SUMMARY OF PRODUCT CHARACTERISTICS

1. NAME OF THE MEDICINAL PRODUCT

TRIBEKSOL 250 mg/250 mg/1 mg film-coated tablet

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Active Substance:

Vitamin B_1 (Thiamine mononitrate)	250 mg
Vitamin B_6 (Pyridoxine hydrochloride)	250 mg
Vitamin B_{12}	1 mg

Excipient(s):

Anhydrous lactose (produced from cow's milk)	14 mg
Ponceau 4R	0,07 mg

For the full list of other excipients, see section 6.1.

3. PHARMACEUTICAL FORM

Film-coated tablet.

Pink, round, slightly curved, film-coated tablet with a characteristic (Vitamin B_1) odor.

4. CLINICAL PARTICULARS

4.1. Therapeutic Indications

- In patients with risk factors for deficiency of vitamins B_1, B_6, B_{12},
- Patients with general clinical symptoms such as mental and physical fatigue, forgetfulness, irritability, weakness, tremors,
- Neuritis, polyneuritis, diabetic neuropathy, neuralgia, shingles, tremor,
- In other painful conditions such as arthritis, periarthritis, sciatica, lumbalgia, arthralgia, myalgia, cramps and rheumatic pains,
- It is used as an adjunct in the treatment of cardiomyopathy, vomiting after surgery, radiation sickness, febrile rheumatism and chronic intoxication, especially during alcoholism.

4.2. Posology and method of administration

Posology/frequency and duration of treatment:

Unless otherwise recommended by a doctor;

For children over 12 years and adults: 1 film-coated tablet per day

The product is usually prescribed for one to several weeks. In some cases, the doctor may extend the treatment period for several months.

Method of administration:

Film-coated tablets should be taken orally and swallowed whole with liquid.

Additional information on special populations:

Renal/Hepatic failure:

Contraindicated in patients with renal or hepatic impairment due to high doses of B vitamins (see section 4.3).

Paediatric population:

Contraindicated in children under 12 years of age due to high doses of B vitamins (see section 4.3).

Geriatric population:

No specific dose recommendation is given.

Other:

Contraindicated during pregnancy and lactation due to high doses of B vitamins (see section. 4.3).

4.3. Contraindications

TRIBEKSOL is contraindicated in patients with known hypersensitivity to any of the substances in the composition of the medicine.

Due to the high dose of vitamin B_6, the product is contraindicated in the following cases:

- Pregnancy and lactation.
- Children under 12 years of age.
- Patients with renal or hepatic impairment.

4.4. Special warnings and precautions for use

The recommended dosage and duration of treatment should not be exceeded.

Due to the high levels of vitamin B_6 (pyridoxine hydrochloride), the product should not be taken at higher dose levels or for longer than recommended. If vitamin B_6 (pyridoxine hydrochloride) is not taken as recommended (overdose hazard, see section "Overdose"), serious neurotoxicity may occur.

Vitamin B_6, accelerates the breakdown of levodopa and reduces its effect. Therefore, in patients treated with levodopa, vitamin B_6 should not be used at a dose several times higher than the 2nhib requirement of 2 mg. This interaction does not occur when the patient is administered a peripheral decarboxylase 2nhibitör or a combination of levodopa and a peripheral decarboxylase 2nhibitör.

Vitamin B_{12} is not recommended for people with Leber's disease as it may increase the risk of optic atrophy.

Patients with severe megaloblastic anemia may develop hypokalemia, thrombocytosis and sudden death when treated intensively with vitamin B_{12}.

In case of decreased concentration of vitamin B_{12} or abnormal decrease in concentration with maximum dose intake, it may cause irreversible neurological damage if inadequate treatment is taken for more than 3 months.

Folate deficiency; not demonstrated but may compromise therapeutic response.

This medicinal product contains anhydrous lactose. Patients with rare hereditary galactose intolerance, Lapp lactose insufficiency or glucose-galactose malabsorption problems should not use this medicine.

May cause allergic reactions due to the ponceau 4R excipient.

4.5. Interaction with other medicinal product and other forms of interaction

Vitamin B_1 (thiamine):

- Thiosemicarbazone and 5-fluorouracil inhibit thiamine activity.
- Antacids inhibit thiamine absorption.

Laboratory test interferences

- Thiamine may cause false positive results in urobilinogen determination using the Ehrlich reagent.
- High thiamine doses may interfere with spectrophotometric determination of serum theophylline concentrations.

Vitamin B_6 (pyridoxine):

Various medicines interact with pyridoxine and can lead to lower pyridoxine levels. These medicines include:

- Cycloserine
- Hydralazines
- Isoniazid
- Desoxypyridoxine
- D-penicillamine
- Oral contraceptives
- Alcohol

High doses of vitamin B_6, inhibit the effect of levodopa. (see section 4.4).

Vitamin B_{12}:

Excessive alcohol intake for more than two weeks, aminosalicylates, colchicine, especially in combination with aminoglycosides, histamine (H2) receptor antagonists, metformin and related biguanides, oral contraceptives and proton pump inhibitors may reduce vitamin B_{12} absorption from the gastrointestinal tract; patients receiving these therapies have increased vitamin B_{12} requirements.

Antibiotics, serum and erythrocyte vitamin B_{12} concentration may affect the microbiological measurement method and cause false low results.

High and continuous doses of folic acid can lower vitamin B_{12} concentrations in the blood. Ascorbic acid can deplete vitamin B_{12}. Large amounts of vitamin C should be avoided 1 hour after oral intake of vitamin B_{12}.

Additional information on special populations:

Paediatric population:

No interaction study with TRIBEKSOL has been performed. Those listed above are based on bibliographic data.
TRIBEKSOL is also contraindicated for children under 12 years of age.

4.6. Pregnancy and lactation

General recommendation

Pregnancy Category: X

Women with childbearing potential/ Birth Control (Contraception)

Women of childbearing potential must use effective contraception during treatment.

Vitamin B_1, B_6, and/or B_{12} have no effect on hormonal contraception. However, there are no studies on other methods of contraception.

Pregnancy period

The medicine is contraindicated for use during pregnancy due to the high dose of vitamin B_6, which greatly exceeds the "Recommended Daily Dietary Allowance".

Lactation period

The product is contraindicated for use during lactation due to the high dose of vitamin B_6, which greatly exceeds the "Recommended Daily Dietary Allowance".

Reproductive ability/Fertility

There are no fertility studies with TRIBEKSOL.

4.7. Effects on ability to drive and use machines

No effect on the ability to drive and operate machinery has been observed.

4.8. Undesirable effects

The evaluation of undesirable effects is based on the following frequencies:
Very common (≥1/10); common (≥1/100 to <1/10); uncommon (≥1/1.000 to <1/100); rare (≥1/10.000 to <1/1.000); very rare (<10.000), unknown (cannot be estimated from available data).

The listed undesirable effects are based on spontaneous reports. Therefore, it is not possible to give frequency information for each of them.

Immune system diseases
Unknown: Allergic reactions may include urticaria, facial edema, wheezing, erythema, rashes and blisters.

Nervous system diseases
Unknown: Dizziness, headache, peripheral neuropathy, somnolence, paresthesia

Respiratory, chest and mediastinal disorders*
Unknown : Wheezing
*Only in the context of an allergic reaction

Gastrointestinal diseases
Unknown: Diarrhea, dyspepsia, nausea, abdominal pain

Skin and subcutaneous tissue diseases
Unknown: Rashes, erythema

Kidney and urinary tract diseases
Unknown: Abnormal urine odor

Studies
Unknown: Increased aspartate aminotransferase, decreased blood folate levels

Reporting suspected adverse reactions
Reporting of suspected adverse reactions after marketing authorisation is of great importance. Reporting allows the benefit/risk balance of the medicine to be monitored continuously. Healthcare professionals should report any suspected adverse reactions to the Turkish Pharmacovigilance Centre (TÜFAM). (www.titck.gov.tr; e-mail: tufam@titck.gov.tr; tel: 0 800 314 00 08; fax: 0 312 218 35 99)

4.9. Overdose and Treatment
There is no evidence that this product may cause an overdose if used as recommended.

Symptoms of an overdose include sensory neuropathy and neuropathy syndromes, nausea, headache, paresthesia, somnolence, increased serum AST levels (SGOT) and decreased serum folic acid concentrations. The effects resolve if treatment is stopped.

5. PHARMACOLOGICAL PARTICULARS
5.1. Pharmacodynamic properties
Pharmacotherapeutic group: Vitamin B complex

ATC code: A11DB
The active ingredients in TRIBEKSOL are vital for cellular energy production, protein and nucleic acid metabolism.

Vitamin B_1:
Thiamine pyrophosphate (TPP), the coenzymatic form of vitamin B_1, is involved in two main types of metabolic reactions: decarboxylation of a-ketoacids (e.g. pyruvate, a-ketoglutarate and branched-chain keto acids) and transketolation (e.g. between hexose and pentose phosphates). Thus, the major physiological role of vitamin B_1 is to act as a coenzyme in carbohydrate metabolism, where TPP is required for several steps in the breakdown of glucose to provide energy.

Besides its metabolic role as a co-enzyme, vitamin B_1 also plays a role in neurotransmitter function and nerve transmission.

Vitamin B_1, in high doses and especially in combination with vitamins B_6 and B_{12}, suppresses the transmission of neural stimuli and may thus exert an analgesic effect.

The early stages of vitamin B_1, deficiency may be accompanied by non-specific symptoms that may be overlooked or easily misinterpreted. Clinical signs of insufficiency include anorexia; weight loss; mental changes such as apathy, short-term memory loss, confusion and irritability; muscle weakness; and cardiovascular effects such as enlarged heart.

Conditions that often accompany marginal vitamin B_1, deficiency and require supplementation are regular alcohol consumption, high carbohydrate intake and heavy physical exertion.

Functional consequences of severe vitamin B_1, deficiency include cardiac failure, muscle weakness and peripheral and central neuropathy. The symptoms of clinical beriberi (severe vitamin B_1 deficiency) change with age. Adults may show dry (paralytic or nervous), wet (cardiac) or cerebral (Wernicke-Korsakoff syndrome) forms of beriberi. These conditions should be treated immediately with vitamin B_1. Severe cases of vitamin B_1, deficiency in industrialized countries are likely to be associated with limited food consumption and high alcohol consumption. In such cases, renal and cardiovascular complications are life- threatening.

Vitamin B_6:
Vitamin B_6 is a coenzyme for more than 100 enzymes involved in amino acid and protein metabolism, including aminotransferases, decarboxylases, racemases and dehydratases. It is a coenzyme for 8-aminolevulinate synthase, which catalyzes the first step in heme biosynthesis, and for cystathionine B-synthase and cystathioninase, enzymes involved in the transsulfuration pathway from homocysteine to cysteine. Most of the total vitamin B_6 in the body is found in the phosphorylase muscle bond.

A high rate of amino acid production and breakdown is an important parameter for the efficient and effective functioning of the central nervous system; therefore, an adequate and appropriate supply of vitamin B_6 is required. It plays an important role in the synthesis of biogenic amines and neurotransmitters in the brain. The conversion of glutamic acid to GABA (an inhibitory neurotransmitter in the central nervous system) requires vitamin B_6, as a coenzyme. Vitamin B_6, is also required for the conversion of tyrosine to dopamine and noradrenaline, tryptophan to 5-hydroxytryptamine and histidine to histamine.

The classic clinical symptoms of vitamin B_6, insufficiency are:
Skin and mucosal lesions, such as seborrheic dermatitis, glossitis and buccal erosions. Peripheral neuritis with nerve degeneration causing sensory disturbances, polyneuropathies.
Cerebral convulsions with electroencephalographic abnormalities.
Hypochromic anemia with microcytosis.
Impaired lymphocyte proliferation and maturation, antibody production and T-cell activity.
Depression and confusion.
Impaired platelet function and coagulation mechanisms.

Vitamin B6 deficiency is also observed because many drugs act as pyridoxine antagonists. These include:

- Cycloserine, an antibiotic
- Hydralazines
- Isoniazid, a tuberculostatic
- Desoxypyridoxine, an antimetabolite
- D-penicillamine, a copper-binding agent
- Oral contraceptives
- Alcohol

Other factors that cause a decrease in vitamin B_6, are diseases and pathological conditions such as asthma, diabetes, renal disorders, heart disease and breast cancer. A number of conditions are treated with higher doses of vitamin B_6 (premenstrual syndrome, carpal tunnel syndrome, depressions and diabetic neuropathy).

Vitamin B_6, available in concentrations between 30-100 mg/day, is often prescribed as a first-line treatment for nausea and vomiting in pregnancy.

Vitamin B_6, administered in appropriate doses also protects patients against the side effects of radiation therapy.

<u>Vitamin B_{12} (cobalamin):</u>
Vitamin B_{12} is a cofactor for two enzymes: Methionine synthase (a reaction important for tetrahydrofolic acid regeneration) and L-methylmalonyl-CoA mutase. Methionine synthase requires methylcobalamin as a cofactor for the methyl transfer from methyltetrahydrofolate to homocysteine, ultimately forming methionine and tetrahydrofolate. L-Methylmalonyl-CoA

mutase requires adenosylcobalamin to ultimately convert L-methylmalonyl-CoA to succinyl-CoA in an isomerization reaction. In B_{12} deficiency, folate may accumulate in the serum as a result of the slowdown of B_{12}-dependent methyltransferase. An appropriate and sufficient supply of B_{12} is important for normal blood formation and neurological function. Vitamin B_{12} is a cofactor for catechol-O-methyl transferase, which plays an important role in the degradation of catecholamines in the synaptic cleft, namely noradrenaline and dopamine.

High doses of vitamin B_{12} (in combination with vitamins B_1 and B_6) have an analgesic effect.

The major cause of clinically observable B_{12} deficiency is pernicious anemia. The hematologic effects of B_{12} are skin coldness associated with a gradual onset of common anemia symptoms such as decreased energy and exercise tolerance, fatig, shortness of breath and palpitations. The underlying mechanism of anemia is an interference with normal deoxyribonucleic acid (DNA) synthesis. Hematologic complications resolve completely with treatment with B_{12} supplementation.

Neurological complications are present in 75-90% of people with clinically observable B_{12} deficiency, and only about 25% of cases may be a clinical manifestation of B_{12} deficiency. Neurological symptoms include sensory disturbances (tingling and numbness) in the extremities (more in the lower extremities). The senses of vibration and position are particularly affected. Motor disorders occur, including gait abnormalities. Cognitive changes may occur, ranging from loss of concentration to memory loss, disorientation and overt dementia, with or without mood changes. There may also be visual disturbances, insomnia, impotence, impaired bowel and bladder control. The progression of neurologic symptoms varies but is usually gradual. Whether neurologic complications improve after treatment depends on the duration of their presence.

Special attention should be paid to patients at risk of vitamin B_{12} deficiency:

- Elderly people
- Vegans and vegetarians
- HIV positive patients
- Patients with gastrointestinal diseases
- Patients with autoimmunity or a family history of pernicious anemia

TRIBEKSOL, which is a combination of vitamins B_1, B_6 and B_{12} in pharmacological doses, shows antalgic, antineuritic, detoxifying and antianemic properties. These vitamins, which are also found together in nature, complement each other in terms of their functions on the metabolism of cells, especially the cells of the nervous system. As a result, TRIBEKSOL has an effect far above the effect that can be achieved by using these vitamins individually.

5.2. Pharmacokinetic properties

General characteristics

Vitamin B_1 (thiamine):

Absorption:

Vitamin B_1, is rapidly absorbed in humans, mostly in the proximal small intestine. There are two mechanisms, one by carrier-based transport at low physiological concentrations (< 2 mcm) and one by passive diffusion at higher concentrations. Absorption is generally high, but intestinal absorption in humans is rate-limited.

The need for vitamin B_1, is directly related to the intake of carbohydrates: 0.5 mg per 1,000 caloriesA high-calorie and especially high-carbohydrate diet increases the associated thiamine requirement.

Distribution:

The total average amount of vitamin B_1, in adult humans is about 30 mg. The organ with the highest content is usually the heart (0.28-0.79 mg per 100 g), followed by the kidney (0.24- 0.58), liver (0.20-0.76) and brain (0.14-0.44). Vitamin B_1, levels in the spinal cord and brain are about twice those in the peripheral nerves. Whole blood vitamin B_1, ranges from 5 to 12 mcg/100 mL; 90% is found in red blood cells and leukocytes. The concentration in leukocytes is 10 times higher than the concentration in red blood cells. The rate of production and breakdown of vitamin B_1, in the body is relatively high and it is never stored in large amounts in tissues. It therefore needs to be in constant supply. Inadequate intake can cause biochemical and then clinical signs of insufficiency. When vitamin B_1, intake is about 60 mcg per 100 g body weight (or 42 mg per 70 kg) and total body vitamin B_1 reaches 2 mcg/g (or 140 mg per 70 kg), a plateau level is reached in most tissues.

Two other mechanisms are involved in vitamin B_1, transport across the blood-brain barrier. However, the saturable mechanism at the blood-brain barrier is different from the energy-dependent mechanism seen in the intestine and the active transport system in the cells of the cerebral cortex, which relies on membrane-bound phosphatases.

Biotransformation:

Oral (or parenteral) thiamine is rapidly converted in tissues to disphosphate and to a lesser extent to triphosphate esters. All vitamin B_1, in excess of tissue needs, binding and storage capacity is rapidly excreted in the urine. In rats, parenteral intake of 10 mcg/100 mg body weight (or 70 mg per 7 kg) of thiamine has proven to be sufficient for proliferation, but less than normal tissue levels. Stimulation of nerves causes release of thiamine or monophosphate with a concomitant decrease in tri and diphosphatases.

Excretion:

Vitamin B_1 is excreted in urine. In humans, there is a small increase in urinary vitamin B_1 excretion when oral doses higher than 2.5 mg are administered. The half-life of vitamin B_1 in the

body is 10-20 days. In addition to free vitamin B_1 and small amounts of thiamine diphosphate, thiochrome, and thiamine disulfide, approximately 20 or more vitamin B_1 metabolites have been reported in the urine of rats and humans.
have been identified, but only six of them have been actually identified and described. The ratio of metabolites relative to excreted vitamin B_1 increases with decreasing vitamin B_1 intake.

Vitamin B_6 (pyridoxine):
Absorption:
The various dietary forms of vitamin B_6, are absorbed by intestinal mucosal cells, mainly in the jejunum and ileum (intestine), via passive diffusion-dependent phosphorylation; hence its large capacity.

Distribution:
B_6 forms are converted to pyridoxal phosphate (PLP) and pyridoxamine phosphate (PMP) in the liver, erythrocytes and other tissues. These compounds are distributed throughout animal tissues, but none are stored. Phosphorylase, the enzyme that converts glycogen to glucose-l- phosphate, contains most of the body's vitamin B_6. About half of the vitamin B_6 present in the body can reflect the phosphorylase of skeletal muscle. PLP can be found in plasma as a PLP- albumin complex and in erythrocytes bound to hemoglobin. The concentration of PL in erythrocytes is four to five times as large as the concentration found in plasma.

Biotransformation:
In particular, PLP and PMP function as coenzymes in transamination reactions; in particular, PLP acts as a cofactor for many enzymes involved in the synthesis or catabolism of amino acids. PLP is also involved in the decarboxylation and racemization of A-amino acids, in other metabolic transformations of amino acids and in the metabolism of lipids and nucleic acids. It is also the essential coenzyme for glycogen phosphorylase (IOM Vitamin B_6, 1998). Pyridoxal phosphate is also required for the synthesis of 8-aminolevulinic acid, a precursor of hemin.

Excretion:
Normally, the major excretion product is 4-pyridoxic acid, which accounts for about half of the B_6, compounds in urine. With higher doses of vitamin B_6, the proportion of other forms of vitamin B_6 also increases. At very high doses of pyridoxine, most of the dose is excreted unchanged in the urine. B_6 is probably also excreted in limited amounts in faeces, but is difficult to quantify due to microbial synthesis of B_6, in the intestine.

Vitamin B_{12} (cobalamin):
Absorption:
As mentioned above, the terms vitamin B_{12} and cobalamin refer to all members of a group of compounds (corrinoids) that contain high amounts of cobalt. Corrinoids can be converted into two cobalamin coenzymes that are active in human metabolism. Cyanocobalamin is a commercially available form of vitamin B_{12}.

Cobalamins are absorbed by two different mechanisms: an active mechanism (protein-based) and a diffusion-type mechanism. A healthy stomach absorbs small amounts of vitamin B_{12} through the active process, which requires intrinsic factor (a glycoprotein secreted by the parietal cells of the stomach after stimulation by food), pancreatic competence and a normally functioning terminal ileum. In the stomach, food-bound B_{12} is dissociated from proteins by acid and pepsin. The released B_{12} is then bound to R proteins (haptocorrins) secreted by salivary glands and gastric mucosa. In the small intestine, pancreatic proteases partially degrade R proteins and the released B_{12} binds to intrinsic factor. The resulting complex of intrinsic factor and B_{12} binds to specific receptors on the ileal mucosa; after internalization of the complex, B_{12} enters the enterocyte. Approximately 3 to 4 hours later, B_{12} enters the circulation. At dose levels up to 10 mcg, the efficiency of this mechanism is approximately 50%. At dose levels higher than 10 mcg, the efficiency and effectiveness of absorption decreases. The second absorption mechanism occurs by diffusion at a very low rate and is approximately It shows a yield and efficacy corresponding to 1% and only at oral doses exceeding 100 meg does it achieve a quantitatively statistically significant result.

If there is a deficiency in intrinsic factor (as in pernicious anemia), B_{12} malabsorption occurs; if this disorder is left untreated, potentially irreversible neurological damage and life- threatening anemia result.

Distribution:

The predominant forms in plasma and tissue are methylcobalamin, adenosylcobalamin and hydroxocobalamin. Methylcobalamin accounts for 60% to 80% of total plasma cobalamin. In normal humans, cobalamins are found mainly in the liver, where the average amount is 1.5 mg. The kidneys, heart, spleen and brain each contain about 20-30 mcg. Average values for total body content calculated for adult humans are 2- 5 mg. The pituitary gland has the highest concentration per gram of any organ/tissue. Adenosylcobalamin is the major cobalamin among all cellular tissuesaccounting for about 60-70% in the liver and about 50% in other organs.

Biotransformation:

As vitamin B_{12} passes through the intestinal mucosait is transferred to the plasma transporter protein transcobalamin II, which delivers the vitamin to cells. The specific biochemical reactions in which cobamide coenzymes are involved are of two types: (1) those containing 5-deoxyadenosine covalently attached to the cobalt atom (adenosylcobalamin) and (2) those with a methyl group attached to the central cobalt atom (methylcobalamin). The coenzyme methylcobalamin catalyzes a transmethylation from a folic acid cofactor to homocysteine, forming methionine. This reaction frees up the unmethylated folate cofactor for other single carbon transfer reactions important for nucleic acid synthesis. The other cobalamin coenzyme, deoxyadenosylcobalamin, catalyzes the conversion of methylmalonyl-coenzyme A to succinyl-coenzyme A, a reaction in the pathway for the degradation of certain amino acids and single-chain fatty acids.

Vitamin B_{12} deficiency causes macrocytic, megaloblastic anemia, neurological symptoms from demyelination of the spinal cord, brain, optic and peripheral nerves, and other less specific symptoms (e.g. tongue pain, weakness). In the absence of anemia, and especially in the elderly, neuropsychiatric manifestations of vitamin B_{12} insufficiency occur.

Excretion:
The urinary, biliary and fecal routes are the main excretion pathways. Only unbound plasma cobalamin is available for urinary excretion and therefore urinary excretion via glomerular filtration of free cobalamin is minimal: Variably at levels up to 0.25 mcg per day. Approximately 0.5-5 mcg of cobalamin per day is secreted into the digestive tract, mainly in bile; at least 65 - 75% is reabsorbed in the ileum via the intrinsic factor mechanism. This effective recycling of B_{12} enterohepatic circulation through bile and other intestinal secretory channels does not function in the presence of pernicious anemia due to a deficiency in intrinsic factor activity. Total loss in the body ranges from 2 to 5 mcg per day. Thus, the daily loss of vitamin B_{12} corresponds to approximately 0.1% of the body pool (range 0.05-0.2%), regardless of the total size.

5.3. Preclinical safety data
There are no specific studies with this product, the preclinical safety of its individual components has been widely documented.

6. PHARMACEUTICAL PARTICULARS
6.1. List of excipients
Povidone K 90 (Plasdone K 90)
Microcrystalline cellulose (Avicel pH 112 SLM)
Croscarmellose sodium (Ac-di-sol)
Anhydrous lactose (produced from cow's milk)
Magnesium stearate

Film coating agents:
Opadry 0Y-24931 pink

- Titanium dioxide
- Polyethylene glycol 400
- Ponceau 4R
- Indigocarmine
- Quinoline yellow

6.2. Incompatibilities
Not relevant.

6.3. Shelf life
48 months

6.4. Special precautions for storage
Store at room temperature under 25°C. Protect from moisture.

6.5. Nature and contents of container
30 and 50 film tablets in PVC/PVDC Al blister pack

6.6. Disposal of residues of human medicinal products and other special precautions
Unused products or waste materials must be disposed of in accordance with the 'Regulation on the control of medical waste' and 'Regulation on the control of packaging and packaging waste'.

7. MARKETING AUTHORISATION HOLDER
DEVA HOLDİNG A.Ş.
Halkalı Merkez Mah.Basın Ekspres Cad. No: 1
34303 Küçükçekmece - İstanbul
Tel: 0 212 692 92 92
Fax: 0 212 697 00 24

8. MARKETING AUTHORISATION NUMBER(S)
139/67

9. DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION
Date of first authorisation: 29.07.1986

10. DATE OF REVISION OF SPC