STARCOX FILM-COATED TABLETS 60MG
STARCOX FILM-COATED TABLETS 90MG
STARCOX FILM-COATED TABLETS 120MG

Name and Strength of Active Ingredient
STARCOX FILM-COATED TABLETS 60MG:
Each film-coated tablet contains Etoricoxib 60mg

STARCOX FILM-COATED TABLETS 90MG:
Each film-coated tablet contains Etoricoxib 90mg

STARCOX FILM-COATED TABLETS 120MG:
Each film-coated tablet contains Etoricoxib 120mg

Product Description

STARCOX FILM-COATED TABLETS 60MG is available as white colored, round shaped, biconvex film coated tablet engraved GP on one side and plain on other side.

STARCOX FILM-COATED TABLETS 90MG is available as reddish brown colored, round shaped, biconvex film coated tablet “GETZ” debossed on one side and plain on other side.

STARCOX FILM-COATED TABLETS 120MG is available as reddish brown colored, round shaped, biconvex film coated tablet with “GP” debossed on one side and plain on other side.

Pharmacodynamics

Etoricoxib is a nonsteroidal anti-inflammatory drug (NSAID) that exhibits anti-inflammatory, analgesic, and antipyretic activities. It is a potent, orally active, highly selective cyclooxygenase-2 (COX-2) inhibitor within and above the clinical dose range. Two isoforms of cyclooxygenase have been identified: cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2). COX-1 is responsible for prostaglandin-mediated normal physiologic functions such as gastric cytoprotection and platelet aggregation. Inhibition of COX-1 by nonselective NSAIDs has been associated with gastric damage and platelet inhibition. COX-2 has been shown to be primarily responsible for the synthesis of prostanoid mediators of pain, inflammation, and fever. Selective inhibition of COX-2 by etoricoxib decreases these clinical signs and symptoms with decreased GI toxicity and without effects on platelet function.

Across reported clinical pharmacology studies, etoricoxib produced dose-dependent inhibition of COX-2 without inhibition of COX-1 at doses up to 150 mg daily.

The influence on gastroprotective COX-1 activity was also assessed in a reported clinical study where prostaglandin synthesis was measured in gastric biopsy samples from subjects administered either etoricoxib 120 mg daily, naproxen 500 mg twice daily, or placebo.

Etoricoxib did not inhibit gastric prostaglandin synthesis as compared to placebo. In contrast, naproxen inhibited gastric prostaglandin synthesis by approximately 80% compared with placebo. These data further support the COX-2 selectivity of etoricoxib.

Platelet Function

It was reported that multiple doses of etoricoxib up to 150 mg administered daily up to nine days had no effect on bleeding time relative to placebo. Similarly, bleeding time was not altered in a single dose study with etoricoxib 250 or 500 mg. There was no inhibition of ex vivo arachidonic acid- or collagen-induced platelet aggregation at steady state with doses of etoricoxib up to 150 mg. These findings are consistent with the COX-2 selectivity of etoricoxib.

Pharmacokinetics

Absorption

The onset of action occurred as early as 24 minutes after dosing.

Orally administered etoricoxib is well absorbed. It was reported that the mean oral bioavailability is approximately 100%. Following 120 mg once-daily dosing to steady state, the peak plasma concentration was observed at approximately 1 hour after administration to fasted adults. The pharmacokinetics of etoricoxib are linear across the clinical dose range.

A standard meal had no clinically meaningful effect on the extent or rate of absorption of a dose of etoricoxib 120 mg.

Distribution

Etoricoxib is approximately 92% bound to human plasma protein over the range of concentrations of 0.05 to 5 mcg/mL. The volume of distribution at steady state (V_{dss}) is approximately 120 L in humans.

Etoricoxib crosses the placenta in rats and rabbits, and the blood-brain barrier in rats.

Metabolism

Etoricoxib is extensively metabolized with <1% of a dose recovered in urine as the parent drug. The major route of metabolism to form the 6'-hydroxymethyl derivative is catalyzed by cytochrome P450 (CYP) enzymes.

Five metabolites have been identified in man. The principal metabolite is the 6'-carboxylic acid derivative of etoricoxib formed by further oxidation of the 6'-hydroxymethyl derivative. These principal metabolites either demonstrate no measurable activity or are only weakly active as COX-2 inhibitors. None of these metabolites inhibit COX-1.

Elimination

Elimination of etoricoxib occurs almost exclusively through metabolism followed by renal excretion. Steady state concentrations of etoricoxib are reached within seven days of once-

daily administration of 120 mg, with an accumulation ratio of approximately 2, corresponding to an accumulation half-life of approximately 22 hours. The plasma clearance is estimated to be approximately 50 mL/min.

Indication

STARCOX tablets are indicated for:
• Acute and chronic treatment of the signs and symptoms of osteoarthritis (OA) and rheumatoid arthritis (RA)
• Treatment of ankylosing spondylitis (AS)
• Treatment of acute gouty arthritis
• Chronic low back pain (60 mg only)
• Treatment of acute pain, including that related to primary dysmenorrhoea and minor dental procedures.
The decision to prescribe a selective COX-2 inhibitor should be based on an assessment of the individual patient's overall risks.

Recommended Dose

STARCOX is administered orally. STARCOX may be taken with or without food.
STARCOX should be administered for the shortest duration possible and the lowest effective daily dose should be used.

Osteoarthritis
The recommended dose is 30 mg or 60 mg once daily.

Rheumatoid Arthritis
The recommended dose is 60 mg once daily. In some patients with insufficient relief from symptoms, an increased dose of 90 mg once daily may increase efficacy. Once the patient is clinically stabilised, down-titration to a 60 mg once daily dose may be appropriate. In the absence of an increase in therapeutic benefit, other therapeutic options should be considered.

Ankylosing Spondylitis
The recommended dose is 60 mg once daily. In some patients with insufficient relief from symptoms, an increased dose of 90 mg once daily may increase efficacy. Once the patient is clinically stabilised, down-titration to a 60 mg once daily dose may be appropriate. In the absence of an increase in therapeutic benefit, other therapeutic options should be considered.

Chronic low back pain
The recommended dose is 60 mg once daily.

Acute Pain
In the following acute painful conditions, STARCOX should be used only for the acute symptomatic period, limited to a maximum of 8 days treatment:

Acute Gouty Arthritis
The recommended dose is 120 mg once daily.

Primary Dysmenorrhea
The recommended dose is 120 mg once daily.

Minor Dental Procedures
The recommended dose is 90 mg once daily.

Doses greater than those recommended for each indication have either not demonstrated additional efficacy or have not been studied. Therefore:

The dose for OA should not exceed 60 mg daily.
The dose for RA should not exceed 90 mg daily.
The dose for ankylosing spondylitis should not exceed 90 mg daily.
The dose for acute gout should not exceed 120 mg daily.
The dose for acute pain and primary dysmenorrhea should not exceed 120 mg daily.
The dose of chronic low back pain should not exceed 60 mg daily.
The dose for minor dental procedures should not exceed 90 mg daily.

Given the association between cardiovascular risk and exposure to COX-2 Inhibitors, doctors are advised to use the lowest effective dose for the shortest possible duration of treatment. The patient's need for symptomatic relief and response to therapy should be re-evaluated periodically.

Elderly, Gender, Race
No dosage adjustment in STARCOX is necessary for the elderly or based on gender or race.

Hepatic Insufficiency
In patients with mild hepatic insufficiency (Child-Pugh score 5-6), a dose of 60 mg once daily should not be exceeded. In patients with moderate hepatic insufficiency (Child-Pugh score 7-9), the dose should be reduced; a dose of 60 mg every other day should not be exceeded, administration of 30 mg once daily can also be considered. There are no clinical or pharmacokinetic data in patients with severe hepatic insufficiency (Child-Pugh score >9).

Renal Insufficiency
In patients with advanced renal disease (creatinine clearance <30 mL/min), treatment with STARCOX is not recommended. No dosage adjustment is necessary for patients with lesser degrees of renal insufficiency (creatinine clearance ≥30 mL/min).

STARCOX is available at the strengths of 60mg, 90mg and 120mg only and may not be able to deliver all the dosing recommendations mentioned above. In such cases, other approved strengths should be used.

Contraindication

Etoricoxib is contra-indicated in patients with:

– hypersensitivity to any component of this product.

– congestive heart failure (NYHA II-IV)

– established ischemic heart disease, peripheral arterial disease, and/or cerebrovascular disease (including patients who have recently undergone coronary artery bypass graft surgery or angioplasty)

– hypertension (high blood pressure) whose blood pressure is not under control

– pregnancy

– increased risk of cardiovascular disease (ischeamic heart disease and stroke).

Warning and Precautions

Reported clinical trials suggest that the selective COX-2 inhibitor class of drugs may be associated with an increased risk of thrombotic events (especially MI and stroke), relative to placebo and some NSAIDs (naproxen). As the cardiovascular risks of selective COX-2 inhibitors may increase with dose and duration of exposure, the shortest duration possible and the lowest effective daily dose should be used. The patient's need for symptomatic relief and response to therapy should be re- evaluated periodically.

Patients with significant risk factors for cardiovascular events (e.g. hypertension, hyperlipidaemia, diabetes mellitus, smoking) should only be treated with etoricoxib after careful consideration.

Selective COX-2 inhibitors are not a substitute for aspirin for cardiovascular prophylaxis because of their lack of effect on platelets. Because etoricoxib, a member of this class, does not inhibit platelet aggregation, antiplatelet therapies should not be discontinued.

There is a further increase in the risk of gastrointestinal adverse effects (gastrointestinal ulceration or other gastrointestinal complications) for etoricoxib, other selective COX-2 inhibitors and NSAIDs, when taken concomitantly with acetylsalicylic acid (even at low doses). The relative difference in gastrointestinal safety between selective COX-2 inhibitors + acetylsalicylic acid vs. NSAIDs + acetylsalicylic acid has not been adequately evaluated in long-term clinical trials.

In patients with advanced renal disease, treatment with etoricoxib is not recommended. Clinical experience in patients with estimated creatinine clearance of <30 mL/min is very limited. If

therapy with etoricoxib must be initiated in such patients, close monitoring of the patient's renal function is advisable.

Long-term administration of NSAIDs has resulted in renal papillary necrosis and other renal injury. Renal prostaglandins may play a compensatory role in the maintenance of renal perfusion. Therefore, under conditions of compromised renal perfusion, administration of etoricoxib may cause a reduction in prostaglandin formation and, secondarily, in renal blood flow, and thereby impair renal function. Patients at greatest risk of this response are those with pre-existing significantly impaired renal function, uncompensated heart failure, or cirrhosis. Monitoring of renal function in such patients should be considered.

Caution should be used when initiating treatment with etoricoxib in patients with considerable dehydration. It is advisable to rehydrate patients prior to starting therapy with etoricoxib.

As with other drugs known to inhibit prostaglandin synthesis, fluid retention, edema and hypertension have been observed in some patients taking etoricoxib. The possibility of fluid retention, edema or hypertension should be taken into consideration when etoricoxib is used in patients with pre-existing edema, hypertension, or heart failure. All Nonsteroidal Antiinflammatory Drugs (NSAIDs), including etoricoxib, can be associated with new onset or recurrent congestive heart failure. (see SIDE EFFECTS.). Etoricoxib may be associated with more frequent and severe hypertension than some other NSAIDs and selective COX-2 inhibitors, particularly at high doses. Therefore, special attention should be paid to blood pressure monitoring during treatment with etoricoxib. If blood pressure rises significantly, alternative treatment should be considered.

Physicians should be aware that individual patients may develop upper gastrointestinal (GI) ulcers/ulcer complications irrespective of treatment. Upper GI ulcers/ulcer complications have occurred in patients treated with etoricoxib. These events can occur at any time during use and without warning symptoms. Independent of treatment, patients with a prior history of GI perforation, ulcers and bleeding (PUB) and patients greater than 65 years of age are known to be at a higher risk for a PUB.

Elevations of alanine aminotransferase (ALT) and/or aspartate aminotransferase (AST) (approximately three or more times the upper limit of normal) have been reported in approximately 1% of patients in clinical trials treated for up to one year with etoricoxib 30, 60 and 90 mg daily.

A patient with symptoms and/or signs suggesting liver dysfunction, or in whom an abnormal liver function test has occurred, should be evaluated for persistently abnormal liver function tests. If persistently abnormal liver function tests (three times the upper limit of normal) are detected, etoricoxib should be discontinued.

Etoricoxib should be used with caution in patients who have previously experienced acute asthmatic attacks, urticaria, or rhinitis, which were precipitated by salicylates or non-selective cyclooxygenase inhibitors. Since the pathophysiology of these reactions is unknown,

physicians should weigh the potential benefits of prescribing etoricoxib versus the potential risks.

When using etoricoxib in the elderly and in patients with renal, hepatic, or cardiac dysfunction, medically appropriate supervision should be maintained. If these patients deteriorate during treatment, appropriate measures should be taken, including discontinuation of therapy.

Serious skin reactions, some of them fatal, including exfoliative dermatitis, Stevens-Johnson syndrome, and toxic epidermal necrolysis, have been reported very rarely in association with the use of NSAIDs and some selective COX-2. These serious events may occur without warning. Patients appear to be at highest risk for these reactions early in the course of therapy: the onset of the reaction occurring in the majority of cases within the first month of treatment. Serious hypersensitivity reactions (such as anaphylaxis and angioedema) have been reported in patients receiving etoricoxib. Some selective COX-2 inhibitors have been associated with an increased risk of skin reactions in patients with a history of any drug allergy. Etoricoxib should be discontinued at the first appearance of skin rash, mucosal lesions, or any other sign of hypersensitivity.

Etoricoxib may mask fever, which is a sign of infection. The physician should be aware of this when using etoricoxib in patients being treated for infection.

Warning to prescriber when prescribing COX-2 Inhibitors to patients with risk factors of heart disease, hypertension (high blood pressure), hyperlipidemia, diabetes, smoking patient and patient with peripheral arterial disease.

Risk of GI Ulceration, Bleeding and Perforation with NSAID

Serious GI toxicity such as bleeding, ulceration and perforation can occur at any time, with or without warning symptoms, in patients treated with NSAID therapy. Although minor upper GI problems (e.g. dyspepsia) are common, usually developing early in therapy, prescribers should remain alert for ulceration and bleeding in patients treated with NSAIDs even in the absence of previous GI tract symptoms.

Studies to date have not identified any subset of patients not at risk of developing peptic ulceration and bleeding. Patients with prior history of serious GI events and other risk factors associated with peptic ulcer disease (e.g. alcoholism, smoking, and corticosteroid therapy) are at increased risk. Elderly or debilitated patients seem to tolerate ulceration or bleeding less than other individuals and account for most spontaneous reports for fatal GI events.

Interactions With Other Medicaments

Warfarin: In subjects stabilized on chronic warfarin therapy, the administration of etoricoxib 120 mg daily was associated with an approximate 13% increase in prothrombin time International Normalized Ratio (INR). Standard monitoring of INR values should be conducted when therapy with etoricoxib is initiated or changed, particularly in the first few days, in patients receiving warfarin or similar agents.

Rifampin: Co-administration of etoricoxib with rifampin, a potent inducer of hepatic metabolism, produced a 65% decrease in etoricoxib plasma area under the curve (AUC). This interaction should be considered when etoricoxib is co-administered with rifampin.

Methotrexate: Etoricoxib at 60 and 90 mg had no effect on methotrexate plasma concentrations or renal clearance. Monitoring for methotrexate-related toxicity should be considered when etoricoxib at doses greater than 90 mg daily and methotrexate are administered concomitantly.

Diuretics, Angiotensin Converting Enzyme (ACE) Inhibitors and Angiotensin II Antagonists (AIIAs): Reports suggest that NSAIDs including selective COX-2 inhibitors may diminish the antihypertensive effect of diuretics, ACE inhibitors and AIIAs. This interaction should be given consideration in patients taking etoricoxib concomitantly with these products.

In some patients with compromised renal function (e.g., elderly patients or patients who are volume-depleted, including those on diuretic therapy) who are being treated with non-steroidal anti-inflammatory drugs, including selective COX-2 inhibitors, the co-administration of ACE inhibitors or AIIAs may result in a further deterioration of renal function, including possible acute renal failure. These effects are usually reversible. Therefore, the combination should be administered with caution, especially in the elderly.

Lithium: Reports suggest that non-selective NSAIDs and selective COX-2 inhibitors may increase plasma lithium levels. This interaction should be given consideration in patients taking etoricoxib concomitantly with lithium.

Aspirin: Etoricoxib can be used concomitantly with low-dose aspirin at doses for cardiovascular prophylaxis. However, concomitant administration of low-dose aspirin with etoricoxib increases the rate of GI ulceration or other complications compared to use of etoricoxib alone.

Oral Contraceptives: It was reported etoricoxib 60mg given concomitantly with an oral contraceptive containing 35 mcg ethinyl estradiol (EE) and 0.5 to 1 mg norethindrone for 21 days increased the steady state $AUC_{0\text{-}24hr}$ of EE by 37%. Etoricoxib 120 mg given with the same oral contraceptive either concomitantly or separated by 12 hours, increased the steady state $AUC_{0\text{-}24hr}$ of EE by 50 to 60%. This increase in EE concentration should be considered when selecting an appropriate oral contraceptive for use with etoricoxib. An increase in EE exposure can increase the incidence of adverse events associated with oral contraceptives (e.g., venous thrombo-embolic events in women at risk).

Hormone Replacement Therapy: There were reported increases in estrogenic concentration when etoricoxib was administered with conjugated estrogen. These should be taken into consideration when selecting post-menopausal hormone therapy for use with etoricoxib.

Other: In drug-interaction studies, etoricoxib did not have clinically important effects on the pharmacokinetics of prednisone/prednisolone or digoxin.

Antacids and ketoconazole (a potent inhibitor of CYP3A4) did not have clinically important effects on the pharmacokinetics of etoricoxib.

Pregnancy, Lactation, Pediatric and Elderly

Pregnancy

The use of etoricoxib, as with any drug substance known to inhibit COX-2, is not recommended in women attempting to conceive. No clinical data on exposed pregnancies are available for etoricoxib. The potential for human risk in pregnancy is unknown. Etoricoxib, as with other medicinal products inhibiting prostaglandin synthesis, may cause uterine inertia and premature closure of the ductus arteriosus during the last trimester. Etoricoxib is contraindicated in pregnancy. If a woman becomes pregnant during treatment, etoricoxib must be discontinued.

Lactation

It is not known whether etoricoxib is excreted in human milk. Because many drugs are excreted in human milk and because of the possible adverse effects of drugs that inhibit prostaglandin synthesis on nursing infants, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use

Safety and effectiveness of etoricoxib in pediatric patients have not been established.

Use in the Elderly

Pharmacokinetics in the elderly (65 years of age and older) are similar to those in the young. It was reported, a higher incidence of adverse experiences was seen in older patients compared to younger patients; the relative differences between etoricoxib and control groups were similar in the elderly and the young. Greater sensitivity of some older individuals cannot be ruled out.

Side Effects

In clinical trials, etoricoxib was evaluated for safety in 9295 individuals, including 5774 patients with OA, RA or chronic low back pain (approximately 600 patients with OA or RA were treated for one year or longer).

The following drug-related adverse experiences were reported in clinical studies in patients with OA, RA, or chronic low back pain treated for up to 12 weeks. These occurred in ≥1% of patients treated with etoricoxib and at an incidence greater than placebo: asthenia/fatigue, dizziness, lower extremity edema, hypertension, dyspepsia, heartburn, nausea, headache, ALT increased, AST increased.

The adverse experience profile was similar in patients with OA or RA treated with etoricoxib for one year or longer.

In the MEDAL Study, an endpoint driven CV outcomes trial involving 23, 504 patients, the safety of etoricoxib 60 or 90 mg daily was compared to diclofenac 150 mg daily in patients

with OA or RA (mean duration of treatment was 20 months). In this large trial, only serious adverse events and discontinuations due to any adverse events were recorded. The rates of confirmed thrombotic cardiovascular serious adverse events were similar between etoricoxib and diclofenac. The incidence of discontinuations for hypertension-related adverse events was less than 3% in each treatment group; however, etoricoxib 60 and 90 mg demonstrated significantly higher rates of discontinuations for these events than diclofenac. The incidence of congestive heart failure adverse events (discontinuations and serious events) and the incidence of discontinuations due to edema occurred at similar rates on etoricoxib 60 mg compared to diclofenac, however, the incidences for these events were higher for etoricoxib 90 mg compared to diclofenac. The incidence of discontinuations due to atrial fibrillation was higher for etoricoxib compared to diclofenac.

The EDGE and EDGE II studies compared the GI tolerability of etoricoxib 90 mg daily (1.5 to 3 times the doses recommended for OA) and diclofenac 150 mg daily in 7111 patients with OA (EDGE Study; mean duration of treatment 9 months) and 4086 patients with RA (EDGE II; mean duration of treatment 19 months). In each of these studies, the adverse experience profile on etoricoxib was generally similar to that reported in the phase IIb/III placebo-controlled clinical studies; however, hypertension and edema-related adverse experiences occurred at a higher rate on etoricoxib 90 mg than on diclofenac 150 mg daily. The rate of confirmed thrombotic cardiovascular serious adverse events occurring in the two treatment groups was similar.

In a combined analysis of phase IIb to V clinical studies of 4 weeks duration or longer (excluding the MEDAL PROGRAM Studies), there was no discernible difference in the rate of confirmed thrombotic cardiovascular serious adverse events between patients receiving etoricoxib ≥30 mg or non-naproxen NSAIDs. The rate of these events was higher in patients receiving etoricoxib compared with those receiving naproxen 500 mg twice daily.

In a clinical study for ankylosing spondylitis, patients were treated with etoricoxib 90 mg once daily for up to 1 year (N=126). In another clinical study for ankylosing spondylitis (N=857), patients were treated with etoricoxib 60 mg or 90 mg once daily for up to 26 weeks. The adverse experience profile in these studies was generally similar to that reported in chronic studies in OA, RA and chronic low back pain.

In a clinical study for acute gouty arthritis, patients were treated with etoricoxib 120 mg once daily for eight days. The adverse experience profile in this study was generally similar to that reported in the combined OA, RA, and chronic low back pain studies.

In initial clinical studies for acute analgesia, patients were treated with etoricoxib 120 mg once daily for one to seven days. The adverse experience profile in these studies was generally similar to that reported in the combined OA, RA, and chronic low back pain studies.

In the combined studies for acute post-operative dental pain, the incidence of post-dental extraction alveolitis (dry socket) reported in patients treated with etoricoxib was similar to that of patients treated with active comparators.

Post-marketing experience

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

Blood and lymphatic system disorders: thrombocytopenia.

Immune system disorders: hypersensitivity reactions, anaphylactic/anaphylactoid reactions including shock.

Metabolism and nutrition disorders: hyperkalemia.

Psychiatric disorders: anxiety, insomnia, confusion, hallucinations, depression, restlessness.

Nervous system disorders: dysgeusia, somnolence, intracranial hemorrhage.

Eye disorders: blurred vision.

Cardiac disorders: congestive heart failure, palpitations, angina, arrhythmia.

Vascular disorders: hypertensive crisis, deep vein thrombosis.

Respiratory, thoracic and mediastinal disorders: bronchospasm, pulmonary embolism.

Gastrointestinal disorders: abdominal pain, oral ulcers, peptic ulcers including perforation and bleeding (mainly in elderly patients), vomiting, diarrhea.

Hepatobiliary disorders: hepatitis, jaundice, hepatic failure.

Skin and subcutaneous tissue disorders: angioedema, pruritus, erythema, rash, Stevens-Johnson syndrome, toxic epidermal necrolysis, urticaria, fixed drug eruption.

Renal and urinary disorders: renal insufficiency, including renal failure

Symptoms and Treatment of Overdose

It has been reported that administration of etoricoxib at single doses up to 500 mg and multiple doses up to 150 mg/day for 21 days did not result in significant toxicity. There have been reports of acute overdosage with etoricoxib, although adverse experiences were not reported in the majority of cases. The most frequently observed adverse experience were consistent with the safety profile for etoricoxib. (e.g. gastrointestinal events, renovascular events).

In the event of overdose, it is reasonable to employ the usual supportive measures, e.g., remove unabsorbed material from the gastrointestinal tract, employ clinical monitoring, and institute supportive therapy, if required.

Etoricoxib is not dialyzable by hemodialysis; it is not known whether etoricoxib is dialyzable by peritoneal dialysis.

Effects on Ability to Drive and Use Machine

No studies on the effect of etoricoxib on the ability to drive or use machines have been performed. However, patients who experience dizziness, vertigo or somnolence while taking etoricoxib should refrain from driving or operating machinery.

Storage Condition
Do not store above 30°C.

Protect from sunlight and moisture.

Dosage form and packaging available

STARCOX FILM-COATED TABLETS 60MG are available in blister pack of 3x10's.

STARCOX FILM-COATED TABLETS 90MG are available in blister pack of 3x10's.

STARCOX FILM-COATED TABLETS 120MG are available in blister pack of 3x10's.

Manufacturer
Getz Pharma (Pvt.) Ltd.,
Plot 29-30, Sector-27, Korangi Industrial Area, 74900 Karachi, Pakistan.

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Arial font with minimum font size of 6.