New Zealand Data Sheet
APO-CLOMIPRAMINE
Clomipramine hydrochloride 10mg and 25mg tablets

Presentation
APO-CLOMIPRAMINE 10mg tablets are triangular, pale yellow, film-coated biconvex tablets, and engraved “10” on one side. Each tablet typically weighs 31mg and contains 10mg Clomipramine hydrochloride.

APO-CLOMIPRAMINE 25mg tablets are round, pale yellow, film-coated biconvex tablets, and engraved “25” on one side. Each tablet typically weighs 78mg and contains 25mg Clomipramine hydrochloride.

Do not halve tablet. Dose equivalence when the tablet is divided has not been established.

Indications
- The treatment of depressive states (in adults only) including endogenous, reactive, neurotic, organic, masked and involutional forms of depression, depression associated with schizophrenia and personality disorders, depressive syndromes due to presenility or senility, chronic painful conditions, and chronic somatic diseases, and depressive mood disorders of a reactive, neurotic or psychopathic nature.
- Obsessive-compulsive syndromes
- Phobias and panic attacks.
- Cataplexy accompanying narcolepsy
- Chronic painful conditions

Dosage and Administration
The dosage and method of administration should be adapted to the individual patient's condition. The aim is to achieve an optimum effect while keeping the doses as low as possible and increasing them cautiously, particularly in elderly patients, who generally show a stronger response to clomipramine than patients of intermediate age groups.

Depression, Obsessive-Compulsive Syndromes, and Phobias
Start treatment with one coated tablet of 25mg 2-3 times daily. Raise the daily dosage stepwise, eg. 25mg every few days (depending on how the medication is tolerated) to 4 to 6 tablets of 25mg during the first week of treatment. In severe cases this dosage can be increased up to a maximum of 250mg daily. Once there is a distinct improvement, adjust the daily dosage to a maintenance level of about 2 to 4 coated tablets of 25mg.

Panic Attacks, Agoraphobia
Start with one tablet of 10mg daily, possibly in combination with a benzodiazepine. Depending on how the medication is tolerated, raise the dosage until the desired response is obtained, while gradually withdrawing the benzodiazepine. The daily dosage required varies greatly from patient to patient and lies between 25 and 100mg. If necessary it can be increased to 150mg. It is advisable for treatment not to be discontinued for at least 6 months and for the maintenance dose to be reduced slowly during this time.

Cataplexy Accompanying Narcolepsy
Daily dose of 25-75mg.
Chronic Painful Conditions
The dosage must be individualised (10 - 150mg daily), while taking account of concomitant analgesic medication (and of the possibility of reducing use of analgesics).

Elderly Patients
Start treatment with 1 tablet of 10mg daily. Gradually raise the dosage to an optimum level of 30 - 50mg daily, which should be reached after about 10 days and then maintained until the end of treatment.

Adolescent Depression
Not recommended for use in adolescent patients 13-18 years of age for the treatment of depression, unless under the supervision of a specialist.

Maximum Tolerated Daily Dose
Maximum daily dose is 250mg

Do not halve tablet. Dose equivalence when the tablet is divided has not been established.

Contraindications
- Clomipramine is contraindicated for the treatment of depression in patients 12 years of age and under.
- Clomipramine is contraindicated for the treatment of nocturnal enuresis.
- Hypersensitivity to clomipramine and any of the excipients, or cross-sensitivity to tricyclic antidepressants of the dibenzazepine group.
- Clomipramine should not be given in combination, or within 14 days before or after treatment, with a MAO inhibitor (see Interactions). The concomitant treatment with selective, reversible MAO-A inhibitors, such as moclobemide, is also contraindicated.
- Recent myocardial infarction.

Warnings and Precautions
Clinical Worsening and Suicide Risk
Patients of any age with Major Depressive Disorder may experience worsening of their depression and/or the emergence of suicidal ideation and behaviour (suicidality), whether or not they are taking antidepressant medications, and this risk may persist until significant remission occurs. Patients should be closely monitored, especially at the beginning of therapy or when the dose is changed, until such improvement occurs.

There has been a long-standing concern that some antidepressants may have a role in the emergence of suicidality in some patients. The possible risk of increased suicidality in patients applies to all classes of antidepressant medicines, as available data are not adequate to exclude this risk for any antidepressant. Therefore, consideration should be given to changing the therapeutic regimen, including possibly discontinuing the medication, in patients whose depression is persistently worse or whose emergent suicidality is severe, abrupt in onset, or was not part of the patient's presenting symptoms. Generally, when stopping an antidepressant, doses should be tapered rather than stopped abruptly.

The following symptoms, anxiety, agitation, panic attacks, insomnia, irritability, hostility (aggressiveness), impulsivity, akathisia (psychomotor restlessness), hypomania, and mania, have been reported in adult and paediatric patients being treated with antidepressants for major depressive disorder as well as for other indications, both psychiatric and non-psychiatric.
Although a causal link between the emergence of such symptoms and either the worsening of depression and/or the emergence of suicidal impulses has not been established, consideration should be given to changing the therapeutic regimen, including possibly discontinuing the medication, in patients for whom such symptoms are severe, abrupt in onset, or were not part of the patient's presenting symptoms.

Because of the possibility of co-morbidity between major depressive disorder and other psychiatric and non-psychiatric disorders, the same precautions observed when treating patients with major depressive disorder should be observed when treating patients with other psychiatric and non-psychiatric disorders.

**Mania and Bipolar Disorder**
A major depressive episode may be the initial presentation of bipolar disorder. It is generally believed (though not established in controlled trials) that treating such an episode with any antidepressant alone may increase the likelihood of a mixed/manic episode in patients at risk for bipolar disorder. Prior to initiating treatment with an antidepressant, patients should be adequately screened to determine if they are at risk for bipolar disorder. It should be noted that clomipramine is not approved for use in treating bipolar depression.

**Information for Patients and Families**
Patients and their families should be alerted about the need to monitor for the emergence of anxiety, agitation, panic attacks, insomnia, irritability, hostility, impulsivity, akathisia, hypomania, mania, worsening of depression, and suicidal ideation, especially early during antidepressant treatment. Such symptoms should be reported to the patient's doctor, especially if they are severe, abrupt in onset, or were not part of the patient's presenting symptoms.

The patient has the right to treatment meeting appropriate ethical and professional standards, and the patient needs to be fully informed with frank discussion of risk/benefit issues relating to the medicines efficacy and safety when used in the treatment regimen proposed.

Tricyclic antidepressants are known to lower the convulsion threshold and clomipramine should, therefore, be used with extreme caution in patients with epilepsy and other predisposing factors, e.g. brain damage of varying aetiology, concomitant use of neuroleptics, withdrawal from alcohol or medicines with anticonvulsive properties (e.g. benzodiazepines). It appears that the occurrence of seizures is dose dependent. Therefore, the recommended total daily dose of clomipramine should not be exceeded.

**Cardiovascular disorders**
Clomipramine should be administered with particular caution in patients with cardiovascular disorders, especially those with cardiovascular insufficiency, conduction disorders (e.g. atrioventricular block grades I to III), or arrhythmias. Monitoring of cardiac function and the ECG is indicated in such patients, as well as in elderly patients. An ECG should be performed prior to starting treatment, at steady state, after an increase in dose or after starting any potentially interacting medicine.

Tricyclic Antidepressant medicines, including clomipramine, particularly when given in high doses, have been reported to product QTc prolongation, arrhythmias (including torsades de pointes (TdP), sinus tachycardia and prolongation of the conduction time). Myocardial Infarction and stroke have been reported with medicines of this class. (see Adverse Effects).

Clomipramine should be used with caution in patients with risk factors for QTc prolongation/TdP including congenital long QT syndrome, age >65 years, female sex, structural heart disease/LV
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dysfunction, medical conditions such as renal or hepatic disease, use of medicines that inhibit the metabolism of clomipramine and the concomitant use of other QTC prolonging medicines (see Interactions). Hypokalaemia and hypomagnesaemia should be corrected prior to treatment.

Consideration should be given to stopping clomipramine treatment or reducing the dose if the QTC interval is >500ms or increases by >60ms.

Because of its anticholinergic properties, clomipramine should be used with caution in patients with a history of increased intraocular pressure, narrow-angle glaucoma, or urinary retention (eg. diseases of the prostate).

Caution is called for when giving tricyclic antidepressants to patients with severe hepatic disease and tumours of the adrenal medulla (eg. phaeochromocytoma, neuroblastoma), in whom they may provoke hypertensive crises.

Many patients with panic disorder experience more marked anxiety at the start of the treatment with clomipramine (see Dosage and Administration). This paradoxical initial increase in anxiety is most pronounced during the first few days of treatment and generally subsides within two weeks.

Before starting treatment with clomipramine it is advisable to check blood pressure, because patients with postural hypotension or a labile circulation may experience a fall in blood pressure.

Caution is indicated in patients with hyperthyroidism or patients receiving thyroid preparations, owing to the possibility of cardiac toxicity.

In patients with liver disease, periodic monitoring of hepatic enzyme levels is recommended.

Although changes in the white blood cell count have been reported with clomipramine only in isolated cases, periodic blood cell counts and monitoring for symptoms such as fever and sore throat are called for, particularly during the first few months of therapy and during prolonged treatment. Like related tricyclic antidepressants, clomipramine should be given with electroconvulsive therapy only under careful supervision.

In predisposed and elderly patients, tricyclic antidepressants may provoke pharmacogenic (delirious) psychoses, particularly at night. These disappear within a few days of withdrawing clomipramine.

Clomipramine has been reported to be associated with fewer deaths following overdose than other tricyclic antidepressants.

Caution is called for in patients with chronic constipation. Tricyclic antidepressants may cause paralytic ileus, particularly in elderly and in bedridden patients.

Before general or local anaesthesia, the anaesthetist should be told that the patient has been receiving clomipramine (see Interactions).

An increase in dental caries has been reported during long-term treatment with tricyclic antidepressants. Regular dental check-ups are therefore advisable during long-term treatment.

Decreased lacrimation and accumulation of mucoid secretions due to the anticholinergic properties of tricyclic antidepressants may cause damage to the corneal epithelium in patients with contact lenses.
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Abrupt withdrawal should be avoided because of possible adverse reactions (see Adverse Effects).

Use in Pregnancy
Category C
Experience with clomipramine in pregnancy is limited. Since there have been isolated reports of a possible connection between the use of tricyclic antidepressants and adverse effects (developmental disorders) on the foetus, treatment with clomipramine should be avoided during pregnancy, unless the anticipated benefits justify the potential risk to the foetus.

Neonates whose mothers had taken tricyclic antidepressants until delivery showed withdrawal symptoms, such as dyspnoea, lethargy, colic, irritability, hypotension or hypertension, and tremor or spasms, during the first few hours or days. To avoid such symptoms, clomipramine should if possible be gradually withdrawn at least 7 weeks before the calculated date of confinement.

Use in Lactation
Since the active substance passes into the breast milk, clomipramine should be gradually withdrawn or the infant weaned if the patient is breastfeeding.

Effects on ability to drive and use machines
Likely to produce severe adverse effects or presumed to be potentially dangerous on the ability to drive or use machinery.

Patients receiving clomipramine should be warned that blurred vision, drowsiness and other CNS symptoms (see Adverse Effects) may occur, in which case they should not drive, operate machinery, or do anything else requiring alertness. Patients should also be warned that alcohol or other medicines may potentiate these effects (see Interactions).

Other
Mutagenicity, Carcinogenicity and Reproduction Toxicity Studies
According to the experimental data available, clomipramine has no mutagenic, carcinogenic, or teratogenic effects.

Adverse Effects
Unwanted effects are usually mild and transient, disappearing under continued treatment or with a reduction in the dosage. They do not always correlate with plasma levels or dose. It is often difficult to distinguish certain undesirable effects from symptoms of depression such as fatigue, sleep disturbances, agitation, anxiety, constipation, and dry mouth.

If severe neurological or psychiatric reactions occur, clomipramine should be withdrawn.

Elderly patients are particularly sensitive to anticholinergic, neurological, psychiatric, or cardiovascular effects. Their ability to metabolise and eliminate medicines may be reduced, leading to a risk of elevated plasma concentrations at therapeutic doses.

Frequency of Adverse Effects
Estimates from clinical trials and spontaneous ADR reports, classified as follows:
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<table>
<thead>
<tr>
<th>Classification</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequent</td>
<td>&gt; 10%</td>
</tr>
<tr>
<td>Occasional</td>
<td>&gt; 1% to 10%</td>
</tr>
<tr>
<td>Rare</td>
<td>&gt; 0.001% to 1%</td>
</tr>
<tr>
<td>Isolated cases</td>
<td>&lt; 0.001%</td>
</tr>
</tbody>
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Central Nervous System
Psychic Effects
*Frequent*: drowsiness, fatigue, restlessness, increased appetite.
*Occasional*: confusion, disorientation, hallucinations (particularly in elderly patients and patients with Parkinson's disease), anxiety states, agitation, sleep disturbances, mania, hypomania, aggressiveness, impaired memory, de-personalisation, aggravated depression, impaired concentration, insomnia, nightmares, yawning.
*Rare*: activation of psychotic symptoms.

Neurological Effects
*Frequent*: dizziness, tremor, headache, myoclonus.
*Occasional*: delirium, speech disorders, paraesthesias, muscle weakness, muscle hypertonia.
*Rare*: convulsions, ataxia.
*Isolated cases*: EEG changes, hyperpyrexia.

Anticholinergic Effects
*Frequent*: dry mouth, sweating, constipation, disorders of visual accommodation, blurred vision, disturbances of micturition.
*Occasional*: hot flushes, mydriasis.
*Isolated cases*: glaucoma.

Cardiovascular System
*Occasional*: tachycardia, palpitations, hypotension, syncope, myocardial infarction, stroke, and ECG changes (including QTc prolongation, non-specific ST and T wave changes and conduction disorders such as heart block, bundle branch block and widened QRS complex) in patients of normal cardiac status.
*Rare*: arrhythmias (including ventricular tachycardia, ventricular fibrillation and Torsades de Pointes), hypertension.
*Isolated cases*: conduction disorders (e.g. widening of QRS complex, PQ changes, bundle-branch block).

Gastrointestinal Tract
*Frequent*: nausea.
*Occasional*: vomiting, abdominal disorders, diarrhoea, anorexia.
*Isolated cases*: gastrointestinal haemorrhage.

Liver
*Occasional*: elevated transaminases.
*Isolated cases*: hepatitis with or without jaundice.

Skin
*Occasional*: allergic skin reactions (skin rash, urticaria), photosensitivity, pruritus.
*Isolated cases*: oedema (local or generalised), hair loss.

Please refer to Medsafe website ([www.medsafe.govt.nz](http://www.medsafe.govt.nz)) for the most recent datasheet
**Endocrine System and Metabolism**

*Frequent:* weight gain, disturbances of libido and potency.
*Occasional:* galactorrhoea, breast enlargement.
*Isolated cases:* SIADH (inappropriate antidiuretic hormone secretion syndrome).

**Hypersensitivity**

*Isolated cases:* allergic alveolitis (pneumonitis) with or without eosinophilia, systemic anaphylactic/anaphylactoid reactions including hypotension.

**Blood**

*Isolated cases:* leucopenia, agranulocytosis, thrombocytopenia, eosinophilia, purpura.

**Sense Organs**

*Occasional:* taste disturbances, tinnitus.

**Others**

The following symptoms occasionally occur after abrupt withdrawal or reduction of the dose: nausea, vomiting, abdominal pain, diarrhoea, insomnia, headache, nervousness, and anxiety.

**Post-marketing Experience**

See Adverse Effects

**Interactions**

**Pharmacokinetics Interactions**

**MAO Inhibitors**

Do not give clomipramine for at least 2 weeks after discontinuation of treatment with MAO inhibitors (there is a risk of severe symptoms such as hypertensive crisis, hyperpyrexia, myoclonus, agitation seizures, delirium and coma). The same applies when giving a MAO inhibitor after previous treatment with clomipramine. In both instances clomipramine or the MAO inhibitor should initially be given in small, gradually increasing doses and its effects monitored.

There is evidence to suggest that clomipramine may be given as little as 24 hours after a reversible MAO-A inhibitor such as moclobemide, but the two-week washout period must be observed if the MAO-A inhibitor is given after clomipramine has been used.

**Adrenergic Neurone Blockers**

Clomipramine may diminish or abolish the antihypertensive effects of guanethidine, betanidine, reserpine, clonidine and alpha-methyldopa. Patients requiring co-medication for hypertension should therefore be given antihypertensives of a different type (eg. diuretics, vasodilators, or beta-blockers).

**Sympathomimetic Agents**

Clomipramine may potentiate the cardiovascular effects of adrenaline, noradrenaline, isoprenaline, ephedrine and phenylephrine (eg. local anaesthetics).

**Medicines that can prolong the QT interval**

The risk of QTc prolongation and/or ventricular arrhythmias (e.g. torsades de pointes) is increased with concomitant use of other medicines which prolong the QTc interval (e.g. some
antipsychotics and antibiotics). Please check the data sheet of other medicines administered for information on their effects on the QTc interval.

**CNS Depressants**
Tricyclic antidepressants may potentiate the effects of alcohol and other central depressant substances (e.g. barbiturates, benzodiazepines, or general anaesthetics).

**Anticholinergic Agents**
Tricyclic antidepressants may potentiate the effects of these medicines (e.g. phenothiazine, antiparkinsonian agents, antihistamines, atropine biperiden) on the eye, central nervous system, bowel and bladder.

**Quinidine**
Tricyclic antidepressants should not be employed in combination with antiarrhythmic agents of the quinidine type.

**Selective Serotonin Reuptake Inhibitors (SSRI)**
Co-medication may lead to additive effects on the serotonergic system. Fluoxetine and fluvoxamine may also increase plasma concentrations of clomipramine, with corresponding adverse effects.

**Liver-Enzyme Inducers**
Agents which activate the hepatic mono-oxygenase enzyme system (e.g. barbiturates, carbamazepine, phenytoin, nicotine, and oral contraceptives) may accelerate the metabolism and lower the plasma concentrations of clomipramine, resulting in decreased efficacy. Plasma levels of phenytoin and carbamazepine may increase, with corresponding adverse effects. It may be necessary to adjust the dosage of these medicines.

**Neuroleptics**
Co-medication may result in increased plasma levels of tricyclic antidepressants, a lowered convulsion threshold, and seizures. Combination with thioridazine may produce severe cardiac arrhythmias.

**Anticoagulants**
Tricyclic antidepressants may potentiate the anticoagulant effect of coumarin agents due to their inhibition of hepatic metabolism. Careful monitoring of plasma prothrombin is therefore advised.

**Cimetidine, Methylphenidate, Oestrogens**
These medicines increase plasma concentrations of tricyclic antidepressants, whose dosage should therefore be reduced.

**Pharmacodynamic Interactions**
N/A

**Overdosage**
The signs and symptoms of overdose with clomipramine are similar to those reported with other tricyclic antidepressants. Cardiac abnormalities and neurological disturbances are the main complications. In children, accidental ingestion of any amount should be regarded as serious and potentially fatal.
Signs and Symptoms
Symptoms generally appear within 4 hours of ingestion and reach maximum severity after 24 hours. Owing to delayed absorption (anticholinergic effect), long half-life, and enterohepatic recycling of the clomipramine, the patient may be at risk for up to 4 - 6 days.

The following signs and symptoms may be seen:
- Central Nervous System: drowsiness, stupor, coma, ataxia, restlessness, agitation, enhanced reflexes, muscular rigidity and choreoathetoid movements, convulsions.
- Cardiovascular System: hypotension, tachycardia, QTc prolongation, arrhythmias (including Torsades de pointes), conduction disorders, shock, heart failure; in very rare cases cardiac arrest.
- Respiratory depression, cyanosis, vomiting, fever, mydriasis, sweating, and oliguria or anuria may also occur.

Treatment
There is no specific antidote, and treatment is essentially symptomatic and supportive.

Anyone suspected of receiving an overdose of clomipramine, particularly children, should be hospitalised and kept under close surveillance for at least 72 hours.

Perform gastric lavage or induce vomiting as soon as possible if the patient is alert. If the patient is not alert, secure the airway with a cuffed endotracheal tube before beginning lavage, and do not induce vomiting. These measures are recommended for up to 12 hours or even longer after the overdose, since the anticholinergic effect of the drug may delay gastric emptying. Administration of activated charcoal may help to reduce drug absorption.

Treatment of symptoms is based on modern methods of intensive care, with continuous monitoring of cardiac function, blood gases, and electrolytes, and if necessary emergency measures such as anticonvulsive therapy, artificial respiration, and resuscitation. Since it has been reported that physostigmine may cause severe bradycardia, asystole, and seizures, its use is not recommended in cases of overdosage with clomipramine. Haemodialysis or peritoneal dialysis are ineffective because of the low plasma concentrations of clomipramine.

Further Information
Actions
Pharmacotherapeutic Group
Tricyclic antidepressant. Noradrenaline and preferential serotonin-reuptake inhibitor.

The therapeutic activity of clomipramine is believed to be based on its ability to inhibit the neuronal re-uptake of noradrenaline (NA) and serotonin (5-HT) released in the synaptic cleft, with inhibition of 5-HT re-uptake being the more important of these activities.

Clomipramine also has a wide pharmacological spectrum of action, which includes alpha₁-adrenolytic, anticholinergic, antihistaminic, and anti-serotoninergic (5-HT-receptor blocking) properties.
Clomipramine acts on the depressive syndrome as a whole, including in particular typical features such as psychomotor retardation, depressed mood, and anxiety. The clinical response usually sets in after 2-3 weeks of treatment.

Clomipramine also exerts a specific effect on obsessive-compulsive disorder distinct from its antidepressant effects. In chronic pain with or without somatic causes, clomipramine acts presumably by facilitating serotonin and noradrenaline neurotransmission.

**Pharmacokinetics**

Clomipramine is completely absorbed from the gastrointestinal tract. The systemic bioavailability of unchanged clomipramine is reduced to about 50% by hepatic first-pass metabolism due to desmethylclomipramine. The bioavailability of clomipramine is not markedly affected by the ingestion of food. Only the onset of absorption may be slightly delayed and therefore time to peak prolonged.

During oral administration of constant daily doses of clomipramine, the steady-state plasma concentrations of clomipramine show a high variability between patients. The dose of 75mg daily, administered as coated tablets of 25mg t.i.d. produces steady-state plasma concentrations ranging from about 20 to 175 ng/mL.

The steady-state plasma concentrations of the active metabolite, desmethylclomipramine, follow a similar pattern.

However, at a dose of 75mg clomipramine per day, they are 40 - 85% higher than those of clomipramine.

Clomipramine is 97.6% bound to plasma proteins. The apparent distribution volume is about 12 to 17 L/kg body weight. Concentrations in cerebrospinal fluid are about 2% of the plasma concentration. Clomipramine passes into maternal milk in concentrations similar to those in plasma.

The major route of biotransformation of clomipramine is demethylation to desmethylclomipramine. In addition, clomipramine and desmethylclomipramine are hydroxylated to 8-hydroxyclomipramine and 8-hydroxy-desmethylclomipramine, but little is known about their activity in vivo. The hydroxylation of clomipramine and desmethylclomipramine is under genetic control similar to that of debrisoquine. In poor metabolisers of debrisoquine this may lead to high concentrations of desmethylclomipramine, whereas those of clomipramine are less influenced.

Clomipramine is eliminated from the blood with a mean half-life of 21h (range: 12-36h), and desmethylclomipramine with a mean half-life of 36h.

About two thirds of a single dose of clomipramine are excreted in the form of water-soluble conjugates in the urine and approximately one third in the faeces. The quantity of unchanged clomipramine and desmethylclomipramine excreted in the urine is about 2% and 0.5% of the dose administered, respectively.

In elderly patients, owing to reduced metabolic clearance, plasma clomipramine concentrations at any given dose are higher than in younger patients. The effects of hepatic and renal impairment on the pharmacokinetics of clomipramine have not been determined.
Other

Chemical Structure

![Chemical Structure Image]

List of excipients
Microcrystalline Cellulose, Lactose Monohydrate, Magnesium stearate, colloidal Silicon Dioxide, Croscarmellose Sodium, Hydroxypropyl Methylcellulose (Hypromellose), Polyethylene Glycol 3350 (Carbowax/Macrogol 3350), Titanium Dioxide, Carnauba Wax and Yellow Ferric Oxide

Tablets are gluten free, contain lactose and yellow ferric oxide as the colourant.

Pharmaceutical Precautions

Instructions for Handling
Nil

Incompatibilities
See Interactions

Shelf-Life
Shelf life:
10mg: 3 years from the date of manufacture.
25mg: 2 years from the date of manufacture.

Special Precautions for Storage
Store at or below 25°C
Protect from heat light and moisture.
Keep out of reach of children

Package Quantities
APO-CLOMIPRAMINE 10mg and 25mg: Blisters containing 100 tablets.

Medicine Schedule
Prescription Medicine
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Sponsor Details
Apotex NZ Ltd
32 Hillside Road
Glenfield
Private Bag 102-995
North Shore Mail Centre
Auckland
Telephone: (09) 444 2073
Fax: (09) 444 2951

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