CENTER FOR DRUG EVALUATION AND RESEARCH

Approval Package for:

Application Number    74929

Trade Name  Etodolac Capsules 300mg

Generic Name  Etodolac Capsules 300mg

Sponsor  Aesgen, Inc.
**APPLICATION 74929**

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APPLICATION FOR DRUG EVALUATION AND RESEARCH

Application Number 74929

APPROVAL LETTER
Aesgen Inc.
Attention: Suzanne Yu; U.S. Agent
5051 New Centre Drive
Suite 103
Wilmington, NC 28403

Dear Madam:

This is in reference to your abbreviated new drug application dated July 23, 1996, submitted pursuant to Section 505(j) of the Federal Food, Drug, and Cosmetic Act, for Etodolac Capsules, 300 mg.


We have completed the review of this abbreviated application and have concluded that the drug is safe and effective for use as recommended in the submitted labeling. Accordingly, the application is approved. The Division of Bioequivalence has determined your Etodolac Capsules, 300 mg to be bioequivalent and, therefore, therapeutically equivalent to the listed drug (Lodine® Capsules, 300 mg of Wyeth Ayerst Laboratories, Inc.). Your dissolution testing should be incorporated into the stability and quality control program using the same method proposed in your application.

Under 21 CFR 314.70, certain changes in the conditions described in this abbreviated application require an approved supplemental application before the change may be made.

Post-marketing reporting requirements for this abbreviated application are set forth in 21 CFR 314.80-81. The Office of Generic Drugs should be advised of any change in the marketing status of this drug.

We request that you submit, in duplicate, any proposed advertising or promotional copy which you intend to use in your initial advertising or promotional campaigns. Please submit all proposed materials in draft or mock-up form, not final print. Submit both copies together with a copy of the proposed or final printed labeling to the Division of Drug Marketing, Advertising, and Communications (HFD-240). Please do not use Form FD-2253 (Transmittal of Advertisements and Promotional Labeling for Drugs for Human Use) for this initial submission.
We call your attention to 21 CFR 314.01(b)(3) which requires that materials for any subsequent advertising or promotional campaign be submitted to our Division of Drug Marketing, Advertising, and Communications (HFD-240) with a completed Form FD-2253 at the time of their initial use.

Sincerely yours,

Douglas L. Sporn
Director
Office of Generic Drugs
Center for Drug Evaluation and Research
CENTER FOR DRUG EVALUATION AND RESEARCH

APPLICATION NUMBER 74929

FINAL PRINTED LABELING
ETODOLAC Capsules

300 mg

CAUTION: Federal law prohibits dispensing without prescription.

100 Capsules

ETODOLAC Capsules

300 mg

CAUTION: Federal law prohibits dispensing without prescription.

500 Capsules
Etodolac is a nonsteroidal anti-inflammatory drug (NSAID) that exhibits anti-inflammatory, analgesic, and antipyretic activities in animal models. The mechanism of action of etodolac, like that of other NSAIDs, is not known but is believed to be associated with the inhibition of cyclooxygenase, the enzyme responsible for converting arachidonic acid to prostaglandins. This mechanism is distinct from the actions of corticosteroids, which do not inhibit cyclooxygenase.

Pharmacology
Etodolac is a nonsteroidal anti-inflammatory drug (NSAID) that exhibits anti-inflammatory, analgesic, and antipyretic activities in animal models. The mechanism of action of etodolac, like that of other NSAIDs, is not known but is believed to be associated with the inhibition of cyclooxygenase, the enzyme responsible for converting arachidonic acid to prostaglandins. This mechanism is distinct from the actions of corticosteroids, which do not inhibit cyclooxygenase.

Each tablet, for oral administration, contains 200 mg of etodolac. In addition, the tablets contain the following inactive ingredients: hypromellose, lactose monohydrate, magnesium stearate, microcrystalline cellulose, polyethylene glycol, povidone, sodium lauryl sulfate, sodium starch glycolate, titanium dioxide, and yellow iron oxide.

Pharmacokinetics
The pharmacokinetics of etodolac have been evaluated in 18 normal subjects, 6 elderly patients (aged 65 years or older), 5 patients with congestive heart failure, and 10 patients with congestive heart failure and hypertension. Etodolac, when administered orally, exhibits kinetics that are well described by a two-compartment model with first-order absorption.

Etodolac has no apparent pharmacokinetic interaction when administered with piroxicam, piroxicam, or indomethacin.

Absorption
Etodolac is well absorbed and has a relative bioavailability of 100% when 200 mg capsules were compared with a 200 mg tablet. The time to reach peak serum concentration is approximately 1.5 to 3 hours. The absolute bioavailability of etodolac from either the tablet or capsule formulation is at least 87%. Etodolac does not undergo significant first-pass metabolism following oral administration. Mean (± SD) peak plasma concentrations range from approximately 14 ± 4 to 37 ± 9 ng/mL, with 500 to 1000 mg single doses and 500 to 1000 mg twice-daily doses, respectively. The bioavailability of etodolac is dose-dependent. The area under the plasma concentration-time curve is linearly dose-dependent up to 600 mg every 12 hours. Peak concentrations are dose-proportional for both the total and free etodolac following doses up to 400 mg every 12 hours, but following a 600 mg dose the area under the plasma concentration-time curve was approximately 20% higher than predicted on the basis of lower doses.

Table 1: Etodolac Steady-State Pharmacokinetic Parameters
(Kn-177)

<table>
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<th>Kinetic Parameters</th>
<th>Mean (± SD)</th>
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<tr>
<td>Exposure of oral absorption</td>
<td>47 ± 16 mg L/mg h</td>
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<tr>
<td>Distribution half-life (t1/2, d)</td>
<td>0.32 (0.28-0.37)</td>
</tr>
<tr>
<td>Terminal half-life (t1/2, d)</td>
<td>1.6 (1.3-1.9)</td>
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Eblexol was well absorbed and had a relative bioavailability of 100% when 250 mg capsules were compared with a solution of etodolac. Based on mean balance studies, the systemic availability of etodolac from either the solution or capsules is at least 90%, which suggests that etodolac is not undergo significant first-pass metabolism following oral administration. Mean (± SD) peak plasma concentrations range from approximately 14 ± 4.4 to 37 ± 9 ng/mL after 500 to 800 mg single doses and are reached in 8.0 ± 3.0 minutes (see Table 1 for summary of pharmacokinetic parameters). The mean peak time was 1.2 ± 1.0 hours (range 0.5 to 3.8 hours). Elimination: The elimination half-life of etodolac is 2.5 ± 0.9 hours. Metabolism: Etodolac is extensively metabolized in the liver, with several metabolites excreted in the urine (see Table 2). The major metabolites were glucuronide and hydroxylation products. The primary metabolic pathways included glucuronidation and hydroxylation. Elimination: The mean plasma clearance of etodolac, following oral dosing at 250 mg, is 47 ± 16 mL/min and terminal disposition half-life is 7.5 ± 4.0 hours. Other effects of etodolac on food absorption were not evaluated. A single dose of etodolac did not alter test meal absorption and absorption was not significantly affected by food intake. The mean bioavailability of etodolac was 92% after a single oral dose of 250 mg administered with food. Effects on QT interval: A single dose of etodolac (250 mg) had no significant effect on the QT interval in a clinical study. Effects on platelet aggregation: The mean bleeding time of 20 healthy volunteers was 3.8 ± 0.5 minutes after placebo and 4.0 ± 0.4 minutes after etodolac 250 mg, demonstrating that etodolac has no significant effect on platelet function. Effects on coagulation: Prothrombin time, activated partial thromboplastin time, and platelet count were not altered by a single oral dose of etodolac. Effects on homocysteine levels: The effects of etodolac on homocysteine levels were evaluated in a randomized, double-blind, placebo-controlled study in patients with coronary artery disease. Eto- dolac did not significantly affect plasma homocysteine levels. Effects on other laboratory tests: Eto- dolac had no significant effect on liver function tests, renal function tests, or electrolyte levels. Effects on hematologic parameters: Eto- dolac had no significant effect on hematologic parameters. Effects on tumor necrosis factor-α (TNF-α): The effects of etodolac on TNF-α levels were evaluated in a randomized, double-blind, placebo-controlled study in patients with moderate to severe ulcerative colitis. Eto- dolac 200 mg was administered for 3 days and TNF-α levels were measured at baseline and at 24, 48, and 72 hours after the start of treatment. Eto- dolac 200 mg significantly reduced TNF-α levels compared to placebo (p < 0.05). Effects on platelet aggregation: The effects of etodolac on platelet aggregation were evaluated in a randomized, double-blind, placebo-controlled study in patients with unstable angina. Eto- dolac 200 mg was administered for 3 days and platelet aggregation was measured at baseline and at 24, 48, and 72 hours after the start of treatment. Eto- dolac 200 mg significantly reduced platelet aggregation compared to placebo (p < 0.05). Effects on coagulation: The effects of etodolac on coagulation parameters were evaluated in a randomized, double-blind, placebo-controlled study in patients with unstable angina. Eto- dolac 200 mg was administered for 3 days and coagulation parameters were measured at baseline and at 24, 48, and 72 hours after the start of treatment. Eto- dolac 200 mg had no significant effect on coagulation parameters. Effects on tumor necrosis factor-α (TNF-α): The effects of etodolac on TNF-α levels were evaluated in a randomized, double-blind, placebo-controlled study in patients with moderate to severe ulcerative colitis. Eto- dolac 200 mg was administered for 3 days and TNF-α levels were measured at baseline and at 24, 48, and 72 hours after the start of treatment. Eto- dolac 200 mg significantly reduced TNF-α levels compared to placebo (p < 0.05).
patients treated chronically with NSAIDs, and the risk is in the absence of previous GI tract symptoms. In patients treated with chronic NSAIDS, especially those with previous GI problems, risk factors for bleeding include: (1) previous bleeding or peptic ulcer disease; (2) concurrent use of aspirin or other NSAIDS; (3) concurrent use of any corticosteroid, alcohol, or tobacco; (4) concurrent use of warfarin or other anticoagulants; (5) concurrent use of heparin or other thrombolytics; and (6) concurrent use of any other medications known to cause GI bleeding. In such patients, the risk of bleeding may be higher, and the use of GI protective agents (e.g., histamine 2 blockers, proton pump inhibitors, or sucralfate) may be considered. It is important to monitor patients for signs of GI bleeding and to stop the medication if bleeding occurs.

Exposure to Other NSAIDS and the Risk of GI Bleeding: Patients who are already taking other NSAIDS should be asked whether they are currently taking any other NSAIDS. If so, the other NSAIDS should be stopped, and the patient should be switched to naproxen sodium. If this is not possible, the dose of each NSAID should be reduced to the lowest effective dose.

Hypersensitivity Reactions: Hypersensitivity reactions, including anaphylactic reactions, have been reported with the use of NSAIDS. These reactions may occur in patients with or without a history of hypersensitivity to aspirin or other NSAIDS. Patients should be monitored for symptoms of anaphylaxis, such as rash, itching, or difficulty breathing. If a hypersensitivity reaction occurs, the medication should be stopped immediately.

Skin Reactions: Skin reactions, including rashes, urticaria, and photosensitivity, have been reported with the use of NSAIDS. Patients should be monitored for these reactions, and the medication should be stopped if a skin reaction occurs.

Urinary Tract Reactions: Urinary tract reactions, including hematuria, proteinuria, and renal insufficiency, have been reported with the use of NSAIDS. Patients should be monitored for these reactions, and the medication should be stopped if a urinary tract reaction occurs.

Liver Reactions: Liver reactions, including jaundice, hepatitis, and liver failure, have been reported with the use of NSAIDS. Patients should be monitored for these reactions, and the medication should be stopped if a liver reaction occurs.

Nervous System Reactions: Nervous system reactions, including headache, dizziness, and somnolence, have been reported with the use of NSAIDS. Patients should be monitored for these reactions, and the medication should be stopped if a nervous system reaction occurs.

Respiratory Reactions: Respiratory reactions, including bronchospasm, asthma, and rhinitis, have been reported with the use of NSAIDS. Patients should be monitored for these reactions, and the medication should be stopped if a respiratory reaction occurs.

Gastrointestinal Reactions: Gastrointestinal reactions, including nausea, vomiting, diarrhea, and constipation, have been reported with the use of NSAIDS. Patients should be monitored for these reactions, and the medication should be stopped if a gastrointestinal reaction occurs.

Other Reactions: Other reactions, including fever, myalgia, arthralgia, and pruritus, have been reported with the use of NSAIDS. Patients should be monitored for these reactions, and the medication should be stopped if an other reaction occurs.

In general, naproxen sodium is well tolerated by most patients. However, as with all medications, patients should be monitored for adverse reactions. If a serious adverse reaction occurs, the medication should be stopped immediately.
About 14% of patients with asthma may have asymptomatic anaphylaxis. The use of sympathomimetics has been associated with severe reactions in patients with acute asthma and a history of anaphylaxis. These reactions include bronchospasm, wheezing, and edematous swelling of the upper airway. In some cases, it has even been observed that patients with asthma who have had an acute asthma attack may experience anaphylactic symptoms during treatment. This is why the use of sympathomimetics should be carefully monitored in patients with asthma who have a history of anaphylaxis.

The treatment of patients with asthma should be based on the severity of the patient's symptoms and the response to previous treatment. In general, patients with mild to moderate asthma can be treated with inhaled corticosteroids or long-acting bronchodilators. In patients with severe asthma, combination therapy with inhaled corticosteroids and long-acting bronchodilators may be necessary. It is important to note that the treatment of asthma should be individualized for each patient, and the choice of medication should be based on the patient's response to treatment and the severity of their symptoms.

In addition to pharmacological treatment, patients with asthma should also be advised to avoid triggers of their asthma, such as smoke, pollution, and allergens. It is important to educate patients about the importance of regular follow-up appointments and the importance of adhering to their prescribed treatment regimen. With proper management, asthma can be effectively controlled and the patient can lead a normal life.
Phenylbutazone capsules increase (by about 50%) the coagulation time in vitro in platelet rich plasma. Phenylbutazone in vitro does not increase the PT/INR and PTT. However, it is recom- mended that these tests be monitored with other anticoagulant therapy.

Strong-Laboratory Test (Intervention).

The area of patients who have taken phenylbutazone can give a labile response in vitro. Phenylbutazone may cause a decrease in the coagulation time in vitro in platelet rich plasma. Phenylbutazone in vivo does not cause a change in the PT/INR or PTT.

Thrombocytopenia has been observed in 15% of patients treated with phenylbutazone. No specific cause has been identified in these patients. Phenylbutazone in vivo does not cause a decrease in the number of platelets. Phenylbutazone in vivo does not cause an increase in the number of platelets. No specific cause has been identified in these patients.

Phenylbutazone has not been associated with other laboratory sig- nals. Phenylbutazone in vivo does not cause a change in the number of leukocytes. Phenylbutazone in vivo does not cause a change in the number of eosinophils.

Phenylbutazone in vivo does not cause an increase in the number of reticulocytes. Phenylbutazone in vivo does not cause an increase in the number of lymphocytes. Phenylbutazone in vivo does not cause an increase in the number of monocytes.

Phenylbutazone in vivo does not cause a change in the number of neutrophils. Phenylbutazone in vivo does not cause a change in the number of basophils. Phenylbutazone in vivo does not cause a change in the number of eosinophils.

Phenylbutazone in vivo does not cause a change in the number of platelets. Phenylbutazone in vivo does not cause a change in the number of lymphocytes. Phenylbutazone in vivo does not cause a change in the number of neutrophils. Phenylbutazone in vivo does not cause a change in the number of eosinophils.

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Phenylbutazone in vivo does not cause a change in the number of eosinophils.
OVERDOSE

Erections following acute NSAID overdose are usually limited to tachycardia, tachypnea, nausea, vomiting, and anorexia; none of which are generally reversible with standard medical therapy. However, a small number of patients may develop respiratory compromise. In such cases, intubation and ventilatory support should be considered. If intubation is necessary, bronchoscopy, hyperventilation, and respiratory support should be provided as needed. In cases of severe respiratory compromise, intubation and mechanical ventilation may be required. Patients with severe respiratory compromise should be referred to a critical care unit for further management.

IN CASE OF OVERDOSE

1. Induce vomiting if done within 1 hour of ingestion.
2. If patient is unresponsive, provide assisted ventilation and respiratory support.
3. Ensure adequate oxygenation and ventilation.
4. If intubation is necessary, perform bronchoscopy and intubation as soon as possible.
5. Monitor renal function closely, as NSAIDs can cause renal failure.
6. Provide supportive care, including intravenous fluids and monitoring for signs of dehydration.
7. Consider hemodialysis if renal failure is severe.
8. Provide symptomatic treatment for any untoward effects.

DOSE AND ADMINISTRATION

As with other NSAIDs, the lowest effective dose and dosage interval should be used for each patient. Dosage adjustments should be made based on the patient's response to therapy and the presence of any untoward effects. If necessary, dosage adjustments can be made to achieve the desired therapeutic effect.

Infants and Children:

- For children weighing less than 10 kg, the recommended starting dose is 0.2 mg/kg/day in two divided doses, with a minimum of 6 hours between doses.
- For children weighing 10 kg to 20 kg, the recommended starting dose is 0.5 mg/kg/day in two divided doses, with a minimum of 6 hours between doses.
- For children weighing more than 20 kg, the recommended starting dose is 1 mg/kg/day in two divided doses, with a minimum of 6 hours between doses.

ADVERSE REACTIONS

The most common adverse reactions associated with ibuprofen are nausea, vomiting, diarrhea, headache, dizziness, and GI disturbances. Other adverse reactions include allergic reactions, skin reactions, and hematological reactions. Patients should be monitored closely for any untoward effects.

Especial: Gastrointestinal bleeding and perforation have been reported with the use of NSAIDs, and the risk is increased with higher doses of ibuprofen. Patients with a history of peptic ulcer disease or with a history of NSAID-induced GI bleeding are at increased risk.

Patient Information

- Instruct patients to take the medicine exactly as prescribed, and to report any untoward effects promptly.
- Advise patients to avoid taking NSAIDs with alcohol or other drugs that may increase the risk of gastrointestinal bleeding.
- Inform patients of the potential for serious adverse reactions, including allergy, anaphylaxis, and anaphylactoid reactions.

Epidemiological and Statistical

The epidemiological and statistical data on the safety and effectiveness of ibuprofen are based on clinical trials involving a large number of patients. The data indicate that ibuprofen is well tolerated and effective in the treatment of acute pain and inflammation.

Patient Counseling

- Inform patients of the potential for patient drug interactions, including those involving aspirin and other NSAIDs.
- Advise patients to report any untoward effects promptly, including those that may be severe or life-threatening.

Manufactured by

Aesas

Princeton, NJ 08540

By

Aesas PHARMACEUTICAL CORPORATION

Capitol, PA 19010, USA

Item #355000000

Rev 10/07

New 10/97

Distributed by

Aesas PHARMACEUTICAL CORPORATION

Capitol, PA 19010, USA

Item #355000000

Rev 10/07

New 10/97
CENTER FOR DRUG EVALUATION AND RESEARCH

APPLICATION NUMBER 74929

CHEMISTRY REVIEW(S)
1. CHEMISTRY REVIEW NO 3

2. ANDA 74-929

3. NAME AND ADDRESS OF APPLICANT
   Aesgen Inc.
   Attention: Jeffrey S. Bauer
   5051 New Centre Drive
   Suite 103
   Wilmington, NC 28403

4. LEGAL BASIS FOR SUBMISSION
   Lodine® Capsules, 300 mg
   (Wyeth Ayerst, NDA 18-922, patent expired 01/31/91;
   exclusivity for treatment of arthritis until 06/28/99.)

5. SUPPLEMENT(s) N/A

6. PROPRIETARY NAME N/A

7. NONPROPRIETARY NAME
   Etodolac Capsules, 300 mg

8. SUPPLEMENT(s) PROVIDE(s) FOR N/A

9. AMENDMENTS AND OTHER DATES

10. PHARMACOLOGICAL CATEGORY
    NSAID

11. Rx or OTC Rx

12. RELATED IND/NDA/DMF(s)

13. DOSAGE FORM
    Capsules

14. POTENCY
    300 mg

15. CHEMICAL NAME AND STRUCTURE
    (±)-1,8-Diethyl-1,3,4,9-tetrahydro-
    pyrano[3,4-b]indole-1-acetic acid

    C₁₉H₁₉NO₃

    M.W. = 287.36

    [41340-25-4]

16. RECORDS AND REPORTS N/A

17. COMMENTS

18. CONCLUSIONS AND RECOMMENDATIONS
    Recommend: APPROVAL. (That is, if it weren't for
    unresolved dissolution issues, and labeling).


cc: ANDA 74-929
DIV FILE
Field Copy
Reading File

Endorsements:
HFD-623/J.Smith/ 1/25/97
HFD-623/V.Sayeed/
HFD-617/J.Wilson, PM/
Y:\NEW\FIRMSAM\AESGEN\LTAS\REV\74929AP3.CD
F/T by: gp/1/2/98