

# Data Sheet

## Madopar<sup>®</sup>

*Levodopa + benserazide capsules 62.5, 125 and 250; dispersible tablet 62.5; HBS capsule 125*

For the treatment of Parkinson's disease

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## Description

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### **Composition**

#### **Active ingredient**

Madopar is a combination of levodopa and the decarboxylase inhibitor benserazide (as hydrochloride) in a ratio of 4:1.

The preparation is available as capsules of three different strengths, as single scored dispersible tablets of one strength and as capsules with a controlled release action.

#### **Excipients**

##### ***Madopar 62.5, 125 and 250 capsules***

All three forms of the capsules contain microcrystalline cellulose, talc, povidone, magnesium stearate, gelatin, and the colourant(s) indigo carmine, titanium dioxide and iron oxide. The Madopar 62.5 capsules also contain mannitol.

##### ***Madopar HBS capsules***

Hypromellose, hydrogenated vegetable oil, calcium hydrogen phosphate, mannitol, povidone, talc, magnesium stearate, gelatin and the colourant(s) indigo carmine, titanium dioxide and iron oxide .

##### ***Madopar 62.5 Dispersible tablets***

The dispersible tablets contain citric acid, maize starch, microcrystalline cellulose and magnesium stearate.

## ***Type of Dosage Form***

### **Standard forms**

Madopar 62.5 capsule

A No. 4 size capsule with a "ROCHE" imprint, an opaque light-grey body and an opaque powder-blue cap. Each capsule contains: 50 mg levodopa and 14.25 mg benserazide hydrochloride (equivalent to 12.5 mg of the base).

#### Madopar 125 capsule

A No. 2 size capsule with a "ROCHE" imprint, an opaque flesh coloured body and an opaque powder-blue cap. Each capsule contains: 100 mg levodopa and 28.5 mg benserazide hydrochloride (equivalent to 25 mg of the base).

#### Madopar 250 Capsule

A No. 1 size capsule with a "ROCHE" imprint, an opaque caramel coloured body and an opaque powder-blue cap. Each capsule contains: 200 mg levodopa and 57 mg benserazide hydrochloride (equivalent to 50 mg of the base).

### Dispersible form

#### Madopar 62.5 dispersible tablet

A white, cylindrical, bi planar tablet with "ROCHE" and "62.5" imprinted on one side and a breakbar on the other side. Each tablet contains: 50 mg levodopa and 14.25 mg benserazide hydrochloride (equivalent to 12.5 mg of the base).

### Controlled release form

#### Madopar HBS (Hydrodynamically Balanced System with controlled release)

A No. 1 size capsule with a "ROCHE" imprint, an opaque light blue body and an opaque dark green cap. Each capsule contains: 100 mg levodopa and 28.5 mg benserazide hydrochloride (equivalent to 25 mg of the base).

Madopar HBS capsules must not be opened before ingestion because the controlled-release characteristics will be lost.

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## Clinical Particulars

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### *Therapeutic Indications*

Madopar is indicated for the treatment of all forms of Parkinson's syndrome with the exception of medicine-induced parkinsonism.

Madopar dispersible is a formulation which is suitable for patients with dysphagia (difficulties in swallowing) or who require a formulation with a more rapid onset of action, e.g. patients suffering from early morning and afternoon akinesia, or who exhibit "delayed on" or "wearing off" phenomena.

Madopar HBS is indicated for patients presenting with all types of fluctuations in response, especially those related to fluctuations in plasma levels (i.e. "peak dose dyskinesia" and "end of dose deterioration") and for better control of nocturnal symptoms.

Further experience is required to determine whether it is also advantageous to use Madopar HBS in new Parkinson patients.

## ***Dosage and Administration***

### **Method of administration**

When taking standard Madopar capsules or Madopar HBS, patients must always ensure that they swallow the whole capsule without chewing it.

Madopar dispersible tablets are to be dispersed in a quarter of a glass of water (approx. 25-50 ml). The tablets disintegrate completely, producing a milky-white dispersion within a few minutes. Because of rapid sedimentation, it is advisable to stir the dispersion before drinking. Madopar dispersible tablets should be taken within half an hour of preparing the dispersion.

Madopar should be taken at least 30 minutes before or 1 hour after meals, whenever possible. Undesirable gastrointestinal effects, which may occur mainly in the early stages of the treatment, can largely be controlled by taking Madopar with a small snack (e.g. biscuits) or liquid or by increasing the dose slowly.

### **Standard dosage**

Treatment with Madopar should be introduced gradually; dosage should be assessed individually and titrated for optimal effect. The following dosage instructions should therefore be regarded as guidelines.

### **Initial therapy**

In the early stages of Parkinson's disease it is advisable to start treatment with one capsule of Madopar 62.5 three to four times daily. As soon as tolerability of the initial dosing schedule is confirmed, the dosage should be increased slowly in accordance with the patient's response.

An optimal effect is generally achieved with a daily dosage of Madopar corresponding to 300 - 800 mg of levodopa + 75 - 200 mg benserazide, to be divided into 3 or more doses. Between 4 and 6 weeks may be needed to achieve the optimal effect. If it proves necessary to further increase the daily dosage, this should be done on a monthly basis.

### **Maintenance therapy**

The average maintenance dosage is 1 capsule of Madopar 125 three to six times daily. The number of individual doses (not less than 3) and their distribution throughout the day must be titrated for optimal effect. Madopar HBS and Madopar dispersible may substitute standard Madopar to achieve an optimal effect.

### **Special dosage instructions**

Dosage must be carefully titrated in all patients (see Therapeutic Indications). Patients on other anti-parkinsonian agents may receive Madopar. However, as treatment with Madopar proceeds and the therapeutic effect becomes apparent, the dosage of the other medication may need to be reduced or these medicines gradually withdrawn.

Madopar dispersible tablets are particularly suitable for patients with dysphagia (difficulties in swallowing) or in situations where a more rapid onset of action is required, e.g. in patients suffering from early morning and afternoon akinesia, or who exhibit "delayed on" or "wearing off" phenomena.

Patients who experience large fluctuations in the medicine's effect in the course of the day (on-off phenomena) should receive smaller, more frequent single doses or be switched to Madopar HBS.

The switch from standard Madopar to Madopar HBS is preferably made from one day to the next, beginning with the morning dose. The daily dose and dosing interval should initially be the same as with standard Madopar.

After 2 - 3 days, the dosage should be gradually increased by about 50%. Patients should be informed that their condition may temporarily deteriorate.

Due to the pharmacokinetic properties of Madopar HBS, the onset of action is delayed. The clinical effect may be achieved more rapidly by administering Madopar HBS together with standard Madopar or Madopar dispersible. This may prove especially useful for the first morning dose, which should preferably be higher than the subsequent daily doses. The individual titration for Madopar HBS must be carried out slowly and carefully, allowing intervals of at least 2 - 3 days between dose changes.

In patients with nocturnal immobility, positive effects have been reported after gradually increasing the last evening dose to 250 mg of Madopar HBS on retiring.

Excessive responses to Madopar HBS (dyskinesia) can be controlled by increasing the interval between doses rather than reducing the single doses.

Treatment with standard Madopar or Madopar dispersible should be resumed if the response to Madopar HBS is inadequate.

Patients should be carefully observed for possible undesirable psychiatric symptoms.

## ***Contraindications***

Madopar must not be given to patients with known hypersensitivity to levodopa or benserazide.

Madopar must not be given in conjunction with non-selective monoamine oxidase (MAO) inhibitors. However, selective MAO-B inhibitors, such as selegiline and rasagiline, or selective MAO-A inhibitors, such as moclobemide, are not contraindicated. Combination of MAO-A and MAO-B inhibitors is equivalent to non-selective MAO inhibition, and hence this combination should not be given concomitantly with Madopar (see Interactions with other Medicinal Products and other Forms of Interaction).

Madopar must not be given to patients with decompensated endocrine, renal or hepatic function, cardiac disorders, psychiatric diseases with a psychotic component or closed angle glaucoma.

Madopar must not be given to patients less than 30 years old (skeletal development must be complete).

Madopar must not be given to pregnant women or to women of childbearing potential in the absence of adequate contraception (see Pregnancy and Nursing mothers). If pregnancy occurs in a woman taking Madopar, the medicine must be discontinued (as advised by the prescribing physician).

## ***Warnings and Precautions***

### **General**

Hypersensitivity reactions may occur in susceptible individuals.

Regular measurement of intraocular pressure is advisable in patients with open-angle glaucoma, as levodopa theoretically has the potential to raise intraocular pressure.

Depression can be part of the clinical picture in patients with Parkinson's disease and may also occur in patients treated with Madopar.

If a patient on levodopa requires a general anaesthetic, the normal Madopar regimen should be continued as close to the surgery as possible, except in the case of halothane. In general anaesthesia with halothane Madopar should be discontinued 12 - 48 hours before surgical intervention as fluctuations in blood pressure and/or arrhythmias may occur in patients on Madopar therapy. Madopar therapy may be resumed following surgery; the dosage should be increased gradually to the preoperative level.

Madopar must not be withdrawn abruptly. Abrupt withdrawal of the preparation may result in a neuroleptic malignant-like syndrome (hyperpyrexia and muscular rigidity, possibly psychological changes and elevated serum creatinine phosphokinase) which may be life-threatening. Should a combination of such symptoms and signs occur, the patient should be kept under medical surveillance, if necessary, hospitalised and rapid and appropriate symptomatic treatment given. This may include resumption of Madopar therapy after an appropriate evaluation.

Levodopa has been associated with somnolence and episodes of sudden sleep onset. Sudden onset of sleep during daily activities, in some cases without awareness or warning signs, has been reported very rarely. Patients must be informed of this and advised to exercise caution while driving or operating machines during treatment with levodopa. Patients who have experienced somnolence and/or an episode of sudden sleep onset must refrain from driving or operating machines. Furthermore a reduction of dosage or termination of therapy may be considered (see Ability to drive and use machines).

#### *Dopaminergic medicines*

Pathological gambling, increased libido and hypersexuality have been reported in patients treated with dopamine agonists for Parkinson's disease. There is no established causal relationship between Madopar, which is not a dopamine agonist, and these events. However, caution is advised as Madopar is a dopaminergic medicine. Health care professionals should inform patients to seek help from their physician if they, their family or their carer notice that their behaviour is unusual.

### **Potential for medicine dependence or abuse**

A small sub-group of Parkinson's disease patients suffer from cognitive and behavioural disturbance that can be directly attributed to taking increasing quantities of medication against medical advice and well beyond the doses required to treat their motor disabilities.

### **Ability to drive and use machines**

Patients being treated with levodopa and presenting with somnolence and/or sudden sleep episodes must be informed to refrain from driving or engaging in activities where impaired alertness may put

themselves or others at risk of serious injury or death (e.g. operating machines) until such recurrent episodes and somnolence have resolved (see Warnings and Precautions - General).

### **Laboratory tests**

Checks of liver function and blood count should be performed during treatment.

Patients with diabetes should undergo frequent blood sugar tests, and the dosage of anti-diabetic agents should be adjusted to blood sugar levels.

## ***Interactions with other Medicinal Products and other Forms of Interaction***

### **Pharmacokinetic interactions**

Co-administration of the anticholinergic agent trihexyphenidyl with standard Madopar reduces the rate, but not the extent, of levodopa absorption. Trihexyphenidyl given concomitantly with Madopar HBS does not affect the pharmacokinetics of levodopa.

Co-administration of antacids with Madopar HBS reduces the extent of levodopa absorption by 32%.

Ferrous sulphate decreases the maximum plasma concentration and the AUC of levodopa by 30-50%. The pharmacokinetic changes observed during co-treatment with ferrous sulphate appear to be clinically significant in some but not all patients.

Metoclopramide increases the rate of levodopa absorption.

There are no pharmacokinetic interactions between levodopa and the following compounds: bromocriptine, amantadine, selegiline and domperidone.

### **Pharmacodynamic interactions**

Neuroleptics, opioids and antihypertensive medications containing reserpine inhibit the action of Madopar.

If Madopar is to be administered to patients receiving irreversible non-selective MAO inhibitors, an interval of at least 2 weeks should be allowed between cessation of the MAO inhibitor and the start of Madopar therapy. Otherwise unwanted effects such as hypertensive crises are likely to occur (see Contraindications). Selective MAO-B inhibitors, such as selegiline and rasagiline and selective MAO-A inhibitors, such as moclobemide, can be prescribed to patients on Madopar therapy; it is recommended to readjust the levodopa dose to the individual patient's needs, in terms of both efficacy and tolerability. Combination of MAO-A and MAO-B inhibitors is equivalent to non-selective MAO inhibition, and hence this combination should not be given concomitantly with Madopar (see Contraindications).

Madopar should not be administered concomitantly with sympathomimetics (agents such as epinephrine, norepinephrine, isoproterenol or amphetamine which stimulate the sympathetic nervous system) as levodopa may potentiate their effects. Should concomitant administration prove necessary, close surveillance of the cardiovascular system is essential, and the dose of the sympathomimetic agents may need to be reduced.

Combination with other agents such as anticholinergics, amantadine and dopamine agonists is permissible, though both the desired and the undesired effects of treatment may be intensified. It may be necessary to reduce the dosage of Madopar or the other substance. When initiating an adjuvant treatment with a COMT inhibitor, a reduction of the dosage of Madopar may be necessary. Anticholinergics should not be withdrawn abruptly when Madopar therapy is instituted, as levodopa does not begin to take effect for some time.

Levodopa may affect the results of laboratory tests for catecholamines, creatinine, uric acid and glucose.

Coombs' tests may give a false-positive result in patients taking Madopar.

A diminution of effect is observed when the medicine is taken with a protein-rich meal.

*General anaesthesia with halothane:* Madopar should be discontinued 12-48 hours before surgical intervention requiring general anaesthesia with halothane as fluctuations in blood pressure and/or arrhythmias may occur.

For general anaesthesia with other anaesthetics (see Warnings and Precautions – General).

## ***Use in Special Populations***

### **Pregnancy – Category B3**

Madopar is contraindicated during pregnancy and in women of childbearing potential in the absence of adequate contraception (see Contraindications, Preclinical Safety).

### **Nursing mothers**

Since it is not known whether benserazide passes into breast milk, mothers requiring Madopar treatment should not nurse their infants, since the occurrence of skeletal malformations in the infants can not be excluded.

### **Renal impairment**

Levodopa and benserazide are both extensively metabolised and less than 10% of levodopa is excreted unchanged through the kidneys. No dose reduction is therefore necessary in case of mild or moderate renal insufficiency.

Pharmacokinetic data with levodopa in renal impaired patients are not available. Madopar is well tolerated by uraemic patients undergoing haemodialysis.

### **Hepatic impairment**

Levodopa is mainly metabolised by the aromatic amino acid decarboxylase that is abundantly present in the intestinal tract, in kidney and heart in addition to the liver.

Pharmacokinetic data with levodopa in hepatic impaired patients are not available.

## **Undesirable Effects**

*Blood and Lymphatic System Disorders:* Haemolytic anaemia, transient leucopenia and thrombocytopenia have been reported in rare cases. Therefore, as in any long-term levodopa-containing treatment, blood count and liver and kidney function should be monitored periodically.

*Metabolic and Nutritional Disorders:* Anorexia has been reported.

*Psychiatric Disorders:* Depression can be part of the clinical picture in patients with Parkinson's disease and may also occur in patients treated with Madopar. Agitation, anxiety, insomnia, hallucinations, delusions and temporal disorientation may occur particularly in elderly patients and in patients with a history of such disorders.

*Nervous System Disorders:* Isolated cases of ageusia or dysgeusia have been reported. At later stages of the treatment, dyskinesia (e.g. choreiform or athetotic) may occur. These can usually be eliminated or be made tolerable by a reduction of dosage. With prolonged treatment, fluctuations in therapeutic response may also be encountered. They include freezing episodes, end-of-dose deterioration and the "on-off" effect. These can usually be eliminated or made tolerable by adjusting the dosage and by giving smaller single doses more frequently. An attempt at increasing the dosage again can subsequently be made in order to intensify the therapeutic effect. Madopar is associated with somnolence and has been associated very rarely with excessive daytime somnolence and sudden sleep onset episodes.

*Cardiac Disorders:* Cardiac arrhythmias may occur occasionally.

*Vascular Disorders:* Orthostatic hypotension may occur occasionally. Orthostatic disorders commonly improve following reduction of the Madopar dosage.

*Gastrointestinal Disorders:* Nausea, vomiting and diarrhoea have been reported with Madopar. Undesirable gastrointestinal effects, which may occur mainly in the early stages of the treatment, can largely be controlled by taking Madopar with some food or liquid or by increasing the dose slowly.

*Skin and Subcutaneous Tissue Disorders:* Allergic skin reactions such as pruritus and rash may occur in rare cases.

*Investigations:* Transient elevation of liver transaminase and alkaline phosphatase may occur. Increased gamma-glutamyltransferase has been reported. Rises in blood urea nitrogen have been noted with Madopar. Urine may be altered in colour, usually acquiring a red tinge which turns dark on standing.

Patients treated with dopamine agonists for treatment of Parkinson's disease, especially at high doses, have been reported as exhibiting signs of pathological gambling, increased libido and hypersexuality. These effects are generally reversible upon reduction of the dose or treatment discontinuation. There is no established causal relationship between Madopar, which is not a dopamine agonist, and these events. However, caution is advised as Madopar is a dopaminergic medicine.

## **Laboratory abnormalities**

See Undesirable Effects – Investigations

## **Overdose**

### ***Symptoms and signs***

Symptoms and signs of overdose are qualitatively similar to the side effects of Madopar in therapeutic doses but may be of greater severity. Overdose may lead to: cardiovascular side effects (e.g. cardiac arrhythmias), psychiatric disturbances (e.g. confusion and insomnia), gastro-intestinal effects (e.g. nausea and vomiting) and abnormal involuntary movements (see Undesirable Effects).

If a patient has taken an overdose of a controlled release form of Madopar (i.e. Madopar HBS capsules), occurrence of symptoms and signs may be delayed due to delayed absorption of the active substances from the stomach.

### ***Treatment***

Monitor the patient's vital signs and institute supportive measures as indicated by the patient's clinical state. In particular patients may require symptomatic treatment for cardiovascular effects (e.g. antiarrhythmics) or central nervous system effects (e.g. respiratory stimulants, neuroleptics).

In addition, for the controlled release formulation further absorption should be prevented using an appropriate method.

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## **Pharmacological Properties and Effects**

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Dopamine, which acts as a neurotransmitter in the brain, is not present in sufficient quantities in the basal ganglia of parkinsonian patients. Levodopa or L-DOPA (3,4-dihydroxy phenylalanine) is an intermediate in dopamine biosynthesis. Levodopa (dopamine precursor) is used as a prodrug to increase dopamine levels since it is able to cross the blood-brain barrier whereas dopamine itself cannot. Once levodopa has entered the central nervous system, it is metabolised to dopamine by aromatic L-amino acid decarboxylase

After administration, levodopa is rapidly decarboxylated to dopamine in extracerebral as well as cerebral tissues. As a result, most of the levodopa administered is not available to the basal ganglia, and the dopamine produced peripherally frequently causes unwanted effects. It is therefore particularly desirable to inhibit extracerebral decarboxylation of levodopa. This can be achieved by simultaneous administration of levodopa and benserazide, a peripheral decarboxylase inhibitor.

Madopar is a combination of these two substances in a ratio of 4:1 - this ratio having proved optimal in clinical trials and therapeutic use - and is just as effective as large doses of levodopa given alone.

## ***Pharmacokinetic Properties***

### **Absorption**

#### ***Standard forms***

Levodopa is mainly absorbed from the upper regions of the small intestine, and absorption there is independent of the site. Maximum plasma concentrations of levodopa are reached approximately one hour after ingestion of standard Madopar.

The maximum plasma concentration of levodopa and the extent of levodopa absorption (AUC) increase proportionally with dose (50-200 mg levodopa).

Food intake reduces the rate and extent of levodopa absorption. The peak levodopa plasma concentration is 30% lower and occurs later when standard Madopar is administered after a standard meal. The extent of levodopa absorption is reduced by 15%.

### ***Dispersible form***

The pharmacokinetic profiles of levodopa following administration of Madopar dispersible in healthy volunteers and parkinsonian patients are very similar to those following administration of standard Madopar, but time to peak concentrations tends to be shorter after Madopar dispersible. There is less interindividual variability in absorption parameters for Madopar dispersible taken as a suspension.

### ***Controlled release form***

The pharmacokinetic properties of Madopar HBS differ from those of standard Madopar (capsules) and dispersible form. The active ingredients are released slowly in the stomach. Maximum plasma concentrations of levodopa, which are 20 – 30% of those achieved with the standard dosage forms, are reached about 3 hours after administration. The plasma concentration-time curve shows a longer 'half-value duration' (time span during which plasma concentrations are equal to or exceed half the maximum concentration) than with standard Madopar, which indicates pronounced controlled-release properties. The bioavailability of Madopar HBS is 50 – 70% of that of standard Madopar and is not affected by food. Maximum plasma concentrations of levodopa are not affected by food, but occur later (5 hours) after postprandial administration of Madopar HBS.

## **Distribution**

Levodopa crosses the blood-brain barrier by a saturable transport system. It is not bound to plasma proteins, and its volume of distribution is 57 litres. The AUC of levodopa in cerebrospinal fluid is 12% of that in plasma.

In contrast to levodopa, benserazide does not penetrate the blood-brain barrier at therapeutic doses. It is concentrated mainly in the kidneys, lungs, small intestine and liver.

## **Metabolism**

Levodopa is metabolised by two major pathways (decarboxylation and O-methylation) and two minor ones (transamination and oxidation).

Aromatic amino acid decarboxylase converts levodopa to dopamine. The major end-products of this pathway are homovanillic acid and dihydroxyphenylacetic acid. Catechol-O-methyltransferase methylates levodopa to 3-O-methyldopa. This major plasma metabolite has an elimination half-life of 15 hours, and it accumulates in patients who receive therapeutic doses of Madopar.

Decreased peripheral decarboxylation of levodopa when it is administered with benserazide is reflected in higher plasma levels of levodopa and 3-O-methyldopa and lower plasma levels of catecholamines (dopamine, noradrenaline) and phenolcarboxylic acids (homovanillic acid, dihydroxyphenylacetic acid).

Benserazide is hydroxylated to trihydroxybenzylhydrazine in the intestinal mucosa and the liver. This metabolite is a potent inhibitor of the aromatic amino acid decarboxylase.

## **Elimination**

In the presence of peripherally inhibited levodopa decarboxylase the elimination half-life of levodopa is approximately 1.5 hours. The elimination half-life is slightly longer (approximately 25%) in elderly patients (65 – 78 years of age) with Parkinson's disease (see Pharmacokinetics in special populations). The clearance of levodopa from plasma is about 430 ml/min.

Benserazide is almost entirely eliminated by metabolism. The metabolites are mainly excreted in the urine (64%) and to a smaller extent in faeces (24%).

## **Pharmacokinetics in special populations**

No pharmacokinetic data are available in uraemic and hepatic patients.

### ***Effect of age on the pharmacokinetics of levodopa***

In older Parkinsonian patients (65-78 years of age) both the elimination half-life and the AUC of levodopa is about 25% higher than in younger patients (34-64 years of age). The statistically significant age effect is clinically negligible and is of minor importance for the dosing schedule of any indication.

## ***Preclinical Safety***

### **Carcinogenicity**

Carcinogenicity studies were not conducted with Madopar.

### **Mutagenicity**

Madopar and its constituents (levodopa and benserazide) were not observed to be mutagenic in the Ames test. No further data are available.

### **Impairment of fertility**

No animal studies on fertility were performed with Madopar.

### **Teratogenicity**

Teratogenicity studies showed no teratogenic effects or effects on skeletal development in mice (400 mg/kg; rats (600 mg/kg; 250 mg/kg), and rabbits (120 mg/kg; 150 mg/kg).

At maternally toxic dose levels, intrauterine deaths increased (rabbits) and/or foetal weight decreased (rats).

### **Other**

General toxicological studies in rats have shown the possibility of disturbed foetal skeletal development.

No further animal data of relevance are available.

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## Stability

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### Madopar 62.5, 125, 250 capsules

Store at or below 30°C.

Keep the bottle tightly closed.

### Madopar 62.5 Dispersible tablets

Store at or below 25°C.

Madopar dispersible should be taken within half an hour of dissolving the tablet.

### Madopar HBS capsules

Store at or below 30°C.

Keep the bottle tightly closed.

This medicine should not be used after the expiry date shown on the pack.

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## Medicine Classification

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Prescription medicine

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## Packs

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### Madopar 62.5

Capsules each containing 50 mg levodopa  
+ 12.5 mg benserazide.

Bottles of 100 capsules

### Madopar Dispersible 62.5

Dispersible tablets each containing  
50 mg levodopa +12.5 mg benserazide.

Bottles of 100 tablets

### Madopar 125

Capsules each containing 100 mg levodopa  
+ 25 mg benserazide.

Bottles of 100 capsules

### Madopar 250

Capsules each containing 200 mg levodopa  
+ 50 mg benserazide.

Bottles of 100 capsules

### Madopar HBS

Capsules each containing 100 mg levodopa  
+ 25 mg benserazide.

Bottles of 100 capsules



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## **Name and Address**

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## **Date of Preparation**

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08 November 2011