INFLAMMATION REDUCTION PACK- inflammation reduction pack
TMIG, Inc.

Inflamation Reduction Pack

Kit Description

Capsaicin Cream 0.025%

Ranitidine Tablet USP 150mg

Diclofenac Sodium Tablet 75mg

Ranitidine hydrochloride

Ranitidine hydrochloride (HCl), is a histamine H2-receptor antagonist. Chemically it is N-[2-[[[5-[(dimethylamino)methyl]-2-furanyl]methyl]thio]ethyl]-N'-methyl-2-nitro-1,1-ethenediamine, HCl.

It has the following structure:

ranitidine hydrochloride chemical structure
The empirical formula is C13H22N4O3S • HCl, representing a molecular weight of 350.87.

Ranitidine HCl is a white to pale yellow, granular substance that is soluble in water. It has a slightly bitter taste and sulfur like odor.

Each tablet, for oral administration contains 168 mg or 336 mg of ranitidine hydrochloride equivalent to 150 mg and 300 mg of ranitidine, respectively. Inactive ingredients: D & C Red #30 Aluminum Lake, hydroxypropyl cellulose, hydroxypropyl methylcellulose, magnesium stearate, microcrystalline cellulose, triethyl citrate, sodium starch glycolate, titanium dioxide and flavoring. The 300 mg also contains: D & C Yellow #10 Aluminum Lake.

Each capsule, for oral administration contains 168 mg or 336 mg of ranitidine hydrochloride equivalent to 150 mg and 300 mg of ranitidine, respectively. Inactive ingredients: Ammonium hydroxide, colloidal silicon dioxide, corn starch, FD & C Blue #1, FD & C Red #40, FD & C Yellow #6, gelatin, magnesium stearate, pharmaceutical glaze, propylene glycol, silicon dioxide, simethicone, sodium lauryl sulfate, sodium starch glycolate, and titanium dioxide.

Diclofenac sodium

Diclofenac sodium delayed-release tablets, USP are a benzeneacetic acid derivative. The chemical name is 2-[(2,6-dichlorophenyl)amino] benzeneacetic acid, monosodium salt. The molecular weight is 318.13. Its molecular formula is C14H10Cl2NNaO2, and it has the following structural formula

78e77986-figure-01
Each enteric-coated tablet for oral administration contains 50 mg or 75 mg of diclofenac sodium, USP. In addition, each tablet contains the following inactive ingredients: aluminum hydrate, colloidal silicon dioxide, hypromellose, lactose monohydrate, magnesium stearate, microcrystalline cellulose, polyethylene glycol, polysorbate 80, polyvinyl acetate phthalate, propylene glycol, silica, sodium alginate, sodium starch glycolate (Type A), stearic acid, synthetic black iron oxide, talc, and titanium dioxide.

Capsaisin 0.025%

For external use only
Read all warnings and directions before use. Test first on small area of skin.

Do not use
on wounds or damaged skin
if you are allergic to capsicum or chili peppers
When using this product
you may experience a burning sensation. The intensity of this reaction varies among individuals and may be severe. With regular use, this sensation generally disappears after several days.
avoid contact with the eyes, lips, nose and mucous membranes
do not tightly wrap or bandage the treated area
do not apply heat to the treated area immediately before or after use
Stop use and ask a doctor if
condition worsens or does not improve after regular use
severe burning persists or blistering occurs
Keep out of reach of children. If swallowed, get medical help or contact a Poison Control Center immediately.

Diclofenac Sodium

Cardiovascular Effects
Cardiovascular Thrombotic Events

Clinical trials of several COX-2 selective and nonselective NSAIDs of up to three years duration have shown an increased risk of serious cardiovascular (CV) thrombotic events, myocardial infarction, and stroke, which can be fatal. All NSAIDs, both COX-2 selective and nonselective, may have a similar risk. Patients with known CV disease or risk factors for CV disease may be at greater risk. To minimize the potential risk for an adverse CV event in patients treated with an NSAID, the lowest effective dose should be used for the shortest duration possible. Physicians and patients should remain alert for the development of such events, even in the absence of previous CV symptoms. Patients should be informed about the signs and/or symptoms of serious CV events and the steps to take if they occur.

There is no consistent evidence that concurrent use of aspirin mitigates the increased risk of serious CV thrombotic events associated with NSAID use. The concurrent use of aspirin and an NSAID does increase the risk of serious GI events (see WARNINGS, GI EFFECTS).

Two large, controlled, clinical trials of a COX-2 selective NSAID for the treatment of pain in the first 10-14 days following CABG surgery found an increased incidence of myocardial infarction and stroke (see CONTRAINDICATIONS).

Hypertension
NSAIDs can lead to onset of new hypertension or worsening of pre-existing hypertension, either of which may contribute to the increased incidence of CV events. Patients taking thiazides or loop diuretics may have impaired response to these therapies when taking NSAIDs. NSAIDs, including diclofenac sodium delayed-release tablets, USP should be used with caution in patients with hypertension. Blood pressure (BP) should be monitored closely during the initiation of NSAID treatment and throughout the course of therapy.

Congestive Heart Failure and Edema
Fluid retention and edema have been observed in some patients taking NSAIDs. Diclofenac sodium delayed-release should be used with caution in patients with fluid retention or heart failure.

Gastrointestinal (GI) Effects - Risk Of GI Ulceration, Bleeding, And Perforation
NSAIDs, including diclofenac sodium delayed-release, can cause serious gastrointestinal (GI) adverse events including inflammation, bleeding, ulceration, and perforation of the stomach, small intestine, or large intestine, which can be fatal. These serious adverse events can occur at any time, with or without warning symptoms, in patients treated with NSAIDs. Only one in five patients, who develop a serious upper GI adverse event on NSAID therapy, is symptomatic. Upper GI ulcers, gross bleeding, or perforation caused by NSAIDs occur in approximately 1% of patients treated for 3-6 months, and in

about 2%-4% of patients treated for one year. These trends continue with longer duration of use, increasing the likelihood of developing a serious GI event at some time during the course of therapy. However, even short-term therapy is not without risk.

NSAIDs should be prescribed with extreme caution in those with a prior history of ulcer disease or gastrointestinal bleeding. Patients with a prior history of peptic ulcer disease and/or gastrointestinal bleeding who use NSAIDs have a greater than 10-fold increased risk for developing a GI bleed compared to patients with neither of these risk factors. Other factors that increase the risk for GI bleeding in patients treated with NSAIDs include concomitant use of oral corticosteroids or anticoagulants, longer duration of NSAID therapy, smoking, use of alcohol, older age, and poor general health status. Most spontaneous reports of fatal GI events are in elderly or debilitated patients and therefore, special care should be taken in treating this population.

To minimize the potential risk for an adverse GI event in patients treated with an NSAID, the lowest effective dose should be used for the shortest possible duration. Patients and physicians should remain alert for signs and symptoms of GI ulceration and bleeding during NSAID therapy and promptly initiate additional evaluation and treatment if a serious GI adverse event is suspected. This should include discontinuation of the NSAID until a serious GI adverse event is ruled out. For high risk patients, alternate therapies that do not involve NSAIDs should be considered.

Renal Effects

Caution should be used when initiating treatment with diclofenac sodium delayed-release in patients with considerable dehydration.

Long-term administration of NSAIDs has resulted in renal papillary necrosis and other renal injury. Renal toxicity has also been seen in patients in whom renal prostaglandins have a compensatory role in the maintenance of renal perfusion. In these patients, administration of a nonsteroidal anti-inflammatory drug may cause a dose-dependent reduction in prostaglandin formation and, secondarily, in renal blood flow, which may precipitate overt renal decompensation. Patients at greatest risk of this reaction are those with impaired renal function, heart failure, liver dysfunction, those taking diuretics and ACE inhibitors, and the elderly. Discontinuation of NSAID therapy is usually followed by recovery to the pretreatment state.

Advanced Renal Disease

No information is available from controlled clinical studies regarding the use of diclofenac sodium delayed-release in patients with advanced renal disease. Therefore, treatment with diclofenac sodium delayed-release is not recommended in these patients with advanced renal disease. If diclofenac sodium delayed-release therapy must be initiated, close monitoring of the patient's renal function is advisable.

Hepatic Effects

Elevations of one or more liver tests may occur during therapy with diclofenac sodium delayed-release. These laboratory abnormalities may progress, may remain unchanged, or may be transient with continued therapy. Borderline elevations (i.e., less than 3 times the ULN [ULN = the upper limit of the normal range]) or greater elevations of transaminases occurred in about 15% of diclofenac-treated patients. Of the markers of hepatic function, ALT (SGPT) is recommended for the monitoring of liver injury.

In clinical trials, meaningful elevations (i.e., more than 3 times the ULN) of AST (GOT) (ALT was not measured in all studies) occurred in about 2% of approximately 5,700 patients at some time during diclofenac treatment. In a large, open-label, controlled trial of 3,700 patients treated for 2-6 months, patients were monitored first at 8 weeks and 1,200 patients were monitored again at 24 weeks. Meaningful elevations of ALT and/or AST occurred in about 4% of patients and included marked elevations (i.e., more than 8 times the ULN) in about 1% of the 3,700 patients. In that open-label study, a higher incidence of borderline (less than 3 times the ULN), moderate (3-8 times the ULN), and marked (>8 times the ULN) elevations of ALT or AST was observed in patients receiving diclofenac when compared to other NSAIDs. Elevations in transaminases were seen more frequently in patients with

osteoarthritis than in those with rheumatoid arthritis.

Almost all meaningful elevations in transaminases were detected before patients became symptomatic. Abnormal tests occurred during the first 2 months of therapy with diclofenac in 42 of the 51 patients in all trials who developed marked transaminase elevations.

In postmarketing reports, cases of drug-induced hepatotoxicity have been reported in the first month, and in some cases, the first 2 months of therapy, but can occur at any time during treatment with diclofenac. Postmarketing surveillance has reported cases of severe hepatic reactions, including liver necrosis, jaundice, fulminant hepatitis with and without jaundice, and liver failure. Some of these reported cases resulted in fatalities or liver transplantation.

Physicians should measure transaminases periodically in patients receiving long-term therapy with diclofenac, because severe hepatotoxicity may develop without a prodrome of distinguishing symptoms. The optimum times for making the first and subsequent transaminase measurements are not known. Based on clinical trial data and postmarketing experiences, transaminases should be monitored within 4 to 8 weeks after initiating treatment with diclofenac. However, severe hepatic reactions can occur at any time during treatment with diclofenac.

If abnormal liver tests persist or worsen, if clinical signs and/or symptoms consistent with liver disease develop, or if systemic manifestations occur (e.g., eosinophilia, rash, abdominal pain, diarrhea, dark urine, etc.), diclofenac sodium delayed-release should be discontinued immediately.

To minimize the possibility that hepatic injury will become severe between transaminase measurements, physicians should inform patients of the warning signs and symptoms of hepatotoxicity (e.g., nausea, fatigue, lethargy, diarrhea, pruritus, jaundice, right upper quadrant tenderness, and "flu-like" symptoms), and the appropriate action patients should take if these signs and symptoms appear.

To minimize the potential risk for an adverse liver related event in patients treated with diclofenac sodium delayed-release, the lowest effective dose should be used for the shortest duration possible. Caution should be exercised in prescribing diclofenac sodium delayed-release with concomitant drugs that are known to be potentially hepatotoxic (e.g., antibiotics, anti-epileptics).

Anaphylactic Reactions
As with other NSAIDs, anaphylactic reactions may occur both in patients with the aspirin triad and in patients without known sensitivity to NSAIDs or known prior exposure to diclofenac sodium delayed-release. Diclofenac sodium delayed-release should not be given to patients with the aspirin triad. This symptom complex typically occurs in asthmatic patients who experience rhinitis with or without nasal polyps, or who exhibit severe, potentially fatal bronchospasm after taking aspirin or other NSAIDs. (See CONTRAINDICATIONS and PRECAUTIONS, PREEXISTING ASTHMA.) Anaphylaxis-type reactions have been reported with NSAID products, including with diclofenac products, such as diclofenac sodium delayed-release. Emergency help should be sought in cases where an anaphylactic reaction occurs.

Skin Reactions
NSAIDs, including diclofenac sodium delayed-release, can cause serious skin adverse events such as exfoliative dermatitis, Stevens-Johnson Syndrome (SJS), and toxic epidermal necrolysis (TEN), which can be fatal. These serious events may occur without warning. Patients should be informed about the signs and symptoms of serious skin manifestations and use of the drug should be discontinued at the first appearance of skin rash or any other sign of hypersensitivity.

Pregnancy
In late pregnancy, as with other NSAIDs, diclofenac sodium delayed-release should be avoided because it may cause premature closure of the ductus arteriosus.

Ranitidine Hydochloride

Capsaisain cream

Rugby® NDC 0536-2525-25

SATISFACTION GUARANTEED Rugby OR YOUR MONEY BACK

Capsaicin Cream, 0.025%

External Analgesic

Penetrating Pain Relief

Net Wt 2.1 oz (60 g)

Rugby® NDC 0536-2525-25

SATISFACTION GUARANTEED Rugby OR YOUR MONEY BACK

Capsaicin Cream, 0.025%

External Analgesic

Penetrating Pain Relief

Net Wt 2.1 oz (60 g)

Drug Facts

Active ingredient — **Purpose**

Capsaicin 0.025%..... External analgesic

Uses temporarily relieves minor aches and pains of muscles and joints due to:
● simple backache ● arthritis ● strains ● sprains

Warnings
For external use only
Read all warnings and directions before use. Test first on small area of skin. ▼

N 3 0536-2525-25 4

Mfd for:
Rugby Laboratories, Inc.
Duluth, Georgia 30097
www.rugbylaboratories.com
R1211
07-057 Made in Canada

Drug Facts (continued)

Do not use ● on wounds or damaged skin ● if you are allergic to capsicum or chili peppers

When using this product ● you may experience a burning sensation. The intensity of this reaction varies among individuals and may be severe. With regular use, this sensation generally disappears after several days. ● avoid contact with the eyes, lips, nose and mucous membranes ● do not tightly wrap or bandage the treated area ● do not apply heat to the treated area immediately before or after use

Stop use and ask a doctor if ● condition worsens or does not improve after regular use ● severe burning persists or blistering occurs

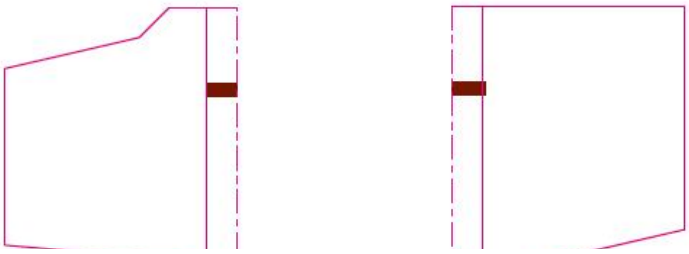
Keep out of reach of children. If swallowed, get medical help or contact a Poison Control Center immediately.

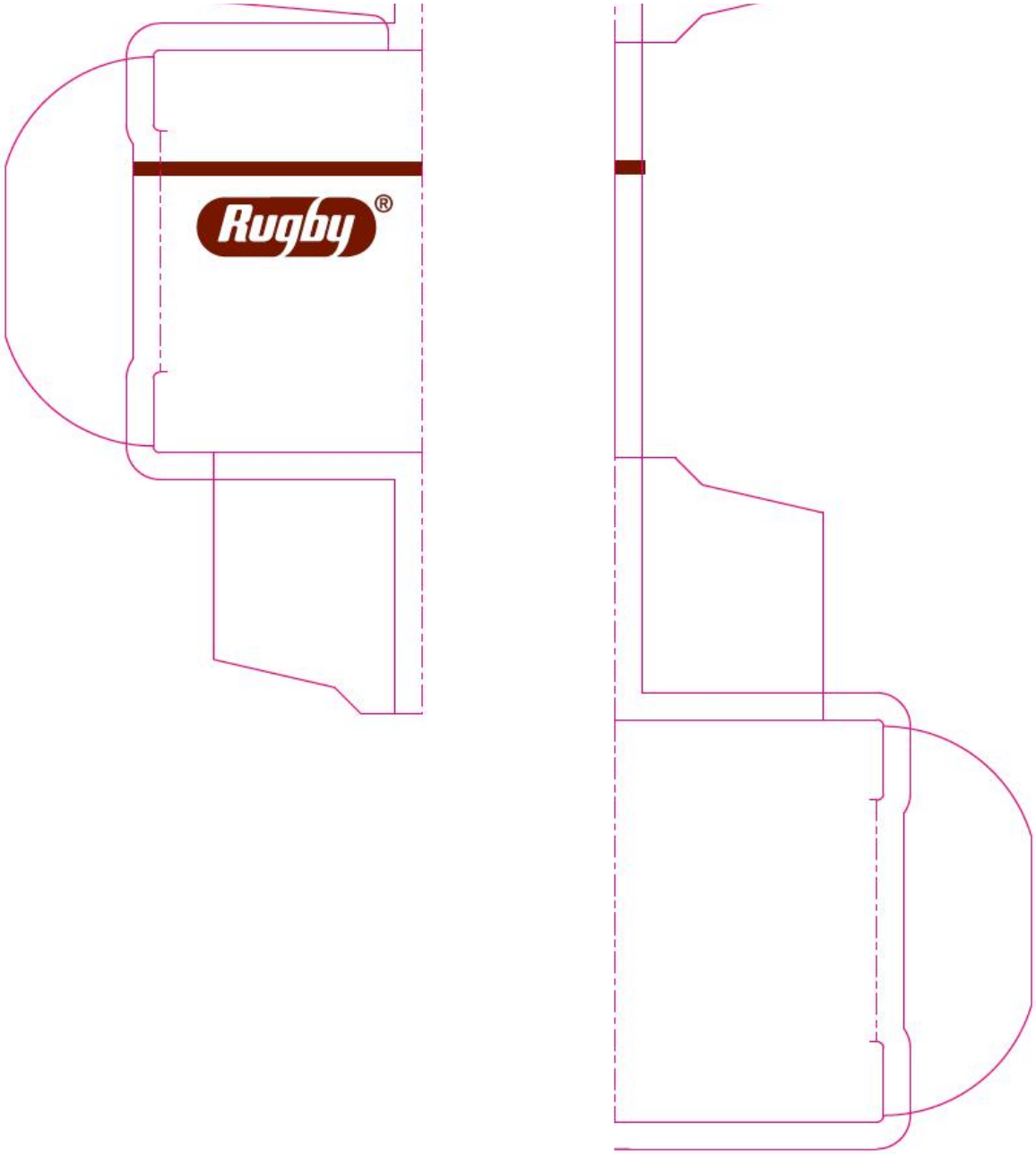
Directions **Adults and children 18 years of age and older:** ● apply a thin film of cream to affected area and gently rub in until fully absorbed ● unless treating hands, wash hands thoroughly with soap and water immediately after application ● for best results, apply 3 to 4 times daily. **Children under 18 years:** ask a doctor

Other information store at room temperature 15° - 30°C (59° - 86°F)

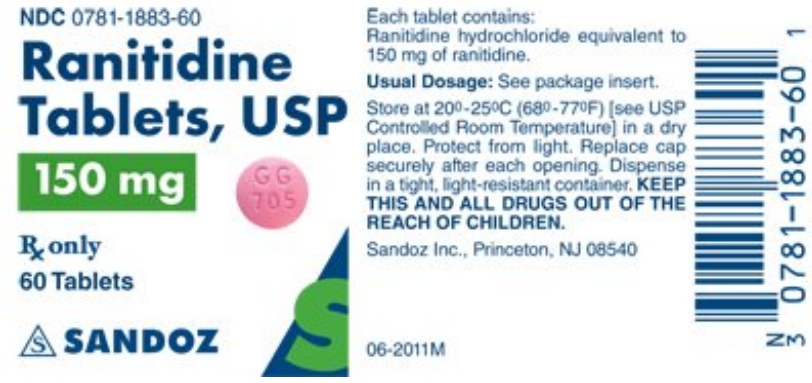
Inactive ingredients acrylates/C10-30 alkyl acrylate crosspolymer, cetyl acetate, methylparaben, PPG-20 methyl glucose ether distearate, propylene glycol, propylparaben, stearate-100, stearic acid, stearyl alcohol, triethanolamine, water

Questions or comments? Call **1-800-645-2158** 9 am - 5 pm ET, Monday - Friday

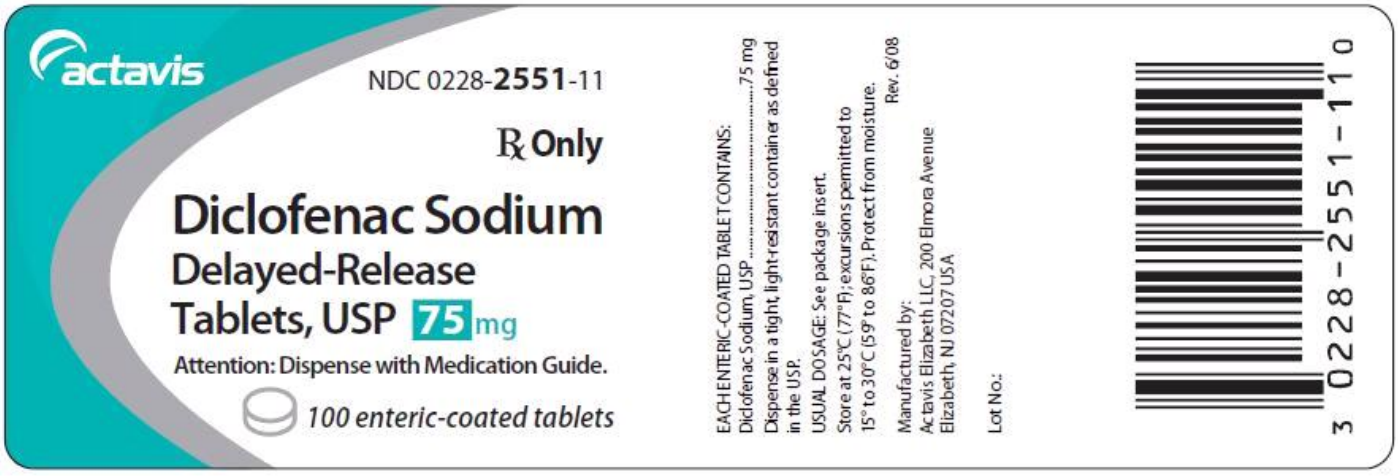




Ranitdine



Diclofenac



Ranitdine

Ranitidine is a competitive, reversible inhibitor of the action of histamine at the histamine H2-receptors, including receptors on the gastric cells. Ranitidine does not lower serum Ca++ in hypercalcemic states. Ranitidine is not a anticholinergic agent.

Pharmacokinetics
Absorption

Ranitidine tablets and ranitidine capsules are 50% absorbed after oral administration, compared to an intravenous (IV) injection with mean peak levels of 440 to 545 ng/mL occurring 2 to 3 hours after a 150 mg dose. Absorption is not significantly impaired by the administration of food or antacids. Propantheline slightly delays and increases peak blood levels of ranitidine, probably by delaying gastric emptying and transit time. In one study, simultaneous administration of high-potency antacid (150 mmol) in fasting subjects has been reported to decrease the absorption of ranitidine.

Distribution

The volume of distribution is about 1.4 L/kg. Serum protein binding averages 15%.

Metabolism

In humans, the N-oxide is the principal metabolite in the urine; however, this amounts to <4% of the dose. Other metabolites are the S-oxide (1%) and the desmethyl ranitidine (1%). The remainder of the administered dose is found in the stool. Studies in patients with hepatic dysfunction (compensated cirrhosis) indicate that there are minor, but clinically insignificant, alterations in ranitidine half-life, distribution, clearance, and bioavailability.

Excretion

The principal route of excretion is the urine, with approximately 30% of the orally administered dose collected in the urine as unchanged drug in 24 hours. Renal clearance is about 410 mL/min, indicating active tubular excretion. The elimination half-life is 2.5 to 3 hours. Four patients with clinically significant renal function impairment (creatinine clearance 25 to 35 mL/min) administered 50 mg of ranitidine intravenously had an average plasma half-life of 4.8 hours, a ranitidine clearance of 29 mL/min, and a volume of distribution of 1.76 L/kg. In general, these parameters appear to be altered in proportion to creatinine clearance (see DOSAGE AND ADMINISTRATION).

Geriatrics

The plasma half-life is prolonged and total clearance is reduced in the elderly population due to a decrease in renal function. The elimination half-life is 3 to 4 hours. Peak levels average 526 ng/mL following a 150 mg twice daily dose and occur in about 3 hours (see PRECAUTIONS: GERIATRIC USE and DOSAGE AND ADMINISTRATION: DOSAGE ADJUSTMENT FOR PATIENTS WITH

IMPAIRED RENAL FUNCTION).

Pediatrics

There are no significant differences in the pharmacokinetic parameter values for ranitidine in pediatric patients (from 1 month up to 16 years of age) and healthy adults when correction is made for body weight. The average bioavailability of ranitidine given orally to pediatric patients is 48% which is comparable to the bioavailability of ranitidine in the adult population. All other pharmacokinetic parameter values (t1/2, Vd, and CL) are similar to those observed with intravenous ranitidine use in pediatric patients. Estimates of Cmax and Tmax are displayed in Table 1.

Ranitdine

Ranitidine is indicated in:

Short-term treatment of active duodenal ulcer. Most patients heal within 4 weeks. Studies available to date have not assessed the safety of ranitidine in uncomplicated duodenal ulcer for periods of more than 8 weeks.
Maintenance therapy for duodenal ulcer patients at reduced dosage after healing of acute ulcers. No placebo-controlled comparative studies have been carried out for periods of longer than 1 year.
The treatment of pathological hypersecretory conditions (e.g., Zollinger-Ellison syndrome and systemic mastocytosis).
Short-term treatment of active, benign gastric ulcer. Most patients heal within 6 weeks and the usefulness of further treatment has not been demonstrated. Studies available to date have not assessed the safety of ranitidine in uncomplicated, benign gastric ulcer for periods of more than 6 weeks.
Maintenance therapy for gastric ulcer patients at reduced dosage after healing of acute ulcers. Placebo-controlled studies have been carried out for 1 year.
Treatment of GERD. Symptomatic relief commonly occurs within 24 hours after starting therapy with ranitidine 150 mg two times a day.
Treatment of endoscopically diagnosed erosive esophagitis. Symptomatic relief of heartburn commonly occurs within 24 hours of therapy initiation with ranitidine 150 mg 4 times daily.
Maintenance of healing of erosive esophagitis. Placebo-controlled trials have been carried out for 48 weeks.
Concomitant antacids should be given as needed for pain relief to patients with active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; GERD; and erosive esophagitis.

Ranitidine

Ranitdine

Active Duodenal Ulcer
The current recommended adult oral dosage of ranitidine for duodenal ulcer is 150 mg twice daily. An alternative dosage of 300 mg once daily after the evening meal or at bedtime can be used for patients in whom dosing convenience is important. The advantages of one treatment regimen compared to the other in a particular patient population have yet to be demonstrated (see CLINICAL PHARMACOLOGY: CLINICAL TRIALS: ACTIVE DUODENAL ULCER). Smaller doses have been shown to be equally effective in inhibiting gastric acid secretion in US studies, and several foreign trials have shown that 100 mg twice daily is as effective as the 150 mg dose.

Antacid should be given as needed for relief of pain (see CLINICAL PHARMACOLOGY: PHARMACOKINETICS).

INFLAMMATION REDUCTION PACK

inflammation reduction pack kit

Product Information			
Product Type	HUMAN PRESCRIPTION DRUG	**Item Code (Source)**	NDC:69176-010

Packaging				
#	**Item Code**	**Package Description**	**Marketing Start Date**	**Marketing End Date**
1	NDC:69176-010-01	1 in 1 KIT; Type 0: Not a Combination Product	08/18/2015	

Quantity of Parts		
Part #	**Package Quantity**	**Total Product Quantity**
Part 1		1
Part 2		1
Part 3		1

Part 1 of 3

RUGBY CAPSAICIN EXTERNAL ANALGESIC

capsaicin cream

Product Information	
Item Code (Source)	NDC:0536-2525
Route of Administration	TOPICAL

Active Ingredient/Active Moiety		
Ingredient Name	**Basis of Strength**	**Strength**
CAPSAICIN (UNII: S07O44R1ZM) (CAPSAICIN - UNII:S07O44R1ZM)	CAPSAICIN	0.025 g in 100 g

Inactive Ingredients	
Ingredient Name	**Strength**
PPG-20 METHYL GLUCOSE ETHER DISTEARATE (UNII: 0057334FAB)	
CETYL ACETATE (UNII: 4Q43814HXS)	
PROPYLENE GLYCOL (UNII: 6DC9Q167V3)	
STEARETH-100 (UNII: 4OH5W9UM87)	
STEARIC ACID (UNII: 4ELV7Z65AP)	
STEARYL ALCOHOL (UNII: 2KR89I4H1Y)	
TROLAMINE (UNII: 9O3K93S3TK)	
WATER (UNII: 059QF0KO0R)	
CARBOMER COPOLYMER TYPE A (ALLYL PENTAERYTHRITOL CROSSLINKED) (UNII: 71DD5V995L)	
PROPYLPARABEN (UNII: Z8IX2SC1OH)	
METHYLPARABEN (UNII: A2I8C7HI9T)	

Product Characteristics			
Color	white	**Score**	
Shape		**Size**	
Flavor		**Imprint Code**	
Contains			

Marketing Information

Marketing Category	Application Number or Monograph Citation	Marketing Start Date	Marketing End Date
OTC monograph not final	part348	03/01/2012	

Part 2 of 3

DICLOFENAC SODIUM

diclofenac sodium tablet, delayed release

Product Information	
Item Code (Source)	NDC:0228-2551
Route of Administration	ORAL

Active Ingredient/Active Moiety		
Ingredient Name	**Basis of Strength**	**Strength**
DICLOFENAC SODIUM (UNII: QTG126297Q) (DICLOFENAC - UNII:144O8QL0L1)	DICLOFENAC SODIUM	75 mg

Inactive Ingredients	
Ingredient Name	**Strength**
POLYSORBATE 80 (UNII: 6OZP39ZG8H)	
SODIUM ALGINATE (UNII: C269C4G2ZQ)	
STEARIC ACID (UNII: 4ELV7Z65AP)	
FERROSOFERRIC OXIDE (UNII: XM0M87F357)	
TITANIUM DIOXIDE (UNII: 15FIX9V2JP)	
HYPROMELLOSES (UNII: 3NXW29V3WO)	
LACTOSE MONOHYDRATE (UNII: EWQ57Q8I5X)	
MAGNESIUM STEARATE (UNII: 70097M6I30)	
POLYETHYLENE GLYCOLS (UNII: 3WJQ0SDW1A)	
PROPYLENE GLYCOL (UNII: 6DC9Q167V3)	
SILICON DIOXIDE (UNII: ETJ7Z6XBU4)	
TALC (UNII: 7SEV7J4R1U)	
SODIUM STARCH GLYCOLATE TYPE A POTATO (UNII: 5856J3G2A2)	
CELLULOSE, MICROCRYSTALLINE (UNII: OP1R32D61U)	

Product Characteristics			
Color	white	**Score**	no score
Shape	ROUND	**Size**	9mm
Flavor		**Imprint Code**	R;551
Contains			

Marketing Information

Marketing Category	Application Number or Monograph Citation	Marketing Start Date	Marketing End Date
ANDA	ANDA074514	03/26/1996	

Part 3 of 3

RANITIDINE HYDROCHLORIDE

ranitidine hydrochloride tablet, film coated

Product Information	
Item Code (Source)	NDC:0781-1883
Route of Administration	TOPICAL

Active Ingredient/Active Moiety		
Ingredient Name	**Basis of Strength**	**Strength**
RANITIDINE HYDROCHLORIDE (UNII: BK76465IHM) (RANITIDINE - UNII:884KT10YB7)	RANITIDINE	150 mg

Inactive Ingredients	
Ingredient Name	**Strength**
PEPPERMINT (UNII: V95R5KMY2B)	
SODIUM STARCH GLYCOLATE TYPE A POTATO (UNII: 5856J3G2A2)	
TRIETHYL CITRATE (UNII: 8Z96QXD6UM)	
D&C RED NO. 30 (UNII: 2S42T2808B)	
HYDROXYPROPYL CELLULOSE (TYPE H) (UNII: RFW2ET671P)	
HYPROMELLOSE 2910 (3 MPA.S) (UNII: 0VUT3PMY82)	
MAGNESIUM STEARATE (UNII: 70097M6I30)	
CELLULOSE, MICROCRYSTALLINE (UNII: OP1R32D61U)	
TITANIUM DIOXIDE (UNII: 15FIX9V2JP)	

Product Characteristics			
Color	pink	**Score**	score with uneven pieces
Shape	ROUND	**Size**	8mm
Flavor		**Imprint Code**	GG;705
Contains			

Marketing Information

Marketing Category	Application Number or Monograph Citation	Marketing Start Date	Marketing End Date
ANDA	ANDA074655	10/22/1997	

Marketing Information

Marketing Category	Application Number or Monograph Citation	Marketing Start Date	Marketing End Date
ANDA	ANDA074514	08/18/2015	

Labeler - TMIG, Inc. (036572986)

Registrant - TMIG, Inc. (036572986)

Establishment

Name	Address	ID/FEI	Business Operations
EPM Packaging		079124340	repack(69176-010)

Establishment

Name	Address	ID/FEI	Business Operations
Garcoa, Inc.		103039178	manufacture(0536-2525)

Establishment

Name	Address	ID/FEI	Business Operations
Sandoz Inc.		110342024	manufacture(0781-1883)

Establishment

Name	Address	ID/FEI	Business Operations
Actavis Elizabeth LLC		623114928	manufacture(0228-2551)

Revised: 8/2015 TMIG, Inc.