

Antacids and alginates

Fast, short-acting acid neutralisers and a physically distinct alginate raft that floats over gastric contents; the class's real teaching point is that its members differ from each other more than most students expect, both in bowel effect and in interaction risk.

Online guide: <https://www.medguideglobal.com/pharmacology/gastrointestinal/antacids-and-alginates>

Content updated: 2026-09-25

Quick revision

One Liner: Antacids neutralise gastric acid directly and act within minutes but only briefly; alginates work by a completely different physical mechanism, forming a floating raft rather than neutralising acid; and the class's cation choice - calcium, magnesium or aluminium - decides both the bowel side effect and the interaction risk, not just the antacid strength.

1. Antacids reduce the acid reaching the duodenum by directly neutralising gastric acid already present in the stomach. [1]
2. Calcium carbonate dissociates and its carbonate ion binds free hydrogen ions, raising gastric pH; it can also cause constipation and, via gastrin stimulation, acid rebound. [2]
3. Magnesium hydroxide neutralises gastric acid. At higher laxative doses, unabsorbed magnesium salts draw water into the bowel; diarrhoea can also occur with antacid use. [3]
4. Aluminium hydroxide neutralises gastric acid. The resulting rise in pH reduces pepsin activity as a secondary effect; constipation and, with excess use, faecal impaction are its characteristic bowel effects. [1]
5. Alginates do not neutralise gastric acid at all. In the presence of gastric acid, sodium alginate gels and, combined with bicarbonate-generated carbon dioxide, forms a foam raft that floats on the stomach contents and physically blocks reflux. [4]
6. Magnesium-, aluminium- and calcium-containing antacids (including calcium carbonate) can bind tetracycline and fluoroquinolone antibiotics in the gut, reducing their absorption. Check each oral medicine for product-specific interactions. [1]
7. Calcium-alkali (milk-alkali) syndrome is the combination of hypercalcaemia, metabolic alkalosis and kidney injury associated with excessive calcium carbonate or other calcium and absorbable alkali intake. [2]
8. Renal failure impairs magnesium excretion and is a specific reason to avoid magnesium-containing antacids, since retained magnesium can progress to hypermagnesemia and, at high levels, cardiac arrest. [3]

Overview

1. Antacids act by directly neutralising acid already present in the stomach, reducing the amount of acid that reaches the duodenum and, at a pH above 4, inhibiting the proteolytic enzyme pepsin. This is a fast but short-acting effect compared with acid-suppressing classes that reduce how much acid is secreted in the first place, which is why antacids are typically used for on-demand symptom relief rather than sustained acid control. [1]
2. The choice of calcium, magnesium or aluminium changes both adverse effects and interactions. Excessive calcium carbonate and absorbable alkali intake can cause calcium-alkali syndrome, with hypercalcaemia, metabolic alkalosis and kidney injury; calcium carbonate may also cause constipation. Magnesium hydroxide neutralises acid, can cause diarrhoea and may accumulate when kidney function is significantly impaired. Aluminium hydroxide neutralises acid and can cause constipation; prolonged use in renal failure can lead to aluminium accumulation and toxicity. Some products combine magnesium with aluminium or calcium to balance bowel effects. [2][3][1]
3. Alginates are mechanistically distinct from every antacid cation above. Sodium alginate is a naturally occurring polysaccharide that gels on contact with gastric acid; combined with sodium bicarbonate, the carbon dioxide released

by that reaction becomes trapped in the gel, turning it into a foam that floats as a raft on top of the stomach contents. Rather than neutralising the bulk of the gastric acid, the raft physically blocks reflux and maintains a locally higher pH within itself - a mechanical barrier rather than a chemical neutraliser, which is why alginate products are marketed specifically for reflux rather than as general antacids. [4]

Classification and drug examples

Intro: Grouped by cation for the antacids, since the cation decides both the bowel side effect and the interaction risk, with alginates kept separate because their mechanism is physical rather than chemical.

1. Name: Calcium-based antacids

1. Note: Neutralise acid and may cause constipation; excessive calcium carbonate and alkali intake can cause calcium-alkali syndrome.

Name: Calcium carbonate

Tums

Route: Oral

Note: May cause constipation; excessive calcium carbonate and alkali intake can cause calcium-alkali syndrome. [2] [2]

2. Name: Magnesium-based antacids

2. Note: Neutralise acid; the osmotic bowel effect that causes diarrhoea at higher doses is the same mechanism that makes magnesium hydroxide a laxative.

Name: Magnesium hydroxide

Milk of magnesia

Route: Oral

Note: Reacts with gastric acid to form water and magnesium chloride; osmotic retention of fluid drives its diarrhoea side effect and its use as a laxative. [3] [3]

3. Name: Aluminium-based antacids

3. Note: Neutralise acid and inhibit pepsin; the characteristic bowel effect runs opposite to magnesium.

Name: Aluminium hydroxide

Route: Oral

Note: Constipation and, in excess, faecal impaction are its characteristic bowel effects; often combined with magnesium hydroxide to offset this. [1] [1]

4. Name: Alginate raft-forming agents

4. Note: A mechanically distinct class: forms a physical, floating barrier rather than neutralising acid chemically.

Name: Sodium alginate (with antacid components)

Gaviscon

Route: Oral

Note: Gels on contact with gastric acid; combined bicarbonate generates CO₂ that turns the gel into a floating raft blocking reflux. [4] [4]

Mechanism of action

Summary: Antacids neutralise gastric acid already present in the stomach through direct acid-base chemistry specific to their cation; alginates instead gel on contact with acid and, with bicarbonate-generated carbon dioxide, form a floating raft that physically blocks reflux rather than neutralising the bulk of the acid.

Target: Gastric acid (chemical neutralisation) for antacids; the gastro-oesophageal junction and gastric contents surface (physical barrier) for alginates

Cidal Static: not applicable (not an antimicrobial class)

Kill Kinetics: not applicable (not an antimicrobial class)

1. Step: 1

1. Title: An antacid cation reacts with gastric acid

1. Detail: Calcium carbonate dissociates into calcium and carbonate ions, with the carbonate binding free hydrogen ions to raise gastric pH; magnesium hydroxide and aluminium hydroxide react with hydrochloric acid to form water and the corresponding chloride salt. [2][3]
2. Step: 2
2. Title: Gastric pH rises and pepsin activity falls
2. Detail: Once pH rises above about 4, pepsin's proteolytic activity is inhibited, reducing mucosal irritation alongside the acid neutralisation itself. [1]
3. Step: 3
3. Title: The antacid effect is short-lived
3. Detail: Because the mechanism is a direct chemical reaction with existing acid rather than suppression of ongoing acid secretion, the effect wears off as new acid is secreted, which is why antacids suit on-demand relief rather than sustained control. [1]
4. Step: 4
4. Title: Alginate takes a different path entirely
4. Detail: Sodium alginate gels on contact with gastric acid; the accompanying sodium bicarbonate reacts with acid to generate carbon dioxide, which becomes trapped inside the gel and converts it into a buoyant foam. [4]
5. Step: 5
5. Title: The raft floats and physically blocks reflux
5. Detail: The resulting raft floats on top of the gastric contents and maintains a locally higher pH within itself, forming a mechanical barrier against reflux rather than neutralising the stomach's overall acid load. [4]

Major clinical uses

1. Drug: Calcium carbonate
1. Indication: On-demand relief of heartburn and acid indigestion
1. Line: first-line/as-needed
1. Note: Fast onset makes it suited to symptom-triggered rather than sustained use. [2]
2. Drug: Magnesium hydroxide
2. Indication: Antacid relief at lower doses; osmotic laxative (milk of magnesia) at higher doses
2. Line: first-line/as-needed
2. Note: The same osmotic mechanism underlies both uses at different dose levels. [3]
3. Drug: Aluminium hydroxide
3. Indication: On-demand relief of acid indigestion; also used to bind phosphate in the gut in hyperphosphataemia
3. Line: first-line/as-needed
3. Note: Phosphate-binding is a separate, non-antacid application of the same chemistry. [1]
4. Drug: Sodium alginate (with antacid components)
4. Indication: Symptomatic gastro-oesophageal reflux, particularly where a physical barrier against reflux is wanted rather than acid neutralisation alone
4. Line: first-line/as-needed
4. Note: Marketed specifically for reflux because the raft mechanism targets the reflux event itself, not just gastric acidity. [4]

Pharmacokinetics

1. This class acts locally within the gastrointestinal tract rather than through systemic absorption and distribution, so conventional pharmacokinetic parameters such as half-life and distribution are not the clinically relevant measure - timing relative to other oral drugs is. [1]
2. Because antacids can bind or alter the absorption of some oral drugs, particularly tetracyclines and fluoroquinolones, a clinician or pharmacist should check product-specific timing instructions for other medicines. [1]

3. This page gives no dose regimens by design. Doses depend on indication, formulation and the treating clinician's or pharmacist's assessment, and belong in a current prescribing reference.

Adverse effects

1. Effect: Diarrhoea (magnesium-based antacids)

1. Detail: An extension of magnesium's osmotic mechanism, the same effect that makes magnesium hydroxide a laxative at higher doses. [3]

2. Effect: Constipation and faecal impaction (aluminium-based antacids)

2. Detail: The characteristic bowel effect of aluminium hydroxide, opposite in direction to magnesium. [1]

3. Effect: Constipation, nausea and vomiting (calcium carbonate)

3. Detail: Documented common adverse effects of calcium carbonate use, alongside acid rebound. [2]

1. Effect: Milk-alkali syndrome

1. Detail: A syndrome of hypercalcaemia, metabolic alkalosis and kidney injury associated with excessive calcium carbonate or other calcium and absorbable alkali intake.

1. Action: Symptoms such as nausea, confusion or reduced urine output during regular calcium carbonate use warrant prompt medical assessment. [2]

2. Effect: Hypermagnesemia

2. Detail: Rare with ordinary antacid use, but serious magnesium accumulation can occur with significant renal impairment, especially when magnesium-containing products are used excessively; severe cases can cause cardiac effects.

2. Action: Magnesium-containing antacids need caution or avoidance in significant renal impairment, and any muscle weakness, confusion or cardiac symptoms in a magnesium user need urgent medical evaluation. [3]

3. Effect: Aluminium accumulation in renal impairment

3. Detail: Prolonged aluminium-containing antacid use in people with renal failure can cause aluminium accumulation, with neurological and bone toxicity including encephalopathy and osteomalacia.

3. Action: People with kidney disease should seek clinician or pharmacist advice before repeated use of aluminium-containing antacids; new confusion or bone symptoms need medical assessment. [1]

Drug: Calcium carbonate

Effect: Excess intake can cause hypercalcaemia and calcium-alkali syndrome; vascular calcification concerns are mainly relevant in chronic kidney disease when calcium carbonate is used as a phosphate binder. [2]

Contraindications, precautions and interactions

Known hypersensitivity to the specific antacid or alginate product or its excipients. [2]

1. Significant renal impairment - magnesium can accumulate with magnesium-containing products, and prolonged aluminium-containing antacid use can increase aluminium toxicity risk; excessive calcium and alkali intake can cause calcium-alkali syndrome. [3][2][1]

2. Heart failure and states requiring sodium restriction, given that some antacid and alginate formulations contain sodium bicarbonate. [1]

3. Existing hypercalcaemia or suspected digoxin toxicity requires clinician assessment before use of calcium-containing products because elevated calcium can worsen toxicity risk. [2][1]

1. With: Tetracycline and fluoroquinolone antibiotics

1. Effect: Magnesium trisilicate, magnesium hydroxide, aluminium salts and calcium carbonate can all bind these antibiotics in the gut, impeding their absorption. [1]

2. With: Other oral medicines with product-specific antacid interactions

2. Effect: Antacids can alter absorption of some oral medicines; check the current prescribing information for the specific medicine rather than assuming a class-wide interaction. [1]

3. With: Drugs cleared renally with pH-dependent excretion

3. Effect: Sodium bicarbonate-containing formulations can alkalinise urine, altering the renal elimination of some drugs. [1]

Comparison tables

Title: Which cation, which bowel effect, which specific risk

Caption: The class's real teaching point: the cation, not just the neutralising strength, decides the side-effect profile. A prescribing reference and the treating clinician govern actual product choice.

1. Key: agent

1. Label: Agent

1. Is Row Header: true

2. Key: bowel_effect

2. Label: Characteristic bowel effect

3. Key: specific_risk

3. Label: Signature serious risk

1. Agent: Calcium carbonate

1. Bowel Effect: Constipation

1. Specific Risk: Milk-alkali syndrome [2]

2. Agent: Magnesium hydroxide

2. Bowel Effect: Diarrhoea (osmotic)

2. Specific Risk: Hypermagnesemia in renal failure [3]

3. Agent: Aluminium hydroxide

3. Bowel Effect: Constipation, faecal impaction

3. Specific Risk: Aluminium accumulation and bone or neurological toxicity with prolonged use in renal failure; phosphate depletion can also occur [1]

4. Agent: Sodium alginate (raft)

4. Bowel Effect: Not a neutraliser; minimal direct bowel effect from the raft mechanism itself

4. Specific Risk: None specific to the raft mechanism beyond its antacid co-ingredients [4]

High-yield exam pearls

1. Pearl: "Antacid" and "alginate" are not the same mechanism wearing different names.

1. Why: Antacids chemically neutralise acid already in the stomach; alginates gel in acid and, with added bicarbonate's CO₂, float as a physical raft over gastric contents without neutralising the bulk of the acid - a mechanical barrier, not a chemical one. [4]

2. Pearl: Magnesium loosens, aluminium tightens.

2. Why: Magnesium hydroxide's osmotic bowel effect causes diarrhoea, while aluminium hydroxide characteristically causes constipation - which is exactly why many combination antacid products pair the two, so one agent's bowel effect offsets the other's. [3][1]

3. Pearl: Calcium-alkali syndrome follows excessive calcium and alkali intake.

3. Why: It is commonly associated with excessive calcium carbonate use and presents with hypercalcaemia, metabolic alkalosis and kidney injury; it is not a general effect of every antacid. [2]

4. Pearl: Antacid timing matters for other drugs, not just for the antacid itself.

4. Why: Because antacids bind or alter the absorption of tetracyclines, fluoroquinolones and other acid-dependent drugs, the practical teaching point is spacing doses apart rather than assuming co-administration is fine. [1]

5. Pearl: Renal function decides whether magnesium is even an option.

5. Why: Magnesium is normally excreted renally; in renal failure that excretion falls, so a magnesium-containing antacid can drive hypermagnesemia toward dangerous cardiac territory in a way a calcium or aluminium product would not. [3]

Common exam traps

1. Trap: "Antacids and alginates both work by neutralising stomach acid."
 1. Correction: Only antacids neutralise acid chemically. Alginates form a physical raft that floats over gastric contents and maintains a locally higher pH inside the raft without neutralising the bulk of the gastric acid - a mechanically distinct mechanism. [4]
 2. Trap: "All antacids cause the same bowel side effect."
 2. Correction: Magnesium-based antacids cause diarrhoea through an osmotic effect; aluminium-based antacids cause constipation. Flattening these into one "antacids upset the bowel" statement loses the exact distinction that makes combination products (magnesium plus aluminium) rational in the first place. [3][1]
 3. Trap: "Milk-alkali syndrome can happen with any antacid."
 3. Correction: It is associated with excessive calcium and absorbable alkali intake, often calcium carbonate, and consists of hypercalcaemia, metabolic alkalosis and kidney injury. [2]

Self-test questions

1. Question: What is the key mechanistic difference between an antacid and an alginate raft-forming agent?
 1. There is no difference; both neutralise gastric acid chemically
 2. Antacids chemically neutralise acid already in the stomach; alginates gel in acid and form a floating physical raft that blocks reflux without neutralising the bulk of the acid
 3. Alginates neutralise acid faster than any antacid
 4. Antacids only work in the oesophagus, alginates only work in the stomach
 1. Explanation: Antacids are direct chemical neutralisers. Alginates instead gel on contact with gastric acid and, with bicarbonate-generated CO₂, form a foam raft that floats on the gastric contents and physically impedes reflux, maintaining a locally higher pH inside the raft rather than neutralising the stomach's overall acid load. [4]
2. Question: A combination antacid product contains both magnesium hydroxide and aluminium hydroxide. What is the pharmacological rationale?
 1. It doubles the neutralising strength with no other purpose
 2. Magnesium's osmotic diarrhoea effect and aluminium's constipating effect tend to offset each other
 3. Aluminium prevents magnesium from being absorbed
 4. There is no rationale; it is a marketing combination only
 2. Explanation: Magnesium hydroxide promotes defecation through osmotic fluid retention, while aluminium hydroxide characteristically causes constipation. Combining them is a deliberate way to reduce the net bowel-habit disturbance of either agent alone. [3][1]
3. Question: Which adverse effect is specifically associated with calcium carbonate rather than magnesium- or aluminium-based antacids?
 1. Diarrhoea
 2. Constipation only
 3. Calcium-alkali syndrome (hypercalcaemia, metabolic alkalosis and kidney injury after excess calcium and alkali intake)
 4. Hypermagnesemia
 3. Explanation: Calcium-alkali syndrome follows excessive calcium and absorbable alkali intake, commonly calcium carbonate, and presents with hypercalcaemia, metabolic alkalosis and kidney injury. Magnesium products instead raise concern for magnesium accumulation in renal impairment. [2]
4. Question: Why should a magnesium-containing antacid be used cautiously in a patient with renal failure?
 1. Renal failure has no effect on magnesium handling
 2. Renal failure decreases magnesium excretion, which can lead to hypermagnesemia and, at high levels, dangerous cardiac effects

3. It increases magnesium absorption from the gut only
4. Renal failure makes magnesium hydroxide chemically inert
4. Explanation: Magnesium is normally cleared by the kidneys. With significant renal impairment, especially alongside excessive magnesium-containing product use, accumulation can cause hypermagnesemia and serious cardiac effects. [3]
5. Question: Why is dose-spacing recommended between an antacid and a tetracycline or fluoroquinolone antibiotic?
1. The antacid destroys the antibiotic in the bloodstream
2. Magnesium-, aluminium- or calcium-containing antacids can bind these antibiotics in the gut, impeding their absorption
3. There is no interaction between antacids and antibiotics
4. The antibiotic neutralises the antacid's effect
5. Explanation: Antacids containing magnesium trisilicate, magnesium hydroxide, aluminium salts or calcium carbonate can chelate or bind tetracycline and fluoroquinolone antibiotics in the gastrointestinal tract, reducing how much of the antibiotic is absorbed - a reason to separate dosing rather than co-administer. [1]

Frequently asked questions

1. Question: What is the difference between an antacid and an alginate product like Gaviscon?
1. Answer: An antacid neutralises gastric acid chemically. An alginate product gels on contact with acid and, with added bicarbonate, forms a foam raft that floats on the stomach contents and physically blocks reflux, without neutralising the bulk of the acid. [4]
2. Question: Why do some antacids cause diarrhoea and others constipation?
2. Answer: It depends on the cation. Magnesium hydroxide has an osmotic effect that draws water into the bowel, causing diarrhoea. Aluminium hydroxide has the opposite tendency, causing constipation, which is why the two are often combined. [3][1]
3. Question: Is it safe to take antacids with other medicines?
3. Answer: Not always at the same time. Magnesium- and aluminium-containing antacids can reduce absorption of tetracycline and fluoroquinolone antibiotics. Timing depends on the specific medicines and products, so a pharmacist or clinician should check the current instructions. [1]
4. Question: What is milk-alkali syndrome?
4. Answer: Calcium-alkali syndrome is the triad of hypercalcaemia, metabolic alkalosis and kidney injury after excessive calcium and absorbable alkali intake, commonly involving calcium carbonate. [2]

References

- [1] Antacids (StatPearls). StatPearls Publishing / NCBI Bookshelf 2023. <https://www.ncbi.nlm.nih.gov/books/NBK526049/>
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- [4] pH-responsive alginate polymeric rafts for controlled drug release, using Box-Behnken response surface design. PubMed Central, National Library of Medicine 2018. <https://pmc.ncbi.nlm.nih.gov/articles/PMC5812168/>

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