**Pharmacodynamics**

Mechanism of Action

Atorvastatin is a synthetic lipid-lowering agent. Atorvastatin is an inhibitor of HMG-CoA reductase, the rate-limiting enzyme that converts 3-hydroxy-3-methyl-glutaryl-coenzyme A to mevalonate, a precursor of sterols, including cholesterol.

Triglycerides (TG) and cholesterol in the liver are incorporated into very low density lipoprotein (VLDL) and released into the plasma for delivery to peripheral tissues. Low density lipoprotein (LDL) is formed from VLDL and is catabolised primarily through the high affinity LDL-receptor.

Atorvastatin lowers plasma cholesterol and lipoprotein levels by inhibiting HMG-CoA reductase and cholesterol synthesis in the liver and by increasing the number of hepatic LDL receptors on the cell-surface to enhance uptake and catabolism of LDL. Atorvastatin reduces LDL production and the number of LDL particles. Atorvastatin produces a marked and sustained increase in LDL receptor activity coupled with a beneficial change in the quality of circulating LDL particles. Atorvastatin concentrations in human liver have demonstrated that cholesterol and lipoprotein levels of total cholesterol (Total-C), low density lipoprotein cholesterol (LDL-C) and apolipoprotein B (apo B) promote human atherosclerosis and are risk factors for developing cardiovascular disease. Similarly, decreased levels of high density lipoprotein cholesterol (HDL-C) are associated with the development of atherosclerosis. Epidemiological investigations have established that cardiovascular morbidity and mortality vary directly with the level of total-C and LDL-C and inversely with the level of HDL-C.

Atorvastatin reduces total-C, LDL-C, and apo B in both normal volunteers and in patients with homocysteinemia and heterozygous familial hypercholesterolaemia (FH), non-familial forms of hypercholesterolaemia, and mixed dyslipidaemia. Atorvastatin also reduces very low density lipoprotein cholesterol (VLDL-C) and TG and produces variable increases in HDL-C and apolipoprotein A-1. Atorvastatin reduces total-C, LDL-C, VLDL-C, apo B and TG, and increases HDL-C in patients with isolated hypertriglyceridaemia. Atorvastatin reduces intermediate density lipoprotein cholesterol (IDL-C) in patients with dysbetalipoproteinemia. In animal models, atorvastatin limits the development of lipid-enriched atherosclerotic lesions and promotes the regression of pre-established atheroma.

**Pharmacokinetics**

**Absorption**

Atorvastatin is rapidly absorbed after oral administration; maximum plasma concentrations occur within 1 to 2 hours. A high-fat meal prior to administration is absorbed intact. The absolute bioavailability is 14%. The low systemic availability is attributed to pre-systemic clearance in gastrointestinal mucosa and/or hepatic first-pass metabolism. Individual variation of drug dose should be based on therapeutic response (see DOSAGE AND ADMINISTRATION).

**Distribution**

The mean volume of distribution of atorvastatin is about 400 litres. Atorvastatin is 98% bound to plasma proteins. A RBG/plasma ratio of approximately 0.25 is considered to occur in general population, with an older age, hs-PCR and renal impairment.

**Metabolism**

In humans, atorvastatin is extensively metabolised to ortho- and para-hydroxylated derivatives. In vitro inhibition of HMG-CoA reductase by ortho- and para-hydroxylated metabolites is equivalent to that of atorvastatin. Approximately 70% of circulating inhibitory activity for HMG-CoA reductase is attributed to active metabolites. In vitro studies suggest the importance of atorvastatin metabolism by cytochrome P450 3A4, consistent with increased plasma concentrations of atorvastatin in humans following co-administration with erythromycin, a known inhibitor of this enzyme (see PRECAUTIONS). In animals, the ortho-hydroxy metabolite undergoes further glucuronidation.

**Elimination**

Atorvastatin is eliminated primarily in bile following hepatic and extrahepatic metabolism; however, the drug does not appear to undergo enterohepatic recirculation. Mean plasma elimination half-life of atorvastatin in humans is approximately 14 hours, but the half-life of inhibitory activity for HMG-CoA reductase is 20 to 30 hours due to the contribution of active metabolites. Less than 2% of a dose of atorvastatin is recovered in urine following oral administration.

**Special populations**

**Elderly**

Plasma concentrations of atorvastatin are higher (approximately 40% for Cmax and 30% for AUC) in healthy elderly subjects (age ≥65 years) than in young adults. Lipid effects are comparable to that seen in younger patient populations given equal doses of atorvastatin.

**Children and Adolescents**

Pharmacokinetic studies have not been conducted in the paediatric population.

**Gender**

Plasma concentrations of atorvastatin in women differ (approximately 20% higher for Cmax and 10% lower for AUC) from those in men; however, there is no clinically significant difference in lipid effects with atorvastatin between men and women.

**Renal Impairment**

Renal disease has no influence on the plasma concentrations or lipid effects of atorvastatin; thus, dose adjustment in patients with renal dysfunction is not necessary (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

**Haemodialysis**

While atorvastatin has not been conducted in patients with end-stage renal disease, haemodialysis is not expected to significantly enhance clearance of atorvastatin since the drug is extensively bound to plasma proteins.

**Hepatic Impairment**

Plasma concentrations of atorvastatin are markedly increased (+approximately 16 fold in Cmax and 11 fold in AUC) in patients with chronic alcoholic liver disease (Childs-Pugh B) (see CONTRAINDICATIONS, PRECAUTIONS and DOSAGE AND ADMINISTRATION).

**CLINICAL TRIALS**

In a multicentre, placebo-controlled, double-blind dose-response study in patients with hypercholesterolaemia, atorvastatin was given as a single daily dose over 6 weeks.

Atorvastatin (10-80 mg) reduced total-C (30%-46%), LDL-C (41%-61%), apolipoprotein B (34%-50%) and triglycerides (14%-33%) while producing variable increases in HDL-C and apolipoprotein A (Table 1). A therapeutic response was seen within 2 weeks, and maximum response achieved within 4 weeks.

**Table 1: Dose-Response in Patients with Primary Hypercholesterolaemia**

<table>
<thead>
<tr>
<th>Atorvastatin dose (mg)</th>
<th>N</th>
<th>Total-C</th>
<th>LDL-C</th>
<th>AUC ( \log_{10} ) B</th>
<th>TG</th>
<th>HDL-C</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>12</td>
<td>4.8</td>
<td>1.6</td>
<td>6.4</td>
<td>0.95</td>
<td>5.9</td>
<td>0.5</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>3.4</td>
<td>0.9</td>
<td>5.0</td>
<td>0.76</td>
<td>8.1</td>
<td>0.7</td>
</tr>
<tr>
<td>20</td>
<td>10</td>
<td>2.6</td>
<td>0.8</td>
<td>4.3</td>
<td>0.80</td>
<td>7.9</td>
<td>0.7</td>
</tr>
</tbody>
</table>

The reduction of fatal and non-fatal strokes did not reach a pre-defined significance level (p=0.01), a favourable trend was observed with a 26% relative risk reduction.

The primary endpoint examined in ASCOT was the rate of fatal coronary heart disease or non-fatal myocardial infarction over 3 years. These coronary events occurred in 1.9% of atorvastatin-treated patients compared to 3% of placebo-treated patients, a relative risk reduction of 36% (p = 0.0055) (Table 2). Although this difference was statistically
significant for the whole trial population, this difference was not statistically significant in specified subgroups such as diabetes, patients with left ventricular hypertrophy (LVH), previous vascular disease, or metabolic syndrome.

Non Insulin Dependant Diabetes Mellitus (NIDDM)

A 26-week randomised, double-blind, comparator study in NIDDM subjects showed that atorvastatin is effective in dyslipidaemic patients with this condition. A total of 744 NIDDM patients were randomised to receive atorvastatin 10 or 80 mg daily or placebo. A significantly greater mean percent decrease in triglycerides was observed in the atorvastatin treated groups compared to placebo (Table 3).

Liver Dysfunction

As with other lipid-lowering agents of the same class, moderate (>3 x upper limit of normal [ULN]) elevations of serum transaminases may occur. In an open-label, randomised, cross-over study in patients with hypertriglyceridaemia (baseline TG ≥ 4.1 mmol/L), atorvastatin 20 mg/day and 80 mg/day produced significantly greater reductions in triglycerides than placebo (Table 3).

Haemorrhagic Stroke

A post-hoc analysis of a clinical study (SPARCL) in patients without known coronary heart disease who had a recent stroke or TIA, showed a higher incidence of haemorrhagic stroke in patients on atorvastatin 80 mg (32/8925, 0.36%) compared to placebo (33/8926, 0.37%). In the study, all cause mortality was numerically higher in the atorvastatin arm than the placebo arm. At study end all cause mortality was 9.1% on atorvastatin vs. 8.9% on placebo.

Coronary Disease

The effects of HMG-CoA reductase inhibitors on male fertility have not been studied in adequate numbers of patients. The effects, if any, on the gonadal and adrenal axes in pre-menopausal women are unknown. Caution should be exercised if an HMG-CoA reductase inhibitor is administered concomitantly with other drugs that may decrease the levels or activity of endogenous steroid hormones such as contraceptive, propranolol and cimetidine.

Intestinal Lung Disease

Carcinogenicity, Mutagenicity, Impairment of Fertility

In a 2-year study in rats given 10, 30 or 100 mg/kg/day, the incidence of hepato-cellular adenoma was marginally, although not significantly, increased in females at 100 mg/kg/day. The maximum dose used was 14 times higher than the human daily dose of 80 mg/kg. HMG-CoA reductase inhibitors have not been shown to cause increases in males in mice and rats. Atorvastatin did not demonstrate mutagenic or clastogenic potential in an appropriate battery of assays. It was negative in the Ames test with Salmonella typhimurium and Escherichia coli, and in the in vitro HGPRT forward mutation assay in Chinese hamster lung cells.

Atorvastatin did not produce significant increases in chromosomal aberrations in the in vitro Chinese hamster lung cell assay and was negative in the in vivo mouse micronucleus test.

Skeletal Muscle

Uncomplicated myalgia has been reported in atorvastatin-treated patients (see ADVERSE EFFECTS). Myopathy, defined as muscular aching or weakness or muscle weakness in conjunction with increases in creatine kinase (CK) values > 10 x ULN, should be considered in any patient with diffuse myalgias, muscle tenderness or weakness and/or marked elevation of CK. Patients should be advised to promptly unexplained muscle pain, tenderness or weakness, particularly during the initial months of therapy and any period of upward dosage titration of either drug. Therefore, lower starting and maintenance doses of atorvastatin should also be considered when taken concomitantly with the aforementioned drugs.

ADVERSE EFFECTS

Liver Dysfunction

As with other lipid-lowering agents of the same class, moderate (>3 x upper limit of normal [ULN]) elevations of serum transaminases may occur. In an open-label, randomised, cross-over study in patients with hypertriglyceridaemia (baseline TG ≥ 4.1 mmol/L), atorvastatin 20 mg/day and 80 mg/day produced significantly greater reductions in triglycerides than placebo (Table 3).

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Effects on Fertility

The effects of atorvastatin on spermatogenesis and human fertility have not been investigated in clinical studies. Dietary administration of 100 mg atorvastatin/kg/day to rats caused a decrease in spermatozoon concentration in the testes, a decrease in sperm motility and an increase in sperm abnormalities. Similar effects, however, were not observed in 17 rat males treated by gavage with 40 mg/kg/day for 17 days (FACG). Atorvastatin did not demonstrate mutagenic or clastogenic potential in an appropriate battery of assays. It was negative in the Ames test with Salmonella typhimurium and Escherichia coli, and in the in vitro HGPRT forward mutation assay in Chinese hamster lung cells.

Atorvastatin did not produce significant increases in chromosomal aberrations in the in vitro Chinese hamster lung cell assay and was negative in the in vivo mouse micronucleus test.

Liver Dysfunction

As with other lipid-lowering agents of the same class, moderate (>3 x upper limit of normal [ULN]) elevations of serum transaminases may have been reported following therapy with atorvastatin.

Liver function tests should be performed before the initiation of treatment and periodically thereafter. Patients who develop increased transaminase levels should be monitored until the abnormalities resolve. Should an increase in ALT or AST of >3 x ULN persist, reduction of dose or withdrawal of atorvastatin is recommended. Atorvastatin should be used with caution in patients who consume substantial quantities of alcohol and/or have a history of liver disease. Active liver disease or unexplained persistent transaminase elevations are contraindications to use (see CONTRAINDICATIONS).
phenytoin) can lead to variable reductions in plasma concentrations of atorvastatin. Due to the dual interaction
between atorvastatin and drugs that compete with it for the same transporters, atorvastatin levels may be decreased.
Concomitant administration of atorvastatin with inducers of cytochrome P450 3A4 (e.g. efavirenz, rifampicin,
erithromycin (500 mg four times a day), or clarithromycin (500 mg twice daily), known inhibitors of cytochrome
P450 3A4, can lead to increases in plasma concentrations of cyclosporin, fibric acid derivatives, erythromycin, azole antifungals, or niacin (see PRECAUTIONS). Based on experience with other HMG-CoA reductase inhibitors caution should be
exercised when atorvastatin is administered with inhibitors of cytochrome P450 3A4 (e.g. cyclosporin, mastoiide antibiots including erythromycin and azole antifungals including itraconazole).
The risk of myopathy during treatment with other HMG-CoA reductase inhibitors is increased with concurrent administration of cyclosporin, rifampicin, fibric acid derivatives, erythromycin, azole antifungals, or niacin (see PRECAUTIONS). Concomitant administration of atorvastatin with inducers of cytochrome P450 3A4 (e.g. efavirenz, rifampicin, phenytoin) can lead to variable reductions in plasma concentrations of atorvastatin. Due to the dual interaction mechanism of rifampicin (cytochrome P450 3A4 inducer) and inhibition of hepatect uptake transporter (P-gp/Bcrp), simultaneous co-administration of atorvastatin with rifampicin is recommended, as delayed administration of atorvastatin after administration of rifampicin has been associated with a significant reduction in atorvastatin plasma concentrations.
Fusidic acid Although interaction studies with atorvastatin and fusidic acid have not been conducted, severe muscle problems such as rhabdomyolysis have been reported in post-marketing experience with this combination. Patients should be closely monitored and temporary suspension of atorvastatin treatment may be appropriate.

**Effects of Other Medicines on Atorvastatin**
The following drugs have been shown to have an effect on the pharmacokinetics or pharmacodynamics of atorvastatin:
- Antacid Co-administration of an oral antacid suspension containing magnesium and aluminium hydroxides with atorvastatin decreased atorvastatin plasma concentrations approximately 35%, however, LDL-C reduction was not altered.
- Colestipol Plasma concentrations of atorvastatin were lower (approximately 25%) when colestipol and atorvastatin were co-administered. However, LDL-C reduction was greater when atorvastatin and colestipol were co-administered than when either drug was given alone.
- Transporter Inhibitors Atorvastatin and atorvastatin-metabolites are substrates of the OATP1B1 transporter. Inhibitors of the OATP1B1 (e.g. cyclosporin) can increase the bioavailability of atorvastatin. Concomitant administration of atorvastatin 10 mg and cyclosporin 5.2 mg/day resulted in an increase in exposure to atorvastatin (see DOSAGE AND ADMINISTRATION).
- Erythromycin/Clarithromycin In healthy individuals, co-administration of atorvastatin (10 mg once daily) and erythromycin (500 mg four times a day) or clarithromycin (500 mg twice daily), known inhibitors of cytochrome P450 3A4, was associated with higher plasma concentrations of atorvastatin (see PRECAUTIONS).
- Protease Inhibitors Co-administration of atorvastatin and protease inhibitors, known inhibitors of cytochrome P450 3A4, was associated with increased plasma concentrations of atorvastatin.
- Dilatazum Hydrochloride Co-administration of atorvastatin (40 mg) with dilatazum (240 mg) was associated with higher plasma concentrations of atorvastatin.
- Artesaoconazole Concomitant administration of atorvastatin (20 to 40 mg) and itraloconazole (200 mg) was associated with an increase in atorvastatin AUC.
- Grapefruit Juice Contains one or more components that inhibit cytochrome P450 3A4 and can increase plasma concentrations of atorvastatin, especially with excessive grapefruit juice consumption (1–2 L per day).

**Effects of Atorvastatin on Other Medicines**
The following medicines have been shown to have their pharmacokinetics or pharmacodynamics affected by atorvastatin:
- Digoxin When multiple doses of digoxin (0.25 mg once daily) and 10 mg atorvastatin were co-administered, steady-state plasma digoxin concentrations were unaffected. However, steady-state plasma digoxin concentrations increased by approximately 20% following administration of digoxin with 80 mg atorvastatin daily. Patients taking digoxin should be monitored appropriately.
- Oral Contraceptives Co-administration of an oral contraceptive containing norethindrone and ethinyl estradiol increased AUC values for norethindrone and ethinyl estradiol by approximately 30% and 20%. These increases should be considered when selecting an oral contraceptive to take atorvastatin.

Medicines Shown Not to Interact with Atorvastatin Cimetidine Atorvastatin plasma concentrations and LDL-C reduction were not altered by co-administration of cimetidine.

**Clinical Adverse Experiences**
Adverse events reported in ≥2% of patients in placebo-controlled clinical studies of atorvastatin, regardless of causality assessment, are shown in the table below.

<table>
<thead>
<tr>
<th>Effect</th>
<th>Body as a Whole</th>
<th>Upper Respiratory Tract</th>
<th>Skin and Appendages</th>
<th>Gastrointestinal System</th>
<th>Musculoskeletal System</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
</tr>
<tr>
<td>Body as a Whole</td>
<td>Infection</td>
<td>Headache</td>
<td>URI</td>
<td>Abdominal Pain</td>
<td>Rash</td>
</tr>
<tr>
<td>Placebo</td>
<td>10.0</td>
<td>10.3</td>
<td>2.8</td>
<td>10.1</td>
<td>1.4</td>
</tr>
<tr>
<td>Atorvastatin 10 mg</td>
<td>10.1</td>
<td>10.3</td>
<td>2.8</td>
<td>10.1</td>
<td>1.4</td>
</tr>
<tr>
<td>Atorvastatin 20 mg</td>
<td>10.1</td>
<td>10.3</td>
<td>2.8</td>
<td>10.1</td>
<td>1.4</td>
</tr>
<tr>
<td>Atorvastatin 40 mg</td>
<td>10.1</td>
<td>10.3</td>
<td>2.8</td>
<td>10.1</td>
<td>1.4</td>
</tr>
</tbody>
</table>

The following additional adverse effects have been reported in clinical trials of atorvastatin:
- Body as a Whole: angioneurotic oedema
- Digestive System: vomiting, anorexia, hepatitis, pancreatitis, cholestatic jaundice
- Nervous System: paraesthesia peripheral neuropathy
- Musculoskeletal System: muscle cramps, myalgia, myopathy
- Skin and Appendages: pruritus, alopecia
- Urogenital System: impotence

Special Senses: deafness
- Metabolic and Nutritional Disorders: hyperglycaemia, hyperglycaemia
- Cerebrovascular System: haemorrhagic stroke
- Not all effects listed have been causally associated with atorvastatin therapy.

In ASCOT [see CLINICAL TRIALS, PREVENTION of Cardiovascular Disease (ASCOT)] involving 10,305 participants treated with atorvastatin 10 mg daily (n=5,148) or placebo (n=5,157), the safety and tolerability profile of the group treated with atorvastatin was comparable to that of the group treated with placebo during a median of 3.3 years of follow-up.

**Post-Marketing Experience**
Rare adverse events that have been reported post-marketing which are not listed above, regardless of causality, include the following:
- Nervous System: tremor
- Musculoskeletal System: myalgia

The following adverse events have been reported with some statins: Excessive cases of interstitial lung disease, especially with long term therapy (see PRECAUTIONS).
DOSAGE AND ADMINISTRATION

TROVAS can be administered within the dosage range of 10–80 mg/day as a single daily dose. Atorvastatin can be taken at any time of the day, with or without food. Therapy should be individualised according to the target lipid levels, the recommended goal of therapy, and the patient’s response. After initiation and/or upon titration of atorvastatin, lipid levels should be re-assessed within 4 weeks and dosage adjusted according to the patient’s response.

Primary Hypercholesterolaemia and Mixed Dyslipidaemia

The majority of patients are controlled with 10 mg atorvastatin once a day. A therapeutic response is evident within two weeks, and the maximum response is usually achieved within four weeks. The response is maintained during chronic therapy.

Homozygous Familial Hypercholesterolaemia

Adults: In the compassionate-use study of patients with homozygous familial hypercholesterolaemia, most patients responded to 80 mg of atorvastatin with a greater than 15% reduction in LDL-C (18%-42%).

Children: Treatment experience in a paediatric population (with doses of atorvastatin up to 80 mg/day) is limited.

Use in Renal Impairment: Renal disease has no influence on the plasma concentrations or on the LDL-C reduction of atorvastatin; thus, no adjustment of the dose is required (see PHARMACOLOGY and PRECAUTIONS).

Use in Hepatic Impairment: Plasma concentrations of atorvastatin are markedly increased in patients with chronic alcoholic liver disease (Childs-Pugh B). The benefits of therapy should be weighed against the risks when atorvastatin is to be given to patients with hepatic insufficiency (see PHARMACOLOGY, CONTRAINDICATIONS and PRECAUTIONS).

Use in Combination with Other Medicinal Compounds: In cases where co-administration of atorvastatin with cyclosporin is necessary, the dose of atorvastatin should not exceed 10 mg. (see PRECAUTIONS, Skeletal Muscle and Interactions with Other Medicines).

OVERDOSAGE

There is no specific treatment for atorvastatin overdose. Should an overdose occur, the patient should be treated symptomatically, and supportive measures instituted as required. In symptomatic patients, monitor serum creatinine, BUN, creatinine phosphokinase, and urine myoglobin for indications of renal impairment secondary to rhabdomyolysis. Liver function tests should be performed in symptomatic patients.

If there has been significant ingestion, consider administration of activated charcoal. Activated charcoal is most effective when administered within 1-hour of ingestion. In patients who are not fully conscious or have impaired gag reflex, consideration should be given to administering activated charcoal via nasogastric tube once the airway is protected. For rhabdomyolysis, administer sufficient 0.9% saline to maintain urine output of 2 to 3 mL/kg/hr. Diuretics may be necessary to maintain urine output. Urinary alkalinisation is not routinely recommended. Due to extensive drug binding to plasma proteins, haemodialysis is not expected to significantly enhance atorvastatin clearance.

Contact the Poisons Information Centre for advice on the management of an overdose.

PRESENTATION AND STORAGE CONDITIONS

TROVAS 10 mg tablets are white to off-white, elliptical, film-coated tablets debossed ‘AS10’ on one side and plain on the other side.

TROVAS 20 mg tablets are white to off-white, elliptical, film-coated tablets debossed ‘AS20’ on one side and plain on the other side.

TROVAS 40 mg tablets are white to off-white, elliptical, film-coated tablets debossed ‘AS40’ on one side and plain on the other side.

TROVAS 80 mg tablets are white to off-white, elliptical, film-coated tablets debossed ‘AS80’ on one side and plain on the other side.

All strengths are supplied in blister packs containing 6 or 30 tablets, and HDPE bottles containing 28, 30 or 100 tablets. Not all presentations and pack sizes may be marketed.

Storage

Store below 25°C in the original container. Protect from light.

NAME AND ADDRESS OF THE SPONSOR

Ranbaxy Australia Pty Ltd.
Suite 4.02, Building D, Level 4
12 – 24 Talavera Road
North Ryde, NSW 2113

POISON SCHEDULE OF THE MEDICINE

Prescription only medicine (S4)

DATE OF APPROVAL

13 September 2011

DATE OF MOST RECENT AMENDMENT

20 January 2012