Data Sheet

RANBAXY-LOSARTAN®
losartan potassium
12.5 mg, 25 mg, 50 mg, & 100 mg tablets

Presentation
Ranbaxy-Losartan 12.5 mg tablets are white to off-white, circular, biconvex, film-coated tablets embossed with “L1” on one side and plain on the other side.

Ranbaxy-Losartan 25 mg tablets are white to off-white, circular, biconvex, film-coated tablets embossed with “L2” on one side and plain on the other side.

Ranbaxy-Losartan 50 mg tablets are white to off-white, circular, biconvex, film-coated tablets embossed with “L3” on one side and plain on the other side.

Ranbaxy-Losartan 100 mg tablets are white to off-white, circular, biconvex, film-coated tablets embossed with “L4” on one side and plain on the other side.

Do not halve the tablets. Dose equivalence when the tablets are divided has not been established.

Therapeutic Class
Ranbaxy-Losartan (losartan potassium), the first of a new class of agents for the treatment of hypertension is an angiotensin II receptor (type AT₁) antagonist. Ranbaxy-Losartan also provides a reduction in the combined risk of cardiovascular death, stroke, and myocardial infarction in hypertensive patients with left ventricular hypertrophy and renal protection for type 2 diabetic patients with proteinuria.

Indications
Hypertension
Ranbaxy-Losartan is indicated for the treatment of hypertension.

Reduction in the Risk of Cardiovascular Morbidity and Mortality in Hypertensive Patients with Left Ventricular Hypertrophy
Ranbaxy-Losartan is indicated to reduce the risk of cardiovascular morbidity and mortality as measured by the combined incidence of cardiovascular death, stroke, and myocardial infarction in hypertensive patients with left ventricular hypertrophy (see Race).

Heart Failure
Ranbaxy-Losartan is indicated for the treatment of heart failure in patients who cannot tolerate an ACE inhibitor. Switching patients with heart failure who are stable on an ACE inhibitor to Ranbaxy-Losartan is not recommended.
Renal Protection in Type 2 Diabetic Patients with Proteinuria
Ranbaxy-Losartan is indicated to delay the progression of renal disease as measured by a reduction in the combined incidence of doubling of serum creatinine, end stage renal disease (need for dialysis or renal transplantation) or death; and to reduce proteinuria.

Dosage and Administration
Ranbaxy-Losartan may be administered with or without food.

Ranbaxy-Losartan may be administered with other antihypertensive agents.

Do not halve the tablets. Dose equivalence when the tablets are divided has not been established.

Hypertension
The usual starting and maintenance dose is 50 mg once daily for most patients. The maximal antihypertensive effect is attained 3-6 weeks after initiation of therapy. Some patients may receive an additional benefit by increasing the dose to 100 mg once daily.

For patients with intravascular volume-depletion (e.g., those treated with high-dose diuretics), a starting dose of 25 mg once daily should be considered (see Warnings and Precautions).

No initial dosage adjustment is necessary for elderly patients or for patients with renal impairment, including patients on dialysis. A lower dose should be considered for patients with a history of hepatic impairment (see Warnings and Precautions).

Reduction in the Risk of Cardiovascular Morbidity and Mortality in Hypertensive Patients with Left Ventricular Hypertrophy
The usual starting dose is 50 mg of Ranbaxy-Losartan once daily. A low dose of hydrochlorothiazide should be added and/or the dose of Ranbaxy-Losartan should be increased to 100 mg once daily based on blood pressure response.

Heart Failure
The initial dose of Ranbaxy-Losartan in patients with heart failure is 12.5 mg once daily. The dose should generally be titrated at weekly intervals (i.e., 12.5 mg daily, 25 mg daily, 50 mg daily) to the usual maintenance dose of 50 mg once daily, as tolerated by the patient.

Ranbaxy-Losartan is usually given in combination with diuretics and digitalis.

Renal Protection in Type 2 Diabetic Patients with Proteinuria
The usual starting dose is 50 mg once daily. The dose may be increased to 100 mg once daily based on blood pressure response. Ranbaxy-Losartan may be administered with other antihypertensive agents (e.g., diuretics, calcium channel blockers, alpha- or beta-blockers, and centrally acting agents) as well as with insulin and other commonly used hypoglycaemic agents (e.g., sulfonylureas, glitazones and glucosidase inhibitors).
**Contraindications**
Ranbaxy-Losartan is contraindicated in patients who are hypersensitive to any component of this product.

**Warnings and Precautions**
Hypersensitivity: Angioedema. (See Adverse Effects.)

**Hypotension and Electrolyte/Fluid Imbalance**
In patients who are intravascularly volume-depleted (e.g., those treated with high-dose diuretics), symptomatic hypotension may occur. These conditions should be corrected prior to administration of Ranbaxy-Losartan, or a lower starting dose should be used (see Dosage and Administration). In type 2 diabetic patients with nephropathy treated with an angiotensin II antagonist, serum potassium levels should be monitored.

Electrolyte imbalances are common in patients with renal impairment, with or without diabetes, and should be addressed. In a clinical study conducted in type 2 diabetic patients with proteinuria, the incidence of hyperkalaemia was higher in the group treated with Ranbaxy-Losartan as compared to the placebo group; however, few patients discontinued therapy due to hyperkalaemia (see Adverse Effects, Laboratory Test Findings).

**Liver Function Impairment**
Based on pharmacokinetic data which demonstrate significantly increased plasma concentrations of losartan in cirrhotic patients, a lower dose should be considered for patients with a history of hepatic impairment (see Dosage and Administration and Pharmacokinetics).

**Renal Function Impairment**
As a consequence of inhibiting the renin-angiotensin system, changes in renal function including renal failure have been reported in susceptible individuals; these changes in renal function may be reversible upon discontinuation of therapy.

Other medicines that affect the renin-angiotensin system may increase blood urea and serum creatinine in patients with bilateral renal artery stenosis or stenosis of the artery to a solitary kidney. Similar effects have been reported with Ranbaxy-Losartan; these changes in renal function may be reversible upon discontinuation of therapy.

**Dual blockade of the renin-angiotensin-aldosterone system:**
As a consequence of inhibiting the renin-angiotensin-aldosterone system, hypotension, syncope, hyperkalaemia and changes in renal function (including acute renal failure) have been reported in susceptible individuals, especially if combining medicinal products that affect this system. Dual blockade of the renin-angiotensin-aldosterone system (e.g. by adding an ACE-inhibitor to an angiotensin II receptor antagonist) is, therefore, not recommended in patients with already controlled blood pressure and should be limited to individually defined cases with close monitoring of renal function.
Pregnancy
When used in pregnancy during the second and third trimesters, medicines that act directly on the renin-angiotensin system can cause injury and even death in the developing foetus. When pregnancy is detected, Ranbaxy-Losartan should be discontinued as soon as possible.

Although there is no experience with the use of Ranbaxy-Losartan in pregnant women, animal studies with losartan potassium have demonstrated foetal and neonatal injury and death, the mechanism of which is believed to be pharmacologically mediated through effects on the renin-angiotensin system. In humans, foetal renal perfusion, which is dependent upon the development of the renin angiotensin system, begins in the second trimester; thus, risk to the foetus increases if Ranbaxy-Losartan is administered during the second or third trimesters of pregnancy.

Nursing Mothers
It is not known whether losartan is excreted in human milk. Because many medicines are excreted in human milk and because of the potential for adverse effects on the nursing infant, a decision should be made whether to discontinue nursing or discontinue the medicine, taking into account the importance of the medicine to the mother.

Paediatric Use
Antihypertensive effects of Ranbaxy-Losartan have been established in hypertensive paediatric patients aged >1 month to 16 years. Use of Ranbaxy-Losartan in these age groups is supported by evidence from adequate and well-controlled studies of Ranbaxy-Losartan in paediatric and adult patients as well as by literature in paediatric patients.

The pharmacokinetics of losartan have been investigated in 50 hypertensive paediatric patients >1 month to <16 years of age following once daily oral administration of approximately 0.54 to 0.77 mg/kg of losartan (mean doses). The active metabolite is formed from losartan in all age groups. Pharmacokinetics of losartan and its active metabolite are generally similar across the studied age groups and consistent with pharmacokinetic historic data in adults.

In a clinical study involving 177 hypertensive paediatric patients 6 to 16 years of age, patients who weighed ≥ 20 kg to <50 kg received either 2.5, 25 or 50 mg of losartan daily and patients who weighed ≥ 50 kg received either 5, 50 or 100 mg of losartan daily. Losartan administration once daily lowered trough blood pressure in a dose-dependent manner. The dose response to losartan was observed across all subgroups (e.g., age, Tanner stage, gender, race). However, the lowest doses studied, 2.5 mg and 5 mg, corresponding to an average daily dose of 0.07 mg/kg, did not appear to offer consistent antihypertensive efficacy. In this study, Ranbaxy-Losartan was generally well tolerated.

For patients who can swallow tablets, the recommended dose is 25 mg once daily in patients ≥ 20 to <50 kg. The dose can be increased to a maximum of 50 mg once daily. In patients ≥ 50 kg, the starting dose is 50 mg once daily. The dose can be increased to a maximum of 100 mg once daily.
In paediatric patients who are intravascularly volume depleted, these conditions should be corrected prior to administration of Ranbaxy-Losartan.

The adverse experience profile for paediatric patients appears to be similar to that seen in adult patients.

Ranbaxy-Losartan is not recommended in paediatric patients with glomerular filtration rate <30 mL/min/1.73 m², in paediatric patients with hepatic impairment, or in neonates as no data are available.

**Use in the Elderly**

In clinical studies there was no age-related difference in the efficacy or safety profile of losartan.

**Race**

Based on the LIFE (Losartan Intervention For Endpoint reduction in hypertension) study, the benefits of Ranbaxy-Losartan on cardiovascular morbidity and mortality compared to atenolol do not apply to Black patients with hypertension and left ventricular hypertrophy although both treatment regimens effectively lowered blood pressure in Black patients. In the overall LIFE study population (n=9193), treatment with Ranbaxy-Losartan resulted in a 13.0% risk reduction (p=0.021) as compared to atenolol for patients reaching the primary composite endpoint of the combined incidence of cardiovascular death, stroke, and myocardial infarction. In this study, Ranbaxy-Losartan decreased the risk of cardiovascular morbidity and mortality compared to atenolol in non-Black, hypertensive patients with left ventricular hypertrophy (n=8660) as measured by the primary endpoint of the combined incidence of cardiovascular death, stroke, and myocardial infarction (p=0.003). In this study, however, Black patients treated with atenolol were at lower risk of experiencing the primary composite endpoint compared with Black patients treated with Ranbaxy-Losartan (p=0.03). In the subgroup of Black patients (n=533; 6% of the LIFE study patients), there were 29 primary endpoints among 263 patients on atenolol (11%, 25.9 per 1000 patient-years) and 46 primary endpoints among 270 patients (17%, 41.8 per 1000 patient-years) on Ranbaxy-Losartan.

**Animal Toxicology**

**Carcinogenesis**

Losartan potassium was not carcinogenic when administered at maximum tolerated dosage levels to rats and mice for 105 and 92 weeks, respectively. These maximum tolerated dosage levels provided respective margins of systemic exposure for losartan and its pharmacologically active metabolite over that achieved in humans treated with 50 mg of losartan of approximately 270- and 150-fold in rats and 45- and 27-fold in mice.

**Mutagenesis**

Losartan potassium was negative in the microbial mutagenesis and V-79 mammalian cell mutagenesis assays. In addition, there was no evidence of direct genotoxicity in the in vitro alkaline elution and in vitro chromosomal aberration assays at concentrations that were approximately 1700 times greater than the maximum plasma level achieved in man at the recommended therapeutic dosage level. Similarly, there was no induction of chromosomal aberrations in bone
marrow cells of male or female mice after the administration of toxic oral doses of up to 1500 mg/kg (4500 mg/m²) (750 times the maximum recommended daily human dose).

In addition, the active metabolite showed no evidence of genotoxicity in the microbial mutagenesis, *in vitro* alkaline elution, and *in vitro* chromosomal aberration assays.

**Reproduction**
Fertility and reproductive performance were not affected in studies with male and female rats given oral doses of losartan potassium up to approximately 150 and 300 mg/kg/day, respectively. These dosages provide respective margins of systemic exposure for losartan and its pharmacologically active metabolite of approximately 150/125-fold in male rats and 300/170-fold in female rats over that achieved in man at the recommended daily dose.

**Development**
Losartan potassium has been shown to produce adverse effects in rat foetuses and neonates. The effects include decreased body weight, mortality and/or renal toxicity. In addition, significant levels of losartan and its active metabolite were shown to be present in rat milk. Based on pharmacokinetic assessments, these findings are attributed to medicine exposure in late gestation and during lactation.

**Effects on the Ability to Drive and Use Machinery**
There are no data to suggest that Ranbaxy-Losartan affects the ability to drive and use machines.

**Adverse Effects**
Ranbaxy-Losartan has been evaluated for safety in more than 2500 patients treated for essential hypertension. In general, treatment with Ranbaxy-Losartan was well tolerated. The overall incidence of adverse experiences reported with Ranbaxy-Losartan was comparable to placebo. For the most part, adverse experiences have been mild and transient in nature and have not required discontinuation of therapy. In controlled clinical trials, discontinuation of therapy due to clinical adverse experiences was required in only 2.3% and 3.7% of patients treated with Ranbaxy-Losartan and placebo, respectively.

In controlled clinical trials for essential hypertension, dizziness was the only adverse effect reported as medicine-related that occurred with an incidence greater than placebo in one percent or more of patients treated with Ranbaxy-Losartan. In addition, dose-related orthostatic effects were seen in less than one percent of patients. Rarely, rash was reported, although the incidence in controlled clinical trials was less than placebo.

In these double-blind controlled clinical trials, for essential hypertension the following adverse experiences reported with Ranbaxy-Losartan occurred in >1 percent of patients, regardless of medicine relationship:
Ranbaxy-Losartan (n=2085)  Placebo (n=535)

**Body as a Whole**
- Abdominal pain 1.7 1.7
- Asthenia/fatigue 3.8 3.9
- Chest pain 1.1 2.6
- Oedema/swelling 1.7 1.9

**Cardiovascular**
- Palpitation 1.0 0.4
- Tachycardia 1.0 1.7

**Digestive**
- Diarrhoea 1.9 1.9
- Dyspepsia 1.1 1.5
- Nausea 1.8 2.8

**Musculoskeletal**
- Back pain 1.6 1.1
- Muscle cramps 1.0 1.1

**Nervous/Psychiatric**
- Dizziness 4.1 2.4
- Headache 14.1 17.2
- Insomnia 1.1 0.7

**Respiratory**
- Cough 3.1 2.6
- Nasal congestion 1.3 1.1
- Pharyngitis 1.5 2.6
- Sinus disorder 1.0 1.3
- Upper respiratory infec 6.5 5.6

Losartan was generally well tolerated in a controlled clinical trial in hypertensive patients with left ventricular hypertrophy. The most common drug-related side effects were dizziness, asthenia/fatigue, and vertigo.

In the LIFE study, among patients without diabetes at baseline, there was a lower incidence of new onset diabetes mellitus with losartan as compared to atenolol (242 patients versus 320 patients, respectively, p<0.001). Because there was no placebo group included in the study, it is not known if this represents a beneficial effect of losartan or an adverse effect of atenolol.

Losartan was generally well tolerated in a controlled clinical trial in type 2 diabetic patients with proteinuria. The most common drug-related side effects were asthenia/fatigue, dizziness, hypotension and hyperkalaemia (see Warnings and Precautions, Hypotension and Electrolyte/Fluid Imbalance.)
Losartan was generally well tolerated in controlled clinical trials for heart failure. Adverse experiences observed were typical of those expected in this population. The most common medicine-related side effects were dizziness and hypotension.

The following additional adverse reactions have been reported in post-marketing experience:

**Hypersensitivity:** Anaphylactic reactions, angioedema including swelling of the larynx and glottis causing airway obstruction and/or swelling of the face, lips, pharynx and/or tongue has been reported rarely in patients treated with losartan; some of these patients previously experienced angioedema with other medicines including ACE inhibitors. Vasculitis, including Henoch-Schoenlein purpura, has been reported rarely.

**Gastrointestinal:** Hepatitis (reported rarely), liver function abnormalities, vomiting

**Haematologic:** Anaemia thrombocytopenia (reported rarely)

**Musculoskeletal:** Myalgia, arthralgia

**Nervous System/Psychiatric:** Migraine, dysgeusia

**Respiratory:** Cough

**Skin:** Urticaria, pruritus, erythroderma

**Laboratory Test Findings**

In controlled clinical trials for essential hypertension, clinically important changes in standard laboratory parameters were rarely associated with administration of losartan. Hyperkalaemia (serum potassium >5.5 mEq/L) occurred in 1.5% of patients in the hypertension clinical trials. In a clinical study conducted in type 2 diabetic patients with proteinuria, 9.9% of patients treated with losartan and 3.4% of patients treated with placebo developed hyperkalaemia (see Warnings and Precautions, Hypotension and Electrolyte/Fluid Imbalance). Elevations of ALT occurred rarely and usually resolved upon discontinuation of therapy.

**Interactions**

In clinical pharmacokinetic trials, no medicine interactions of clinical significance have been identified with hydrochlorothiazide, digoxin, warfarin, cimetidine, phenobarbital and ketoconazole and erythromycin. Rifampin and fluconazole have been reported to reduce levels of active metabolite. The clinical consequences of these interactions have not been evaluated.

As with other medicines that block angiotensin II or its effect, concomitant use of potassium sparing diuretics (e.g., spironolactone, triamterene, amiloride), potassium supplements, or salt substitutes containing potassium may lead to increases in serum potassium.

As with other medicines which affect the excretion of sodium, lithium excretion may be reduced. Therefore, serum lithium levels should be monitored carefully if lithium salts are to be co-administered with angiotensin II receptor antagonists.
Non-steroidal anti-inflammatory drugs (NSAIDs) including selective cyclooxygenase-2 inhibitors (COX-2 inhibitors) may reduce the effect of diuretics and other antihypertensive drugs. Therefore, the antihypertensive effect of angiotensin II receptor antagonists may be attenuated by NSAIDs including selective COX-2 inhibitors.

In some patients with compromised renal function who are being treated with non-steroidal anti-inflammatory drugs, including selective cyclooxygenase-2 inhibitors, the co-administration of angiotensin II receptor antagonists may result in a further deterioration of renal function. These effects are usually reversible.

**Overdosage**

Significant lethality was observed in mice and rats after oral administration of 1000 mg/kg (3000 mg/m2) and 2000 mg/kg (11,800 mg/m2), respectively.

Limited data are available in regard to overdosage in humans. The most likely manifestation of overdosage would be hypotension and tachycardia; bradycardia could occur from parasympathetic (vagal) stimulation. If symptomatic hypotension should occur, supportive treatment should be instituted.

Neither losartan nor the active metabolite can be removed by haemodialysis.

**Actions**

Losartan, the first of a new class of agents for the treatment of hypertension is an angiotensin II receptor (type AT1) antagonist.

Angiotensin II, a potent vasoconstrictor, is the primary active hormone of the renin-angiotensin system, and a major determinant of the pathophysiology of hypertension. Angiotensin II binds to the AT1 receptor found in many tissues (e.g., vascular smooth muscle, adrenal gland, kidneys, and the heart) and elicits several important biological actions, including vasoconstriction and the release of aldosterone. Angiotensin II also stimulates smooth muscle cell proliferation. A second angiotensin II receptor has been identified as the AT2 receptor subtype, but it plays no known role in cardiovascular homeostasis.

Losartan is a potent, synthetic, orally active compound. Based on binding and pharmacological bioassays, it binds selectively to the AT1 receptor. *In vitro* and *in vivo*, both losartan and its pharmacologically active carboxylic acid metabolite (E-3174) block all physiologically relevant actions of angiotensin II, regardless of the source or route of synthesis. In contrast to some peptide antagonists of angiotensin II, losartan has no agonist effects.

Losartan binds selectively to the AT1 receptor and does not bind to or block other hormone receptors or ion channels important in cardiovascular regulation. Furthermore, losartan does not inhibit ACE (kininase II), the enzyme that degrades bradykinin.

Consequently, effects not directly related to blocking the AT1 receptor, such as the potentiation of bradykinin-mediated effects or the generation of oedema (losartan 1.7%, placebo 1.9%), are not associated with losartan.
Pharmacokinetics

Absorption
Following oral administration, losartan is well absorbed and undergoes first-pass metabolism, forming an active carboxylic acid metabolite and other inactive metabolites. The systemic bioavailability of losartan tablets is approximately 33%. Mean peak concentrations of losartan and its active metabolite are reached in 1 hour and in 3-4 hours, respectively. There was no clinically significant effect on the plasma concentration profile of losartan when the medicine was administered with a standardised meal.

Distribution
Both losartan and its active metabolite are >99% bound to plasma proteins, primarily albumin. The volume of distribution of losartan is 34 litres. Studies in rats indicate that losartan crosses the blood-brain barrier poorly, if at all.

Metabolism
About 14% of an intravenously- or orally-administered dose of losartan is converted to its active metabolite. Following oral and intravenous administration of 14C-labelled losartan potassium, circulating plasma radioactivity primarily is attributed to losartan and its active metabolite. Minimal conversion of losartan to its active metabolite was seen in about one percent of individuals studied.

In addition to the active metabolite, inactive metabolites are formed, including two major metabolites formed by hydroxylation of the butyl side chain and a minor metabolite, an N-2 tetrazole glucuronide.

Elimination
Plasma clearance of losartan and its active metabolite is about 600mL/min and 50mL/min, respectively. Renal clearance of losartan and its active metabolite is about 74mL/min and 26mL/min, respectively. When losartan is administered orally, about 4% of the dose is excreted unchanged in the urine, and about 6% of the dose is excreted in the urine as active metabolite. The pharmacokinetics of losartan and its active metabolite are linear with oral losartan potassium doses up to 200 mg.

Following oral administration, plasma concentrations of losartan and its active metabolite decline polyexponentially with a terminal half-life of about 2 hours and 6-9 hours, respectively. During once-daily dosing with 100 mg, neither losartan nor its active metabolite accumulates significantly in plasma.

Both biliary and urinary excretion contribute to the elimination of losartan and its metabolites. Following an oral dose of 14C-labelled losartan in man, about 35% of radioactivity is recovered in the urine and 58% in the faeces. Following an intravenous dose of 14C-labelled losartan in man, about 43% of radioactivity is recovered in the urine and 50% in the faeces.

Characteristics in Patients
The plasma concentrations of losartan and its active metabolite observed in elderly male hypertensives are not significantly different from those observed in young male hypertensives.
Plasma concentrations of losartan were up to 2-fold higher in female hypertensives as compared to male hypertensives. Concentrations of the active metabolite were not different in males and females. This apparent pharmacokinetic difference is not judged to be of clinical significance.

Following oral administration in patients with mild to moderate alcoholic cirrhosis of the liver, plasma concentrations of losartan and its active metabolite were, respectively, 5-fold and 1.7-fold greater than those seen in young male volunteers.

Plasma concentrations of losartan are not altered in patients with creatinine clearance above 10 mL/min. Compared to patients with normal renal function, the AUC for losartan is approximately 2-fold greater in haemodialysis patients. Plasma concentrations of the active metabolite are not altered in patients with renal impairment or in haemodialysis patients. Neither losartan nor the active metabolite can be removed by haemodialysis.

Pharmaceutical Precautions
Store at temperatures below 25°C. Keep container tightly closed. Protect from light.

Medicine Classification
Prescription Medicine

Package Quantities
Ranbaxy-Losartan 12.5 mg tablets are available in blister packs of 30 ‘s and 90’s
Ranbaxy-Losartan 25 mg tablets are available in blister packs of 30 ‘s and 90’s.
Ranbaxy-Losartan 50 mg tablets are available in blister packs of 30’s and 90’s
Ranbaxy-Losartan 100 mg tablets are available in blister packs of 30 ‘s and 90’s.

Further Information
Chemistry
Losartan potassium, a non-peptide molecule, is chemically described as 2-butyl-4-chloro-1-[2’-(1H-tetrazol-5-yl)[1,1’-biphenyl]-4-yl]methyl]-1H-imidazole -5-methanol monopotassium salt.

Its empirical formula is C22H22ClKN6O, and its structural formula is:

![Chemical Structure of Losartan Potassium]

Losartan potassium is a white to off-white free-flowing crystalline powder with a molecular weight of 461.01. It is freely soluble in water, soluble in alcohols, and
slightly soluble in common organic solvents, such as acetonitrile and methyl ethyl ketone.

Oxidation of the 5-hydroxymethyl group on the imidazole ring results in the active metabolite of losartan.

**Inactive Ingredients**
Each tablet contains the following inactive ingredients: microcrystalline cellulose, lactose, pregelatinized starch, magnesium stearate, hydroxypropyl cellulose, hypromellose and titanium dioxide.

**Name and Address**
Douglas Pharmaceuticals Ltd,
Central Park Drive, Lincoln,
P.O.Box 45027, Auckland, NEW ZEALAND

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25 August 2010