

Laxatives and antidiarrhoeals

Two opposite clinical problems, transit that is too slow and transit that is too fast, answered by drugs grouped by how they act rather than by the symptom: laxatives are split into bulk-forming, osmotic, stimulant and stool-softening mechanisms, while the leading antidiarrhoeal, loperamide, is a peripherally restricted opioid whose entire safety story is what happens when that restriction is defeated by overdose.

Online guide: <https://www.medguideglobal.com/pharmacology/gastrointestinal/laxatives-and-antidiarrhoeals>

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Quick revision

One Liner: Laxatives are organised by mechanism, not by brand: bulk-forming agents add stool volume, osmotic agents pull water into the lumen, stimulants drive colonic contraction directly, and stool softeners let water into the stool passively - each with its own onset. Loperamide, the leading antidiarrhoeal, is a mu-opioid agonist that normally cannot cross into the brain, and its entire boxed safety story is about what happens when that restriction is deliberately overridden by high-dose misuse.

1. Bulk-forming agents such as psyllium husk add fibre volume to the stool and depend on the patient drinking enough fluid with them; taking psyllium without adequate liquid is itself a labelled choking risk. [10]
2. Osmotic agents (polyethylene glycol 3350, magnesium citrate, magnesium hydroxide, lactulose) work by drawing water into the intestinal lumen; onset ranges from about 30 minutes to 6 hours for the saline agents to 1 to 3 days for polyethylene glycol. [8][1]
3. Stimulant laxatives (bisacodyl, senna) act directly on the colonic mucosa to trigger peristalsis; bisacodyl's oral onset is 6 to 12 hours, but its rectal suppository acts within 15 to 60 minutes because it bypasses the time needed for colonic conversion to the active metabolite. [11]
4. Docusate is a stool softener, not a stimulant: it lets water and fat penetrate the stool passively, which is why it is cramp-free but also why it is a weaker single agent for established constipation than a stimulant or osmotic drug. [4]
5. Loperamide slows gut transit by agonising mu-opioid receptors in the enteric plexus; at labelled OTC and prescription doses it does not produce central opioid effects because P-glycoprotein at the blood-brain barrier keeps it out of the CNS. [12]
6. That same P-glycoprotein restriction is saturable: taking loperamide well above the labelled dose lets it cross into the brain, and separately produces dose-dependent cardiac sodium- and potassium-channel blockade, producing QT prolongation, ventricular arrhythmia including Torsades de Pointes, syncope and cardiac arrest, some of it fatal. [5][12]
7. Bismuth subsalicylate is both an antidiarrhoeal and an antisecretory/mild antimicrobial agent used for upset stomach and travellers' diarrhoea; it is a salicylate, so it carries the same Reye syndrome caution in children and teenagers recovering from viral illness as aspirin. [6]
8. Chronic overuse of osmotic or stimulant laxatives can produce electrolyte disturbance - the saline magnesium agents in particular are limited to short-term use and to patients with normal renal function because retained magnesium is renally cleared. [8][9]

Overview

1. Constipation and diarrhoea sit at opposite ends of the same physiological axis, transit time through the gut, and the drugs used for each are grouped by mechanism rather than by which end of that axis they treat. On the laxative side there are four genuinely different ways to speed transit: adding bulk to the stool so the bowel wall stretches and contracts reflexively (bulk-forming agents), drawing water into the lumen osmotically (osmotic agents), stimulating colonic smooth muscle and secretion directly (stimulant agents), and softening the stool by letting water penetrate it

passively (stool softeners). Each mechanism has a different onset, which is the single most exam-relevant fact about this group: a patient told to expect relief "tonight" needs a stimulant or a rectal formulation, not an osmotic agent that may take a day or more. [11][1]

2. Polyethylene glycol 3350, the most widely used osmotic agent, is an inert, non-absorbed polymer that simply holds water in the colon; its own label states it generally produces a bowel movement in 1 to 3 days and instructs against use beyond 7 days without medical advice. Magnesium citrate and magnesium hydroxide are faster osmotic agents, producing a bowel movement in as little as 30 minutes to 6 hours, but the retained magnesium is cleared renally, so their labelling restricts them to short-term use and flags patients with kidney disease specifically. Lactulose, a non-absorbable synthetic disaccharide, works the same osmotic way but is fermented by colonic bacteria into acidic byproducts, which is also why gaseous distension, flatulence and cramping are common at effective doses. [1][8][7]

3. Stimulant laxatives illustrate how administration route changes onset even for the same drug. Bisacodyl is a prodrug that must reach the colon and be converted there to its active metabolite before it stimulates the mucosa and drives peristalsis; taken orally that conversion takes 6 to 12 hours, which is why bisacodyl tablets are typically dosed at bedtime for a morning effect. Given rectally as a suppository, the drug acts directly on the rectal mucosa without needing that transit time, producing a bowel movement in as little as 15 to 60 minutes. Senna works by a comparable colon-stimulant mechanism after bacterial activation of its glycosides. Both stimulants and the saline osmotic agents carry the same caution against habitual, long-term self-use because sustained overstimulation of the bowel and repeated fluid/electrolyte shifts are the recognised harms of chronic misuse, distinct from their intended short-term role. [11][2][3]

4. On the antidiarrhoeal side, loperamide is the dominant agent precisely because of a pharmacokinetic accident that became a design feature: it is a synthetic opioid that agonises mu-opioid receptors in the myenteric plexus of the gut wall, slowing propulsive contractions and increasing anal sphincter tone, but at labelled doses it is actively pumped back out of the brain by P-glycoprotein at the blood-brain barrier, so it produces essentially no central opioid effect. That barrier is dose-saturable. Deliberately taking loperamide at many multiples of the labelled dose, historically attempted by people trying to self-treat opioid withdrawal or chase euphoria, overwhelms the efflux pump and lets the drug reach the CNS, while independently causing direct cardiac sodium- and potassium-channel blockade that prolongs the QT interval and can precipitate ventricular arrhythmia, including Torsades de Pointes, syncope and cardiac arrest. This is the reason loperamide's own OTC label carries a plain-language "Heart alert" stating that taking more than directed can cause serious heart problems or death, a form of on-label consumer warning unusual for an OTC antidiarrhoeal. [12][5]

Classification and drug examples

Intro: Laxatives are grouped here by mechanism (bulk-forming, osmotic, stimulant, stool softener); antidiarrhoeals are grouped separately by target (peripheral opioid receptor, or the antisecretory/mild-antimicrobial action of bismuth subsalicylate).

1. Name: Bulk-forming laxatives

1. Note: Increase stool volume and water content mechanically; require adequate co-administered fluid to work safely.

1. Name: Psyllium husk

Metamucil

1. Route: Oral

1. Note: Must be taken with at least 8 oz of liquid; taking it dry is a labelled choking hazard. [10]

2. Name: Methylcellulose

Citrucel

2. Route: Oral

2. Note: A semisynthetic bulk-forming fibre with a similar mechanism to psyllium; not separately profiled on this site.

[10] [10]

2. Name: Osmotic laxatives

2. Note: Draw water into the intestinal lumen by osmosis; onset ranges from under an hour (saline agents) to 1 to 3 days (polyethylene glycol).

1. Name: Polyethylene glycol 3350

MiraLAX

1. Route: Oral

1. Note: Non-absorbed, isosmotic polymer; label states not for use beyond 7 days without medical supervision. [1]

2. Name: Lactulose

2. Route: Oral

2. Note: Non-absorbable disaccharide fermented by colonic bacteria; gaseous distension and cramping are common at effective doses. [7]

3. Name: Magnesium citrate

3. Route: Oral

3. Note: Saline osmotic agent; bowel movement typically in 30 minutes to 6 hours, restricted to short-term use. [8]

4. Name: Magnesium hydroxide

Milk of Magnesia

4. Route: Oral

4. Note: Also used as an antacid at lower doses; laxative effect is dose- and concentration-dependent. [9] [1][8]

3. Name: Stimulant laxatives

3. Note: Act directly on colonic mucosa/myenteric plexus to trigger peristalsis; oral onset is markedly slower than rectal onset for the same drug.

1. Name: Bisacodyl

Dulcolax

1. Route: Oral or rectal

1. Note: Oral onset 6 to 12 hours; rectal suppository onset 15 to 60 minutes because colonic prodrug conversion is bypassed. [11]

2. Name: Senna

Senokot

2. Route: Oral

2. Note: Sennoside glycosides are activated by colonic bacteria before stimulating peristalsis. [3] [11]

4. Name: Stool softeners (emollient laxatives)

4. Note: Let water and fat passively penetrate the stool rather than stimulating the bowel; generally the gentlest and slowest class.

Name: Docusate sodium

Colace

Route: Oral

Note: Cramp-free and stimulant-free by design; onset typically 12 to 72 hours. [4] [4]

5. Name: Peripheral mu-opioid agonist (antidiarrhoeal)

5. Note: Acts on gut opioid receptors without labelled-dose central effect; the class's defining safety issue is what happens when that restriction is defeated by overdose.

Name: Loperamide

1. Imodium

2. Imodium A-D

Route: Oral

Note: OTC label carries an explicit consumer-facing cardiac "Heart alert" against exceeding the directed dose. [5] [12]

6. Name: Antisecretory salicylate (antidiarrhoeal)

6. Note: A bismuth salt of salicylic acid with antisecretory, mild antimicrobial and mucosal-protective action.

Name: Bismuth subsalicylate

Pepto-Bismol

Route: Oral

Note: Contains a salicylate moiety; the same Reye syndrome caution in children/teenagers with viral illness applies as for aspirin. [6] [6]

Mechanism of action

Summary: Laxative mechanisms fall into four categories that act at different points along the same pathway to a bowel movement: bulk-forming agents add mechanical stretch, osmotic agents add luminal water, stimulant agents add direct mucosal/neural drive, and stool softeners add passive water penetration. The antidiarrhoeals covered here act by an entirely different logic: loperamide slows the gut down by agonising the same mu-opioid receptor family used by systemic opioid analgesics, but confined to the gut wall by an efflux transporter at the blood-brain barrier, while bismuth subsalicylate reduces intestinal fluid secretion and has mild antimicrobial and mucosal-protective action.

Target: Intestinal lumen and mucosa (laxatives, by mechanism); enteric mu-opioid receptors (loperamide); intestinal secretory processes and, to a lesser extent, mucosal bacteria (bismuth subsalicylate)

Cidal Static: Not applicable - none of these agents act on a pathogen

Kill Kinetics: Onset varies by mechanism from minutes (rectal stimulant) to days (bulk-forming, polyethylene glycol); not applicable in the cidal/static sense

1. Step: 1

1. Title: Bulk-forming agents absorb water and expand

1. Detail: Psyllium husk and methylcellulose absorb water in the gut, increasing stool volume and softness, which stretches the bowel wall and triggers the normal defecation reflex; adequate co-administered fluid is required for this mechanism to work rather than to cause obstruction. [10]

2. Step: 2

2. Title: Osmotic agents pull water into the lumen

2. Detail: Polyethylene glycol 3350 is an inert, non-absorbed polymer that is isosmotic and simply retains the water it is dissolved in; magnesium salts and lactulose are poorly absorbed solutes that osmotically draw additional water into the bowel lumen, softening and increasing the volume of stool. [1][7]

3. Step: 3

3. Title: Stimulant agents act on the colon directly

3. Detail: Bisacodyl, after conversion to its active metabolite in the colon, and senna, after bacterial activation of its glycosides, act directly on the colonic mucosa and myenteric plexus to stimulate peristaltic contraction and alter fluid and electrolyte movement across the mucosa. [11]

4. Step: 4

4. Title: Stool softeners let water and fat penetrate passively

4. Detail: Docusate is a surfactant that lowers stool surface tension, allowing water and lipids to mix into the stool mass more easily without directly stimulating colonic contraction. [4]

5. Step: 5

5. Title: Loperamide agonises mu-opioid receptors confined to the gut

5. Detail: Loperamide binds mu-opioid receptors on the myenteric plexus of the intestinal wall, reducing propulsive peristalsis, increasing segmental contractions and raising anal sphincter tone, which together slow transit and reduce stool frequency and volume. [12]

6. Step: 6

6. Title: P-glycoprotein normally excludes loperamide from the brain

6. Detail: At labelled doses, loperamide that reaches the systemic circulation is actively transported back out of the CNS by the P-glycoprotein efflux pump at the blood-brain barrier, which is why a peripherally acting opioid can be sold over the counter without producing central opioid effects at normal use. [12]

7. Step: 7

7. Title: Overdose saturates that exclusion and adds direct cardiotoxicity

7. Detail: At supratherapeutic doses the P-glycoprotein efflux mechanism is saturated, allowing CNS penetration, while loperamide independently blocks cardiac sodium and potassium channels in a dose-dependent way, prolonging the QT interval and predisposing to ventricular arrhythmia including Torsades de Pointes. [5][12]

Major clinical uses

1. Drug: Psyllium husk / methylcellulose

1. Organism: Not applicable

1. Indication: Occasional constipation; fibre supplementation

1. Line: first-line for mild, chronic constipation, especially with adequate hydration

1. Note: Gentlest mechanism; slowest onset among laxative classes when used alone. [10]

2. Drug: Polyethylene glycol 3350 / magnesium citrate / magnesium hydroxide / lactulose

2. Organism: Not applicable

2. Indication: Occasional constipation; bowel preparation (higher-dose PEG regimens); hepatic encephalopathy (lactulose, via colonic acidification and ammonia trapping)

2. Line: first- or second-line depending on urgency and comorbidity

2. Note: Saline magnesium agents are limited to short-term use, particularly with renal impairment. [1][8][7]

3. Drug: Bisacodyl / senna

3. Organism: Not applicable

3. Indication: Occasional constipation; bowel preparation before colonoscopy; opioid-induced constipation

3. Line: used short-term or bedtime-dosed for a morning effect; rectal bisacodyl for faster, procedure-timed relief

3. Note: Not intended for prolonged, habitual self-use. [11][3]

4. Drug: Docusate sodium

4. Organism: Not applicable

4. Indication: Occasional constipation, particularly where straining should be avoided (e.g. after surgery or myocardial infarction)

4. Line: adjunct or first-line for mild constipation

4. Note: Cramp-free but a comparatively weak single agent for established constipation. [4]

5. Drug: Loperamide

5. Organism: Not applicable

5. Indication: Acute nonspecific diarrhoea, travellers' diarrhoea, chronic diarrhoea associated with inflammatory bowel disease, and reducing ileostomy output

5. Line: first-line symptomatic antidiarrhoeal at labelled dose

5. Note: Contraindicated in bloody or black stool of unclear cause without medical advice, per OTC labelling. [5]

6. Drug: Bismuth subsalicylate

6. Organism: Not applicable

6. Indication: Upset stomach, nausea, indigestion and travellers'/acute diarrhoea

6. Line: OTC first-line for mild self-limited GI upset

6. Note: Salicylate content means the paediatric Reye syndrome caution applies. [6]

Pharmacokinetics

1. Drug: Polyethylene glycol 3350

1. Route: Oral

1. Absorption: Minimal to none; acts locally in the gut lumen

1. Csf Penetration: Not applicable

1. Metabolism: Not metabolised

1. Elimination: Excreted unchanged in stool

1. Dose Adjust: Caution in kidney disease; not for use beyond 7 days without medical supervision [1]
2. Drug: Bisacodyl
2. Route: Oral or rectal
2. Absorption: Minimal systemic absorption; acts locally after colonic conversion to its active metabolite
2. Csf Penetration: Not applicable
2. Metabolism: Hydrolysed to the active metabolite BHPM in the colon
2. Elimination: Largely faecal
2. Dose Adjust: None specific beyond avoiding prolonged habitual use [11]
3. Drug: Loperamide
3. Route: Oral
3. Absorption: Well absorbed but undergoes extensive first-pass hepatic metabolism, limiting systemic exposure
3. Csf Penetration: Normally excluded by P-glycoprotein at labelled dose; penetration occurs when this efflux is saturated at supratherapeutic dose
3. Metabolism: Hepatic, oxidative N-demethylation
3. Elimination: Faecal, as unchanged drug and metabolites; elimination half-life roughly 11 hours
3. Dose Adjust: Hepatic impairment increases systemic exposure; strict avoidance of exceeding labelled maximum daily dose [12]
4. Drug: Bismuth subsalicylate
4. Route: Oral
4. Absorption: Salicylate moiety is absorbed; bismuth itself is poorly absorbed
4. Csf Penetration: Not applicable
4. Metabolism: Hydrolysed in the gut to salicylate and bismuth salts
4. Elimination: Salicylate renally eliminated; bismuth largely faecal
4. Dose Adjust: Avoid with concurrent aspirin/salicylate use; caution in renal impairment [6]
1. This page gives no dose regimens by design. Dose selection depends on age, indication, renal function and duration of use, and belongs in a current prescribing/OTC labelling reference.
2. Onset of action, not potency, is usually the deciding factor in choosing a laxative class clinically: bulk-forming and polyethylene glycol act over days, saline osmotic and rectal stimulant agents act within hours to an hour, which matters when a bowel movement is needed on a schedule (e.g. before a procedure). [1][8][11]

Adverse effects

1. Effect: Bloating, flatulence and abdominal cramping
1. Detail: Reported across bulk-forming, osmotic and stimulant agents; fermentation of lactulose by colonic bacteria makes gaseous distension particularly common with that drug. [7][2]
2. Effect: Diarrhoea with excessive laxative dosing
2. Detail: Any of these classes can overshoot into loose stool if dosed above what is needed for the individual patient. [7][1]
3. Effect: Constipation with loperamide
3. Detail: The mirror-image effect of loperamide's intended mechanism; listed as a common adverse reaction on ondansetron-adjacent antiemetic labels as well, reflecting how often slowed gut transit is the flip side of a drug's intended action elsewhere in this hub. [5]
1. Effect: Fluid and electrolyte disturbance
1. Detail: Overuse of saline osmotic agents (magnesium citrate, magnesium hydroxide) or stimulant laxatives can produce clinically significant dehydration, hypokalaemia or, for the magnesium agents specifically, hypermagnesaemia in patients with reduced renal clearance.
1. Action: Saline laxatives are limited to short-term use and used cautiously, or avoided, in renal impairment; persistent or severe diarrhoea from any laxative warrants medical review. [8][9]

2. Effect: Cardiac arrhythmia with loperamide overdose or misuse
2. Detail: Supratherapeutic loperamide dosing has produced QT-interval prolongation, ventricular tachycardia, Torsades de Pointes, syncope and cardiac arrest, some fatal; most reported cases involved deliberate high-dose misuse, often to self-treat opioid withdrawal or seek euphoria.
2. Action: The OTC label carries an explicit consumer "Heart alert" against exceeding the directed dose; new palpitations, fainting or chest symptoms during loperamide use warrant urgent evaluation. [5][12]
3. Effect: Choking or oesophageal obstruction with bulk-forming agents taken without enough fluid
3. Detail: Psyllium and methylcellulose swell on contact with water; taken dry or with too little liquid they can obstruct the oesophagus.
3. Action: Always taken with at least the labelled volume of fluid; avoided in patients with swallowing difficulty unless medically supervised. [10]
4. Effect: Reye syndrome risk with bismuth subsalicylate in children/teenagers
4. Detail: Because bismuth subsalicylate contains a salicylate, it carries the same caution as aspirin regarding Reye syndrome in children and teenagers recovering from chickenpox or flu-like illness.
4. Action: Avoided in that population; a non-salicylate antidiarrhoeal is used instead if needed. [6]
1. Drug: Polyethylene glycol 3350
1. Effect: Generally well tolerated; label explicitly limits use to 7 days without medical supervision and cautions in kidney disease. [1]
2. Drug: Bisacodyl
2. Effect: Oral onset 6 to 12 hours versus rectal onset 15 to 60 minutes is a labelled, clinically important distinction for the same drug. [11]
3. Drug: Loperamide
3. Effect: The only drug in this class with a documented, dose-dependent cardiac sodium/potassium-channel blockade mechanism distinct from its intended gut effect. [12]
4. Drug: Bismuth subsalicylate
4. Effect: Can cause harmless temporary darkening (blackening) of the tongue and stool, which is sometimes mistaken by patients for gastrointestinal bleeding. [6]

Contraindications, precautions and interactions

1. Loperamide: known hypersensitivity to loperamide; not used where constipation must be avoided, such as in acute dysentery with blood in the stool and high fever, without medical advice. [5]
2. Bismuth subsalicylate: not used in children or teenagers with or recovering from chickenpox or flu-like symptoms, because of the Reye syndrome association with salicylates. [6]
1. Bulk-forming agents are used cautiously, or avoided, in patients with dysphagia, oesophageal stricture or bowel obstruction, and always with adequate co-administered fluid. [10]
2. Saline osmotic agents (magnesium citrate, magnesium hydroxide) are used with caution or avoided in renal impairment because retained magnesium is renally cleared. [8][9]
3. Stimulant and osmotic laxatives are not intended for prolonged, habitual self-use without medical guidance; new or worsening abdominal pain, rectal bleeding or persistent altered bowel habit is a reason to seek medical evaluation rather than to self-treat further. [1][2]
4. Loperamide dosing is never exceeded beyond the labelled maximum, and hepatic impairment increases systemic exposure and cardiac risk. [12][5]
1. With: Other QT-prolonging drugs or CYP3A4/P-glycoprotein inhibitors
1. Effect: Can raise loperamide plasma levels or add to QT prolongation risk, increasing the chance of arrhythmia even without frank overdose. [12]
2. With: Oral medicines taken close in time to bulk-forming fibre
2. Effect: Psyllium and similar fibres can bind or slow absorption of other oral drugs; labelling recommends separating

dosing by roughly 2 hours. [10]

3. With: Aspirin or other salicylates

3. Effect: Concurrent use with bismuth subsalicylate raises combined salicylate exposure and toxicity risk. [6]

Comparison tables

Title: Same clinical goal, four different mechanisms and four different onsets

Caption: Choice of laxative class is driven mostly by how quickly relief is needed and by comorbidity (renal function, dysphagia), not by potency alone.

1. Key: class

1. Label: Class

1. Is Row Header: true

2. Key: example

2. Label: Example

3. Key: mechanism

3. Label: Mechanism

4. Key: onset

4. Label: Typical onset

1. Class: Bulk-forming

1. Example: Psyllium husk

1. Mechanism: Adds fibre volume and water, stretching the bowel wall

1. Onset: 12 to 72 hours [10]

2. Class: Osmotic (polymer)

2. Example: Polyethylene glycol 3350

2. Mechanism: Isosmotic, non-absorbed polymer retains luminal water

2. Onset: 1 to 3 days [1]

3. Class: Osmotic (saline)

3. Example: Magnesium citrate

3. Mechanism: Poorly absorbed magnesium salt draws water osmotically

3. Onset: 30 minutes to 6 hours [8]

4. Class: Stimulant (oral)

4. Example: Bisacodyl tablet

4. Mechanism: Colonic conversion to active metabolite, then direct mucosal stimulation

4. Onset: 6 to 12 hours [11]

5. Class: Stimulant (rectal)

5. Example: Bisacodyl suppository

5. Mechanism: Direct rectal mucosal stimulation, bypassing colonic conversion delay

5. Onset: 15 to 60 minutes [11]

6. Class: Stool softener

6. Example: Docusate sodium

6. Mechanism: Surfactant action lets water and fat penetrate stool passively

6. Onset: 12 to 72 hours [4]

High-yield exam pearls

1. Pearl: Same drug, two very different onsets: oral bisacodyl (6-12h) versus rectal bisacodyl (15-60min).

1. Why: It is a classic exam point that route, not the drug itself, is what determines onset when a prodrug needs colonic conversion. [11]

2. Pearl: Loperamide is an opioid that is not supposed to reach the brain - until it is taken in overdose.

2. Why: P-glycoprotein efflux explains why an opioid agonist is sold over the counter, and its saturability at high dose explains the entire misuse/cardiotoxicity story. [12]
3. Pearl: Loperamide's cardiac risk is a direct sodium/potassium-channel effect, separate from its opioid mechanism.
3. Why: It is easy to assume the danger of loperamide overdose is purely "too much opioid"; the cardiotoxicity is an independent ion-channel action seen at high plasma levels. [12]
4. Pearl: Saline osmotic laxatives (magnesium salts) are the ones most tied to renal caution among the laxatives.
4. Why: Because retained magnesium is renally cleared, hypermagnesaemia risk - not simply diarrhoea - is the reason these are short-term/renal-caution agents. [8]
5. Pearl: Bismuth subsalicylate is a salicylate, so the Reye syndrome caution in febrile children/teens applies to it exactly as it does to aspirin.
5. Why: It is easy to overlook that an antidiarrhoeal carries an aspirin-family paediatric warning because the drug is not marketed as a pain reliever. [6]

Common exam traps

1. Trap: Assuming all laxatives work by the same osmotic mechanism.
1. Correction: Four distinct mechanisms exist - bulk-forming, osmotic, stimulant, and stool-softener - and mixing them up misassigns both onset and adverse-effect profile. [11][1]
2. Trap: Believing loperamide is simply "too weak an opioid" to cause harm.
2. Correction: At labelled dose it is peripherally restricted by P-glycoprotein, not weak; at supratherapeutic dose it both penetrates the CNS and independently blocks cardiac ion channels, which is a different and more dangerous mechanism than opioid excess alone. [12][5]
3. Trap: Treating bulk-forming laxatives as risk-free because they are "just fibre."
3. Correction: Taken without adequate fluid they carry a labelled choking/obstruction risk and can also bind co-administered oral drugs. [10]
4. Trap: Assuming any antidiarrhoeal is safe for febrile children with viral illness.
4. Correction: Bismuth subsalicylate specifically is a salicylate and carries the Reye syndrome caution in that population, unlike loperamide. [6]

Self-test questions

1. Question: Why does oral bisacodyl take 6 to 12 hours to work while rectal bisacodyl works in 15 to 60 minutes?
1. The rectal dose is much higher
2. Bisacodyl is a prodrug that needs time to reach and be converted in the colon; the rectal route acts directly on rectal mucosa without that delay
3. Rectal administration bypasses the liver entirely, changing the drug's structure
4. There is no real difference; timing is coincidental
1. Explanation: Bisacodyl must be converted to its active metabolite in the colon before it can stimulate peristalsis when taken orally; rectal administration acts locally and skips that transit and conversion delay. [11]
2. Question: At labelled OTC doses, why doesn't loperamide cause central opioid effects like sedation or euphoria?
1. It does not bind opioid receptors at all
2. P-glycoprotein at the blood-brain barrier actively pumps it back out of the CNS
3. It is destroyed completely by stomach acid before absorption
4. It only works after conversion to a non-opioid metabolite
2. Explanation: Loperamide is a mu-opioid agonist that is normally excluded from the CNS by P-glycoprotein efflux transport at the blood-brain barrier, confining its effect to the gut at labelled doses. [12]
3. Question: What is the mechanism behind loperamide's cardiac toxicity in overdose?
1. Excessive central opioid depression of the brainstem
2. Direct blockade of cardiac sodium and potassium channels, prolonging the QT interval

3. An allergic hypersensitivity reaction
4. Severe dehydration from diarrhoea rebound
3. Explanation: At supratherapeutic plasma concentrations, loperamide directly blocks cardiac sodium and potassium channels, producing QT prolongation and predisposing to ventricular arrhythmia including Torsades de Pointes. [12][5]
4. Question: Which laxative class is most specifically limited by renal function?
 1. Bulk-forming agents like psyllium
 2. Saline osmotic agents like magnesium citrate
 3. Stool softeners like docusate
 4. Stimulant agents like senna
4. Explanation: Saline osmotic laxatives contain magnesium that is cleared renally; in renal impairment this can accumulate and cause hypermagnesaemia, so these agents are limited to short-term use and used cautiously in that population. [8]
5. Question: Why must bulk-forming laxatives such as psyllium always be taken with adequate fluid?
 1. Otherwise the drug is destroyed by stomach acid
 2. The fibre swells on contact with water and can cause choking or oesophageal obstruction if taken without enough liquid
 3. Fluid is needed to activate an enzyme in the fibre
 4. It has no real safety reason, only a taste reason
5. Explanation: Psyllium and similar bulk-forming agents absorb and swell with water; without adequate co-administered fluid this swelling can obstruct the oesophagus, a labelled choking hazard. [10]
6. Question: Why does bismuth subsalicylate carry a Reye syndrome caution in children and teenagers with viral illness?
 1. It contains a corticosteroid
 2. It contains a salicylate moiety, the same class of compound implicated in Reye syndrome with aspirin
 3. It suppresses the immune system
 4. It is metabolised to a compound structurally identical to acetaminophen
6. Explanation: Bismuth subsalicylate is hydrolysed to release salicylate, so it carries the same Reye syndrome caution in children and teenagers recovering from chickenpox or flu-like illness as aspirin does. [6]

Frequently asked questions

1. Question: Which laxative works fastest?
 1. Answer: Among the oral options, saline osmotic agents such as magnesium citrate act fastest, typically within 30 minutes to 6 hours. Rectal bisacodyl acts even faster, within 15 to 60 minutes, because it bypasses the colonic transit time an oral stimulant needs for conversion to its active form. Bulk-forming agents and polyethylene glycol are the slowest, generally taking a day or more. [8][11][1]
 2. Question: Is it dangerous to take more loperamide than directed to stop diarrhoea faster?
 2. Answer: Yes. Exceeding the labelled dose is specifically associated with serious heart problems, including QT-interval prolongation, ventricular arrhythmia and cardiac arrest, some of them fatal. This is not a theoretical risk - it is significant enough that the OTC label carries a direct consumer-facing "Heart alert." The labelled maximum dose should never be exceeded. [5][12]
 3. Question: Why do some laxatives cause more gas and cramping than others?
 3. Answer: Agents that are fermented by colonic bacteria, such as lactulose, or that stimulate the bowel directly, such as bisacodyl and senna, are more prone to producing gas, bloating and cramping than gentler mechanisms like stool softeners or non-fermented osmotic agents such as polyethylene glycol 3350. [7][2]
 4. Question: Is it safe to use laxatives every day long-term?
 4. Answer: Most laxative labelling, across bulk-forming, osmotic and stimulant classes, cautions against prolonged, habitual self-use without medical supervision, both because chronic constipation may have an underlying cause that

needs evaluation and because sustained overuse - particularly of saline or stimulant agents - can produce fluid and electrolyte disturbance. Ongoing constipation needing daily laxative use is a reason to see a clinician rather than to self-manage indefinitely. [1][8]

References

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