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Brand Names

Heartburn Relief, Pepcid, Pepcid AC, Pepcid AC Maximum Strength, Zantac 360

Indication Specific Dosing

Famotidine injection is intended for use in adult and pediatric hospitalized individuals, or as an alternative to oral famotidine.
Discontinue famotidine injection as soon as the patient is able to tolerate oral treatment and switch to an appropriate oral medication.

For self-medication of pyrosis (heartburn), acid dyspepsia (acid indigestion), or sour stomach, either for prophylaxis or for symptomatic relief

Oral dosage (tablets)

Adults

10 to 20 mg PO twice daily as needed. Max: 40 mg/day.

Children and Adolescents 12 to 17 years

10 to 20 mg PO twice daily as needed. Max: 40 mg/day.

For the treatment of non-erosive gastroesophageal reflux disease (GERD)

For the treatment of non-erosive gastroesophagel reflux disease (GERD)

Oral dosage

Adults

20 mg PO twice daily for up to 6 weeks.

Children and Adolescents

0.5 mg/kg/dose PO twice daily (Max: 40 mg PO twice daily). Treat for 6 to 12 weeks. While famotidine may be effective in patients with less severe GERD, proton pump inhibitors (PPIs) offer more rapid symptom relief and better healing.

Infants 3 to 11 months

0.5 mg/kg/dose PO twice daily initially, in addition to conservative measures such as thickened feedings. May increase the dose to 1 mg/kg/dose PO twice daily if needed. Treat for up to 8 weeks. While famotidine may be effective in patients with less severe GERD, proton pump inhibitors (PPIs) offer more rapid symptom relief and better healing.

Infants 1 to 2 months

0.5 mg/kg/dose PO once daily initially, in addition to conservative measures such as thickened feedings. May increase the dose to 1 mg/kg/dose PO once daily if needed. Treat for up to 8 weeks. While famotidine may be effective in patients with less severe GERD, proton pump inhibitors (PPIs) offer more rapid symptom relief and better healing.

Neonates

0.5 mg/kg/dose PO once daily initially, in addition to conservative measures such as thickened feedings. May increase the dose to 1 mg/kg/dose PO once daily if needed. Treat for up to 8 weeks. While famotidine may be effective in patients with less severe GERD, proton pump inhibitors (PPIs) offer more rapid symptom relief and better healing.

Intravenous dosage

Adults

20 mg IV every 12 hours.

Children and Adolescents

0.25 mg/kg/dose IV every 12 hours initially (Max: 40 mg/day); doses up to 0.5 mg/kg/dose IV every 12 hours have been used. A dose of 0.5 mg/kg/dose IV every 8 to 12 hours is supported by pharmacokinetic and pharmacodynamic data.

Infants 4 to 11 months†

Safety and efficacy have not been established; however, doses of 0.25 to 0.5 mg/kg/dose IV twice daily have been used in 1 small study.

Infants 1 to 3 months†

Safety and efficacy have not been established; however, doses of 0.25 to 0.5 mg/kg/dose IV once daily have been used in 1 small study.

Neonates†

Safety and efficacy have not been established; however, doses of 0.25 to 0.5 mg/kg/dose IV once daily have been used in 1 small study.

For the treatment of erosive esophagitis due to gastroesophagel reflux disease (GERD)

Oral dosage

Adults

20 or 40 mg PO twice daily for up to 12 weeks.

Children and Adolescents

0.5 mg/kg/dose PO twice daily (Max: 40 mg PO twice daily). Treat for 6 to 12 weeks. While famotidine may be effective in patients with less severe GERD, proton pump inhibitors (PPIs) offer more rapid symptom relief and better healing.

Infants 3 to 11 months

0.5 mg/kg/dose PO twice daily initially, in addition to conservative measures such as thickened feedings. May increase the dose to 1 mg/kg/dose PO twice daily if needed. Treat for up to 8 weeks. While famotidine may be effective in patients with less severe GERD, proton pump inhibitors (PPIs) offer more rapid symptom relief and better healing.

Infants 1 to 2 months

0.5 mg/kg/dose PO once daily initially, in addition to conservative measures such as thickened feedings. May increase the dose to 1 mg/kg/dose PO once daily if

needed. Treat for up to 8 weeks. While famotidine may be effective in patients with less severe GERD, proton pump inhibitors (PPIs) offer more rapid symptom relief and better healing.

Neonates

0.5 mg/kg/dose PO once daily initially, in addition to conservative measures such as thickened feedings. May increase the dose to 1 mg/kg/dose PO once daily if needed. Treat for up to 8 weeks. While famotidine may be effective in patients with less severe GERD, proton pump inhibitors (PPIs) offer more rapid symptom relief and better healing.

Intravenous dosage

Adults

20 mg IV every 12 hours.

Children and Adolescents

0.25 mg/kg/dose IV every 12 hours initially (Max: 40 mg/day); doses up to 0.5 mg/kg/dose IV every 12 hours have been used. A dose of 0.5 mg/kg/dose IV every 8 to 12 hours is supported by pharmacokinetic and pharmacodynamic data.

Infants 4 to 11 months†

Safety and efficacy have not been established; however, doses of 0.25 to 0.5 mg/kg/dose IV twice daily have been used in 1 small study.

Infants 1 to 3 months†

Safety and efficacy have not been established; however, doses of 0.25 to 0.5 mg/kg/dose IV once daily have been used in 1 small study.

Neonates†

Safety and efficacy have not been established; however, doses of 0.25 to 0.5 mg/kg/dose IV once daily have been used in 1 small study.

For the treatment of peptic ulcer disease (duodenal ulcer or gastric ulcer)

For acute treatment

Oral dosage

Adults

40 mg PO once daily at bedtime. Most individuals with a duodenal ulcer heal within 4 weeks and full-dose therapy is rarely needed for longer than 6 to 8 weeks.

Adolescents and Children

0.5 mg/kg/day PO at bedtime or 0.25 mg/kg/dose PO twice daily initially (Max: 40 mg/day). Doses up to 1 mg/kg/day (Max: 40 mg/day) PO have been used. Individualize treatment duration and dose based on clinical response and/or pH determination (gastric or esophageal) and endoscopy. In one small study, a treatment duration of 8 weeks was effective for the treatment of gastric or duodenal ulcer.

Intravenous dosage

Adults

20 mg IV every 12 hours.

Adolescents and Children

0.25 mg/kg/dose IV every 12 hours (Max: 40 mg/day). Titrate to a maximum dosage of 0.5 mg/kg every 12 hours (Max: 40 mg/day) based on clinical response and/or gastric pH determination and endoscopy.

For maintenance therapy of duodenal ulcer after the initial treatment phase has been completed

Oral dosage

Adults

20 mg PO once daily at bedtime.

Intravenous dosage

Adults

20 mg IV every 12 hours.

For pathologic GI hypersecretory conditions such as Zollinger-Ellison syndrome, systemic mastocytosis, or multiple endocrine adenoma

syndrome

Oral dosage

Adults

20 mg PO every 6 hours, initially. May titrate. Maximum: 160 mg PO every 6 hours per the manufacturer. Clinically, adjust to patient response. Doses as high as 200 mg PO every 6 hours have been reported.

Intravenous dosage

Adults

20 mg IV every 12 hours; titrate the dosage to individual treatment needs.

For NSAID-induced ulcer prophylaxis†

Oral dosage

Adults

40 mg PO twice daily. Data from clinical trials note that only higher doses of H2-blockers are effective at reducing risk for gastric ulceration from NSAIDs. In one trial, patients receiving a NSAID for either rheumatoid arthritis or osteoarthritis were given famotidine 20 mg PO twice daily, famotidine 40 mg PO twice daily, or placebo for 24 weeks. The cumulative incidence of gastric ulcer was significantly reduced by famotidine 40 mg but not the lower dose compared to placebo. The cumulative incidence of duodenal ulcer was significantly reduced by both famotidine doses relative to placebo.

For stress gastritis prophylaxis† in critically ill patients

Oral dosage

Adults

20 mg PO every 12 hours.

Intravenous dosage

Adults

20 mg IV every 12 hours.

Infants, Children, and Adolescents

1 to 2 mg/kg/day IV divided every 8 to 12 hours (Max: 40 mg/day).

Continuous Intravenous Infusion dosage

Adults

10 mg IV bolus, followed by 1.7 mg/hour (40 mg/day) continuous IV infusion.

For acid aspiration prophylaxis† prior to anesthesia

Oral dosage

Adults

20 mg PO as a single dose given 3 hours prior to the time of surgery, before induction of anesthesia. Alternatively, 40 mg PO the night prior to elective surgery. According to guidelines of the American Society of Anesthesiologists, routine preoperative use is NOT recommended in patients who have no apparent increased risk for pulmonary aspiration. However, some guidelines recommend an H2-receptor antagonist (PO or IV) for all women presenting for cesarean delivery.

Intravenous dosage

Adults

20 mg IV in the morning prior to surgery, before induction of anesthesia. According to guidelines of the American Society of Anesthesiologists, routine preoperative use is NOT recommended in patients who have no apparent increased risk for pulmonary aspiration. However, some guidelines recommend an H2-receptor antagonist (PO or IV) for all women presenting for cesarean delivery.

For the treatment of anaphylaxis†

Intravenous dosage

Adults

20 mg IV as a single dose.

Infants, Children, and Adolescents

0.25 mg/kg/dose (Max: 20 mg/dose) IV as a single dose.

Oral dosage

Adults

20 mg PO as a single dose.

Infants, Children, and Adolescents

0.25 mg/kg/dose (Max: 20 mg/dose) PO as a single dose.

For the treatment of short bowel syndrome†

Oral dosage

Adults

20 to 40 mg PO twice daily for at least 6 months.

Intravenous dosage

Adults

20 to 40 mg IV twice daily for at least 6 months.

Contraindications And Precaution

Drug Interactions

The coadministration of certain medications may lead to harm and require avoidance or therapy modification; review all drug interactions prior to concomitant use of other medications.

Hypersensitivity

This medication is contraindicated in patients with a history of hypersensitivity to it or any of its components.

General Information

Symptomatic response to therapy with famotidine does not preclude the presence of gastric cancer. Individuals who have a suboptimal response or early symptomatic relapse after completing therapy should consider evaluation for gastric malignancy. In the individual who is self-medicating with nonprescription (OTC) formulations, the

continuation of heartburn, acid indigestion, or dyspepsia beyond 2 weeks signals the need to consult a health care provider for evaluation.

renal failure, renal impairment

Famotidine should be used cautiously in patients with renal impairment or renal failure as there is a close relationship between the drug's elimination half-life and creatinine clearance. Dosages of famotidine should be adjusted in patients with moderate or severe renal impairment. Central nervous system (CNS) adverse reactions, including confusion, delirium, hallucinations, disorientation, agitation, seizures, and lethargy, have been reported in older adults and patients with moderate and severe renal impairment treated with famotidine; in some cases, the dosages were not adjusted as recommended for renal impairment.

geriatric

No special precautions have been advised for famotidine use in geriatric adults vs. younger adults, but dosage adjustments are necessary in older adults with reduced renal function. If critically ill, the older adults in some uncontrolled studies have appeared to be more likely to exhibit central nervous system (CNS) reactions to the H2-antagonists. According to the Beers Criteria, H2-receptor antagonists are considered potentially inappropriate medications (PIMs) in geriatric patients with delirium/high risk of delirium (potential for new-onset or worsening delirium) and should be avoided in this patient population. Dosage reduction of H2-receptor antagonists is recommended in geriatric adults with a creatinine clearance less than 50 mL/minute due to the potential for mental status changes.

neonates

Famotidine injection multidose vials contain benzyl alcohol as a preservative and should be avoided in neonates. There have been reports of fatal 'gasping syndrome' in neonates (less than 1 month of age) after the administration of parenteral solutions containing the preservative benzyl alcohol at dosages more than 99 mg/kg/day. The minimum amount of benzyl alcohol to cause toxicity is unknown. Therefore, use preservative-free famotidine injectable formulations in neonates.

pregnancy

Available data on the use of famotidine during pregnancy are insufficient to establish a drug-associated risk of major birth defects, miscarriage, or adverse outcomes. Epidemiologic data show no evidence that use of famotidine or other histamine 2-receptor antagonists (H2RAs) during pregnancy increases fetal malformations compared

with the general population, including first-trimester exposure. In human studies, H2RA exposure was not associated with a higher risk of congenital malformations (adjusted OR 1.03, 95% CI 0.8 to 1.32); results were similar when therapeutic pregnancy terminations were included (adjusted OR 1.17, 95% CI 0.93 to 1.46). No association was found with perinatal mortality, preterm delivery, low birth weight, or low Apgar scores. In animal reproduction studies, no adverse developmental effects were observed with oral famotidine at doses up to approximately 243 and 122 times, respectively, the human dose of 80 mg/day. Guidelines recommend a trial of lifestyle modifications as first-line therapy for heartburn and gastroesophageal reflux disease (GERD) during pregnancy, followed by antacids if lifestyle adjustments are ineffective. For ongoing symptoms, an H2RA can be used. Other medications should be reserved for pregnant individuals who fail H2RA therapy. Self-medication with over-the-counter H2RAs during pregnancy is not recommended. Pregnant individuals should see their health care professional for a proper diagnosis and for treatment recommendations. Famotidine has been used in limited circumstances at term in single doses to prevent acid aspiration during labor; some guidelines recommend an H2RA (oral or intravenous) for all individuals presenting for cesarean delivery.

breast-feeding

Famotidine is compatible with breast-feeding. Famotidine is present in human milk in minimal amounts, with a relative infant dose of 1.9%. This limited amount of famotidine exposure via human milk is not expected to cause adverse effects in the breastfed child.

Pregnancy And Lactation

Available data on the use of famotidine during pregnancy are insufficient to establish a drug-associated risk of major birth defects, miscarriage, or adverse outcomes. Epidemiologic data show no evidence that use of famotidine or other histamine 2-receptor antagonists (H2RAs) during pregnancy increases fetal malformations compared with the general population, including first-trimester exposure. In human studies, H2RA exposure was not associated with a higher risk of congenital malformations (adjusted OR 1.03, 95% CI 0.8 to 1.32); results were similar when therapeutic pregnancy terminations were included (adjusted OR 1.17, 95% CI 0.93 to 1.46). No association was found with perinatal mortality, preterm delivery, low birth weight, or low Apgar scores. In animal reproduction studies, no adverse developmental effects were observed with oral famotidine at doses up to approximately 243 and 122 times, respectively, the human dose of 80 mg/day. Guidelines recommend a trial of lifestyle modifications as first-line therapy for heartburn and gastroesophageal reflux disease (GERD) during pregnancy, followed by antacids if lifestyle adjustments are ineffective.

For ongoing symptoms, an H2RA can be used. Other medications should be reserved for pregnant individuals who fail H2RA therapy. Self-medication with over-the-counter H2RAs during pregnancy is not recommended. Pregnant individuals should see their health care professional for a proper diagnosis and for treatment recommendations. Famotidine has been used in limited circumstances at term in single doses to prevent acid aspiration during labor; some guidelines recommend an H2RA (oral or intravenous) for all individuals presenting for cesarean delivery.

Interactions

Acalabrutinib: (Moderate) The risk for a drug interaction varies by acalabrutinib dosage form. Administer acalabrutinib capsules 2 hours before an H2-blocker; simultaneous coadministration is expected to decrease capsule absorption and reduce acalabrutinib efficacy. Acalabrutinib tablets are unaffected by gastric acid reducing agents, such as H2-blockers, and may be administered without regard to the H2-blocker time of administration. Consider using acalabrutinib tablets in patients requiring a gastric acid reducing agent.
Alogliptin; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Amphetamine: (Moderate) Use amphetamine; dextroamphetamine and H2-blockers concomitantly with caution. Gastrointestinal alkalinizing agents may increase exposure to amphetamine; dextroamphetamine and exacerbate its actions.
Amphetamine; Dextroamphetamine Salts: (Moderate) Use amphetamine; dextroamphetamine and H2-blockers concomitantly with caution. Gastrointestinal alkalinizing agents may increase exposure to amphetamine; dextroamphetamine and exacerbate its actions.
Amphetamine; Dextroamphetamine: (Moderate) Use amphetamine; dextroamphetamine and H2-blockers concomitantly with caution. Gastrointestinal alkalinizing agents may increase exposure to amphetamine; dextroamphetamine and exacerbate its actions.
Atazanavir: (Major) Coadministration of H2-blockers with atazanavir reduces serum atazanavir concentrations; however, H2-blockers can be used under specific administration restrictions. Although data are insufficient to recommend atazanavir dosing in children < 40 kg receiving concomitant H2-blockers, the same recommendations regarding timing and maximum doses of concomitant H2-blockers should be followed. In treatment-naive patients >= 40 kg, do not exceed an H2- blocker dose equivalent to famotidine 40 mg twice daily, and give atazanavir 300 mg with

ritonavir 100 mg once daily with food. Give atazanavir simultaneously with and/or at least 10 hours after the H2- blocker. If a treatment-naive adult or adolescent (>= 40 kg) cannot tolerate ritonavir, do not exceed an H2- blocker dose equivalent to famotidine 20 mg twice daily, and the atazanavir dose should be increased to 400 mg once daily with food given at least 2 hours before or 10 hours after the H2- blocker. Data are insufficent to recommend atazanavir dosing in children or adolescents < 40 kg not receiving ritonavir boosting. In treatment-naive patients on a cobicistat-boosted regimen, cobicistat and atazanavir may be administered without dosage adjustment if given at the same time or a minimum of 10 hours after dosing of the H2-blocker. The H2-blocker dose should not exceed a dose that is comparable to 40 mg/day of famotidine in treatment-naive patients. In treatment-experienced patients >= 40 kg, do not exceed an H2- blocker dose equivalent to famotidine 20 mg twice daily, and give atazanavir 300 mg with ritonavir 100 mg once daily with food. Give atazanavir simultaneously with and/or at least 10 hours after the H2- blocker. In treatment-experienced patients >= 40 kg receiving H2-antagonists and tenofovir, atazanavir should be dosed 400 mg with ritonavir 100 mg once daily with food. In antiretroviral-experienced patients on a cobicistat-boosted regimen, the dosage of cobicistat with atazanavir needs to be increased if administered with H2-blockers; the recommended dose is cobicistat 150 mg/day with atazanavir 400 mg/day and 20 mg/day or less of famotidine or other comparably dosed H2-blocker. Significant reductions in atazanavir serum concentrations may lead to therapeutic failure and the development of HIV resistance. Closely monitor patients for antiretroviral therapeutic failure and resistance development during treatment with an H2- blocker.
Atazanavir; Cobicistat: (Major) Coadministration of H2-blockers with atazanavir reduces serum atazanavir concentrations; however, H2-blockers can be used under specific administration restrictions. Although data are insufficient to recommend atazanavir dosing in children < 40 kg receiving concomitant H2-blockers, the same recommendations regarding timing and maximum doses of concomitant H2-blockers should be followed. In treatment-naive patients >= 40 kg, do not exceed an H2- blocker dose equivalent to famotidine 40 mg twice daily, and give atazanavir 300 mg with ritonavir 100 mg once daily with food. Give atazanavir simultaneously with and/or at least 10 hours after the H2- blocker. If a treatment-naive adult or adolescent (>= 40 kg) cannot tolerate ritonavir, do not exceed an H2- blocker dose equivalent to famotidine 20 mg twice daily, and the atazanavir dose should be increased to 400 mg once daily with food given at least 2 hours before or 10 hours after the H2- blocker. Data are insufficent to recommend atazanavir dosing in children or adolescents < 40 kg not receiving ritonavir boosting. In treatment-naive patients on a cobicistat-boosted regimen, cobicistat and atazanavir may be administered without dosage adjustment if given at the same time or a minimum of 10 hours after dosing of the H2-blocker. The H2-blocker dose should not exceed a dose that is comparable to 40 mg/day of famotidine in

treatment-naive patients. In treatment-experienced patients >= 40 kg, do not exceed an H2- blocker dose equivalent to famotidine 20 mg twice daily, and give atazanavir 300 mg with ritonavir 100 mg once daily with food. Give atazanavir simultaneously with and/or at least 10 hours after the H2- blocker. In treatment-experienced patients >= 40 kg receiving H2-antagonists and tenofovir, atazanavir should be dosed 400 mg with ritonavir 100 mg once daily with food. In antiretroviral-experienced patients on a cobicistat-boosted regimen, the dosage of cobicistat with atazanavir needs to be increased if administered with H2-blockers; the recommended dose is cobicistat 150 mg/day with atazanavir 400 mg/day and 20 mg/day or less of famotidine or other comparably dosed H2-blocker. Significant reductions in atazanavir serum concentrations may lead to therapeutic failure and the development of HIV resistance. Closely monitor patients for antiretroviral therapeutic failure and resistance development during treatment with an H2- blocker.
Avutometinib; Defactinib: (Major) Avoid concomitant use of defactinib and H2-blockers. Concurrent use interferes with defactinib absorption which may decrease defactinib exposure and efficacy.
Bisacodyl: (Minor) The concomitant use of bisacodyl tablets with H2-blockers can cause the enteric coating of the bisacody tablet to dissolve prematurely, leading to possible gastric irritation or dyspepsia. Avoid H2-blockers within 1 hour before or after the bisacodyl dosage.
Bismuth Subsalicylate: (Minor) H2-blockers may increase the systemic absorption of bismuth from bismuth-containing compounds like bismuth subsalicylate.
Bismuth Subsalicylate; metroNIDAZOLE; Tetracycline: (Minor) H2-blockers may increase the systemic absorption of bismuth from bismuth-containing compounds like bismuth subsalicylate.
Bosutinib: (Moderate) Bosutinib displays pH-dependent aqueous solubility; therefore, concomitant use of bosutinib and H2-blockers may result in decreased plasma exposure of bosutinib. Separate the administration of bosutinib and H2-blockers by more than 2 hours.
Cabotegravir; Rilpivirine: (Moderate) Administer rilpivirine at least 4 hours before or 12 hours after an H2-receptor antagonist (H2RA), like famotidine. The gastric pH alterations associated with H2RAs may interfere with rilpivirine absorption and reduce rilpivirine bioavailability and efficacy. Rilpivirine peak and overall exposure decreased by 85% and 76%, respectively, when rilpivirine was taken 2 hours after famotidine. Rilpivirine peak and overall exposure increased 1.21- and 1.13- fold, respectively, when rilpivirine was administered 4 hours before famotidine and decreased by 1% and 9%, respectively, when rilpivirine was taken 12 hours after famotidine.
Canagliflozin; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine

has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Cefpodoxime: (Moderate) H2-blockers should be avoided during treatment with cefpodoxime. Coadministration could result in antibiotic failure. H2-blockers increase gastric pH. Cefpodoxime proxetil requires low gastric pH for dissolution. While the rate of absorption is not affected, coadministration reduces cefpodoxime AUC, peak plasma concentration (by 42%), and extent of absorption (by 32%).
Cefuroxime: (Major) Avoid the concomitant use of H2-blockers and cefuroxime. Drugs that reduce gastric acidity, such as H2-blockers, can interfere with the oral absorption of cefuroxime axetil and may result in reduced antibiotic efficacy.
Cysteamine: (Major) Monitor white blood cell (WBC) cystine concentration closely when administering delayed-release cysteamine (Procysbi) with H2-blockers. Drugs that increase the gastric pH may cause the premature release of cysteamine from delayed-release capsules, leading to an increase in WBC cystine concentration.
Dacomitinib: (Moderate) Administer dacomitinib at least 6 hours before or 10 hours after famotidine. Taking these medications at the same time may reduce dacomitinib absorption and decrease its efficacy.
Dapagliflozin; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Dasatinib: (Major) Avoid the concomitant use of H2-blockers with dasatinib film-coated oral tablets, such as Sprycel. Consider using an alternative dasatinib dosage form, such as Phyrago, or antacids. Separate the administration of all dasatinib oral tablet dosage forms and antacids by at least 2 hours. H2-blockers alter gastric pH and interfere with the absorption of some dasatinib dosage forms which may reduce dasatinib efficacy. The use of an H2-blocker with Sprycel reduced dasatinib overall exposure by 61%.
Desogestrel: Ethinyl Estradiol; Ferrous Bisglycinate: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Dextroamphetamine: (Moderate) Use amphetamine; dextroamphetamine and H2-blockers concomitantly with caution. Gastrointestinal alkalinizing agents may increase exposure to amphetamine; dextroamphetamine and exacerbate its actions.

diphenhydrAMINE; Naproxen: (Moderate) Avoid concomitant use of enteric-coated, delayed-release naproxen and H2-blockers due to the gastric pH elevating effects of H2-blockers. Enteric-coated, delayed-release naproxen tablets are designed to dissolve at a pH of 6 or more.
Dolutegravir; Rilpivirine: (Moderate) Administer rilpivirine at least 4 hours before or 12 hours after an H2-receptor antagonist (H2RA), like famotidine. The gastric pH alterations associated with H2RAs may interfere with rilpivirine absorption and reduce rilpivirine bioavailability and efficacy. Rilpivirine peak and overall exposure decreased by 85% and 76%, respectively, when rilpivirine was taken 2 hours after famotidine. Rilpivirine peak and overall exposure increased 1.21- and 1.13- fold, respectively, when rilpivirine was administered 4 hours before famotidine and decreased by 1% and 9%, respectively, when rilpivirine was taken 12 hours after famotidine.
Empagliflozin; Linagliptin; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Empagliflozin; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Emtricitabine; Rilpivirine; Tenofovir alafenamide: (Moderate) Administer rilpivirine at least 4 hours before or 12 hours after an H2-receptor antagonist (H2RA), like famotidine. The gastric pH alterations associated with H2RAs may interfere with rilpivirine absorption and reduce rilpivirine bioavailability and efficacy. Rilpivirine peak and overall exposure decreased by 85% and 76%, respectively, when rilpivirine was taken 2 hours after famotidine. Rilpivirine peak and overall exposure increased 1.21- and 1.13- fold, respectively, when rilpivirine was administered 4 hours before famotidine and decreased by 1% and 9%, respectively, when rilpivirine was taken 12 hours after famotidine.
Emtricitabine; Rilpivirine; Tenofovir Disoproxil Fumarate: (Moderate) Administer rilpivirine at least 4 hours before or 12 hours after an H2-receptor antagonist (H2RA), like famotidine. The gastric pH alterations associated with H2RAs may interfere with rilpivirine absorption and reduce rilpivirine bioavailability and efficacy. Rilpivirine peak and overall exposure decreased by 85% and 76%, respectively, when rilpivirine was taken 2 hours after famotidine. Rilpivirine peak and overall exposure increased 1.21-

and 1.13- fold, respectively, when rilpivirine was administered 4 hours before famotidine and decreased by 1% and 9%, respectively, when rilpivirine was taken 12 hours after famotidine.
Erlotinib: (Moderate) Administer erlotinib at least 2 hours before and 10 hours after an H2-receptor antagonist (H2RA), like famotidine. The gastric pH alterations associated with H2RAs may interfere with erlotinib absorption and reduce erlotinib bioavailability and efficacy. Erlotinib peak and overall exposure decreased by 54% and 33%, respectively, when it was administered 2 hours after a single dose of another H2RA. When administered at least 2 hours before and 10 hours after another H2RA, erlotinib peak and overall exposure decreased by 17% and 15%, respectively.
Ertugliflozin; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Ferric Maltol: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Fosamprenavir: (Moderate) Monitor for decreased fosamprenavir efficacy if coadministered with H2-blockers. Concurrent use may decrease the plasma concentrations of fosamprenavir leading to a reduction of antiretroviral efficacy and the potential development of viral resistance.
Gefitinib: (Moderate) Administer gefitinib at least 6 hours before or after an H2-receptor antagonist (H2RA), like famotidine. The gastric pH alterations associated with H2RAs may interfere with gefitinib absorption and reduce gefitinib bioavailability and efficacy. Coadministration with another H2RA and antacid to raise gastric pH above 5.0 reduced gefitinib overall exposure by 47%.
glipiZIDE; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
glyBURIDE; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has

been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Infigratinib: (Moderate) Separate the administration of infigratinib and H2-receptor antagonists if concomitant use is necessary. Coadministration may decrease infigratinib exposure resulting in decreased efficacy. Administer infigratinib two hours before or ten hours after an H2-receptor antagonist.
Iron Salts: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Iron Salts: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Iron: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Itraconazole: (Moderate) When administering H2-blockers with the 100 mg itraconazole capsule and 200 mg itraconazole tablet formulations, systemic exposure to itraconazole is decreased. Conversely, exposure to itraconazole is increased when H2-blockers are administered with the 65 mg itraconazole capsule. Administer H2-blockers at least 2 hours before or 2 hours after the 100 mg capsule or 200 mg tablet. Monitor for increased itraconazole-related adverse effects if H2-blockers are administered with itraconazole 65 mg capsules.
Ketoconazole: (Major) Avoid use of H2-blockers with ketoconazole. Medications that increase gastric pH may impair ketoconazole absorption.
Ledipasvir; Sofosbuvir: (Major) Solubility of ledipasvir decreases as gastric pH increases; thus, coadministration of ledipasvir; sofosbuvir with H2-blockers may result in lower

ledipasvir plasma concentrations. Ledipasvir; sofosbuvir can be administered with H2-blockers if given simultaneously or separated by 12 hours. The H2-blocker dose should not exceed a dose that is comparable to famotidine 40 mg twice daily.
Levoketoconazole: (Major) Avoid use of H2-blockers with ketoconazole. Medications that increase gastric pH may impair ketoconazole absorption.
Levonorgestrel; Ethinyl Estradiol; Ferrous Bisglycinate: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Levonorgestrel; Ethinyl Estradiol; Ferrous Fumarate: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Linagliptin; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Mefloquine: (Moderate) H2-blockers may increase plasma concentrations of mefloquine. Patients on chronic mefloquine therapy might be at increased risk of adverse reactions, especially patients with a neurological or psychiatric history. In a small study involving 6 healthy subjects and 6 peptic ulcer patients, cimetidine increased the Cmax and AUC of mefloquine. In the study, the pharmacokinetics of mefloquine were determined after receiving a single oral mefloquine 500 mg dose alone and after 3-days of cimetidine 400 mg PO twice daily. In both healthy subjects and peptic ulcer patients, Cmax was increased 42.4% and 20.5%, respectively. The AUC was increased by 37.5% in both groups. Elimination half-life, total clearance, and volume of distribution were not significantly affected. An increase in adverse reactions was not noted.
metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one

another but may vary based on other individual patient characteristics.
metFORMIN; sAXagliptin: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
metFORMIN; SITagliptin: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Naproxen: (Moderate) Avoid concomitant use of enteric-coated, delayed-release naproxen and H2-blockers due to the gastric pH elevating effects of H2-blockers. Enteric-coated, delayed-release naproxen tablets are designed to dissolve at a pH of 6 or more.
Naproxen; Esomeprazole: (Moderate) Avoid concomitant use of enteric-coated, delayed-release naproxen and H2-blockers due to the gastric pH elevating effects of H2-blockers. Enteric-coated, delayed-release naproxen tablets are designed to dissolve at a pH of 6 or more.
Naproxen; Pseudoephedrine: (Moderate) Avoid concomitant use of enteric-coated, delayed-release naproxen and H2-blockers due to the gastric pH elevating effects of H2-blockers. Enteric-coated, delayed-release naproxen tablets are designed to dissolve at a pH of 6 or more.
Neratinib: (Major) Take neratinib at least 2 hours before the next dose of an H2-blocker or 10 hours after the last dose of an H2-blocker due to decreased absorption and systemic exposure of neratinib; the solubility of neratinib decreases with increasing pH of the GI tract. The Cmax and AUC of neratinib were reduced by 57% and 48%, respectively, when administered 2 hours after a daily dose of ranitidine 300 mg. The Cmax and AUC of neratinib were reduced by 44% and 32%, respectively, when administered 2 hours before ranitidine 150 mg twice daily (given approximately 12 hours apart).
Nilotinib: (Moderate) If concomitant use of these agents is necessary, administer the H2-blocker approximately 10 hours before and approximately 2 hours after the nilotinib dose. Nilotinib displays pH-dependent solubility with decreased solubility at a higher pH. The concomitant use of nilotinib and H2-blockers that elevate the gastric pH may reduce the bioavailability of nilotinib. In a study in healthy subjects, there was no significant change in nilotinib pharmacokinetics when a single 400-mg nilotinib dose was given 10 hours after and 2 hours prior to famotidine.
Nirogacestat: (Major) Avoid concomitant use of nirogacestat and H2 receptor blockers.

Concurrent use may impair nirogacestat absorption which may decrease nirogacestat exposure and reduce its efficacy. Antacids may be used with nirogacestat but administration should be separated by at least 2 hours.
Norethindrone Acetate; Ethinyl Estradiol; Ferrous fumarate: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Norethindrone; Ethinyl Estradiol; Ferrous fumarate: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Octreotide: (Moderate) Coadministration of oral octreotide with H2-blockers may require increased doses of octreotide. Coadministration of oral octreotide with drugs that alter the pH of the upper GI tract, including H2-blockers, may alter the absorption of octreotide and lead to a reduction in bioavailability.
PAZOPanib: (Major) Avoid coadministration of pazopanib with H2-blockers due to decreased absorption of pazopanib, which may decrease efficacy. If concomitant administration with a gastric acid-reducing agent is unavoidable, consider the use of a short-acting antacid in place of an H2-blocker; separate administration of the short-acting antacid and pazopanib by several hours to avoid a reduction in pazopanib exposure. Concomitant use of pazopanib with a proton pump inhibitor decreased pazopanib exposure (AUC and Cmax) by approximately 40%.
Pexidartinib: (Moderate) Administer pexidartinib 2 hours before or 10 hours after H2-blockers as concurrent administration may reduce pexidartinib exposure. Although the effects of H2-blockers on pexidartinib pharmacokinetics have not been studied, other acid-reducing agents have been shown to decrease pexidartinib exposure by 50%.
Pioglitazone; metFORMIN: (Minor) Monitor for metformin-related adverse effects and a decrease in metformin efficacy during concomitant use of famotidine. Famotidine has been observed to have a net neutral effect on metformin overall exposure. Famotidine has counteracting effects on metformin plasma concentrations: it increases metformin's bioavailability and improves elimination. These effects are expected to offset one another but may vary based on other individual patient characteristics.
Polyethylene Glycol; Electrolytes; Bisacodyl: (Minor) The concomitant use of bisacodyl tablets with H2-blockers can cause the enteric coating of the bisacody tablet to dissolve

prematurely, leading to possible gastric irritation or dyspepsia. Avoid H2-blockers within 1 hour before or after the bisacodyl dosage.
Rilpivirine: (Moderate) Administer rilpivirine at least 4 hours before or 12 hours after an H2-receptor antagonist (H2RA), like famotidine. The gastric pH alterations associated with H2RAs may interfere with rilpivirine absorption and reduce rilpivirine bioavailability and efficacy. Rilpivirine peak and overall exposure decreased by 85% and 76%, respectively, when rilpivirine was taken 2 hours after famotidine. Rilpivirine peak and overall exposure increased 1.21- and 1.13- fold, respectively, when rilpivirine was administered 4 hours before famotidine and decreased by 1% and 9%, respectively, when rilpivirine was taken 12 hours after famotidine.
Rilzabrutinib: (Moderate) Separate the administration of rilzabrutinib and H2-blockers if concomitant use is necessary: administer rilzabrutinib at least 2 hours before the H2-blocker. Simultaneous coadministration may decrease rilzabrutinib exposure and reduce rilzabrutinib efficacy. Rilzabrutinib is pH soluble and H2-blockers alter gastric pH. Administering rilzabrutinib 2 hours before an H2-blocker mitigates this interaction and has been observed to reduce rilzabrutinib overall exposure by 28%.
Risedronate: (Major) Use of H2-blockers with delayed-release risedronate tablets (Atelvia) is not recommended. Co-administration of drugs that raise stomach pH increases risedronate bioavailability due to faster release of the drug from the enteric coated tablet. This interaction does not apply to risedronate immediate-release tablets.
Secretin: (Major) Discontinue H2-blockers at least 2 days before performing a secretin stimulation test for gastrinoma. H2-blockers may cause a hyperresponse in gastrin secretion and interfere with the test's diagnostic accuracy.
Selpercatinib: (Moderate) Administer selpercatinib at least 2 hours before or 10 hours after an H2-receptor antagonist (H2RA), like famotidine. The gastric pH alterations associated with H2RAs may interfere with selpercatinib absorption and reduce selpercatinib bioavailability and efficacy. Selpercatinib pharmacokinetics were unaffected by another H2RA when selpercatinib was administered 2 hours before or 10 hours after the H2RA under fasted conditions.
Sodium Ferric Gluconate Complex; Ferric Pyrophosphate Citrate: (Minor) The bioavailability of oral iron salts is influenced by gastric pH, and the concomitant administration of H2-blockers can decrease iron absorption. The non-heme ferric form of iron needs an acidic intragastric pH to be reduced to ferrous and to be absorbed. Iron salts and polysaccharide-iron complex provide non-heme iron. H2-blockers have long-lasting effects on the secretion of gastric acid and thus, increase the pH of the stomach. The increase in intragastric pH can interfere with the absorption of iron salts.
Sofosbuvir; Velpatasvir: (Major) H2-blockers may be administered simultaneously with or 12 hours apart from velpatasvir. H2-blocker doses should not exceed doses comparable to famotidine 40 mg twice daily. Velpatasvir solubility decreases as pH increases; therefore, drugs that increase gastric pH are expected to decrease the concentrations of

velpatasvir, potentially resulting in loss of antiviral efficacy.
Sofosbuvir; Velpatasvir; Voxilaprevir: (Major) H2-blockers may be administered simultaneously with or 12 hours apart from velpatasvir. H2-blocker doses should not exceed doses comparable to famotidine 40 mg twice daily. Velpatasvir solubility decreases as pH increases; therefore, drugs that increase gastric pH are expected to decrease the concentrations of velpatasvir, potentially resulting in loss of antiviral efficacy.
Sotorasib: (Major) Avoid coadministration of sotorasib and gastric acid-reducing agents, such as H2-receptor antagonists. Coadministration may decrease sotorasib exposure resulting in decreased efficacy. If necessary, sotorasib may be administered 4 hours before or 10 hours after a locally acting antacid. Coadministration with an H2-receptor antagonist decreased sotorasib exposure by 38% under fed conditions.
Sparsentan: (Major) Avoid concurrent use of sparsentan and H2 receptor antagonists due to the risk for decreased sparsentan exposure which may reduce its efficacy. Medications that affect gastric pH may reduce sparsentan absorption.
SUMAtriptan; Naproxen: (Moderate) Avoid concomitant use of enteric-coated, delayed-release naproxen and H2-blockers due to the gastric pH elevating effects of H2-blockers. Enteric-coated, delayed-release naproxen tablets are designed to dissolve at a pH of 6 or more.
Taletrectinib: (Major) Avoid concomitant use of taletrectinib and H2-receptor antagonists (H2RAs). Concurrent use may decrease taletrectinib exposure, which may reduce its efficacy. Taletrectinib oral absorption is pH dependent and H2RAs alter gastric pH.
Thalidomide: (Moderate) Thalidomide and other agents that slow cardiac conduction such as H2-blockers should be used cautiously due to the potential for additive bradycardia.
Theophylline, Aminophylline: (Minor) Monitor for aminophylline-related adverse effects in patients with additional risk factors for aminophylline-related harm. The majority of data demonstrate famotidine has no effect on theophylline concentrations, however, some data suggest the potential for famotidine to increase theophylline exposure. Aminophylline is a prodrug of theophylline. (Minor) Monitor for theophylline-related adverse effects in patients with additional risk factors for theophylline-related harm. The majority of data demonstrate famotidine has no effect on theophylline concentrations, however, some data suggest the potential for famotidine to increase theophylline exposure.
tiZANidine: (Major) Avoid concomitant use of tizanidine and famotidine as increased tizanidine exposure may occur. If use together is necessary, initiate tizanidine at 2 mg and increase by 2 to 4 mg per day based on clinical response. Discontinue tizanidine if hypotension, bradycardia, or excessive drowsiness occurs.
Warfarin: (Moderate) Closely monitor the INR if coadministration of warfarin with famotidine is necessary as concurrent use may increase the exposure of warfarin

leading to increased bleeding risk.
Ziftomenib: (Moderate) Separate the administration of ziftomenib and an H2-blocker if concomitant use is necessary: administer ziftomenib at least 2 hours before or 10 hours after the H2-blocker. Simultaneous coadministration may decrease ziftomenib absorption and reduce ziftomenib exposure and efficacy. Ziftomenib is pH soluble and H2-blockers alter gastric pH. Administering ziftomenib 2 hours before or 10 hours after an H2-blocker is expected to mitigate this interaction.

Adverse Reaction

anorexia, cholestasis, constipation, diarrhea, dizziness, dysgeusia, elevated hepatic enzymes, headache, hepatitis, jaundice, nausea, vomiting, xerostomia

Similar to other H2-antagonists, famotidine causes infrequent adverse reactions. In controlled clinical trials, the incidence of adverse reactions in people who received famotidine 40 mg at bedtime was similar to that in the placebo group. The following reactions have occurred in greater than 1% of people and may be causally related to the drug: headache (4.7%), dizziness (1.3%), constipation (1.2%), and diarrhea (1.7%). Other gastrointestinal adverse events reported in clinical trials or post approval include: cholestasis with jaundice, hepatitis, elevated hepatic enzymes, vomiting, nausea, abdominal discomfort, anorexia, dysgeusia (reported as taste disorder), and xerostomia. The relationship between these events and famotidine therapy has been unclear in many cases.

agitation, anxiety, confusion, delirium, depression, drowsiness, hallucinations, insomnia, libido decrease, paranoia, paresthesias, seizures

Reversible mental status changes, including agitation, confusion, delirium, hallucinations, hostility, paranoia, depression, insomnia, and disorientation have been reported rarely following famotidine therapy. Anxiety, libido decrease, paresthesias, and drowsiness have also been reported. A review of central nervous system reactions to H2-antagonists revealed that the incidence varies widely depending on the specific report, and that no single H2-antagonist is more likely to induce CNS reactions than another. Central nervous system reactions are more likely to occur in elderly patients and/or those with renal impairment. Seizures have been rarely reported in patients with renal impairment. Additionally, in a clinical study of famotidine in 35 infants < 1 year of age with GERD symptoms, agitation was reported in 5 patients that resolved when famotidine was discontinued.

acne vulgaris, alopecia, anaphylactoid reactions, angioedema, edema, flushing, ocular inflammation, pruritus, rash, Stevens-Johnson syndrome, toxic epidermal necrolysis, urticaria, xerosis

Few cases of toxic epidermal necrolysis and Stevens-Johnson syndrome have been reported with famotidine therapy. Anaphylactoid reactions have also been reported. Additional hypersensitivity and dermatological reactions reported for famotidine in clinical trials or post approval include: orbital or facial edema, angioedema, urticaria, rash, conjunctival injection or ocular inflammation, alopecia, acne vulgaris, pruritus, xerosis, and flushing. The relationship between these events and famotidine therapy has been unclear in many cases.

infection

Increasing evidence suggests a link between acid-suppression therapy and respiratory infection, specifically pneumonia (community- and hospital-acquired). Several mechanisms have been proposed to account for this association. One such mechanism states that gastric pH serves as a barrier against pathogenic colonization of the gastrointestinal tract. An increase in gastric pH allows for bacterial and viral invasion which, in theory, can precipitate respiratory infections. Another proposed mechanism accounts for the role that gastric acid may have on stimulating the cough reflex that allows for the clearing of infectious agents from the respiratory tract. Finally, the fact that acid-suppressive therapy may impair white blood cell function, which in turn may lead to a depressed immune response to an infection, is listed among possible mechanisms. Regardless of the mechanism, the use of H2-blockers and/or PPIs has been associated with the development of pneumonia. Data from a large epidemiological trial, including 364,683 individuals who developed 5551 first occurrences of community-acquired pneumonia (CAP), suggest an increased risk of developing CAP among users of acid-suppressive therapy compared to those who stopped therapy. After adjusting for confounders, the adjusted relative risk (RR) for CAP among PPI users compared to those who stopped therapy was 1.89 (95% CI, 1.36—2.62). Likewise, users of H2-blockers had an adjusted RR of 1.63 (95% CI, 1.07—2.48) compared to those who stopped therapy. In a second large cohort trial, including 63,878 hospital admissions, acid-suppressive therapy was ordered in 52% (83% PPI and 23% H2- blocker, with some patients exposed to both) of new admissions. Hospital-acquired pneumonia occurred in 2,219 admissions (3.5%) with a higher incidence recorded among acid-suppressive therapy exposed patients compared to non-exposed patients. A subset analysis found a statistically significant association between PPI use (OR, 1.3; 95% CI, 1.1—1.4) and pneumonia. A non-significant association was found with H2-blockers (OR, 1.2; 95% CI, 0.98—1.4); however, the lack of significance was attributed to the studies lack of power to detect

significance for an OR of less than 1.3. Until more is known about the relationship between acid-suppression and pneumonia, clinicians are encouraged to carefully select patients before empirically initiating acid-suppressive therapy with H2-blockers or PPIs. A causal relationship between the use of famotidine and pneumonia has not been established.

atrophic gastritis

Atrophic gastritis, a precursor for gastric cancer, has been associated with prolonged acid suppression with high dose H2-blockers like famotidine in patients who are H. pylori positive. A 'test and treat' approach for baseline H. pylori infections is recommended for patients with reflux esophagitis on long term acid suppression therapy. Treatment of baseline infection decreases inflammation and may reverse corpus gastritis.

arrhythmia exacerbation, AV block, palpitations, QT prolongation

Cardiovascular adverse events reported for famotidine in clinical trials or post approval include: arrhythmia exacerbation, AV block, and palpitations. The relationship between these events and famotidine therapy has been unclear in many cases. In addition, QT prolongation has been reported in patients with impaired renal function whose dose/dosing interval of famotidine may not have been adjusted appropriately.

agranulocytosis, leukopenia, pancytopenia, thrombocytopenia

Hematological adverse events have been reported for famotidine in clinical trials or post approval and include few cases of agranulocytosis, pancytopenia, leukopenia, and thrombocytopenia. The relationship between these events and famotidine therapy has been unclear in many cases.

arthralgia, asthenia, bronchospasm, fatigue, fever, gynecomastia, impotence (erectile dysfunction), lethargy, muscle cramps, musculoskeletal pain, rhabdomyolysis, tinnitus

Miscellaneous adverse events reported for famotidine in clinical trials or post-marketing include: fever, asthenia, fatigue, lethargy, rhabdomyolysis, musculoskeletal pain including muscle cramps, arthralgia, bronchospasm, interstitial pneumonia, and tinnitus. In addition, few cases of impotence (erectile dysfunction) and gynecomastia have been reported; however, in controlled clinical trials, the incidences were not greater than those seen with placebo. The relationship between these events and famotidine therapy has been unclear in many cases.

pernicious anemia, vitamin B12 deficiency

Long-term (e.g., generally > 3 years) treatment with acid-suppressing agents can lead to malabsorption of vitamin B12 (cyanocobalamin). One large case-controlled study compared patients with and without an incident diagnosis of vitamin B12 deficiency (n = 25,956 and 184,199, respectively). A correlation was demonstrated between vitamin B12 deficiency and gastric acid-suppression therapy. Patients receiving >= 2 years of a proton pump inhibitor (PPI)(OR, 1.65 [95% CI, 1.58—1.73]) or >= 2 years of a H2-receptor antagonist (OR, 1.25 [95% CI, 1.17—1.34]) were associated with having an increased risk for vitamin B12 deficiency. A dose-dependant relationship was evident, as daily doses > 1.5 PPI pills/day were more strongly associated with vitamin B12 deficiency (OR, 1.95 [95% CI, 1.77—2.15]) compared to daily doses < 0.75 pills/day (OR, 1.63 [95% CI, 1.48—1.78]; p = 0.007 for interaction). The possibility of cyanocobalamin deficiency and pernicious anemia should be considered if clinical symptoms are observed. Neurological manifestations of pernicious anemia can occur in the absence of hematologic changes.

Description

Famotidine is an oral and parenteral histamine type 2-receptor antagonist (H2RA) used in the treatment of gastrointestinal disorders such as peptic ulcer and gastroesophageal reflux disease (GERD). The actions and indications of famotidine differ little from the other H2RAs, except that famotidine is less likely than cimetidine to interact with other drugs. Nonprescription (OTC) products are available for the prophylaxis and treatment of heartburn and acid indigestion. Both proton pump inhibitors (PPIs) and H2RAs provide symptom control in non-erosive GERD. However, PPIs are preferred over H2RAs for the healing and maintenance treatment of erosive GERD (i.e., erosive esophagitis) due to superior healing rates. H2RAs may be selected when PPIs are not tolerated, are contraindicated, or are not available. H2RAs are often used for stress ulcer prophylaxis in critically ill patients; however, evidence for benefit is lacking, and use may increase the risk of adverse reactions such as pneumonia and Clostridioides difficile infection. Famotidine and other H2-blockers are also first-line treatment options, along with PPIs, for stress-ulcer prophylaxis in critically ill patients with selected risk factors for upper GI bleeding. Famotidine was initially FDA approved in 1986.
NOTE: Some famotidine 10 mg and 20 mg tablets are marketed as Zantac 360, which may create confusion since the brand name was previously associated with a ranitidine product.

Mechanism Of Action

Famotidine competitively inhibits the binding of histamine to H2-receptors on the gastric basolateral membrane of parietal cells, reducing basal and nocturnal gastric acid secretions. The drug also decreases the gastric acid response to stimuli such as food, caffeine, insulin, betazole, or pentagastrin. Famotidine reduces the total volume of gastric juice, thus indirectly decreasing pepsin secretion. The drug does not appear to alter gastric motility, gastric emptying, esophageal pressures, biliary secretions, or pancreatic secretions. Famotidine may aid in gastromucosal healing, and it may protect the mucosa from the irritant effects caused by aspirin and nonsteroidal antiinflammatory agents.

Pharmacokinetics

Famotidine is administered orally and parenterally. Plasma protein binding is approximately 15% to 20%. There is no cumulative effect with repeat doses; plasma concentrations after multiple doses are similar to those after single doses. Famotidine undergoes minimal first-pass metabolism. The majority (65 to 70%) of a famotidine dose is excreted in the urine; 30 to 35% of the dose is metabolized by the liver. The S-oxide metabolite is the only 1 identified in humans. Famotidine elimination half-life is 2.5 to 3.5 hours in adults with normal renal function.

Affected cytochrome P450 isoenzymes and drug transporters: OAT1, OAT3
I In vitro studies indicate that famotidine is a substrate for OAT1 and OAT3. Following coadministration of probenecid (1,500 mg), an inhibitor of OAT1 and OAT3, with a single dose of famotidine 20 mg PO in 8 healthy subjects, the serum AUC0-10h of famotidine increased from 424 to 768 ng x hour/mL and the maximum serum concentration (Cmax) increased from 73 to 113 ng/mL. Renal clearance, urinary excretion rate and amount of famotidine excreted unchanged in urine were decreased. The clinical relevance of this interaction is unknown. Famotidine is considered a weak CYP1A2 inhibitor, but drug interactions due to this activity have not been studied clinically. Although not studied clinically, famotidine may lead to substantial increases in blood concentrations of tizanidine, a CYP1A2 substrate. No data have confirmed or refuted a potential interaction. Other formal studies in man, animal models and in vitro have signaled no significant interference with the disposition of compounds metabolized by the cytochrome P450 hepatic microsomal isoenzyme system. Compounds tested in man include warfarin, theophylline, phenytoin, diazepam, aminopyrine and antipyrine. Indocyanine green as an index of hepatic drug extraction has been tested, and no significant effects have been found. An in vitro study showed that famotidine is an inhibitor of MATE-1. However, no clinically significant interaction with metformin, a substrate for MATE-1, was observed.

Route-Specific Pharmacokinetics

- **Oral Route**

Bioavailability of famotidine is approximately 40% to 45% in adults. Famotidine tablets and oral suspension are bioequivalent. Food may slightly increase and antacids may slightly decrease the bioavailability of famotidine; however, the effects are considered clinically insignificant. The onset of action is usually within 1 hour after oral administration with maximum effects occurring within 1 to 3 hours depending on the dose. The duration of action is roughly 10 to 12 hours. Twenty-five to 30% of an oral dose is excreted in urine as unchanged drug.

- **Intravenous Route**

Sixty-five to 70% of an intravenous dose of famotidine is excreted in urine as unchanged drug.

- **Renal Impairment**

In adult patients with severe renal impairment (creatinine clearance less than 30 mL/minute), the systemic exposure (AUC) of famotidine increased at least 5-fold. In adult patients with moderate renal impairment (creatinine clearance between 30 to 60 mL/minute), the AUC of famotidine increased at least 2-fold.

- **Pediatrics**

Children and Adolescents

The mean oral bioavailability in 8 pediatric patients 11 to 15 years of age was 50%. The mean half-life was 2 to 3.4 hours in pediatric patients 1 to 15 years of age.

Neonates and Infants

After a single administration of 0.5 mg/kg orally in patients from birth to 12 months of age, the bioavailability was approximately 42%. The AUC increased 1.4-fold after a single oral dose of 1 mg/kg compared to 0.5 mg/kg and 2.7-fold after multiple oral doses of 1 mg/kg compared to 0.5 mg/kg. The half-life was 5.82 in infants birth to 12 months of age. Plasma clearance was reduced, and elimination half-life was prolonged in pediatric patients from birth to 3 months of age compared to older pediatric patients. After intravenous administration of 0.5 mg/kg, total clearance was 0.13 +/- 0.06 L/hour/kg, 0.21 +/- 0.06 L/hour/kg, and 0.49 +/- 0.17 L/hour/kg in pediatric patients younger than 1 month of age, younger than 3 months of age, and 4 to 12 months of age, respectively. Elimination half-life was 10.5 hours, 8.1 hours, and 4.5 hours in pediatric patients younger than 1 month of age, younger than 3 months of age, and 4 to 12 months of age, respectively. The duration of acid suppression was longer in pediatric patients from birth to 3 months of age compared to older pediatric patients.

- **Geriatric**

There are no clinically significant age-related changes in famotidine pharmacokinetics in elderly patients versus younger adults. However, in elderly patients with decreased renal

function, the clearance of famotidine may be reduced.

Administration

For storage information, see the specific product information within the How Supplied section.

Oral Administration

May be administered without regard to meals.

Oral Solid Formulations

Tablets: May administer with food, water, or milk to minimize gastric irritation.
Orally disintegrating tablets: No water is needed for administration. Instruct patients to open the tablet blister pack with dry hands, place the tablet on the tongue, allow to disintegrate, then swallow with saliva.

Oral Liquid Formulations

Oral suspension
Reconstitute by slowly adding 46 mL of purified water. Shake vigorously for 5 to 10 seconds after adding water.
After reconstitution, each 5 mL contains 40 mg of famotidine.
Shake suspension well before each use. Measure dosage with a calibrated device for accuracy.
Storage of reconstituted suspension: Store at room temperature for up to 30 days.

Injectable Administration

Visually inspect parenteral products for particulate matter and discoloration prior to administration whenever solution and container permit.
Updates for coronavirus disease 2019 (COVID-19): The FDA is allowing famotidine 10 mg/mL to be used beyond the labeled in-use time to help ensure access during COVID-related drug shortages. This period should be as short as possible, and for a maximum of 2 hours at room temperature or 4 hours when refrigerated. In-use time is defined as the maximum amount of time allowed to elapse between penetration of a closed-container system or after reconstitution of a lyophilized drug before patient administration.

Intravenous Administration

Intermittent IV Injection
Dilute 20 mg of famotidine injection to a total of 5 or 10 mL with 0.9% Sodium Chloride

Injection or other compatible solution to give concentrations of 4 or 2 mg/mL, respectively.
Inject appropriate dose over 2 minutes or more and at a rate of 10 mg/minute or less.

Intermittent IV Infusion (vials)
Dilute 20 mg of famotidine in 100 mL of 5% Dextrose Injection or 0.9% Sodium Chloride Injection or other compatible IV solution to give a final concentration of 0.2 mg/mL.
Infuse over 15 to 30 minutes.
Storage: If not used immediately, store diluted solutions under refrigeration and use within 48 hours of preparation. Although when diluted in common compatible solutions (e.g., 0.9% Sodium Chloride Injection, 5% Dextrose Injection, Lactated Ringer's Injection), famotidine is stable for 7 days at room temperature, there are no data available to confirm sterility under these conditions.

Intermittent IV Infusion (Galaxy containers)
The premixed infusion container contains famotidine 20 mg per 50 mL 0.9% Sodium Chloride Injection.
Check the container for leaks before use by squeezing the bag firmly. If leaks are found, discard solution as sterility may be impaired.
Do not add supplementary medication.
Do not use unless solution is clear, and the seal is intact.
Do not use plastic containers in series connections. Such use could result in air embolism due to residual air being drawn from the primary container before administration of the fluid from the secondary container is complete.
Suspend container from eyelet support.
Remove plastic protector from outlet port at bottom of container.
Attach administration set. Refer to complete directions accompanying set.
Infuse over 15 to 30 minutes.

Continuous IV Infusion
For adults, dilute 40 mg of famotidine in 250 mL of 5% Dextrose Injection or 0.9% Sodium Chloride Injection or other compatible solution. Infuse over 24 hours at a rate of 11 mL/hour or as specified by a physician.
Use a controlled-rate infusion device.
Storage: The diluted solution is stable for up to 48 hours at room temperature. Alternatively, the dosage may be added to a compatible TPN solution for administration over 24 hours.

Maximum Dosage Limits

- **Adults**
  40 mg/day PO or IV for active duodenal or benign gastric ulcer; 20 mg/day PO for ulcer maintenance; 40 mg/day PO for GERD; 80 mg/day PO for esophagitis; doses may go as high as 640 mg/day or 800 mg/day (rare) PO or 80 mg/day IV for hypersecretory conditions such as Zollinger-Ellison; 40 mg/day PO for self-medication (OTC).
- **Geriatric**
  40 mg/day PO or IV for active duodenal or benign gastric ulcer healing; 20 mg/day PO for ulcer maintenance; 40 mg/day PO for GERD; 80 mg/day PO for esophagitis; doses may go as high as 640 mg/day or 800 mg/day (rare) PO or 80 mg/day IV for hypersecretory conditions such as Zollinger-Ellison; 40 mg/day PO for self-medication (OTC).
- **Adolescents**
  1 mg/kg/day PO (Max: 80 mg/day); 1 mg/kg/day IV (Max: 40 mg/day); however, doses up to 2 mg/kg/day IV have been used off-label.
- **Children**
  1 mg/kg/day PO (Max: 80 mg/day); 1 mg/kg/day IV (Max: 40 mg/day); however, doses up to 2 mg/kg/day IV have been used off-label.
- **Infants**
  3 to 11 months: 2 mg/kg/day PO. Safety and efficacy of IV have not been established; however, doses up to 2 mg/kg/day IV have been used off-label.
  1 to 2 months: 1 mg/kg/day PO. Safety and efficacy of IV have not been established; however, doses up to 2 mg/kg/day IV have been used off-label.
- **Neonates**
  1 mg/kg/day PO. Safety and efficacy of IV have not been established; however, 0.5 mg/kg/day IV has been used off-label.

Dosage Forms

- CVS Acid Controller 10mg Tablet
- CVS Acid Controller Maximum Strength 20mg Tablet
- CVS Complete Dual Action Acid Reducer Plus Antacid Chewable Tablet (Berry)
- CVS Complete Dual Action Acid Reducer Plus Antacid Chewable Tablet (Mint)
- Duo Fusion Dual Action Chewable Tablet (Berry)
- Duo Fusion Dual Action Chewable Tablet (Cool Mint)
- Duo Fusion Dual Action Chewable Tablet (Wild Berry)
- Equaline Complete Dual Action Chewable Tablet (Berry)
- Equate Acid Reducer and Antacid Chewable Tablet (Cool Mint)

- Equate Dual Action Acid Reducer Complete 10mg-800mg-165mg Chewable Tablet (Berry)
- Equate Famotidine Original Strength 10mg Tablet
- Equate Famotidine Original Strength 10mg Tablet
- Famotidine 10mg Oral tablet
- Famotidine 10mg/1mL Solution for injection
- Famotidine 20mg Oral tablet
- Famotidine 20mg/50mL Solution for injection
- Famotidine 40mg Oral tablet
- Famotidine 40mg/5mL Powder for oral suspension
- Famotidine 4mg/1mL Solution for injection
- Famotidine Bulk powder
- Foster & Thrive Acid Reducer Maximum Strength 20mg Tablet
- Foster & Thrive Acid Reducer Original Strength 10mg Tablet
- Foster & Thrive Dual Action Acid Reducer Plus Antacid Chewable Tablet (Berry)
- GNP Acid Reducer 10mg Tablet
- GNP Acid Reducer 10mg Tablet
- GNP Acid Reducer 20mg Maximum Strength Tablet
- GNP Acid Reducer Maximum Strength 20mg Tablet
- GNP Acid Reducer Maximum Strength 20mg Tablet
- GNP Dual Action Complete Acid Reducer Plus Antacid Chewable Tablet (Berry)
- GNP Dual Action Complete Acid Reducer Plus Antacid Chewable Tablet (Cool Mint)
- GNP Famotidine 10mg Tablet
- GNP Original Strength Acid Reducer 10mg Tablet
- GoodSense Acid Reducer 10mg Tablet
- GoodSense Acid Reducer Maximum Strength 20mg Tablet
- GoodSense Complete Dual Action Chewable Tablet (Berry)
- GoodSense Dual Action Complete Acid Reducer Plus Antacid Chewable Tablet (Berry)
- Heartburn Relief 10mg Tablet
- Heartburn Relief 20mg Tablet
- Kirkland Acid Controller Maximum Strength 20mg Tablet
- Leader Acid Reducer 10mg Tablet
- Leader Acid Reducer 10mg Tablet
- Leader Acid Reducer 20mg Maximum Strength Tablet
- Leader Acid Reducer Maximum Strength 20mg Tablet
- Leader Complete Dual Action Acid Reducer + Antacid Chewable Tablet (Berry)
- Leader Complete Dual Action Acid Reducer and Antacid Chewable Tablet (Cool Mint)
- Member's Mark Acid-Pep Maximum Strength 20mg Tablet
- Pepcid 20mg Tablet

- Pepcid 20mg Tablet
- Pepcid 20mg Tablet
- Pepcid 40mg Tablet
- Pepcid AC 10mg Tablet
- Pepcid AC Maximum Strength 20mg Tablet
- Pepcid Complete Chewable Tablet (Berry)
- Pepcid Complete Chewable Tablet (Mint)
- Pepcid Complete Chewable Tablet (Tropical Fruit)
- Pepcid Complete Dual Action On-The-Go Chewable Tablet (Berry)
- Premier Value Acid Controller 10mg Tablet
- Premier Value Acid Controller 20mg Tablet
- Publix Acid Reducer 10mg Tablet
- Publix Acid Reducer Maximum Strength 20mg Tablet
- Publix Dual Action Acid Reducer + Antacid Chewable Tablet (Berry)
- Publix Dual Action Acid Reducer + Antacid Chewable Tablet (Mint)
- RITE AID Acid Reducer 10mg Tablet
- RITE AID Acid Reducer Maximum Strength 20mg Tablet
- RITE AID Complete Dual Action Acid Reducer Plus Antacid Chewable Tablet (Berry)
- RITE AID Dual Action Acid Reducer Plus Antacid Chewable Tablet (Berry)
- RITE AID Dual Action Acid Reducer Plus Antacid Chewable Tablet (Mint)
- Today's Health Famotidine 10mg Tablet
- Top Care Acid Reducer 10mg Tablet
- Top Care Acid Reducer Maximum Strength 20mg Tablet
- Top Care Complete Dual Action Chewable Tablet (Berry)
- Top Care Complete Dual Action Chewable Tablet (Mint)
- Walgreens Acid Controller 10mg Tablet
- Walgreens Acid Controller 20mg Tablet
- Walgreens Acid Controller Maximum Strength 20mg Tablet
- Walgreens Acid Controller Maxium Strength 20mg Tablet
- Walgreens Acid Reducer Maximum Strength 20mg Tablet
- Zantac 360 10mg Tablet
- Zantac 360 20mg Tablet

Dosage Adjustment Guidelines

Hepatic Impairment

Specific guidelines for dosage adjustments in hepatic impairment are not available; it appears that no dosage adjustments are needed.

Renal Impairment

General Recommendations

Pediatrics

The following has been recommended based on a dose of 0.5 to 1 mg/kg/day divided every 12 hours in pediatric patients with normal renal function:
eGFR 30 to 50 mL/minute/1.73 m2: 0.5 mg/kg/dose every 24 hours.
eGFR 10 to 29 mL/minute/1.73 m2: 0.25 mg/kg/dose every 24 hours.
eGFR less than 10 mL/minute/1.73 m2: 0.125 mg/kg/dose every 24 hours.

Intravenous Dosage

Adults

Active duodenal or gastric ulcer:
CrCl 30 to 60 mL/minute: 20 mg IV once daily.
CrCl less than 30 mL/minute: 10 mg IV once daily.
Symptomatic nonerosive GERD:
CrCl 30 to 60 mL/minute: 20 mg IV once daily.
CrCl less than 30 mL/minute: 10 mg IV once daily.
Erosive esophagitis diagnosed by endoscopy:
CrCl 30 to 60 mL/minute: 20 mg IV once daily.
CrCl less than 30 mL/minute: 10 mg IV once daily.
Risk reduction for duodenal ulcer recurrence:
CrCl 30 to 60 mL/minute: 20 mg IV once daily.
CrCl less than 30 mL/minute: 10 mg IV once daily.
Pathological hypersecretory conditions: Avoid use.

Pediatrics

The safe and effective dosage has not been established in pediatric patients with renal impairment for the treatment of peptic ulcer disease. The following guidance has been recommended based on a pharmacokinetic study of pediatric patients with stable chronic renal insufficiency (n = 18):
CrCl 50 mL/minute/1.73m2 or more: 0.5 mg/kg/dose (Max: 20 mg) IV every 12 to 24 hours.
CrCl 11 to 49 mL/minute/1.73m2: 0.5 mg/kg/dose (Max: 20 mg) IV every 36 to 48 hours.
CrCl 10 mL/minute/1.73m2 or less: 0.5 mg/kg/dose (Max: 20 mg) IV every 72 to 96 hours or 0.25 mg/kg/dose (Max: 10 mg) IV every 36 to 48 hours.

Oral Dosage

Adults and Pediatric patients weighing 40 kg or more (maximum recommended dosage)
Active duodenal or gastric ulcer:
CrCl 30 to 60 mL/minute: 20 mg PO once daily or 40 mg PO every other day.
CrCl less than 30 mL/minute: 10 mg PO once daily or 20 mg PO every other day.

Symptomatic nonerosive GERD:
CrCl 30 to 60 mL/minute: 20 mg PO once daily.
CrCl less than 30 mL/minute: 10 mg PO once daily or 20 mg PO every other day.
Erosive esophagitis diagnosed by endoscopy:
CrCl 30 to 60 mL/minute: 20 mg PO once daily, 40 mg PO once daily, or 40 mg PO every other day.
CrCl less than 30 mL/minute: 10 mg PO once daily, 20 mg PO once daily, or 20 mg PO every other day.
Risk reduction for duodenal ulcer recurrence:
CrCl 30 to 60 mL/minute: 10 mg PO once daily or 20 mg PO every other day.
CrCl less than 30 mL/minute: 10 mg PO every other day.
Pathological hypersecretory conditions: Avoid use.

Intermittent hemodialysis
Adults
Dose after dialysis.
Pediatrics
0.125 mg/kg/dose every 24 hours.

Peritoneal dialysis
Adults
Administer 10% of normal dose.
Pediatrics
0.125 mg/kg/dose every 24 hours.

Continuous renal replacement therapy (CRRT)
Adults
Administer 10% to 50% of normal dose.
Pediatrics
0.5 mg/kg/dose every 24 hours.

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