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DRUG INTERACTIONS

Veterinary Drug Interactions: The Combinations to Avoid in Practice (PDF)

Most harmful veterinary drug interactions are not exotic — they are ordinary combinations assembled one sensible decision at a time. Inside: Two labelled figures and five reference tables.

Contents

The 8 sections of this guide, in order.

IN SHORT

Most harmful **veterinary drug interactions** are not exotic — they are ordinary combinations assembled one sensible decision at a time. The ones worth knowing cold: **NSAID with a corticosteroid** (never overlap — wash out first), **two drugs pulling the same lever** (two ulcerogenics, two nephrotoxins), **serotonergic stacking**, where trazodone, tramadol, ondansetron and metoclopramide are all on the same list, the one true contraindication of **an MAOI** (selegiline, or an amitraz collar) with any of them, **CYP inhibitors** such as ketoconazole and cimetidine that quietly raise a partner drug's concentration, and **phenobarbital**, which does the opposite and causes silent treatment failure. A useful sorting rule: interactions at *absorption* usually need only a **2-hour gap**; interactions at the *target* need you to choose a different drug. Free printable chart included.

KEY POINTS

1. **Know the level and you know the fix.** In the syringe → do not mix. At absorption → separate the doses. In the liver → adjust and monitor. At the target → avoid.
2. **NSAID plus corticosteroid is the one to never get wrong.** Both injure gastric mucosa, and the effect is explicitly described as exacerbated.
3. **Serotonergic drugs stack invisibly.** Trazodone, tramadol, ondansetron, metoclopramide, mirtazapine, SSRIs and clomipramine are all on the same list — four can accumulate in one hospitalised patient, though the reuptake inhibitors drive it far more than the 5-HT_3 antagonists.
4. **MAO inhibitors are the one absolute.** Selegiline and amitraz — including collars — are contraindicated with any serotonergic drug, with a 14-day washout, not merely monitored.
5. **Enzyme inhibitors cause toxicity; inducers cause failure.** Inhibition looks like an overdose; induction looks like the disease coming back.
6. **Chelation interactions are a scheduling problem, not a prescribing one** — a 2-hour gap usually solves them.
7. **Ask about the breed.** MDR1 status changes every P-glycoprotein conversation.
8. **Ask what the owner gives at home.** Supplements, antacids and leftover analgesics are the drugs missing from the record.

About this article

Every interaction below is taken from the *Drug Interactions* section of the relevant monograph in *Plumb's Veterinary Drug Handbook*, 10th ed., or from *Veterinary Pharmacology and Therapeutics*, 10th ed. Where a statement is a widely taught clinical convention rather than a handbook statement, it is labelled as such. This is a practice aid, not a substitute for checking the specific pair in front of you.

Almost no clinically important drug interaction begins with a bad decision. It begins with four reasonable ones. The dog is anxious, so it gets trazodone. It has surgery, so it gets tramadol. It vomits afterwards, so it gets ondansetron. It keeps vomiting, so metoclopramide is added for prokinesis. Every step is defensible, and the patient is now on four drugs that appear on the same serotonergic list.

That is what **veterinary drug interactions** actually look like in practice — not exotic pairs from a textbook, but ordinary combinations assembled one sensible decision at a time, usually by more than one person. This guide is organised the way you meet them: by the

combination you are about to create, with the mechanism, the severity, and what to do about it.

01 The combinations that matter

If you read nothing else, read this table. Everything after it is the reasoning.

COMBINATION	SEVERITY	WHAT HAPPENS	WHAT TO DO
NSAID + corticosteroid	AVOID	Both injure gastric mucosa by different routes. Plumb's: giving ulcerogenic drugs with glucocorticoids **may increase the risk for GI ulceration**; the pharmacology text is blunter — glucocorticoid lesions are **exacerbated** by simultaneous NSAIDs	Do not overlap. Stop the NSAID and allow a **washout** before starting the steroid. See our NSAIDs guide
Two NSAIDs, or an NSAID started straight after another	AVOID	Additive COX inhibition on gastric and renal prostaglandins	Never overlap. Observe the washout interval for the drug being stopped
Serotonergic stacking trazodone, tramadol, ondansetron, metoclopramide, mirtazapine, SSRIs, clomipramine	CAUTION	Plumb's lists all of these as **serotonergic agents**. Three or four accumulate easily in one hospitalised patient without anyone prescribing a "serotonin drug"	**Count them on the chart** before adding another. Watch for agitation, tremor, hyperthermia and tachycardia
MAO inhibitor + any serotonergic drug selegiline (Anipryl), amitraz — *including tick collars*	AVOID	The most severe interaction on this page. Plumb's: there is a potential risk for **life-threatening serotonin syndrome**, and concurrent use is **contraindicated** — not merely cautioned	**A 14-day washout** between the two drugs, in either direction. Ask whether the dog wears an amitraz collar before dispensing tramadol, trazodone or an SSRI
NSAID + ACE inhibitor	CAUTION	ACE inhibitors work partly through vasodilatory prostaglandins on renal function, so **NSAIDs may decrease their effectiveness** and **reduce the effect on blood pressure**. Plumb's notes one canine study with tepoxalin showed no adverse effects	Not an absolute bar, but avoid in the dehydrated, hypotensive or renally compromised. Monitor renal values and blood pressure
ACE inhibitor + potassium-sparing diuretic benazepril or enalapril + spironolactone	CAUTION	Two drugs pulling the same lever on potassium. Plumb's: **hyperkalaemia (hyperkalemia) may develop**, and notes that the UK label makes the combination with veterinary-labelled enalapril **contraindicated**	Widely and deliberately co-prescribed in cardiac cases — the point is not to avoid it but to **check potassium**, and to think again if the patient is also on an ACE-sparing potassium supplement or becomes azotaemic
Aminoglycoside + furosemide	AVOID	Loop diuretics **may increase the nephrotoxic or ototoxic potential of aminoglycosides** — two of the classic nephrotoxins together in a patient who is being actively dehydrated	Choose another antibiotic. If genuinely unavoidable, ensure volume repletion and monitor renal values closely
CYP inhibitor + narrow-therapeutic-index drug ketoconazole, itraconazole, cimetidine, erythromycin, fluconazole, diltiazem	CAUTION	These inhibit cytochrome P450, so the partner drug's clearance falls and its half-life rises. Plumb's example: this group **increases the half-life and decreases the clearance** of alfentanil, prolonging effects and raising the risk of respiratory depression	Anticipate a **dose reduction** in the partner drug and monitor. The effect builds over days, not minutes
P-glycoprotein inhibitor + ivermectin	AVOID	Inhibitors block the **P-glycoprotein (MDR1)** transporter and **increase ivermectin concentrations** — the transporter that normally keeps it out of the CNS	Especially in **MDR1-mutant** herding breeds. Avoid the combination; ask about breed and prior reactions to MDR1 substrates
Phenobarbital + almost anything hepatically cleared	CAUTION	The opposite problem: phenobarbital **induces** microsomal enzymes, so partner drugs are cleared faster and **fail quietly**. Plumb's cites increased aspirin metabolism and reduced progestin activity	Expect **reduced efficacy**, not toxicity. Reassess doses of co-administered drugs after a few weeks
Digoxin + anything that raises its level or drops potassium	CAUTION	A narrow therapeutic index drug. Plumb's: serum concentrations may be increased, and hypokalaemia (hypokalemia) raises the risk of toxicity	**Monitor serum digoxin** and watch for arrhythmias, vomiting, diarrhoea (diarrhea) and inappetence

02 The four levels of interaction

There is one idea that makes the whole subject manageable: **work out which level the interaction is happening at, and the correct response follows automatically**. Two drugs that bind each other in the gut need a gap between doses. Two drugs that damage the same tissue need one of them removed. The two problems look similar on a warning label and are managed completely differently.

Levels 2 and 3 are **pharmacokinetic** — something has changed how much drug is present. Level 4 is **pharmacodynamic** — the concentrations are exactly what you intended, and the two drugs are simply doing the same thing to the same tissue. That single distinction decides whether the answer is a clock or a different prescription.

LEVEL	WHAT IS HAPPENING	TYPICAL EXAMPLE	THE FIX
1 · In the syringe pharmaceutical	Physical or chemical incompatibility before the drug ever reaches the patient	Two drugs mixed in one syringe, or run into the same giving set, that precipitate	AVOID Never co-mix without checking compatibility
2 · At absorption pharmacokinetic	One drug binds, chelates or alters the pH around the other in the gut, so less of it gets in	Fluoroquinolone + calcium; sucralfate + doxycycline	SEPARATE Usually a **2-hour gap** — same drugs, different clock
3 · Metabolism and transport pharmacokinetic	CYP450 inhibition or induction in the liver, or P-glycoprotein blockade at the barrier — the partner drug's concentration moves	Ketoconazole or cimetidine raising a partner drug; phenobarbital lowering one	CAUTION Adjust the dose and monitor; the effect builds over days
4 · At the target pharmacodynamic	Two drugs pulling the same physiological lever, with normal concentrations of both	NSAID + steroid on gastric mucosa; serotonergic stacking; two nephrotoxins	AVOID Choose a different drug — **scheduling cannot help here**

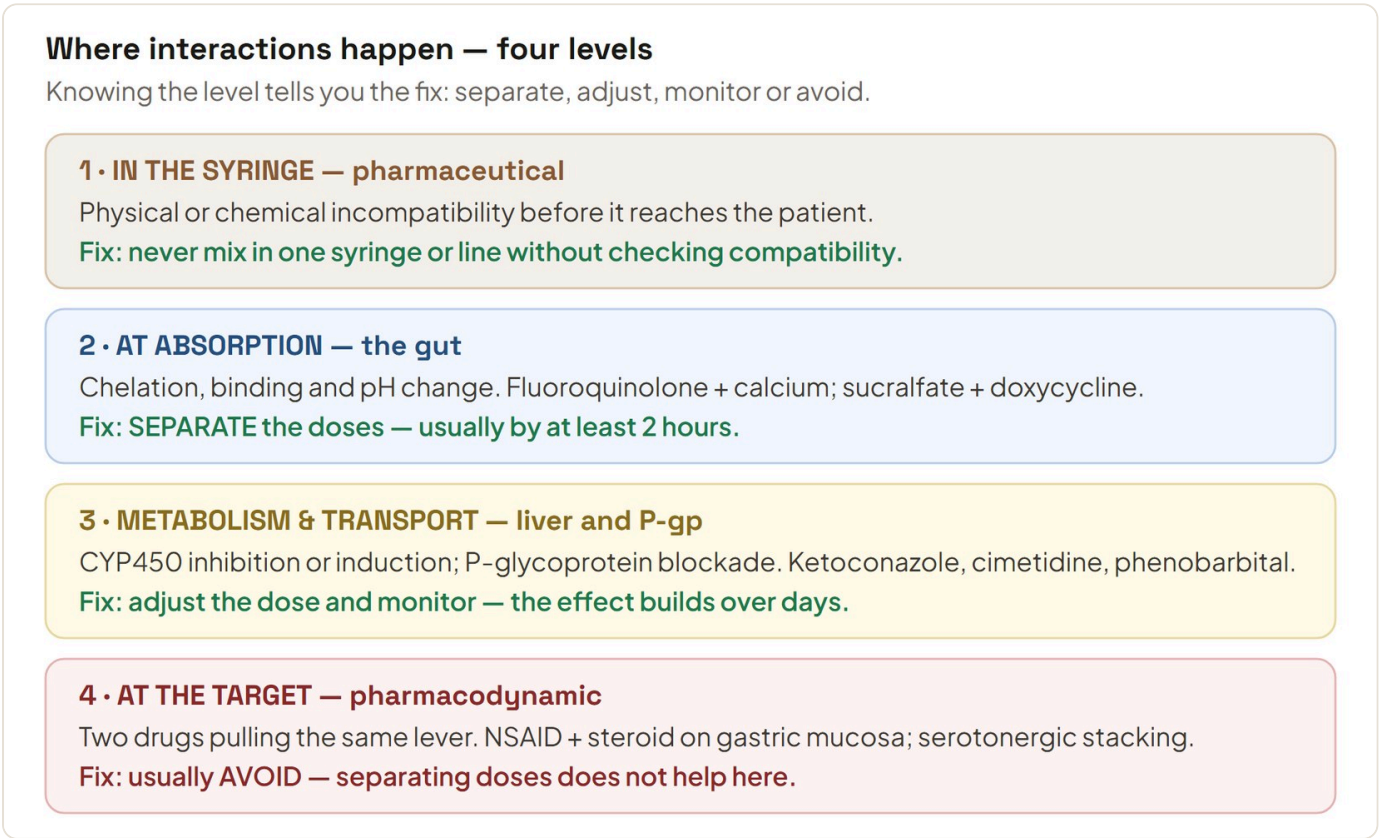

Fig 1 — The four levels, and the fix that goes with each. The distinction that saves the most trouble is between level 2 and level 4: an **absorption** problem is solved by a two-hour gap, while a **target** problem is not solved by any amount of scheduling.

03 NSAID combinations

NSAIDs generate more clinically significant interactions in first-opinion practice than any other class, simply because they are prescribed so often and to patients who are frequently on something else.

With a corticosteroid this is the combination to never get wrong. Plumb's puts it as giving ulcerogenic drugs with glucocorticoids *may increase the risk for GI ulceration*; the pharmacology text is blunter, stating that glucocorticoid lesions are **exacerbated** by simultaneous NSAIDs. Remember that steroids are ulcerogenic by themselves — prednisolone at only 0.25 mg/kg *subcutaneously* every 12 hours for three days produced endoscopically visible gastric lesions in dogs, which also tells you the injury is systemic rather than a local effect of a tablet sitting on the mucosa. Adding an NSAID compounds an injury that is already under way. Our corticosteroids guide covers the steroid side and our NSAIDs guide the washout intervals.

With an ACE inhibitor the picture is more nuanced and worth stating fairly. ACE inhibitors act partly through vasodilatory prostaglandins on renal function, so NSAIDs may reduce their effectiveness and blunt the effect on blood pressure. But Plumb's also records a canine study using tepoxalin that showed no adverse effects. Treat it as a real concern in the patient whose renal perfusion is already marginal — dehydrated, hypotensive, anaesthetised, or in established kidney disease — rather than an absolute prohibition in a healthy dog.

> **The triple whammy** *Clinical convention rather than a handbook entry — neither source lists the three-drug combination as such, though the underlying renal pharmacology is in both.* NSAID, ACE inhibitor and diuretic together is the classic renal-injury combination: one removes the prostaglandin-mediated afferent vasodilation that normally opposes systemic vasoconstriction, one removes angiotensin-II-mediated efferent tone, and one reduces circulating volume. Each is reasonable alone. Together, in a patient who then becomes hypotensive under anaesthesia (anesthesia), they remove most of the kidney's ability to protect its own perfusion. Our fluid therapy guide covers why perfusion is the thing being defended.

04 Serotonin stacking

This is the interaction that has quietly become common, because the drug list has grown while the awareness has not. Plumb's lists as serotonergic agents: **clomipramine, metoclopramide, mirtazapine, ondansetron, SSRIs such as fluoxetine and paroxetine, tramadol and trazodone**.

Read that list again with a hospitalised patient in mind. Two of those — **ondansetron and metoclopramide** — are the drugs you reach for when a dog vomits. Two more are standard for anxiety and pain. None of them is thought of as "a serotonin drug".

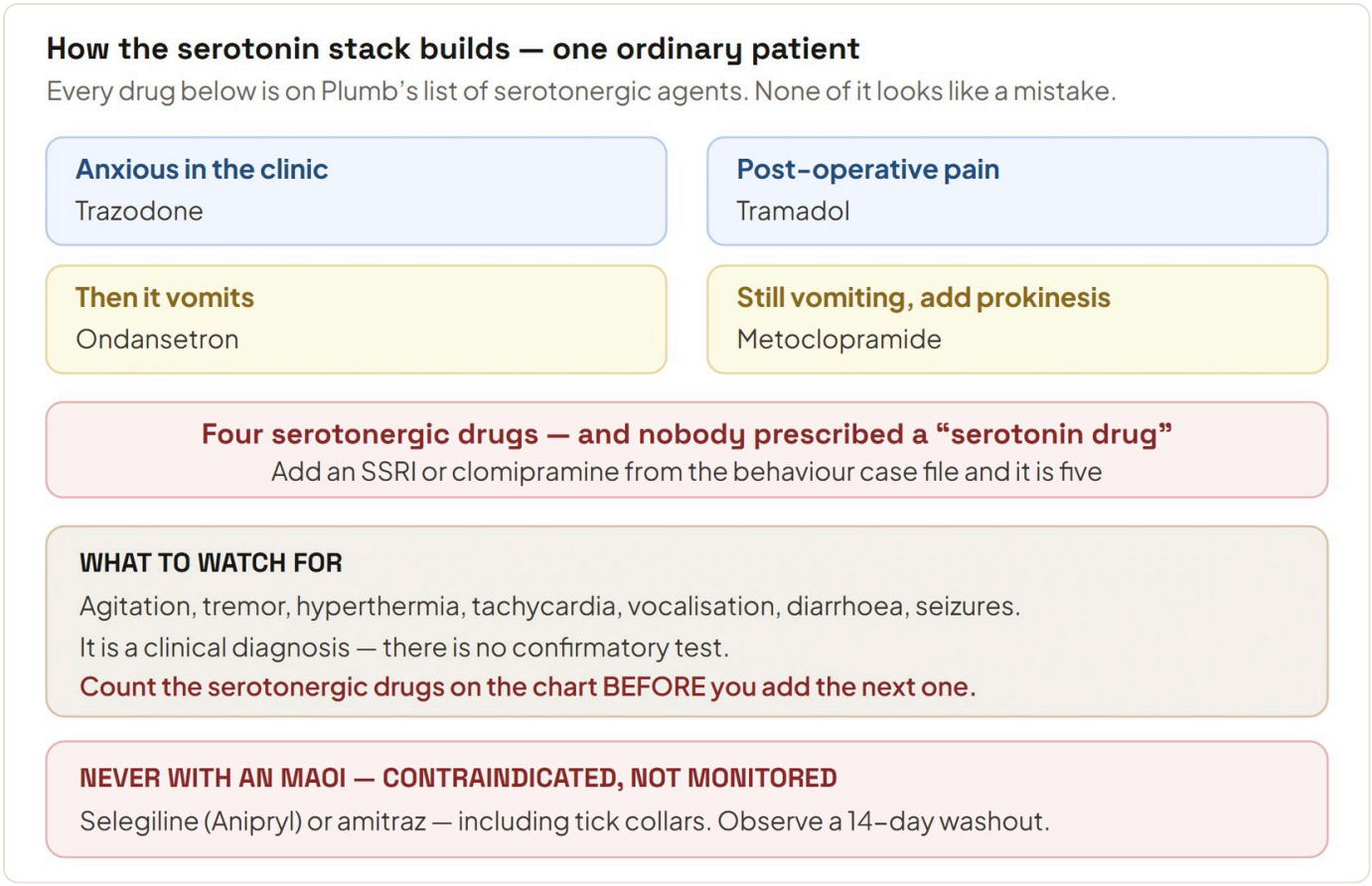

Fig 2 — Four drugs, four sensible decisions, one stack. Every agent shown appears on Plumb's serotonergic list. Serotonin syndrome is a **clinical diagnosis** — there is no confirmatory test — so the defence is counting the drugs on the chart before adding the next one. The red bar is the exception to all of that: with an **MAOI** there is nothing to count, because the combination is contraindicated outright.

They do not all carry equal weight, and it helps to know which end you are at. The drugs that actually drive serotonin excess are the ones that raise synaptic serotonin — the SSRIs, clomipramine, tramadol, trazodone and mirtazapine. Ondansetron and metoclopramide sit at the other end: the pharmacology text describes ondansetron as a 5-HT_3 *antagonist*, and metoclopramide as a 5-HT_4 agonist and weak 5-HT_3 antagonist that blocks 5-HT_3 only at high doses. The distinction shows up in Plumb's own monographs — under trazodone, metoclopramide is flagged for serotonin syndrome while ondansetron is flagged for *QT prolongation* instead. Count every drug on the list, but read a stack of four that includes an SSRI and tramadol very differently from a stack of four that is mostly antiemetics.

The practical habit is simple and takes five seconds: **before adding an antiemetic or an analgesic, count the serotonergic drugs already on the chart**. If the answer is two or more, choose something off the list where you can. Our antiemetics guide sets out which antiemetic suits which cause, and maropitant — an NK_1 antagonist — is not on the serotonergic list.

There is one absolute in this section, and it is the one most easily missed. Everything above is a matter of counting and judgement; **monoamine oxidase inhibitors are not**. Plumb's states that combining an MAOI with a serotonergic drug carries a potential risk for **life-threatening serotonin syndrome** and that concurrent use is **contraindicated**, with a **14-day washout** observed between the two — in either direction. The reason this matters in general practice is that the veterinary MAOIs do not look like psychiatric drugs: **selegiline** (Anipryl) is given for canine cognitive dysfunction and for pituitary-dependent hyperadrenocorticism, and **amitraz** is an acaricide that Plumb's names explicitly *including collars*. A dog can therefore be wearing an interacting drug around its neck while the record shows nothing at all.

Ask before you dispense Before starting tramadol, trazodone, an SSRI or clomipramine, ask two questions: is this dog on selegiline, and is it wearing a tick collar? Both are MAOIs, both are commonly invisible on the clinical record, and both need a 14-day gap rather than monitoring.

05 Enzyme inhibitors and inducers

Most metabolic interactions come down to one question: is the second drug pressing the brake or the accelerator on hepatic metabolism? The two failure modes look nothing alike.

DIRECTION	DRUGS	EFFECT ON THE PARTNER DRUG	CLINICAL CONSEQUENCE
INHIBITORS the brakes	Ketoconazole, itraconazole, fluconazole, cimetidine, erythromycin, diltiazem, chloramphenicol chloramphenicol is a CYP2B11 inhibitor in dogs; Plumb's notes cats may be *particularly* susceptible to its effect on the metabolism of other drugs	Clearance **falls**, half-life **rises**, concentration **climbs**	**Toxicity** from a dose that was previously fine. Onset over days as the partner accumulates
INDUCERS the accelerator	Phenobarbital, rifampin	Clearance **rises**, concentration **falls**	**Silent treatment failure** — the drug simply stops working, and it looks like the disease relapsing

Inhibition is the one people think of. Ketoconazole, itraconazole, fluconazole, cimetidine, erythromycin, diltiazem and chloramphenicol all inhibit cytochrome P450, so a partner drug is cleared more slowly and accumulates. Plumb's gives the pattern precisely for this group with alfentanil: increased half-life, decreased clearance, prolonged effect and a greater risk of respiratory depression. The same logic applies to any narrow-therapeutic-index partner.

Induction is the one that gets missed, because nothing dramatic happens. Phenobarbital induces microsomal enzymes, so co-administered drugs are cleared faster — Plumb's cites increased aspirin metabolism and reduced progestin activity through exactly this route. The patient does not become toxic. The other drug simply stops working, weeks later, and it looks like relapse rather than a pharmacokinetic problem.

> **The tell**An interaction that appears *days to weeks* after a drug was added, in a patient who was previously stable, is far more likely to be metabolic than anything else. Ask what changed on the chart a fortnight ago, not yesterday.

06 P-glycoprotein and MDR1

P-glycoprotein is an efflux transporter that pumps drugs back out of cells — including out of the central nervous system at the blood–brain barrier. Block it, and drugs that are normally excluded get in.

The clinical case that matters is **ivermectin**. Plumb's states that inhibitors block the P-glycoprotein (MDR1) transporter and **increase ivermectin concentrations**. In a dog with the **MDR1 (ABCB1) mutation** — collies and related herding breeds — the transporter is already deficient, so the margin that normally protects the brain is gone before any interacting drug is added. Plumb's also warns that animals with MDR1 mutations may experience neurotoxicity and that certain parasiticides should be used cautiously in patients with a history of adverse reaction to MDR1 substrates such as ivermectin.

> **One question, asked early**"Is there collie in this dog?" changes the entire P-glycoprotein conversation, and it is free to ask. A history of a previous odd reaction to a macrocyclic lactone is the other flag worth taking seriously.

07 The ones you can schedule around

Not every interaction requires a different drug. A whole category is solved by putting time between the doses — and this is where the handbooks give real numbers that summaries tend to omit.

PAIR	WHY	SEPARATE BY
Fluoroquinolone + calcium also aluminium, magnesium, iron, zinc, antacids, dairy	Di- and trivalent cations chelate the fluoroquinolone in the gut. Plumb's: oral calcium-containing products **can reduce absorption**	**At least 2 hours** — Plumb's states this explicitly for calcium carbonate
Sucralfate + doxycycline	Sucralfate binds the partner drug in the gut. Plumb's: it **significantly reduces peak doxycycline concentration and overall exposure in dogs**	**At least 2 hours**, and give the antibiotic first
Sucralfate + other oral drugs	The same binding effect; Plumb's notes reduced GI absorption of oral phenothiazines among others	**At least 2 hours** as a general habit

PAIR	WHY	SEPARATE BY
Oral antacid + budesonide	Plumb's: **the dissolution of the drug's coating is pH dependent**. Note the limit of this one — the same monograph states that famotidine and omeprazole, which also raise gastric pH, **do not significantly affect** budesonide's oral pharmacokinetics	**At least 2 hours** for oral antacids. No separation needed for famotidine or omeprazole

The recurring answer is **at least two hours**, and the recurring cause is a drug being bound or chelated in the gut before it can be absorbed. The failure mode here is not toxicity but **treatment failure**: the antibiotic was given, the owner complied, and it never reached the bloodstream. When a course of doxycycline or a fluoroquinolone appears not to have worked, ask what else was going down the same throat at the same time.

08 The pre-prescribing check

Six questions, none of which takes longer than a few seconds, and between them they catch the large majority of what matters.

ASK	WHY IT CATCHES THINGS
What else is on the chart — including what the owner gives at home, and what the dog is wearing?	Supplements, antacids, calcium, behaviour medication and leftover analgesics are the ones missing from the clinical record — and an **amitraz tick collar** is an MAOI nobody writes down
Am I adding a second drug that pulls the same lever?	Two ulcerogenics, two nephrotoxins, two serotonergics. This is the category where separating doses does not help
Is either drug narrow-therapeutic-index?	Digoxin, phenobarbital, ciclosporin, insulin. Small changes in clearance become clinical events
Is either an enzyme inhibitor or inducer?	The azoles, cimetidine and erythromycin inhibit; phenobarbital induces. Both change the partner drug's exposure
Is this an oral absorption problem I can simply schedule around?	Chelation and binding interactions usually need only a **2-hour gap**, not a different drug
Is it a herding breed?	MDR1 status changes the P-glycoprotein conversation entirely

The single highest-yield item is the first. **Polypharmacy is the underlying risk factor** — every additional drug multiplies the number of possible pairs — and the drugs most often missing from the clinical record are the ones the owner administers at home without thinking of them as medication: joint supplements, antacids, calcium, and analgesics left over from a previous problem.

09 Veterinary drug interactions — frequently asked questions

What is the most dangerous drug interaction in veterinary practice?

It depends on whether you mean the most *frequent* or the most *severe*, and they are different pairs. The one most likely to cause harm in ordinary first-opinion work is an **NSAID given with a corticosteroid**, simply because it comes up so often: both damage gastric mucosa by different mechanisms, and the pharmacology literature describes glucocorticoid lesions as **exacerbated** by simultaneous NSAIDs. Never overlap them — stop one and allow a washout before starting the other. The most *severe* single combination is a **monoamine oxidase inhibitor with any serotonergic drug** — selegiline or an amitraz collar together with tramadol, trazodone or an SSRI — which Plumb's calls a risk of life-threatening serotonin syndrome and lists as contraindicated, with a 14-day washout.

Can a dog take an NSAID and prednisolone together?

No. It is the classic combination to avoid. Steroids alone are ulcerogenic — prednisolone at 0.25 mg/kg SC every 12 hours for three days produced endoscopically visible gastric lesions in dogs — and adding an NSAID compounds it. If a dog on an NSAID needs a steroid, stop the NSAID and observe a washout first.

Which common veterinary drugs are serotonergic?

Plumb's lists **clomipramine, metoclopramide, mirtazapine, ondansetron, SSRIs (fluoxetine, paroxetine), tramadol and trazodone**. Because two of them are routine antiemetics and two are routine for anxiety and pain, three or four can accumulate in a single hospitalised patient without anyone intending it. Count them before adding another — while remembering that the reuptake inhibitors do more of the work than the 5-HT_3 antagonists. Separately, **selegiline and amitraz are monoamine oxidase inhibitors**, and combining either with any of the drugs above is contraindicated rather than merely counted.

How long should you separate a fluoroquinolone from calcium?

At least two hours. Plumb's states this explicitly: oral calcium-containing products reduce fluoroquinolone absorption, and if both calcium carbonate and a fluoroquinolone are needed, the doses should be separated by at least two hours. The same applies to aluminium, magnesium, iron and zinc products, antacids and dairy.

What does ketoconazole interact with?

Ketoconazole is a cytochrome P450 inhibitor, so it raises the concentration of many co-administered drugs by slowing their clearance. The practical rule is to treat any narrow-therapeutic-index partner as needing a **dose reduction and monitoring** rather than to memorise a list. It also inhibits P-glycoprotein, which is a separate route to the same outcome.

Why does phenobarbital make other drugs stop working?

Because it **induces** hepatic microsomal enzymes, so co-administered drugs are metabolised faster and their concentrations fall. Plumb's records this for aspirin metabolism and for reduced progestin activity. It presents as gradual loss of efficacy weeks after starting, which is easily mistaken for the original disease relapsing.

Is serotonin syndrome diagnosed with a blood test?

No — it is a **clinical diagnosis**. Look for agitation, tremor, hyperthermia, tachycardia, vocalisation, diarrhoea and, in severe cases, seizures, in a patient on two or more serotonergic drugs. Because there is no confirmatory test, prevention by counting the drugs is worth more than vigilance after the fact.

Which antiemetic is safest if a dog is already on serotonergic drugs?

Maropitant, which is an NK_1 receptor antagonist and does not appear on the serotonergic list — unlike ondansetron and metoclopramide, which both do. See our antiemetics guide for which drug suits which cause of vomiting.

When to refer

- **NSAID + steroid** — never overlap. Wash out first.
- **Two ulcerogenics or two nephrotoxins** — avoid; scheduling does not help.
- **Serotonergic stack** — count them on the chart before adding another.
- **MAOI + anything serotonergic** — contraindicated. Selegiline and amitraz collars; 14-day washout.
- **NSAID + ACE inhibitor** — avoid in the dehydrated or hypotensive; monitor renal values.
- **ACE inhibitor + spironolactone** — deliberate in cardiac cases; check potassium.
- **Aminoglycoside + furosemide** — choose another antibiotic.
- **CYP inhibitors** (azoles, cimetidine, erythromycin) — expect toxicity; reduce and monitor.
- **Phenobarbital** — expect silent failure; reassess partner doses.
- **P-gp inhibitor + ivermectin** — ask about herding breeds first.
- **Chelation and binding** — separate by at least 2 hours.
- **Ask what the owner gives at home.**

Sources

1. *Plumb's Veterinary Drug Handbook*, 10th ed. — the *Drug Interactions* block of the relevant monographs: the NSAID/glucocorticoid and NSAID/ACE-inhibitor statements, the serotonergic agent list, the monoamine oxidase inhibitor contraindication and 14-day washout (including the naming of amitraz collars under clomipramine), the aminoglycoside/loop diuretic warning, the ACE-inhibitor/potassium-sparing diuretic hyperkalaemia statement and UK-label note, the fluoroquinolone–calcium and sucralfate–doxycycline separation advice, the budesonide antacid statement and its famotidine/omeprazole exemption, the CYP-inhibitor group effect, chloramphenicol as a canine CYP2B11 inhibitor, phenobarbital and rifampin enzyme induction, the P-glycoprotein/MDR1 and ivermectin statements, and the digoxin monitoring advice.
2. Riviere JE, Papich MG (eds). *Veterinary Pharmacology and Therapeutics*, 10th ed. Wiley-Blackwell — Chapter 29 for the statement that glucocorticoid GI lesions are exacerbated by simultaneous NSAIDs (Dow et al. 1990; Narita et al. 2007) and for the low-dose gastric-lesion data (Boston et al. 2003); the gastrointestinal chapter for the receptor pharmacology of metoclopramide (5-HT_4 agonist, weak 5-HT_3 antagonist) and ondansetron (5-HT_3 antagonist); and the description of serotonin syndrome and its clinical signs.
3. Plumb's — Top 10 Drugs Involved in Drug Interactions in Veterinary Medicine — open-access overview organised by drug.
4. University of Illinois — Pharmacist's Corner: Common Drug Interactions — practitioner summary of the ketoconazole, phenobarbital and fluoroquinolone interactions.

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