Telsat
Tablet

Compositions:
TELSAT 40 mg
Each tablet contains
Telmisartan 40 mg

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Telmisartan 80 mg

Product descriptions:
TELSAT 40 mg: white to off-white colored, round shaped uncoated tablets with diameter 9 mm, and having marking "FPP" on one side and breakline on the other side.
Excipients:
Spray dried mannitol, sodium hydroxide, meglumine, povidone K-30, purified water, magnesium stearate.

TELSAT 80 mg: white to off-white colored, round shaped uncoated tablets with diameter 11 mm, and having marking "FPP" on the one side, and plain on the other side.
Excipients:
Spray dried mannitol, sodium hydroxide, meglumine, povidone K-30, purified water, magnesium stearate.

Pharmacology:
Telmisartan is an orally effective and specific angiotensin II receptor (type AT1) antagonist. Telmisartan displaces angiotensin II with very high affinity from its binding site at the AT1 receptor subtype, which is responsible for the known actions of angiotensin II. Telmisartan does not exhibit any partial agonist activity at the AT1 receptor. Telmisartan selectively binds the AT1 receptor. The binding is long lasting. Telmisartan does not show affinity for other receptors, including AT2 and other less characterized AT receptors. The functional role of these receptors is not known, nor is the effect of their possible overstimulation by angiotensin II, whose levels are increased by telmisartan. Plasma aldosterone levels are decreased by telmisartan. Telmisartan does not inhibit human plasma renin or block ion channels. Telmisartan does not inhibit angiotensin converting enzyme (kininase II), the enzyme which also degrades bradykinin. Therefore, it is not expected to potentiate bradykinin-mediated adverse effects. In man, an 80 mg dose of telmisartan almost completely inhibits the angiotensin II evoked blood pressure increase. The inhibitory effect is maintained over 24 hours and still measurable up to 48 hours.

Treatment of essential hypertension
After the first dose of telmisartan, the antihypertensive activity gradually becomes evident within 3 hours. The maximum reduction in blood pressure is generally attained 4 weeks after the start of treatment and is sustained during long-term therapy.
The antihypertensive effect persists constantly over 24 hours after dosing and includes the last 4 hours before the next dose as shown by ambulatory blood pressure measurements. This is confirmed by trough to peak ratios consistently above 80% seen after doses of 40 and 80 mg of telmisartan in placebo controlled clinical studies.
There is an apparent trend to a dose relationship to a time to recovery of baseline systolic blood pressure (SBP). In this respect data concerning diastolic blood pressure (DBP) are inconsistent. In patients with hypertension telmisartan reduces both systolic and diastolic blood pressure without affecting pulse rate. The antihypertensive efficacy of telmisartan has been compared to antihypertensive drugs such as amlodipine, atenolol, enalapril, hydrochlorothiazide, losartan, lisinopril, ramipril and valsartan.
Upon abrupt cessation of treatment with telmisartan, blood pressure gradually returns to pre-treatment values over a period of several days without evidence of rebound hypertension.
Telmisartan treatment has been shown in clinical trials to be associated with statistically significant reductions in Left Ventricular Mass and Left Ventricular Mass Index in patients with hypertension and Left Ventricular Hypertrophy.
Telmisartan treatment has been shown in clinical trials (including comparators like losartan, ramipril and valsartan) to be associated with statistically significant reductions in proteinuria (including microalbuminuria and macroalbuminuria) in patients with hypertension and diabetic nephropathy.
The incidence of dry cough was significantly lower in patients treated with telmisartan than in those given angiotensin converting enzyme inhibitors in clinical trials directly comparing the two antihypertensive treatments.

Reduction of cardiovascular morbidity
Support for use to reduce the risk of cardiovascular events was obtained in a pair of studies. Both enrolled subjects age ≥55 years, at high cardiovascular risk as evidenced by coronary artery disease (75%), diabetes mellitus (27%) accompanied with end-organ damage (e.g., retinopathy, left ventricular hypertrophy, and in ONTARGET (**ON** going **T**elmisartan **A**lone and in combination with **R**amipril **G**lobal **E**ndpoint **T**rial) only, macro- or microalbuminuria), stroke (16%), peripheral vascular disease (13%), or transient ischemic attack (4%).
Patients without a history of intolerance to ACE inhibitors entered ONTARGET, and those with such a history, usually cough (90%), entered TRANSCEND (**T**elmisartan **R**andomized **A**ssessme**N**t **S**tudy in a**CE** i**N**tolerant subjects with cardiovascular **D**isease), but patients with >1+ proteinuria on dipstick were excluded from TRANSCEND. For both ONTARGET and TRANSCEND trials, the primary 4-components composite endpoint was death from cardiovascular causes, myocardial infarction, stroke, and hospitalization for heart failure. The secondary 3-components composite endpoint was death from cardiovascular causes, myocardial infarction, and stroke.
ONTARGET was a randomized, active-controlled, multinational, double-blind study in 25,620 patients who were randomized to telmisartan 80 mg, ramipril 10 mg, or their combination. The population studied was 73% male, 74% caucasian, 14% asian, and 57% were 65 years of age or older. Baseline therapy included acetylsalicylic acid (76%), lipid lowering agents (64%), beta-blockers (57%), calcium channel blockers (34%), nitrates (29%), and diuretics (28%). The mean duration of follow up was about 4 years and 6 months. During the study, 22.0% (n=1,878) of telmisartan patients discontinued the active treatment, compared to 24.4% (n=2,095) of ramipril patients and 25.3% (n=2,152) of telmisartan/ramipril patients.
TRANSCEND randomized patients to telmisartan 80 mg (n=2,954) or placebo (n=2,972). The mean duration of follow up was 4 years and 8 months. The population studied was 57% male, 62% caucasian, 21% asian, and 60% were 65 years of age or older. Baseline therapy included acetylsalicylic acid (75%), lipid lowering agents (58%), beta-blockers (58%), calcium channel blockers (41%), nitrates (34%) and diuretics (33%). During the study, 17.7% (n=523) of telmisartan patients discontinued the active treatment, compared to 19.4% (n=576) of placebo patients.

The results for the TRANSCEND trial are summarized in Table 1, and the results for ONTARGET are summarized in Table 2, as follows. (See **Tables 1** and **Tables 2**)

Table 1. Incidence of the primary and secondary outcomes from TRANSCEND

	Telmisartan vs Placebo (n=2954) (n=2972)		
	Number of events Telmisartan/Placebo	**Hazard ratio 95% CI**	**p-value**
*Composite of CV death, myocardial infarction, stroke, or hospitalization for heart failure	465 (15.7%)/ 504 (17.0%)	0.92 (0.81-1.05)	0.2129
*Composite of CV death, myocardial infarction, or stroke	384 (13.0%)/ 440 (14.8%)	0.87 (0.76-1.00)	0.0483
Individual components of the primary composite endpoint	**Number of events Telmisartan/Placebo**	**Hazard ratio 95% CI**	**p-value**
**All non-fatal MI	114 (3.9%)/ 145 (4.9%)	0.79 (0.62-1.01)	0.0574
**All non-fatal strokes	112 (3.8%)/ 136 (4.6%)	0.83 (0.64-1.06)	0.1365

*The primary endpoint was defined as the time to first event. In case of multiple simultaneous events, all individual events were considered; the sum of patients with individual outcomes may exceed the number of patients with composite (primary or secondary) outcomes.
**For individual components of the primary composite endpoints, all events, regardless whether or not they were the first event, were considered. Therefore, they are more than the first events considered for the primary or secondary composite endpoint.

Table 2. Incidence of the primary and secondary outcomes from ONTARGET

	Telmisartan vs Ramipril (n=8542) (n=8576)	
	Number of events Telmisartan/Ramipril	**Hazard ratio 97.5% CI**
Composite of CV death, myocardial infarction, stroke, or hospitalization for heart failure	1,423 (16.7%)/1,412 (16.5%)	1.01 (0.93-1.10)
Composite of CV death, myocardial infarction, or stroke	1,190 (13.9%)/1,210 (14.1%)	0.99 (0.90-1.08)

Although the event rates in ONTARGET were similar on telmisartan and ramipril, the results did not unequivocally rule out that telmisartan may not preserve a meaningful fraction of the effect of ramipril in reducing cardiovascular events. However, the results of both ONTARGET and TRANSCEND do adequately support telmisartan being more effective than placebo would be in this setting, particularly for the end point of time to cardiovascular death, myocardial infarction, or stroke.
In ONTARGET, there was no evidence that combining ramipril and telmisartan reduced the risk of death from cardiovascular causes, myocardial infarction, stroke, or hospitalization for heart failure greater than ramipril alone; instead, patients who received the combination of ramipril and telmisartan in ONTARGET experienced an increased incidence of clinically important renal dysfunction (e.g., acute renal failure) compared to patients receiving telmisartan or ramipril alone.
Multiple sub-group analyses did not demonstrate any differences in the 4-components composite primary endpoint based on age, gender, or ethnicity for either ONTARGET or TRANSCEND trial.

Pharmacokinetics:
Telmisartan is well absorbed following oral administration although the amount absorbed varies. The mean absolute bioavailability for telmisartan is about 50%. When telmisartan is taken with food, the reduction in the area under the plasma concentration-time curve (AUC) of telmisartan varies from approximately 6% (40 mg dose) to approximately 19% (160 mg dose). By 3 hours after administration plasma concentrations are similar whether telmisartan is taken fasting or with food. The small reduction in AUC is not expected to cause a reduction in the therapeutic efficacy. Gender differences in plasma concentrations were observed, C_{max} and AUC being approximately 3- and 2-fold higher, respectively, in females compared to males without relevant influence on efficacy. Telmisartan is largely bound to plasma protein (>99.5%), mainly albumin and alpha-1 acid glycoprotein. The mean steady state apparent volume of distribution (V_{ss}) is approximately 500 L. Telmisartan is metabolized by conjugation to the glucuronide of the parent compound. No pharmacological activity has been shown for the conjugate. Telmisartan is characterized by biexponential decay pharmacokinetics with a terminal elimination half-life of >20 hours. The maximum plasma concentration (C_{max}) and, to a smaller extent, area under the plasma concentration-time curve (AUC) increase disproportionately with dose. There is no evidence of clinically relevant accumulation of telmisartan. After oral (and intravenous) administration telmisartan is nearly exclusively excreted with the feces, exclusively as unchanged compound. Cumulative urinary excretion is <2% of dose. Total plasma clearance (CL_{tot}) is high (approximately 900 ml/min compared with hepatic blood flow (about 1500 ml/minute).
TELSAT tablet has been studied in a randomized, open-label, three-period, three-sequence, partial replicate design study under fasting conditions which included 30 healthy adult male and female subjects.
After oral administration of 80 mg TELSAT tablet, the mean of AUC_{0-72h} was 5089.1 ng.h/ml. The mean of maximum plasma concentration (C_{max}) was 1018.44 ng/ml and reached within 0.8 hours (0.50-2.00 hours). The mean elimination half-life ($t_{½}$) of TELSAT tablet was 24.36 hours.

Elderly patients
The pharmacokinetics of telmisartan do not differ between younger and elderly patients.

Patients with renal impairment
Lower plasma concentrations were observed in patients with renal insufficiency undergoing dialysis. Telmisartan is highly bound to plasma protein in renal-insufficient patients and cannot be removed by dialysis. The elimination half-life is not changed in patients with renal impairment.

Patients with hepatic impairment
Pharmacokinetic studies in patients with hepatic impairment showed an increase in absolute bioavailability up to nearly 100%. The elimination half-life is not changed in patients with hepatic impairment.

Indications:
- Treatment of essential hypertension.
- Reduction of the risk of non-fatal stroke or non-fatal myocardial infarction in patients 55 years or older at high risk of developing major cardiovascular events who cannot tolerate an angiotensin converting enzyme inhibitor (ACEI).
  High risk of cardiovascular events includes evidence of coronary artery disease, peripheral arterial disease, stroke, transient ischemic attack, or diabetes mellitus with evidence of end-organ damage.

Contraindications:
- Hypersensitivity to the active ingredient or any of the excipients.
- Second and third trimesters of pregnancy.
- Lactation.
- Biliary obstructive disorders.
- Severe hepatic impairment.
- The concomitant use of telmisartan with aliskiren is contraindicated in patients with diabetes mellitus or renal impairment (GFR <60 ml/min/1.73 m^2).

Dosage and administration:
Adults
Treatment of essential hypertension
The recommended dose is 40 mg once daily. Some patients may already benefit at a daily dose of 20 mg. In cases where the target blood pressure is not achieved, telmisartan dose can be increased to a maximum of 80 mg once daily. Alternatively, telmisartan may be used in combination with thiazide-type diuretics such as hydrochlorothiazide, which has been shown to have an additive blood pressure lowering effect with telmisartan. When considering raising the dose, it must be borne in mind that the maximum antihypertensive effect is generally attained four-eight weeks after the start of treatment.
In patients with severe hypertension treatment with telmisartan at doses up to 160 mg alone and in combination with hydrochlorothiazide 12.5-25 mg daily was well tolerated and effective.

Reduction of cardiovascular morbidity
The recommended dose is 80 mg once daily. It is not known whether doses lower than 80 mg of telmisartan are effective in reducing cardiovascular morbidity.
When initiating telmisartan therapy for the reduction of cardiovascular morbidity, monitoring of blood pressure is recommended, and if appropriate adjustment of medications that lower blood pressure may be necessary.

TELSAT may be taken with or without food.

Renal impairment
No posology adjustment is required for patients with renal impairment, including those on hemodialysis. Telmisartan is not removed from blood by hemofiltration.

Hepatic impairment
In patients with mild to moderate hepatic impairment the posology should not exceed 40 mg once daily.

Elderly
No dosing adjustment is necessary.

Children and adolescents
TELSAT is not recommended for use in children below 18 years due to limited data on safety and efficacy.

Warnings and precautions:
TELSAT tablets should not be divided into halves since no studies have been performed on halved tablets.
Pregnancy
Angiotensin II receptor antagonists should not be initiated during pregnancy.
Unless continued angiotensin II receptor antagonist therapy is considered essential, patients planning pregnancy should be changed to alternative antihypertensive treatments which have an established safety profile for use in pregnancy.
When pregnancy is diagnosed, treatment with angiotensin II receptor antagonists should be stopped immediately, and if appropriate, alternative therapy should be started.

Renovascular hypertension
There is an increased risk of severe hypotension and renal insufficiency when patients with bilateral renal artery stenosis or stenosis of the artery to a single functioning kidney are treated with medicinal products that affect the renin-angiotensin-aldosterone system.

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Renal impairment and kidney transplant
When telmisartan is used in patients with impaired renal function, a periodic monitoring of potassium and creatinine serum levels is recommended. There is no experience regarding the administration of telmisartan in patients with a recent kidney transplant.

Intravascular volume depletion
Symptomatic hypotension, especially after the first dose, may occur in patients who are volume and/or sodium depleted by vigorous diuretic therapy, dietary salt restriction, diarrhea or vomiting. Such conditions, especially volume and/or sodium depletion, should be corrected before the administration of telmisartan.

Dual blockade of the renin-angiotensin-aldosterone system (RAAS)
There is evidence that the concomitant use of ACE-inhibitors, angiotensin II receptor blockers or aliskiren increases the risk of hypotension, hyperkalemia and decreased renal function (including acute renal failure).
Dual blockade of RAAS through the combined use of ACE-inhibitors, angiotensin II receptor blockers or aliskiren is therefore not recommended (see Interactions).
If dual blockade therapy is considered absolutely necessary, this should only occur under specialist supervision and subject to frequent close monitoring of renal function, electrolytes and blood pressure.
ACE-inhibitors and angiotensin II receptor blockers should not be used concomitantly in patients with diabetic nephropathy.

Other conditions with stimulation of the renin-angiotensin-aldosterone system
In patients whose vascular tone and renal function depend predominantly on the activity of the renin angiotensin-aldosterone system (e.g. patients with severe congestive heart failure or underlying renal disease, including renal artery stenosis), treatment with medicinal products that affect this system has been associated with acute hypotension, hyperazotemia, oliguria, or rarely acute renal failure.

Primary aldosteronism
Patients with primary aldosteronism generally will not respond to antihypertensive medicinal products acting through inhibition of the renin-angiotensin system. Therefore, the use of telmisartan is not recommended.

Aortic and mitral valve stenosis, obstructive hypertrophic cardiomyopathy
As with other vasodilators, special caution is indicated in patients suffering from aortic or mitral stenosis, or obstructive hypertrophic cardiomyopathy.

Hyperkalemia
During treatment with medicinal products that affect the renin-angiotensin-aldosterone system, hyperkalemia may occur, especially in the presence of renal impairment and/or heart failure. Monitoring of serum potassium in patients at risk is recommended.
Based on experience with the use of medicinal products that affect the renin-angiotensin system, concomitant use with potassium-sparing diuretics, potassium supplements, salt substitutes containing potassium or other medicinal products that may increase the potassium level (heparin, etc.) may lead to an increase in serum potassium and should therefore be co-administered cautiously with telmisartan.

Hepatic impairment
Telmisartan is mostly eliminated in the bile. Patients with biliary obstructive disorders or hepatic insufficiency can be expected to have reduced clearance. Telmisartan should be used with caution in these patients.

Diabetes mellitus
In diabetic patients with an additional cardiovascular risk, i.e. patients with diabetes mellitus and coexistent coronary artery disease (CAD), the risk of fatal myocardial infarction and unexpected cardiovascular death may be increased when treated with blood pressure lowering agents such as ARBs or ACE-inhibitors. In patients with diabetes mellitus CAD may be asymptomatic and therefore undiagnosed. Patients with diabetes mellitus should undergo appropriate diagnostic evaluation, e.g. exercise stress testing, to detect and to treat CAD accordingly before initiating treatment with telmisartan.

Other
As observed for angiotensin converting enzyme inhibitors, angiotensin receptor blockers including telmisartan are apparently less effective in lowering blood pressure in black people than in non-blacks, possibly because of higher prevalence of low-renin states in the black hypertensive population. As with any antihypertensive agent, excessive reduction blood pressure in patients with ischemic cardiopathy or ischemic cardiovascular disease could result in a myocardial infarction or stroke.

Warning: fetal/neonatal morbidity and mortality
Drugs that act directly on the renin-angiotensin system can cause fetal and neonatal morbidity and death when administered to pregnant women. Several dozen cases have been reported in the world literature in patients who were taking angiotensin converting enzyme inhibitors. When pregnancy is detected, telmisartan tablets should be discontinued as soon as possible.
The use of drugs that act directly on the renin-angiotensin system during the second and third trimesters of pregnancy has been associated with fetal and neonatal injury, including hypotension, neonatal skull hypoplasia, anuria, reversible or irreversible renal failure, and death. Oligohydramnios has also been reported, presumably resulting from decreased fetal renal function; oligohydramnios in this setting has been associated with fetal limb contractures, craniofacial deformation, and hypoplastic lung development.
Prematurity, intrauterine growth retardation, and patent ductus arteriosus have also been reported, although it is not clear whether these occurrences were due to exposure to the drug.
These adverse effects do not appear to have resulted from intrauterine drug exposure that has been limited to the first trimester. Mothers whose embryos and fetuses are exposed to an angiotensin II receptor antagonist only during the first trimester should be so informed. Nonetheless, when patients become pregnant, physicians should have the patients discontinue the use of telmisartan tablets as soon as possible.
Rarely (probably less often than once in every thousand pregnancies), no alternative to an angiotensin II receptor antagonist will be found. In these rare cases, the mothers should be apprised of the potential hazards to their fetuses, and serial ultrasound examinations should be performed to assess the intraamniotic environment.
If oligohydramnios is observed, telmisartan tablets should be discontinued unless they are considered life-saving for the mother. Contraction stress testing (CST), a non-stress test (NTS), or biophysical profiling (BPP) may be appropriate, depending upon the week of pregnancy. Patients and physicians should be aware, however, that oligohydramnios may be appear until after the fetus has sustained irreversible injury. Infants with histories of *in utero* exposure to an angiotensin II receptor antagonist should be closely observed for hypotension, oliguria, and hyperkalemia. If oliguria occurs, attention should be directed toward support of blood pressure and renal perfusion. Exchange transfusion or dialysis may be required as a means of reversing hypotension and/or substituting for disordered renal function.
There is no clinical experience with the use of telmisartan tablets in pregnant women. No teratogenic effects were observed when telmisartan was administered to pregnant rats at oral doses of up to 50 mg/kg/day and to pregnant rabbits at oral doses up to 45 mg/kg/day. In rabbits, embryolethality associated with maternal toxicity (reduced body weight gain and food consumption) was observed at 45 mg/kg/day [about 6.4 times the maximum recommended human dose (MRHD) of 80 mg on a mg/m^2 basis]. In rats, maternally toxic (reduction in body weight gain and food consumption) telmisartan doses of 15 mg/kg/day (about 1.9 times the MRHD on a mg/m^2 basis), administered during late gestation and lactation, were observed to produce adverse effects in neonates, including reduced viability, low birth weight, delayed maturation, and decreased weight gain. Telmisartan has been shown to be present in rat fetuses during late gestation and in rat milk. The no observed effect doses for developmental toxicity in rats and rabbits, 5 and 15 mg/kg/day, respectively, are about 0.64 and 3.7 times, on a mg/m^2 basis, the maximum recommended human dose of telmisartan (80 mg/day).

Fertility, pregnancy and lactation
Pregnancy and lactation
The use of angiotensin II receptor antagonists is not recommended during the first trimester of pregnancy and should not be initiated during pregnancy. When pregnancy is diagnosed, treatment with angiotensin II receptor antagonists should be stopped immediately, and, if appropriate, alternative therapy should be started.
The use of angiotensin II receptor antagonists is contraindicated during the second and third trimester of pregnancy. Preclinical studies with telmisartan do not indicate teratogenic effect, but have shown fetotoxicity. Angiotensin II receptor antagonists exposure during the second and third trimesters is known to induce human fetotoxicity (decreased renal function, oligohydramnios, skull ossification retardation) and neonatal toxicity (renal failure, hypotension, hyperkalemia). Unless continued angiotensin II receptor antagonist therapy is considered essential, patients planning pregnancy should be changed to alternative antihypertensive treatments which have an established safety profile for use in pregnancy.
Should exposure to angiotensin II receptor antagonists have occurred from the second trimester of pregnancy, ultrasound check of renal function and skull is recommended.
Infants whose mothers have taken angiotensin II receptor antagonists should be closely observed for hypotension.
Telmisartan is contraindicated during lactation since it is not known whether it is excreted in human milk. Animal studies have shown excretion of telmisartan in breast milk.

Fertility
No studies on fertility in humans have been performed.
In preclinical studies, an effect of telmisartan on male and female fertility was not observed.

Effects on ability to drive and use machines:
No studies on the effect on the ability to drive and use machines have been performed. However, when driving vehicles or operating machinery it should be taken into account that dizziness or drowsiness may occasionally occur when taking antihypertensive therapy.

Adverse reactions:
The incidence of adverse events was not dose related and showed no correlation with gender, age or race of the patients. The adverse drug reactions listed below have been accumulated from patients treated with telmisartan:
Infections and infestations
Urinary tract infections (including cystitis), upper respiratory tract infections, sepsis including fatal outcome.

Blood and lymphatic system disorders
Anemia, eosinophilia, thrombocytopenia.

Immune system disorders
Anaphylactic reaction, hypersensitivity.

Metabolism and nutrition disorders
Hyperkalemia, hypoglycemia (in diabetic patients).

Psychiatric disorders
Insomnia, depression, anxiety.

Nervous system disorders
Syncope (faint).

Eye disorders
Visual disturbance.

Ear and labyrinth disorders
Vertigo.

Cardiac disorders
Bradycardia, tachycardia.

Vascular disorders
Hypotension, orthostatic hypotension.

Respiratory disorders
Dyspnea.

Gastrointestinal disorders
Abdominal pain, diarrhea, dyspepsia, flatulence, vomiting, dry mouth, stomach discomfort.

Hepatobiliary disorders
Hepatic function abnormal/liver disorder*
*Most cases of hepatic function abnormal/liver disorder with telmisartan occurred in patients in Japan.

Skin and subcutaneous tissue disorders
Pruritus, hyperhidrosis, rash, angioedema (with fatal outcome), eczema, erythema, urticaria, drug eruption, toxic skin eruption.

Musculoskeletal, connective tissue and bone disorders
Back pain, muscle spasms (cramps in legs), myalgia, arthralgia, pain in extremity (leg pain), tendon pain (tendinitis like symptoms).

Renal and urinary tract disorders
Renal impairment including acute renal failure (see also under Warnings and precautions).

General disorders and administration site conditions
Chest pain, asthenia (weakness), influenza-like illness.

Investigations
Blood creatinine increased, hemoglobin decreased, blood uric acid increased, hepatic enzymes increased, blood creatine phosphokinase (CPK) increased.

Drug interactions:
Telmisartan may increase the hypotensive effect of other antihypertensive agents. Other interactions of clinical significance have not been identified.
Co-administration of telmisartan did not result in a clinically significant interaction with digoxin, warfarin, hydrochlorothiazide, glibenclamide, ibuprofen, paracetamol, simvastatin and amlodipine. For digoxin, a 20% increase in median plasma digoxin trough concentration has been observed (39% in a single case), monitoring of plasma digoxin levels should be considered.
In one study the co-administration of telmisartan and ramipril led to an increase of up to 2.5 fold in the AUC_{0-24} and C_{max} of ramipril and ramiprilat. The clinical relevance of this observation is not known.
Reversible increases in serum lithium concentrations and toxicity have been reported during concomitant administration of lithium with angiotensin converting enzyme inhibitors.
Cases have also been reported with angiotensin II receptor antagonists including telmisartan. Therefore, serum lithium level monitoring is advisable during concomitant use.
Treatment with NSAIDs (i.e. ASA at anti-inflammatory dosage regimens, COX-2 inhibitors and non-selective NSAIDS) is associated with the potential for acute renal insufficiency in patients who are dehydrated.
Compounds acting on the renin-angiotensin-system like telmisartan may have synergistic effects. Patients receiving NSAIDs and telmisartan should be adequately hydrated and be monitored for renal function at the beginning of combined treatment.
A reduced effect of antihypertensive drugs like telmisartan by inhibition of vasodilating prostaglandins has been reported during combined treatment with NSAIDs.
Dual blockade of the renin-angiotensin-aldosterone-system (RAAS) through the combined use of ACE-inhibitors, angiotensin II receptor blockers or aliskiren is associated with a higher frequency of adverse events such as hypotension, hyperkalemia and decreased renal function (including acute renal failure) compared to the use of a single RAAS-acting agent (see Contraindications and Warnings and precautions).

Clinical laboratory findings
Clinically relevant changes in standard laboratory test parameters were rarely associated with administration of telmisartan tablets.
Hemoglobin
A greater than 2 g/dl decrease in hemoglobin was observed in 0.8% telmisartan patients compared with 0.3% placebo patients. No patients discontinued therapy due to anemia.

Creatinine
A 0.5 mg/dl rise or greater in creatinine was observed in 0.4% telmisartan patients compared with 0.3% placebo patients. One telmisartan-treated patient discontinued therapy due to increases in creatinine and blood urea nitrogen.

Liver enzymes
Occasional elevations of liver chemistries occurred in patients treated with telmisartan; all marked elevations occurred at a higher frequency with placebo. No telmisartan-treated patients discontinued therapy due to abnormal hepatic function.

Overdosage:
Limited information is available with regard to overdose in humans. The most prominent manifestations of telmisartan overdose were hypotension and tachycardia, bradycardia also occurred. If symptomatic hypotension should occur, supportive treatment should be instituted. Telmisartan is not removed by hemodialysis.

Presentations:
TELSAT 40 mg: Box, 3 Alu/Alu blisters x 10 tablets; SIN16506P
TELSAT 80 mg: Box, 3 Alu/Alu blisters x 10 tablets; SIN16505P

ON MEDICAL PRESCRIPTION ONLY.

STORE AT TEMPERATURE BELOW 30ºC.

Manufactured by
PT Dexa Medica
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