blood glucose control in treated patients may be affected by St. John's wort (*Hypericum perforatum*) preparations (See **4.5 Interactions with other medicines and other forms of interactions**), fever, trauma, infection or surgical intervention. It may be necessary to discontinue treatment and to administer insulin in these cases.

The efficacy of oral antidiabetic agents often decreases in the long term. This may be due to progression in the severity of the diabetes, or to a reduced response to treatment. This phenomenon is known as secondary failure and should be distinguished from primary failure, when the drug is ineffective as first-line treatment. However, before classifying the patient as a secondary failure, dose adjustment and reinforcement of dietary measures should be considered.

Use in Renal and Hepatic Impairment

Severe renal or hepatic impairment may affect the distribution of gliclazide and hepatic impairment may also reduce the capacity for neoglucogenesis. These two effects increase the risk of severe hypoglycaemic reactions. A hypoglycaemic episode in these patients may be prolonged and appropriate management should be initiated.

Glucose-6-phosphate dehydrogenase deficiency (G6PD)

Treatment of patients with G6PD-deficiency with sulfonylurea agents can lead to haemolytic anaemia. Since gliclazide belongs to the chemical class of sulfonylurea drugs, caution should be used in patients with G6PD-deficiency and a non-sulfonylurea alternative should be considered.

Use in the elderly

See 4.2 Dose and method of administration

Paediatric use

See 4.2 Dose and method of administration.

Laboratory Tests

Glycated haemoglobin should be monitored regularly. Blood glucose measurement may also be useful.

4.5 INTERACTIONS WITH OTHER MEDICINES AND OTHER FORMS OF INTERACTIONS

Blood glucose monitoring during and after treatment is necessary when gliclazide is used with medicines which can interact with gliclazide. It may also be necessary to adjust the dose of gliclazide MR during and after treatment with such medicines.

The following medications are likely to increase the risk of hypoglycaemia

Concomitant use which is contraindicated:

Miconazole (*systemic route*, *oromucosal gel*):

Increases the hypoglycaemic effect with possible onset of hypoglycaemia symptoms or even coma.

Concomitant use which is not recommended:

Phenylbutazone (systemic route):

Increases the hypoglycaemic effect of sulphonylureas (displaces their binding to plasma proteins and/or reduces their elimination).

It is preferable to use a different anti-inflammatory agent, or else to warn the patient and emphasise the importance of self-monitoring. Where necessary, adjust the dose during and after treatment with the anti-inflammatory agent.

Alcohol:

Acute alcohol intoxication potentiates the hypoglycaemic action of all sulfonylurea agents by inhibiting compensatory reactions. This can lead to the onset of hypoglycaemic coma. Ingestion of alcohol may also cause a disulfiram-like reaction with characteristic flushing of the face, throbbing headache, giddiness, tachypnoea, tachycardia or angina pectoris.

Chronic alcohol abuse may, as a result of liver enzyme induction, increase the metabolism of sulfonylurea drugs, shortening the plasma half-life and duration of action.

Avoid alcohol or medicines containing alcohol.

Concomitant use which requires special care:

Potentiation of the blood glucose lowering effect and therefore in some instances, hypoglycaemia may occur when one of the following medications is taken:

- other antidiabetic agents (insulins, acarbose, biguanides, metformin, thiazolidinediones, dipeptidyl peptidase-4 inhibitors, GLP-1 receptor agonists)
- sulfonamides
- clarithromycin
- clofibrate
- salicylates (high doses)
- Chloramphenicol
- MAOIs
- β-blockers
- H_2-receptor antagonists
- ACE inhibitors
- fluconazole
- nonsteroidal anti-inflammatory agents
- quinolone antibiotics.

The following medications may cause an increase in blood glucose levels

Advise the patient and emphasise the importance of glucose monitoring.

Concomitant use which is not recommended:

Danazol:

If the use of danazol cannot be avoided, it may be necessary to adjust the dose of gliclazide MR during and after treatment with danazol.

Concomitant use which requires special care:

Chlorpromazine:

High doses (>100 mg per day of chlorpromazine) can increase blood glucose levels (reduced insulin release).

Advise the patient and emphasise the importance of glucose monitoring. It may be necessary to adjust the dose of gliclazide MR during and after treatment with chlorpromazine.

Glucocorticoids (systemic and local route: intra-articular, cutaneous and rectal preparations) and tetracosactrin:

Concomitant use may increase blood glucose levels with possible ketosis (glucocorticoids cause reduced tolerance to carbohydrates). Emphasise the importance of blood glucose monitoring, particularly at the start of treatment. It may be necessary to adjust the dose of Gliclazide MR during and after treatment with glucocorticoids.

Salbutamol, terbutaline (intravenous):

May cause increased blood glucose levels due to β_2 agonist effects. If necessary, switch to insulin.

Barbiturates, oestrogens and progestogens:

May adversely affect blood sugar control with hypoglycaemic agents in some patients by causing increased blood glucose levels.

St John's wort (Hypericum perforatum) preparations:

Gliclazide exposure is decreased by St John's wort (*Hypericum perforatum*).

Concomitant use to be taken into consideration:

Anticoagulant therapy (warfarin):

Sulphonylureas may lead to potentiation of anticoagulation during concurrent treatment. Adjustment of warfarin may be necessary.

4.6 FERTILITY, PREGNANCY AND LACTATION

Effect on Fertility

No data available.

Use in Pregnancy (Category C)

The sulfonylureas may enter the foetal circulation and cause neonatal hypoglycaemia. In animal studies embryo-toxicity and/or birth defects have been demonstrated with some sulfonylureas.

Gliclazide should not be used in pregnant women although animal studies of gliclazide have not shown any teratogenic effect. From a clinical point of view, there are no adequate data to allow evaluation of the possible malformative or foetotoxic effects of gliclazide, when administered during pregnancy.

Gliclazide is contraindicated during pregnancy and insulin is the drug of first choice for treatment of diabetes during pregnancy. Treatment should be changed from gliclazide to insulin therapy before pregnancy is attempted, or as soon as pregnancy is discovered. Control of diabetes should be achieved before the time of conception to reduce the risk of congenital abnormalities linked to uncontrolled diabetes.

Use in Lactation

It is not known whether gliclazide or its metabolites are excreted in breast milk., Given the risk of neonatal hypoglycaemia, breastfeeding is contraindicated during treatment with this product. A risk to newborns/infants cannot be excluded.

4.7 EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

Patients should be made aware of the signs and symptoms of hypoglycaemia and should be careful if driving or operating machinery, especially at the beginning of treatment.

4.8 ADVERSE EFFECTS (UNDESIRABLE EFFECTS)

Good clinical acceptability of gliclazide, has been established in many studies as well as in medical practice.

The safety of gliclazide MR has been evaluated in controlled clinical trials in 955 patients, of which 728 patients were treated in long-term comparative trials, against an immediate release formulation of gliclazide 80 mg tablets, for up to 10 months. In these comparative trials, the overall incidence and type of adverse events were similar in both gliclazide MR and gliclazide 80 mg groups.

Adverse events were generally mild and transient, not requiring discontinuation of therapy.

However, where patients did discontinue due to adverse events, the percentage was lower in the gliclazide MR group (2.9%) than in the immediate release group (4.5%).

Serious reactions which have been reported with sulfonylureas are pancytopenia and gastrointestinal haemorrhage. (See also **Class Effects**, near the end of this section).

Hypoglycaemia (see 4.4 Special Warnings and Precautions for Use and 4.9 Overdose)

The most frequent adverse reaction with gliclazide is hypoglycaemia.

As is the case with all sulfonylurea drugs, hypoglycaemic reactions have been reported following gliclazide administration. However, a number of studies have shown that hypoglycaemia is less common with gliclazide than with glibenclamide.

Possible symptoms of hypoglycaemia are: headache, intense hunger, nausea, vomiting, lassitude, sleep disorders, agitation, aggression, poor concentration, reduced awareness and slowed reactions, depression, confusion, visual and speech disorders, aphasia, tremor, paresis, sensory disorders, dizziness, feeling of powerlessness, loss of self-control, delirium, convulsions, shallow respiration, bradycardia, drowsiness and loss of consciousness, possibly resulting in coma and/or death.

In addition, signs of adrenergic counter-regulation may be observed: sweating, clammy skin, anxiety, tachycardia, hypertension, palpitations, angina pectoris and cardiac arrhythmia.

Usually, symptoms disappear after intake of carbohydrate such as sugar (artificial sweeteners have no effect).

Experience with other sulphonylureas shows that hypoglycaemia can recur even when these measures are initially effective. If a hypoglycaemic episode is severe or prolonged, and even if it is temporarily controlled by intake of sugar, immediate medical treatment or even hospitalisation is required.

In long-term comparative studies, the percentage of patients experiencing hypoglycaemic episodes was similar between patients treated with gliclazide MR (11.6%) and those treated with the immediate release formulation of gliclazide (11.1%). However, the number of hypoglycaemic episodes per 100 patient months was lower in the gliclazide MR group (3.5) than in the immediate release group (4.8).

Analysis of elderly patients (over 65 years old) showed less hypoglycaemia than in the general population, with a prevalence of hypoglycaemic episodes lower in the gliclazide MR group (2.6 hypoglycaemic episodes for 100 patient months) than in the immediate release group (4.1).

The percentage of patients experiencing hypoglycaemic episodes in the sub-population with renal failure was similar to that observed in the general population.

Other Adverse Events

Adverse events reported during controlled clinical trials with gliclazide MR were those expected in an ageing population with diabetes.

Adverse events that were reported in at least 2.0% of patients, in long-term controlled clinical studies, are presented in the following table. The most frequent adverse events were not specifically related to the disease (such as respiratory infections or back pain).

Treatment emergent adverse events* (listed by body system) occurring in ≥ 2.0% of patients in long-term controlled clinical trials

	Gliclazide MR (n=728) %	Gliclazide 80 mg (n=734) %
Resistance mechanism		
Viral infection	7.7	5.6
Respiratory		
Rhinitis	4.4	4.6
Bronchitis	4.4	4.6
Pharyngitis	4.3	3.5
Upper respiratory infection	3.3	3.7
Coughing	2.1	2.0
Musculo-skeletal		
Back pain	5.2	4.1
Arthralgia	3.0	3.5
Arthrosis	2.2	2.2
Secondary term		
Inflicted injury	4.3	4.5
Body as a whole		
Headache	3.8	4.6
Asthenia	2.2	2.6
Cardiovascular		
Hypertension	3.2	3.7
Angina pectoris	2.1	2.2
Urinary		
Urinary tract infections	2.6	3.0
Gastrointestinal		
Diarrhoea	2.5	2.0
Central, peripheral, nervous system		
Dizziness	2.2	2.3
Metabolism & nutrition		
Hyperglycaemia	1.9	2.2

* whatever the relationship to treatment

Analysis of adverse events in sub-populations showed a similar pattern to that seen in the general population. Gender, age and renal impairment had no significant influence on the safety profile of gliclazide MR.

Other adverse effects

Gastrointestinal disturbances (reported with gliclazide), including nausea, dyspepsia, diarrhoea, abdominal pain, vomiting and constipation may be avoided or minimised if gliclazide is taken with breakfast.

The following adverse effects have been rarely reported:

Skin and Subcutaneous Tissue Disorders:

Pruritus, urticaria, maculopapular rashes, rash, angioedema, erythema and bullous reactions (such as Stevens-Johnson Syndrome [SJS] and toxic epidermal necrolysis [TEN]) (as with other sulfur-containing medications) and exceptionally, drug rash with eosinophilia and systemic symptoms (DRESS).

Blood and Lymphatic System Disorders (as with other sulphonylurea medications):

Anaemia, leucopenia, thrombocytopenia and agranulocytosis. These are in general reversible upon discontinuation of medication.

Hepatobiliary Disorders:

Elevations of serum bilirubin and hepatic enzymes (AST, ALT, alkaline phosphatase) levels, and exceptionally, hepatitis (isolated reports). Treatment should be discontinued if cholestatic jaundice appears. These symptoms usually disappear after discontinuation of treatment.

Investigations:

Occasional elevations of serum creatinine, blood urea nitrogen

Eye Disorders:

Transient visual disturbances may occur due to changes in blood glucose levels, particularly on initiation of treatment. As with any glucose-lowering medication, transient visual disturbances may occur on initiation of treatment due to changes in blood glucose levels.

Class Effects

The following adverse events have been observed with sulfonylureas: cases of erythrocytopenia, agranulocytosis, haemolytic anaemia, pancytopenia and allergic vasculitis, hyponatremia, elevated liver enzyme levels and even impairment of liver function (e.g. with cholestasis and jaundice) and hepatitis, which regressed after withdrawal of the sulphonylurea or led to life-threatening liver failure in isolated cases.

Reporting suspected adverse reactions after registration of the medicinal product is important. It allows continued monitoring of the benefit-risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions at http://www.tga.gov.au/reporting-problems.

4.9 OVERDOSE

Symptoms

Overdose of sulphonylureas may cause hypoglycaemia.

Moderate symptoms of hypoglycaemia (without loss of consciousness or neurological signs), should be corrected by carbohydrate intake, dose adjustment and/or modification of diet.

Strict monitoring should be continued until the doctor is sure that the patient is out of danger.

Severe hypoglycaemic reactions are possible (with coma, convulsions or other neurological disorders) and must be treated as a medical emergency, requiring immediate hospitalisation.

Treatment

If hypoglycaemic coma is diagnosed or suspected, the patient should be given a rapid I.V. injection of 50 mL of concentrated glucose solution (20 to 30%). This should be followed by continuous infusion of a more dilute glucose solution (10%) at a rate necessary to maintain blood glucose levels above 5 mmol/L. It is recommended that patients should be monitored closely for a 48 hour period at least.

Plasma clearance of gliclazide may be prolonged in patients with hepatic disease. However, due to the strong binding of gliclazide to proteins, dialysis is not effective in these patients.

For information on the management of overdose, contact the Poison Information Centre on 131126 (Australia).

5 PHARMACOLOGICAL PROPERTIES

5.1 PHARMACODYNAMIC PROPERTIES

Mechanism of action

Gliclazide is an oral hypoglycaemic sulfonylurea which differs from other related compounds. It has an N-containing heterocyclic ring with an endocyclic bond. Gliclazide reduces blood glucose levels by stimulating insulin secretion from the beta-cells of the Islets of Langerhans. Gliclazide shows high affinity, strong selectivity and reversible binding to the β-cell K_{ATP} channels with a low affinity for cardiac and vascular K_{ATP} channels. Increased postprandial insulin and C-peptide secretion persists after two years of treatment.

In type II diabetes, gliclazide restores the first peak of insulin secretion in response to glucose and increases the second phase of insulin secretion. A significant increase in insulin release is seen in response to stimulation induced by a meal or glucose.

Gliclazide also has extra-pancreatic effects and haemovascular properties.

It has been shown to increase peripheral insulin sensitivity:

- In muscle, euglycaemic hyperinsulinaemic clamp studies with gliclazide have demonstrated significantly increased (35%) insulin mediated glucose uptake which may improve diabetes control. Gliclazide potentiates insulin action on muscle glycogen synthase. These effects are consistent with a post-transcriptional action of gliclazide on GLUT4 glucose transporters.

- Studies on glucose turnover have further shown that gliclazide decreases hepatic glucose production, leading to an improvement in fasting blood glucose levels.

Gliclazide has been shown in some studies to have actions independent of that on glucose levels. These haemovascular effects of gliclazide include:

- Partial inhibition of platelet aggregation and adhesion with a decrease in markers of platelet activation (beta thromboglobulin, thromboxane B2).

- Increased vascular endothelial fibrinolytic activity (increased tPA activity).

- Anti-oxidant properties, notably a reduction in plasma lipid peroxides and increased erythrocyte superoxide dismutase activity.

- Inhibition of the increased adhesiveness of type II diabetic patient's monocytes to endothelial cells in *vitro*.

The anti-oxidant, platelet inhibiting and fibrinolytic actions of gliclazide involve processes which have been implicated in the pathogenesis of vascular complications of type II diabetes.

There is no clinical evidence that the haemovascular effects of gliclazide are of therapeutic benefit in type II diabetes patients.

Clinical trials

No data available.

5.2 PHARMACOKINETIC PROPERTIES

Absorption

Hydration of the tablets induces formation of a gel to activate drug release.

Plasma levels increase progressively, resulting in a plateau-shaped curve from the sixth to the twelfth hour after administration. Intra-individual variability is low. Gliclazide is completely absorbed and food intake does not affect the rate or degree of absorption.

Distribution

Plasma protein binding is approximately 95%. The relationship between the dose administered and the area under the concentration curve as a function of time is linear for doses of gliclazide up to 90 mg/day. At the highest evaluated dose (135 mg/day), the AUC increases slightly more than proportionally to the dose.

Metabolism

Gliclazide is mainly metabolised in the liver, the products of which are extensively excreted in the urine.

Excretion

Less than 1% of unchanged drug is recovered in the urine. No active metabolites have been detected in plasma.

The clearance of gliclazide has been found to be slightly reduced as a function of age. This reduction, however, is not considered to be clinically significant.

The elimination half-life of gliclazide is approximately 16 hours.

No clinically significant modifications in the pharmacokinetic parameters have been observed in elderly patients.

5.3 PRECLINICAL SAFETY DATA

Genotoxicity

No data available

Carcinogenicity

No data available

6 PHARMACEUTICAL PARTICULARS

6.1 LIST OF EXCIPIENTS

Refer to section 2 and 3 – Qualitative and quantitative composition and pharmaceutical form.

6.2 INCOMPATIBILITIES

See Section 4.5-Interactions with other medicines and other forms of interactions.

6.3 SHELF LIFE

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

6.4 SPECIAL PRECAUTIONS FOR STORAGE

Store below 30°C. Protect from moisture.

6.5 NATURE AND CONTENTS OF CONTAINER

30 mg Tablets

Blister pack (PVC/PVDC/Aluminium silver foil) of 100 tablets.

AUST R 151303

Bottle (HDPE bottle/CR-III PP cap with LDPE foam liner) of 100 tablets.

AUST R 151307

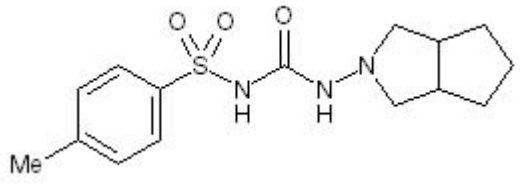
*Not all packs sizes and/or pack types may be available.

6.6 SPECIAL PRECAUTIONS FOR DISPOSAL

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

6.7 PHYSICOCHEMICAL PROPERTIES

Chemical structure



Chemical Name: 1-(3-azabicyclo[3.3.0]oct-3-yl)-3-*p*-tolylsulphonylurea.

Chemical Formula: $C_{15}H_{21}N_3O_3S$

Molecular Weight: 323.4

CAS number

21187-98-4

7 MEDICINE SCHEDULE (POISONS STANDARD)

S4 – Prescription Only Medicine

8 SPONSOR

Apotex Pty Ltd

16 Giffnock Avenue

Macquarie Park NSW 2113

Australia

Tel: +61 2 8877 8333

Web: www.apotex.com/au

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10 DATE OF REVISION

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Section Changed	Summary of new information
All	Reformatted product information
2, 3	Minor Editorial Changes