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NSAIDs IN DOGS & CATS

NSAIDs in Dogs & Cats: Safe Use & Doses (PDF)

Safe use & doses — how NSAIDs work, canine and feline dose tables, why cats are different, what must never be given, and how to recognise and treat NSAID toxicity. Companion to the article at vet-ebooks.com.

Contents

The 16 sections of this guide, in order.

IN SHORT

NSAIDs in dogs and cats are the most-used analgesics in small-animal practice — and the drugs most likely to harm a patient when used carelessly. They work by blocking **cyclo-oxygenase (COX)** — which is also why they injure gut, kidney and liver. This guide covers **how they work, the licensed doses for dogs and cats, why cats are a special case, what must never be given**, and how to recognise and treat NSAID toxicity.

KEY POINTS

1. NSAIDs block **COX**, cutting prostaglandin production. The same prostaglandins protect the **stomach, kidney and platelets** — so efficacy and toxicity share one mechanism.
2. **COX-2 selectivity reduces but does not remove risk.** COX-2 is constitutive in the kidney, so no NSAID is inherently "kidney-sparing" — the gate is **hydration and perfusion**, not the label.
3. **Cats are not small dogs:** deficient glucuronidation means slower clearance, a narrow margin, lower doses and shorter licensed courses.
4. **Never** combine an NSAID with a corticosteroid or another NSAID, and never give **ibuprofen or naproxen**, and **never give paracetamol to a cat** (it is licensed for dogs in some markets, but is lethal to cats).
5. The biggest risk factor is a patient that is **dehydrated, hypotensive or anaesthetised** — prostaglandins are what keep renal perfusion going.
6. Every course needs a **plan**: patient selection, the lowest effective dose, baseline bloods, a recheck, and an owner told exactly when to stop.

Professional reference — check the label

This guide is written for veterinary professionals and students. Doses are collated from *Papich Handbook of Veterinary Drugs* (5th ed.) and cross-checked against *Plumb's Veterinary Drug Handbook* (10th ed.), but **licensing, formulations and approved durations vary by country**. Always confirm against the current product label and your national formulary before prescribing. NSAIDs are prescription medicines and must never be given to an animal except under veterinary direction.

About the images

The three diagrams are reproduced from *Handbook of Veterinary Pain Management, 3rd ed.* for educational reference and are credited beneath each figure; copyright remains with the original authors and publisher.

01 Why NSAIDs deserve respect

Non-steroidal anti-inflammatory drugs are the backbone of analgesia in small-animal practice: they treat osteoarthritis, perioperative pain, soft-tissue injury and fever, they are given by mouth, and they are cheap. They are also, consistently, among the drugs most often implicated in avoidable patient harm. The reason is simple and worth stating plainly — **the mechanism that makes an NSAID work is the mechanism that makes it dangerous**. Understanding that one idea turns **NSAIDs in dogs** and cats from a memorised dose list into a set of decisions you can reason through.

02 How NSAIDs work: the COX cascade

Tissue injury frees **arachidonic acid** from membrane phospholipids via phospholipase A_2. Arachidonic acid is then handled down two arms: **cyclo-oxygenase (COX)** produces the prostaglandins and thromboxane, and **5-lipoxygenase** produces the leukotrienes. The prostaglandins made by COX drive the cardinal signs — vasodilation, oedema, and above all **hyperalgesia**, the sensitisation of nociceptors that makes inflamed tissue hurt. NSAIDs work by blocking COX, so less prostaglandin is made and the pain and inflammation subside. Look at the far-left of the cascade, though, and you can already see the problem: the very same PGE_2 that causes hyperalgesia also delivers **gastric cytoprotection** and renal salt and water handling.

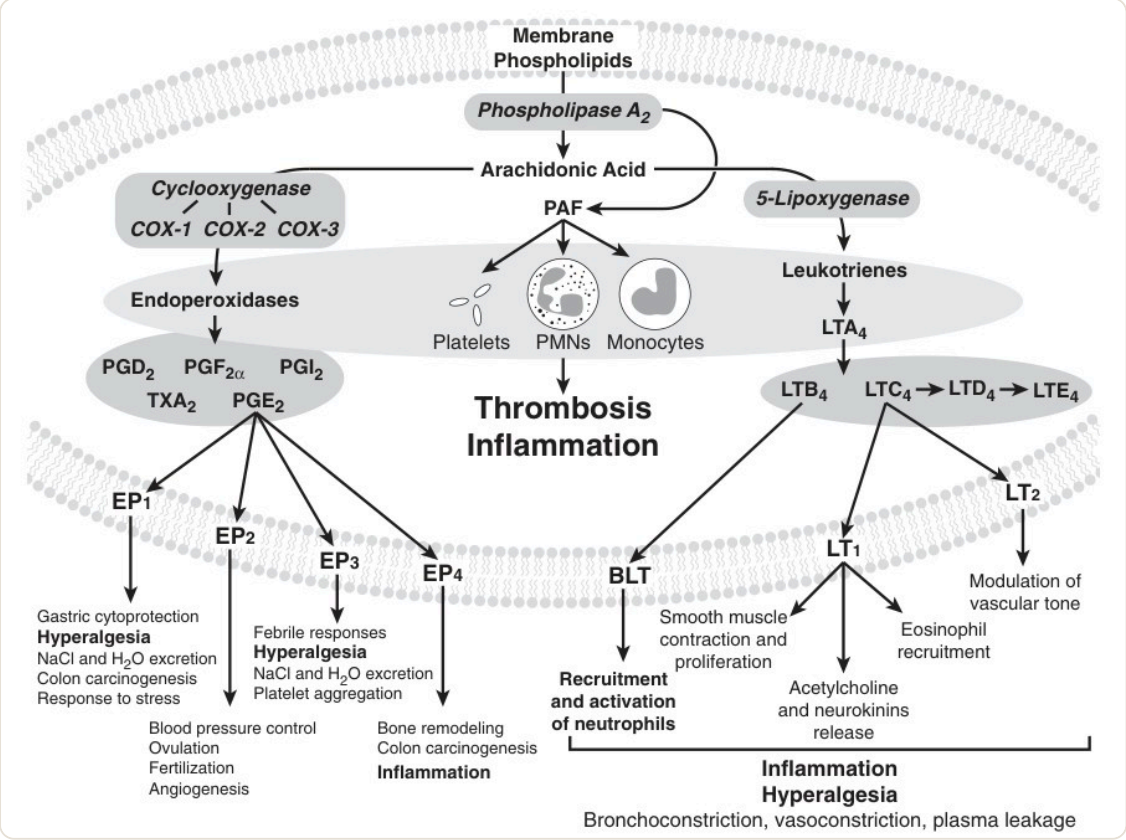

Fig 1 — The arachidonic-acid cascade. Membrane phospholipids → arachidonic acid → COX (prostaglandins, thromboxane) and 5-lipoxygenase (leukotrienes). Note that the PGE_2 branch produces both hyperalgesia *and* gastric cytoprotection — efficacy and toxicity from one pathway.

Image — Budsberg SC. In: Gaynor JS, Muir WW (eds), Handbook of Veterinary Pain Management, 3rd ed. , Figure 8-1. Reproduced for educational reference; copyright remains with the original authors and publisher.

03 COX-1 vs COX-2 — and why "selectivity" is oversold

COX exists in two main isoforms. **COX-1** is largely *constitutive* — switched on all the time, maintaining gastric mucus and bicarbonate, mucosal blood flow, platelet aggregation (via thromboxane) and renal perfusion. **COX-2** is largely *inducible*, upregulated at sites of inflammation by cytokines and endotoxin. That contrast produced the coxibs: block COX-2 for analgesia, spare COX-1 and spare the stomach. It works, but only partly. **COX-2 is also constitutive in the kidney** (macula densa, podocytes) and is expressed in the CNS, and COX-1 contributes to inflammatory pain too. So a COX-2–selective NSAID has a better gastrointestinal profile — it is *not* a drug without risk, and it offers no special protection to the kidney of a hypotensive patient.

04 NSAID doses in dogs (mg/kg and mg/lb)

The following are the commonly cited canine doses. Use the **lowest effective dose**, give with or after food, and never exceed the label. Deracoxib is the one to watch: its post-operative and chronic regimens differ, and confusing them is a recognised cause of toxicity.

DRUG	DOSE (DOG)	ROUTE	NOTES
Carprofen (Rimadyl)	4.4 mg/kg PO q24h (≈2 mg/lb), *or* 2.2 mg/kg PO q12h (≈1 mg/lb)	4.4 mg/kg SC ~2 h pre-op	The practice workhorse; propionic acid class
Meloxicam (Metacam)	0.2 mg/kg day 1 (≈0.09 mg/lb), then **0.1 mg/kg q24h** (≈0.045 mg/lb)	PO, SC or IV	Loading dose then maintenance — don't repeat the 0.2
Firocoxib (Previcox)	5 mg/kg PO q24h (≈2.27 mg/lb)	PO	Coxib; highly COX-2 selective
Deracoxib (Deramaxx)	Post-op 3–4 mg/kg q24h (≤7 days); chronic **1–2 mg/kg q24h** (≈0.45–0.9 mg/lb)	PO	Two different regimens — a classic dosing error
Robenacoxib (Onsior)	Peri-op **2 mg/kg q24h, max 3 days** (label, dogs & cats); chronic OA 1 mg/kg q24h (range 1–2)	PO or SC	Short tissue half-life, long action at inflamed sites
Ketoprofen	2 mg/kg once SC/IM/IV, then 1 mg/kg PO q24h (≤5 days)	SC/IM/IV then PO	Non-selective; short courses only. Some references do *not* recommend it in dogs given bleeding risk
Grapiprant (Galliprant)	2 mg/kg PO q24h (≈0.9 mg/lb)	PO	**Not a COX inhibitor** — an EP4 (piprant) antagonist; no duration limit. Reduce to 1 mg/kg (ABCB1/MDR1 homozygous) or 1.5 mg/kg (heterozygous)

05 NSAID doses in cats — lower, shorter, riskier

Feline dosing is lower, the licensed courses are shorter, and the margin for error is smaller. Note especially that **robenacoxib tablet and injectable doses are not the same**, and that repeat injectable meloxicam is not recommended by the manufacturer in cats.

DRUG	ORAL (CAT)	INJECTABLE / NOTES
Meloxicam	0.05 mg/kg PO q24h; chronic reduce to 0.03 mg/kg q24h or 0.05 mg/kg q48–72h	Single 0.15–0.3 mg/kg SC — manufacturer advises **no second injection**
Robenacoxib	Tablet 1 mg/kg PO q24h	Injection 2 mg/kg SC q24h, max 3 days — **tablet and injection doses differ**
Carprofen	0.5 mg/kg PO q24h (long-term safety *not* established)	4 mg/kg once by injection
Firocoxib	1.5 mg/kg PO once	Long-term safety not determined in cats
Deracoxib	1 mg/kg PO, single dose only	Not for repeat dosing in cats
Ketoprofen	1 mg/kg PO q24h (≤5 days)	2 mg/kg once SC/IM/IV
Grapiprant	Not established	Safe and effective doses not established in cats

06 Why cats are different

Cats are not small dogs, and with NSAIDs the difference is metabolic. Cats have **limited glucuronyl transferase activity**, so drugs cleared by glucuronidation are eliminated slowly; half-lives lengthen, drug accumulates on repeat dosing, and the therapeutic index narrows. Add a species prone to subclinical **chronic kidney disease** — often undiagnosed in the older cat you are about to treat for osteoarthritis — and the case for lower doses, shorter courses and baseline bloodwork becomes obvious. It is also why the licensed feline options are few: in most markets only **meloxicam and robenacoxib** hold meaningful feline licences, and in the United States **no NSAID is approved for long-term use in cats** — only single-dose injectable meloxicam and a maximum three-day course of robenacoxib. For chronic feline use, the **2024 ISFM/AAFP consensus guidelines** are the current reference point.

07 Which NSAID? COX selectivity compared

"Which is the safest NSAID?" is the question every clinician asks, and the honest answer is that **no licensed veterinary NSAID has been shown to be reliably more effective, or reliably safer, than another** in head-to-head clinical use. Selection is driven by the patient, the licence, the formulation and the cost — not by a league table. What *is* useful is knowing where each drug sits on the COX-selectivity spectrum, and that selectivity is both assay-dependent and **species-dependent**: carprofen behaves as a COX-2–selective drug in the dog and cat but is non-selective in the horse, and ketoprofen is COX-1–selective in the cat yet non-selective in the dog.

SELECTIVITY CLASS	EXAMPLES	WHAT IT MEANS
COX-1 preferential/selective	Aspirin, ketoprofen (cat), tepoxalin, flunixin (cow)	COX-1 inhibitory potency at least 5× greater than COX-2
Non-selective	Flunixin, ketoprofen (dog), phenylbutazone, tolfenamic acid, S-carprofen (horse)	No meaningful biological difference between COX-1 and COX-2 inhibition
Slightly / moderately COX-2 selective	S-carprofen (dog, cat), deracoxib, etodolac, meloxicam, mavacoxib	COX-2 potency 5–30× greater than COX-1; some COX-1 inhibition still occurs
Highly COX-2 selective	Firocoxib, robenacoxib, cimicoxib	More than 50× greater potency for COX-2; limited COX-1 inhibition

Grapiprant sits outside this scheme entirely. It is a **piprant — an EP4 prostaglandin-receptor antagonist** (technically classed by some references as a non-COX-inhibiting NSAID) — so it blocks the receptor that mediates PGE_2 pain signalling rather than blocking prostaglandin synthesis. That spares the housekeeping prostaglandins and gives it a different safety profile, but it is still not to be combined with an NSAID or a corticosteroid. In Europe, **mavacoxib** and **enflicoxib** offer extended dosing intervals (enflicoxib is given weekly), which can help compliance in chronic osteoarthritis.

08 The never-give list: ibuprofen, naproxen, paracetamol & aspirin

More NSAID harm comes from the wrong drug than the wrong dose — usually a well-meaning owner reaching into the human medicine cupboard. These are absolute.

NEVER GIVE**Ibuprofen** — a very narrow margin in dogs. Reported thresholds: gastrointestinal signs from ~25–125 mg/kg with **ulceration likely above 50 mg/kg**, **renal failure above ~100–175 mg/kg**, CNS signs (depression, seizures, coma) above ~400 mg/kg. Cats are more sensitive still. Even 16 mg/kg/day chronically produced gastric ulcers in every dog in one study.**Naproxen** — eliminated far more slowly by dogs (half-life ~**35–74 hours** vs ~12–15 hours in humans), so it accumulates; severe, sometimes fatal, GI ulceration.**Paracetamol / acetaminophen** — **never in cats at any dose**: deficient glucuronidation leads to methaemoglobinaemia, Heinz-body anaemia (anemia), facial/paw oedema and death. (Not an NSAID, but the commonest fatal 'painkiller' error.)**Aspirin** — irreversible platelet COX-1 inhibition for the life of the platelet and a long feline half-life; superseded for routine analgesia.**NSAID + corticosteroid** — substantially increases GI ulceration risk for no extra analgesia. Never concurrently; observe a washout in either direction.**NSAID + a second NSAID** — no additional analgesia, markedly more toxicity. Switch only after a washout.

09 Contraindications & patient selection

Choosing the patient matters more than choosing the molecule. Prostaglandins are what preserve renal blood flow when perfusion falls, so any patient whose kidneys are already relying on that compensation is the wrong patient for an NSAID.

DO NOT USE — OR USE ONLY WITH GREAT CARE**Dehydration, hypovolaemia or hypotension** — including the anaesthetised patient without blood-pressure support. The single biggest avoidable risk.**Hepatic disease**, and **uncontrolled or unstable renal disease** — check the older cat before, not after. (Stable, well-hydrated CKD is *not* an absolute bar — see the FAQ.)**Current or recent GI ulceration**, melaena, or protracted vomiting.**Coagulopathy or thrombocytopenia**, and patients with ongoing haemorrhage (hemorrhage).**Concurrent corticosteroids or another NSAID** (see above), and caution with ACE inhibitors, diuretics and other nephrotoxics.**Pregnancy, lactation, and very young animals**; and known hypersensitivity to the drug class.

10 When NSAIDs are contraindicated: the alternatives

If the patient in front of you is on the wrong side of that list, analgesia does not stop — it changes shape. The options with the best evidence in small animals are:

ALTERNATIVES & ADJUNCTS**Anti-NGF monoclonal antibodies** — bedinvetmab (dogs) and frunevetmab (cats), given by monthly injection for osteoarthritis pain. They do not rely on prostaglandin inhibition, which makes them attractive where NSAIDs are contraindicated.**Gabapentin** — useful for neuropathic and chronic pain, and for situational anxiety in cats; sedation is the usual dose-limiting effect.**Amantadine** — an NMDA antagonist used as an add-on for chronic pain that has stopped responding to an NSAID alone.**Paracetamol (acetaminophen)** — licensed for **dogs** in some markets, often combined with codeine. **Absolutely never in cats.Omega-3 fatty acids and disease-modifying agents** (e.g. polysulfated glycosaminoglycans) — modest but genuine adjunctive benefit in osteoarthritis.**Weight management, physiotherapy and environmental modification** — the most under-used and most effective long-term interventions in canine and feline osteoarthritis.

In practice the strongest chronic-pain plans are **multimodal**: a smaller NSAID dose (where it is safe) combined with one or more of the above, rather than a single drug pushed to its ceiling.

11 NSAID side effects: the gastrointestinal tract

The commonest adverse effects are gastrointestinal, because blocking COX-1 removes the prostaglandin-driven defences of the stomach: mucus and bicarbonate secretion fall, mucosal blood flow drops, and epithelial turnover slows. Clinically this runs from inappetence, vomiting and diarrhoea (diarrhea) through to **melaena, ulceration, perforation and peritonitis**. Owners should be told in plain language to stop the drug and phone the practice at the first vomit, black stool, or refused meal — a message that prevents far more harm than any dose calculation.

12 NSAID side effects: the kidney

Under normal perfusion the kidney makes little prostaglandin and NSAIDs are well tolerated. When perfusion falls — dehydration, haemorrhage, anaesthesia (anesthesia), heart failure — renal prostaglandins are recruited to dilate the afferent arteriole and defend glomerular filtration. Give an NSAID at that moment and you remove the compensation, and filtration falls. Note in the figure that **COX-2 is constitutively expressed** at the macula densa and podocytes: this is precisely why COX-2 selectivity does not make an NSAID renally safe.

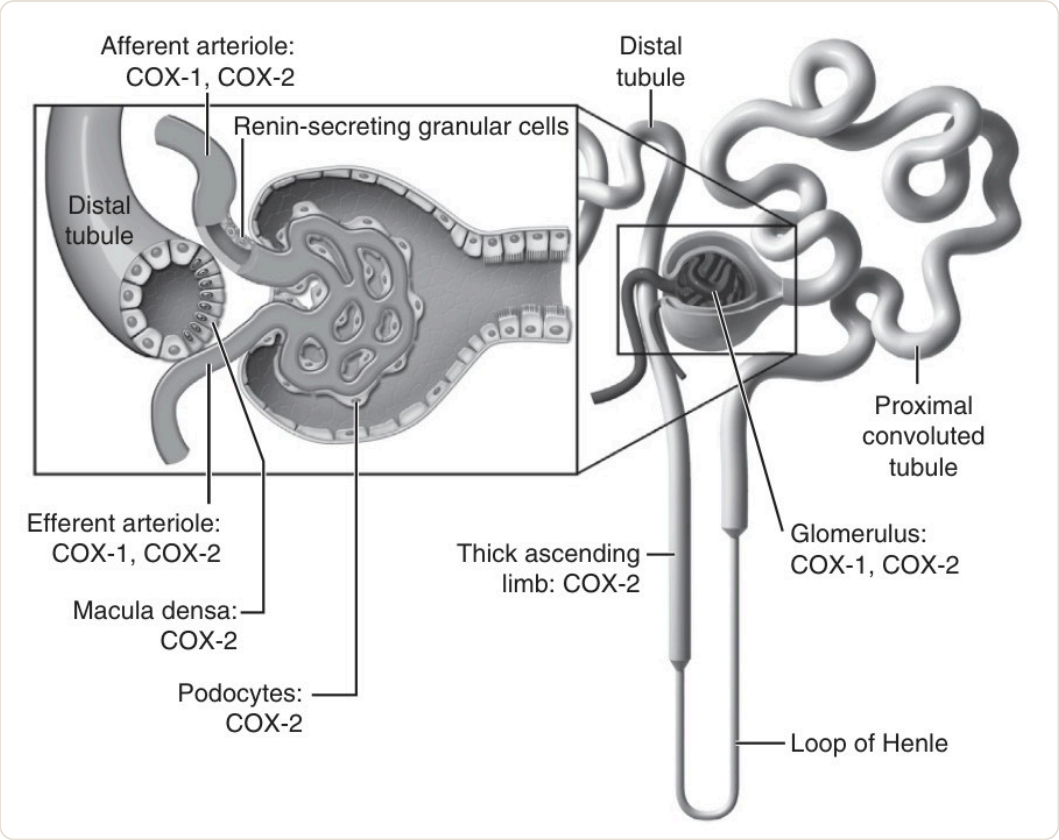

Fig 2 — Where COX lives in the kidney. COX-1 and COX-2 at the afferent and efferent arterioles, COX-2 at the macula densa and podocytes. Because COX-2 is constitutive here, no NSAID — selective or not — protects the kidney of an underperfused patient.

13 Pain, central sensitization & multimodal analgesia

NSAIDs are not purely peripheral drugs. Prostaglandins released within the spinal cord by activated **microglia and astrocytes** amplify transmission at the dorsal horn, contributing to **central sensitization** — the state that produces hyperalgesia (exaggerated pain from a painful stimulus) and allodynia (pain from a normally innocuous one). This is why NSAIDs are effective for inflammatory pain but also why they work best as one component of a **multimodal** plan alongside opioids, local blocks and, where appropriate, adjuncts — and why giving analgesia *before* the surgical stimulus is widely advocated — though for NSAIDs specifically the evidence rests largely on a single canine study.

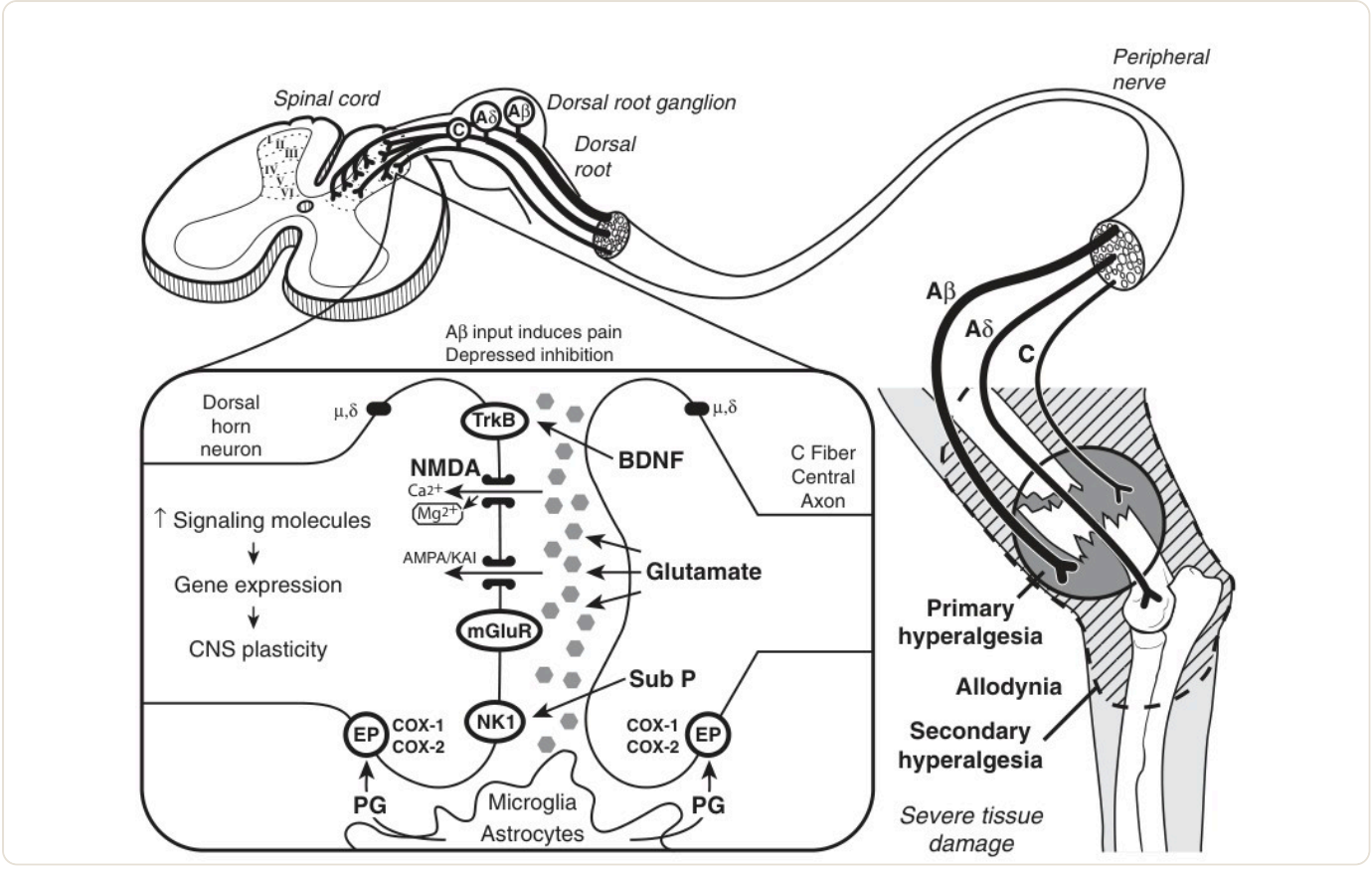

Fig 3 — Prostaglandins in the spinal cord. Prostaglandins from microglia and astrocytes amplify dorsal-horn transmission, driving central sensitization — primary and secondary hyperalgesia and allodynia. NSAIDs act centrally as well as peripherally.

14 Switching NSAIDs: washout periods

If one NSAID has not worked, another sometimes will — individual response genuinely varies. What must not happen is overlap. Convention is a **washout of about 5–7 days** (some sources say 5–10) when changing from one NSAID to another, or between an NSAID and a corticosteroid, in either direction. The more defensible rule is **at least five half-lives of the drug being stopped**, because a flat week is far too long for robenacoxib (canine half-life around an hour) and dangerously too short for long-acting drugs — **mavacoxib needs about a month**. It is worth knowing that Plumb's regards the evidence for a routine NSAID-to-NSAID washout as weak, except after aspirin. After **aspirin**, allow roughly **7–10 days** (3–10 in dogs, ~7–10 in cats), because platelet COX-1 inhibition is irreversible and recovery waits for new platelets. If the patient has had any GI signs, extend the washout and re-examine before restarting.

15 Monitoring & owner communication

Before a long course, take a **baseline**: physical examination, **CBC, a serum chemistry profile including liver enzymes (ALT, ALP) and renal parameters, and urinalysis** — **serial urine specific gravity** is what reveals the early kidney patient a normal creatinine will miss. Recheck at around **2–4 weeks**, then every **3–6 months** for chronic osteoarthritis therapy, and any time the dose changes or the patient becomes unwell. Just as important is the conversation: give the owner the stop signs in writing, tell them never to add a human painkiller, and confirm no one else in the household is also medicating the animal.

16 NSAID toxicity: recognition & management

Suspect NSAID toxicity in any animal with acute vomiting (with or without blood), melaena, abdominal pain, anorexia, polyuria/polydipsia or acute azotaemia (azotemia) — and always ask directly what the owner has given. Management follows the organ at risk.

MANAGING SUSPECTED NSAID OVERDOSE**Decontaminate** if ingestion was recent and the patient is stable and neurologically normal.**Protect the gut** — a proton-pump inhibitor (e.g. omeprazole or pantoprazole), with sucralfate for existing ulceration; misoprostol is the prostaglandin analogue used specifically for NSAID-induced injury.**Protect the kidney** — intravenous fluid therapy to restore and maintain renal perfusion, with serial renal values and urine output.**Watch the liver** — idiosyncratic hepatotoxicity (classically reported with carprofen) is not dose-dependent; check ALT if the patient is unwell.**Stop the NSAID** and do not substitute another. Record the exposure and inform the owner of the washout before any future NSAID.

17 NSAIDs in dogs & cats — frequently asked questions

Can I give my dog ibuprofen?

No. Ibuprofen has a very narrow safety margin in dogs and causes gastric ulceration, perforation and acute kidney injury; cats are even more sensitive. Human NSAIDs should never be given to pets. If a dose has already been given, contact a veterinarian immediately — it is a treatable emergency if caught early.

Can cats have paracetamol (acetaminophen)?

Never. Cats lack the glucuronidation capacity to detoxify it, and even a single tablet can be fatal through methaemoglobinaemia and Heinz-body anaemia. This is the single most important "never" in feline analgesia.

What is the meloxicam dose for a dog?

Commonly 0.2 mg/kg on day one (PO, SC or IV) followed by **0.1 mg/kg once daily**. The loading dose is given once — not repeated daily. In cats the dose is far lower, around 0.05 mg/kg once daily, reduced further for long-term use.

How long can a cat stay on an NSAID?

Longer-term meloxicam is used in cats at reduced doses (for example 0.03 mg/kg daily, or 0.05 mg/kg every 48–72 hours), but licensed durations vary by country and product. Robenacoxib carries a three-day maximum on its label in *both* dogs and cats. Any prolonged course needs baseline and repeat renal monitoring.

Can NSAIDs be given with steroids?

No. Combining an NSAID with a corticosteroid substantially increases the risk of gastrointestinal ulceration and perforation. Use one or the other, with a washout of roughly 5–7 days when switching.

Can you give an NSAID to a cat or dog with kidney disease?

Often, yes — provided the disease is **stable and the patient is well hydrated**. No NSAID is inherently "kidney-sparing", because COX-2 is constitutively expressed at the macula densa and podocytes. But the evidence does not support a blanket ban: low-dose meloxicam has been given long term to cats with pre-existing kidney disease without deterioration in renal values, and robenacoxib produced no change in renal parameters in feline CKD studies. The real gate is **perfusion, not the diagnosis**. The absolute contraindication is the **dehydrated, hypovolaemic, hypotensive or anaesthetised** patient — that is when renal prostaglandins are holding filtration up, and an NSAID removes the compensation.

How long can a dog stay on an NSAID?

For chronic osteoarthritis, treatment is often **indefinite** at the lowest effective dose, with liver and renal values rechecked at 2–4 weeks and then every 3–6 months. Short-course products are different and have hard caps — ketoprofen up to 5 days, post-operative deracoxib up to 7 days, and robenacoxib a maximum of 3 days on label.

> **When to refer**
>
> - **The anaesthetised patient** — give the NSAID once blood pressure is supported and the patient is normovolaemic, not before.
> - **The older arthritic cat** — screen renal function first; use the lowest effective dose and recheck.
> - **The dog that "got into the tablets"** — ibuprofen or naproxen ingestion is an emergency: decontaminate, protect gut and kidney.
> - **The patient switching NSAIDs** — wash out; never overlap, and never add a steroid on top.

Sources

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