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CORTICOSTEROIDS IN DOGS

Corticosteroids in Dogs: Which Steroid, What Dose, How to Taper (PDF)

Corticosteroids in dogs come down to two decisions: which band and which molecule. Inside: Two labelled figures and six reference tables.

Contents

The 11 sections of this guide, in order.

IN SHORT

Corticosteroids in dogs come down to two decisions: *which band* and *which molecule*. The bands for prednisolone are **0.2–0.4 mg/kg/day** physiological, **0.5–1 mg/kg** anti-inflammatory and **2–4 mg/kg** immunosuppressive — with two ceilings almost nobody applies: **never exceed 40 mg per dog** for anti-inflammatory use in large breeds, and for immunosuppression in dogs **over 25 kg use 50–60 mg/m²**, because mg/kg systematically overdoses a big dog. For the molecule, potency and duration travel together: prednisolone is 4× hydrocortisone and short-acting; dexamethasone is 30× and long-acting, with no mineralocorticoid activity and the deepest HPA suppression. Even a physiological dose suppresses the adrenal axis, so every course longer than a fortnight is tapered. Free one-page dose chart included.

KEY POINTS

1. **Choose the band before the number.** Physiological, anti-inflammatory and immunosuppressive are three different jobs, not one dose range.
2. **Anti-inflammatory dosing has a ceiling in milligrams, not mg/kg** — do not exceed **40 mg per dog** in large breeds.
3. **Dogs over 25 kg should be dosed by body surface area** for immunosuppression — 50–60 mg/m². Straight mg/kg overdoses them.
4. **Climbing above 2 mg/kg/day is widely taught to add no further immunosuppression** — only more adverse effects. Add a second agent instead. (A specialist consensus position; the handbooks give the band as 2–4 mg/kg.)
5. **Potency and duration are the same axis.** Dexamethasone's 30× potency comes with +++ HPA suppression and no alternate-day option.
6. **Even a physiological 0.22 mg/kg dose suppresses the HPA axis**, and 0.5 mg/kg q12h does it within two weeks. Recovery takes about two weeks after withdrawal.
7. **Never run a steroid alongside an NSAID.** Glucocorticoid GI lesions are made worse by concurrent NSAIDs.

About the doses

Every dose and potency figure here is transcribed from the two standard references — *Veterinary Pharmacology and Therapeutics*, 10th ed. (Riviere & Papich) and *Plumb's Veterinary Drug Handbook*, 10th ed. Doses are for **dogs**. Confirm against the current label and your own formulary before prescribing.

Steroids are the most used and most misused drug class in small-animal practice. They are cheap, they work within hours, and they will improve almost any inflammatory presentation — which is exactly why they get reached for before a diagnosis and continued after the reason has been forgotten.

Getting **corticosteroids in dogs** right is not complicated, but it does require two decisions in the right order. First, *which dose band* — are you replacing a hormone, damping inflammation, or suppressing an immune response? Those are three different jobs with three different doses. Second, *which molecule* — and that choice is really a choice about duration and adrenal suppression, not about strength.

This guide covers both, with the canine doses in full and the two dose ceilings that are widely ignored.

01 The 30-second answer

For most dogs, most of the time, the answer is **prednisolone** — and the only real question is which band you are in.

THE JOB	REACH FOR	WHY
Routine anti-inflammatory course	**Prednisolone** 0.5–1 mg/kg	Short-acting, so it clears between doses and permits alternate-day therapy. Potency 4× with only mild mineralocorticoid effect
Immune-mediated disease	**Prednisolone** 2 mg/kg (or 50–60 mg/m² if >25 kg)	Same drug, different band. Adding a second agent beats pushing the dose higher
A cat, or a dog with liver disease	**Prednisolone**, never prednisone	Prednisone is a prodrug that the liver must reduce to prednisolone. Give the active drug and remove the variable
Long-term maintenance	**Prednisolone on alternate days**	Only the short-acting group can do this. The anti-inflammatory effect outlasts the HPA suppression just enough to let the axis recover on the off day
Maximum potency or duration	**Dexamethasone**	30× potency, no mineralocorticoid activity, long-acting — but +++ HPA suppression and no alternate-day option
Chronic inflammatory enteropathy	**Budesonide** — with a caveat	Orally administered and **locally active**, for dogs intolerant of systemic steroids — but evidence of fewer adverse effects is **lacking** and HPA suppression is **documented**. Not a free pass
Addisonian crisis	**Not dexamethasone alone**	It has **zero mineralocorticoid activity**. It is useful when you must not disturb an ACTH stimulation test, but it will not replace aldosterone

02 The potency chart

Every glucocorticoid is compared against hydrocortisone (cortisol) on a milligram-for-milligram basis. What the number does *not* tell you is the cost: potency, duration of action and depth of adrenal suppression all rise together.

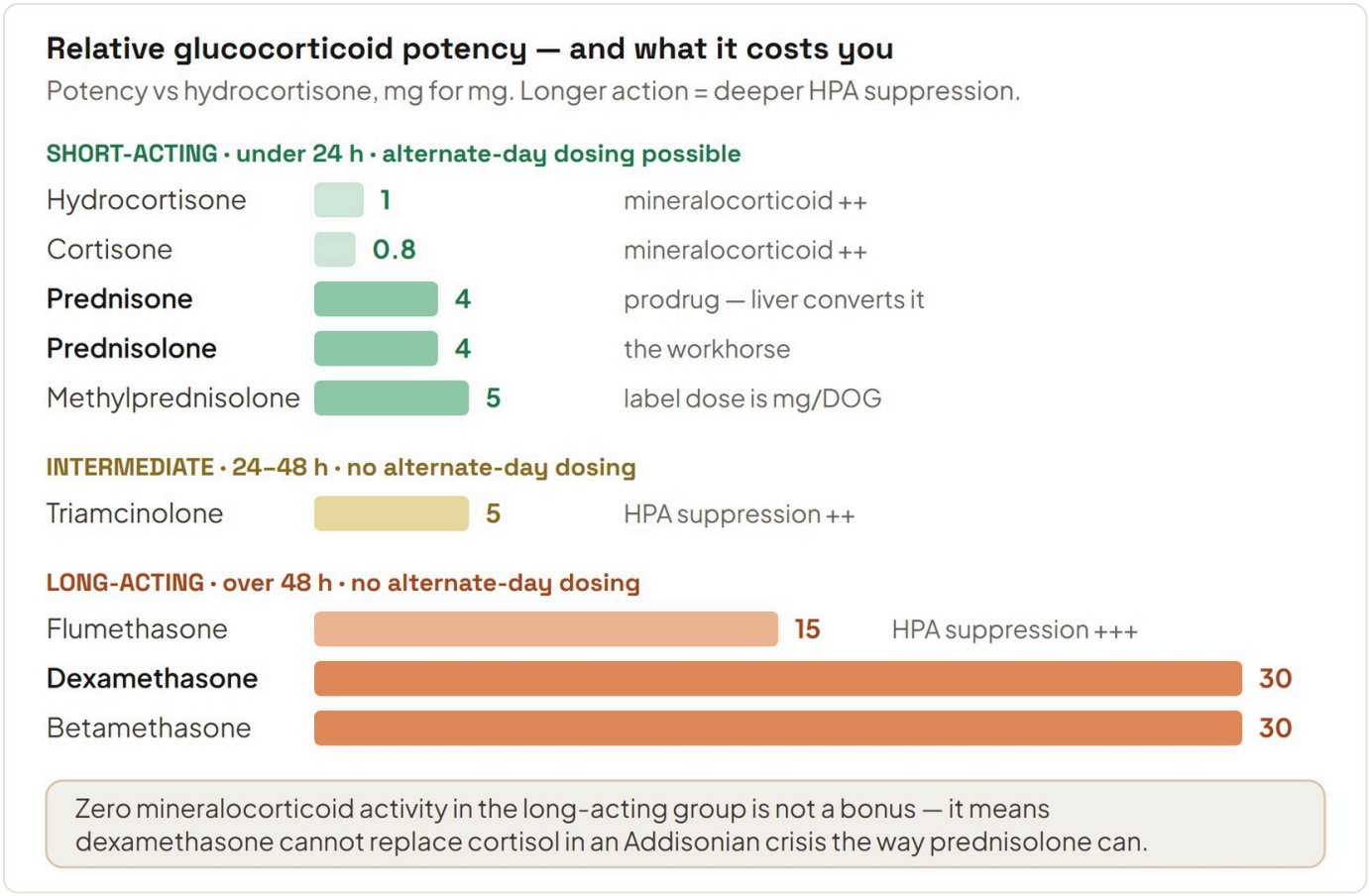

Fig 1 — Relative potency, duration and adrenal cost. Potency is expressed against hydrocortisone, mg for mg. Notice that the long-acting drugs have **zero mineralocorticoid activity** and the deepest HPA suppression, and that only the short-acting group can be given on alternate days.

GLUCOCORTICOID	POTENCY	MINERALOCORTICOID	HPA SUPPRESSION	ALTERNATE-DAY?
Hydrocortisone Short-acting (<24 h)	1×	++	+	No (too short)
Cortisone	0.8×	++	+	Yes (not ideal)
Prednisone	4×	+	+	Yes
Prednisolone	4×	+	+	Yes
Methylprednisolone	5×	+	+	Yes
Triamcinolone Intermediate-acting (24–48 h)	5×	0	++	No
Flumethasone Long-acting (>48 h)	15×	0	+++	No
Dexamethasone	30×	0	+++	No
Betamethasone	30×	0	+++	No

Reading the chart Potency tells you how many milligrams to give; *duration* tells you what it will cost the adrenal axis. A drug that is 30× as potent is not 30× better — it simply means 1 mg of dexamethasone does the work of 30 mg of hydrocortisone, while suppressing the axis for far longer.

One correction worth flagging: the source table prints the long-acting band as "duration of action <48 hours", which is a typographical error — a long-acting drug cannot act for less time than the intermediate band it sits below. It should read **>48 hours**, and that is how it is presented above.

03 The three dose bands

This is the part that matters most, and the part most often collapsed into a single vague "0.5 to 2 mg/kg". They are separate bands for separate purposes.

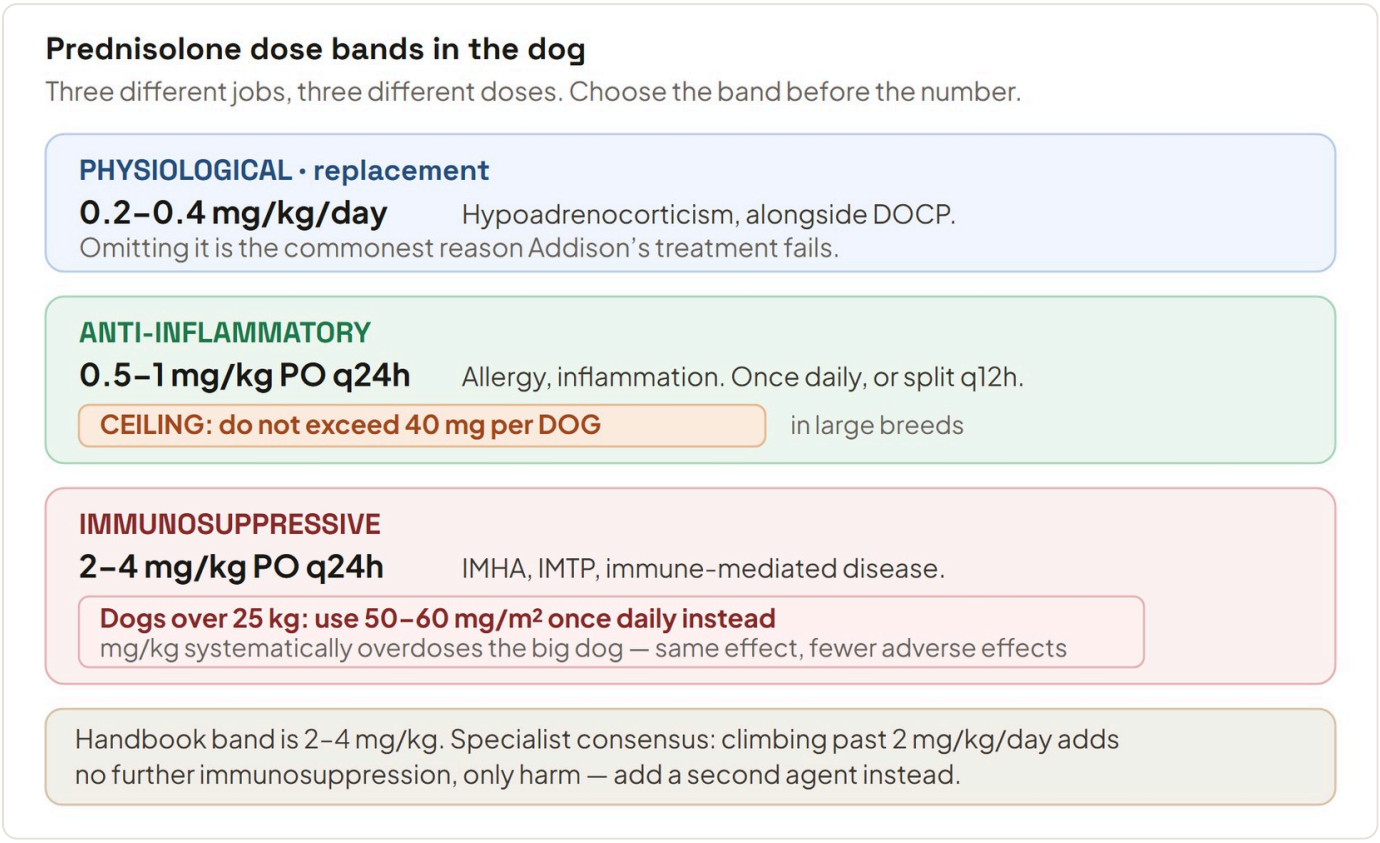

Fig 2 — The three bands, with their ceilings. The two boxed limits are the ones that get missed: a **40 mg-per-dog** cap on anti-inflammatory dosing in large breeds, and **body-surface-area dosing** for immunosuppression in dogs over 25 kg.

BAND	PREDNISOLONE DOSE (DOG)	WHAT IT IS FOR	THE CATCH
Physiological	**0.2–0.4 mg/kg/day**	Replacement in hypoadrenocorticism, given alongside DOCP	Leaving it out is the commonest reason Addison's treatment fails. Even this dose suppresses the HPA axis
Anti-inflammatory	**0.5–1 mg/kg PO q24h**	Allergic and inflammatory disease. Once daily, or the total split q12h	**Do not exceed 40 mg per dog** in large breeds — a ceiling in mg, not mg/kg
Immunosuppressive	**2–4 mg/kg PO q24h**	IMHA, immune thrombocytopenia and other immune-mediated disease	For dogs **over 25 kg use 50–60 mg/m²** instead. The handbook band is 2–4 mg/kg; specialist consensus is that climbing past **2 mg/kg/day** adds no further immunosuppression, only harm
Dexamethasone for comparison	Anti-inflammatory: **0.1 mg/kg IM** Tablets: **0.05–2 mg/kg/day PO** Initial: **0.14–0.28 mg/kg/day**	When you want potency and duration — or a dose that will not interfere with an ACTH stimulation test	**30×** the potency of hydrocortisone and **no mineralocorticoid activity**, so it cannot replace cortisol in a crisis

04 Why big dogs get overdosed

Here is the arithmetic that makes the case. Take a 55 kg dog needing immunosuppression. Dosed at **2 mg/kg**, that is **110 mg** of prednisolone a day. Dosed by surface area at **60 mg/m²** — a 55 kg dog is roughly 1.5 m² — it is about **90 mg**. At 50 mg/m² it is closer to **75 mg**.

The gap widens with size, and it matters because the adverse effects scale with total dose while the immunosuppression does not. Plumb's is explicit that the body-surface method is used in dogs over 25 kg specifically to *provide immunosuppression and minimise adverse effects* — not as an academic refinement.

> **The same logic caps anti-inflammatory dosing**At 1 mg/kg a 55 kg dog would receive 55 mg — well past the **40 mg-per-dog ceiling** Plumb's sets for large breeds. Two dogs of the same weight can need very different milligram totals, and the giant breeds are where mg/kg quietly stops being safe.

05 Which steroid for which job

Once the band is chosen, the molecule follows from three questions: how long do you want it to act, do you need mineralocorticoid activity, and does this patient need testing?

Prednisolone answers most cases. It is short-acting, which sounds like a weakness and is actually the point — it clears between doses, permits alternate-day therapy, and lets the adrenal axis recover. **Dexamethasone** is the tool when you want potency and duration, or when the patient must be treated *and* tested: it does not cross-react in the cortisol assay, so it will not invalidate an ACTH stimulation test.

> **The trap in the long-acting group**Dexamethasone and betamethasone have **zero mineralocorticoid activity**. That is useful when you want a clean glucocorticoid effect — and dangerous if you reach for dexamethasone alone in a suspected **Addisonian crisis**, where the patient needs mineralocorticoid support as well. Potency is not the same as completeness.

06 Prednisone vs prednisolone

They are not interchangeable, and the reason is one metabolic step. **Prednisone is a prodrug**: the liver must reduce its 11-carbon ketone to the ketol before it becomes prednisolone and does anything at all. The same is true of cortisone, which the liver converts to cortisol.

In a dog with a healthy liver the conversion is rapid and the distinction is largely academic — which is why the potency table gives both a value of 4. It stops being academic in **hepatic disease**, and in **cats**, where conversion is unreliable. The safe habit is simple: **prescribe prednisolone** and remove the variable entirely.

> **Where the potency comes from**The jump from hydrocortisone (1×) to prednisolone (4×) is a single structural change — the **1,2 double bond**, which alone gives a fourfold increase in glucocorticoid activity. Adding **16-methylation or fluorination** strips out the mineralocorticoid activity and pushes glucocorticoid potency higher, which is how dexamethasone reaches 30× with a mineralocorticoid score of zero.

07 Budesonide: the "local" steroid, and why it is not a free pass

Budesonide is one of a group sometimes called **"soft" steroids** — molecules designed to act near their site of delivery and then be metabolised to inactive products, the aim being to keep the effect local and the side effects away. In small animals it is used orally, chiefly for **chronic inflammatory enteropathies**, and it is often reached for in the dog that cannot tolerate systemic prednisolone.

> **The caveat that matters**The reasoning is appealing and the evidence is thinner than the reputation. Plumb's states plainly that budesonide "has been proposed for treating patients that are intolerant of systemic steroids" but that **evidence of fewer adverse effects compared with other oral glucocorticoids is lacking, and HPA axis suppression has been documented**. Treat it as a steroid with a useful distribution, *not* as a steroid without systemic consequences — and monitor the patient exactly as you would on prednisolone.

Practical points: it interacts with **CYP3A inhibitors and antacids**, it is embryocidal and teratogenic in laboratory species, and cost and the need to compound down to small dosage strengths are real constraints in general practice.

08 Tapering and the HPA axis

Every clinician knows to taper. Fewer can say *why*, or from what point it becomes necessary — and the published canine data are more sobering than the habit suggests.

DOSE GIVEN	WHAT HAPPENED TO THE ADRENAL AXIS	SOURCE
0.22 mg/kg/day prednisolone PO — a "physiological" dose	Still produced **HPA axis suppression**: a reduced ACTH-stimulated cortisol increment and a reduced zona fasciculata-to-glomerulosa ratio	VPT 10e
0.5 mg/kg q12h prednisolone PO	**Adrenal suppression within 2 weeks** of starting therapy	Chastain & Graham, 1979
The same dose for **1 month**	Profound suppression of ACTH and cortisol — but the axis **returned to normal within 2 weeks** of withdrawal	Moore & Hoenig, 1992
Hydrocortisone 10 mg/kg q12h for 4 months	Ultrasonographically evident **adrenal atrophy**, reversible after 1 month off the drug	Pey et al., 2012

Read that table and two things follow. First, **suppression begins far earlier and at far lower doses than most people assume** — a nominally physiological 0.22 mg/kg daily dose already blunts the axis. Second, and more reassuringly, **recovery is reliable**: after a month of anti-inflammatory dosing the axis returned to normal within about two weeks of withdrawal, and even four months of hydrocortisone produced adrenal atrophy that reversed within a month.

The practical rule: a course of more than about two weeks should be tapered rather than stopped, and the taper exists to let the axis wake up, not to wean the disease. If signs return as you taper, that is the disease telling you the dose was doing work — slow the taper rather than abandoning it.

> **Why alternate-day dosing works at all**It looks like it should not: if a drug suppresses the axis, giving it every other day should suppress it every other day. The reason it works is that for some short-acting preparations the **anti-inflammatory action lasts slightly longer than the HPA-suppressing action**. That narrow gap is the whole basis of alternate-day therapy — and it is why only the short-acting group can be used this way.

09 Adverse effects

Most are dose- and duration-dependent, and most are predictable enough to warn the owner about in advance. Doing so is the difference between an owner who perseveres and an owner who quietly stops the tablets.

SYSTEM	WHAT YOU WILL SEE	NOTES
The obvious three	**Polyuria, polydipsia and polyphagia**, with panting and weight gain	Expected rather than adverse. Warn the owner before the first dose or they will stop the drug
Gastrointestinal	Gastric ulceration	Prednisolone at only **0.25 mg/kg SC q12h for 3 days** produced endoscopically visible gastric lesions in dogs. Made worse by NSAIDs — see below
Endocrine	Iatrogenic hyperadrenocorticism; precipitation of **diabetes mellitus**	Case reports of diabetes in dogs after corticosteroid and methylprednisolone pulse therapy
Thyroid testing	**Lowered serum thyroid hormone concentrations**	Well documented in the dog via TSH suppression. A steroid-treated dog can look hypothyroid on a panel without being hypothyroid — do not diagnose on treatment
Skin	Alopecia, thinning, and **calcinosis cutis**	No specific dose or set of factors has been shown to consistently cause calcinosis cutis
Cardiovascular	Hypertension	Via renin–angiotensin activation and increased pressor response to angiotensin II and noradrenaline
Musculoskeletal	Muscle wasting, weakness	A long-course problem; usually improves once the dose comes down

> **An easy diagnostic trap**Glucocorticoids **lower serum thyroid hormone concentrations** in the dog by suppressing pituitary TSH. A steroid-treated dog can therefore look hypothyroid on a panel while having normal thyroid function — the source is explicit that a true hypothyroid state is not believed to result. **Do not diagnose hypothyroidism on a dog that is currently on steroids.**

10 The NSAID interaction

If you take one safety message from this article, take this one. Glucocorticoids are ulcerogenic in their own right: prednisolone at just **0.25 mg/kg SC every 12 hours for three days** produced endoscopically visible gastric lesions in dogs. That is a low dose over a short course.

Combine that with an NSAID and the effect compounds — **glucocorticoid lesions are exacerbated by simultaneous administration of NSAIDs**. Chronic prednisolone increased gastric mucosal permeability by 50% when given alongside aspirin. The two drug classes injure the same tissue by different routes, and the patient who most tempts you to use both — the painful, inflamed dog — is the one least able to tolerate it.

In practice: **never run the two together**, wash out before switching, and if a dog on an NSAID needs a steroid, stop the NSAID first. Our NSAIDs in dogs and cats guide covers the washout intervals and the toxicity picture in detail. If the patient is already vomiting, our antiemetics guide covers what to do about the sign while you deal with the cause.

11 When NOT to reach for one

Steroids are so effective that the discipline lies in not giving them. These are the situations where reaching for the bottle costs you something.

SITUATION	WHY IT MATTERS
Already on an NSAID	The single most important interaction on this page. Glucocorticoid GI lesions are **exacerbated by concurrent NSAIDs**. Never overlap the two — wash out first
Undiagnosed lymphoma or suspected neoplasia	Standard oncology teaching, and the reason to pause: a few days of steroid can partially treat lymphoma, render the diagnostic cytology uninterpretable and compromise later chemotherapy. **Sample first, treat after**
Before an ACTH stimulation test	Plumb's is explicit that **cortisone cross-reacts with cortisol** in the ACTH response test, and that hydrocortisone and cortisone must not be given on test day — but that **dexamethasone does not cross-react** and does not interfere with cortisol assays. That is why dexamethasone is the steroid of choice when a patient must be treated *and* tested; prednisolone is conventionally withheld too
Active infection	Immunosuppressive doses increase infection risk. Establish the diagnosis before suppressing the response to it
Diabetic patients	Steroids antagonise insulin. If unavoidable, expect the insulin requirement to change and monitor closely
Stopping abruptly after a long course	The suppressed axis cannot respond. Abrupt withdrawal can precipitate an Addisonian crisis

The one that catches people out most often is **lymphoma**. A few days of prednisolone in an undiagnosed case will shrink nodes, partially treat the disease, render cytology uninterpretable and may compromise the response to proper chemotherapy later. Our guide to how cancer develops in animals covers why that matters. **Sample first, treat afterwards** — the delay costs the patient far less than the lost diagnosis.

12 Corticosteroids in dogs — frequently asked questions

What is the steroid dose for a dog?

It depends entirely on the job. For **prednisolone**: **0.2–0.4 mg/kg/day** for physiological replacement, **0.5–1 mg/kg once daily** for anti-inflammatory effect, and **2–4 mg/kg once daily** for immunosuppression. Two ceilings apply: do not exceed **40 mg per dog** for anti-inflammatory use in large breeds, and use **50–60 mg/m²** rather than mg/kg for immunosuppression in dogs over 25 kg.

What is the difference between an anti-inflammatory and an immunosuppressive dose?

Roughly a fourfold difference in milligrams and a completely different intent. Anti-inflammatory dosing (0.5–1 mg/kg) damps inflammation; immunosuppressive dosing (2–4 mg/kg) suppresses the immune response itself, for diseases such as IMHA and immune thrombocytopenia. The handbooks give the band as **2–4 mg/kg**; specialist consensus holds that climbing past **2 mg/kg/day adds no further immunosuppression**, only more adverse effects.

Prednisone or prednisolone for dogs?

Prednisolone. Prednisone is a prodrug that the liver must convert to prednisolone before it works. In a dog with normal liver function the two are near-equivalent, which is why both are rated 4× potency — but in hepatic disease, and in cats, conversion is unreliable. Prescribing prednisolone removes the variable at no cost.

How much stronger is dexamethasone than prednisolone?

About **seven to eight times**. On the standard scale, prednisolone is **4×** hydrocortisone and dexamethasone is **30×**. Dexamethasone is also long-acting with zero mineralocorticoid activity and the deepest HPA suppression, so the extra potency is not free.

How long can a dog stay on steroids?

There is no fixed limit, but adrenal suppression is measurable within about **two weeks** at anti-inflammatory doses, so any course beyond that should be tapered rather than stopped. For genuine long-term use, move to the lowest effective dose on **alternate days** using a short-acting drug — the only group that permits it.

Why can't you stop steroids suddenly?

Because the adrenal axis has been suppressed and cannot immediately resume producing cortisol. Abrupt withdrawal after a prolonged course risks an **Addisonian crisis**. The reassuring part is that recovery is dependable: after a month of anti-inflammatory dosing, the axis returned to normal within about two weeks of stopping.

Can a dog take steroids and an NSAID together?

No. Both injure the gastric mucosa, and glucocorticoid lesions are specifically **worsened by concurrent NSAIDs**. Prednisolone alone at 0.25 mg/kg twice daily for three days already produced visible gastric lesions in dogs. Stop the NSAID and allow a washout before starting a steroid.

Do steroids affect thyroid or ACTH testing?

Yes, both. Glucocorticoids **lower serum thyroid hormone concentrations** in dogs, so a steroid-treated dog can look falsely hypothyroid. For adrenal testing, Plumb's states that **cortisone cross-reacts with cortisol** in the ACTH response test and that hydrocortisone and cortisone must not be given on test day, while **dexamethasone does not cross-react and does not interfere with cortisol assays**. That is why dexamethasone is the steroid of choice when a patient must be treated and tested; prednisolone is conventionally withheld beforehand as well.

Can steroids give a dog Cushing's disease?

They can produce something that looks exactly like it. Naturally occurring **Cushing's disease** (hyperadrenocorticism) is the adrenal glands making too much cortisol; **iatrogenic Cushing's** is the same clinical picture created by the steroid you prescribed. The signs are the ones you would predict from the drug — **polyuria and polydipsia**, panting, a pot-bellied appearance, muscle wasting, thin skin and coat change, sometimes **calcinosis cutis**. The important difference is what the adrenal glands are doing underneath: in the iatrogenic form they are *suppressed*, not overactive, which is exactly why the drug can never simply be stopped. It also means an ACTH stimulation test in a dog on steroids will look flat, and reads as hypoadrenocorticism if you forget what the patient is taking.

When should steroids be avoided altogether?

Whenever suppressing the immune response is the opposite of what the patient needs, or where the drug will make an existing problem worse. That includes undiagnosed infection, corneal ulceration, **demodicosis** and other conditions where immunosuppression lets the organism proliferate; poorly controlled diabetes mellitus; and any patient already on an **NSAID**, where the combined risk of gastrointestinal ulceration is the reason the two are never overlapped. Give the diagnosis before the steroid wherever you can — a course of prednisolone into an undiagnosed lymphoma makes the disease harder to confirm and the later chemotherapy less effective.

What are the most common side effects of steroids in dogs?

Polyuria, polydipsia and polyphagia, with panting and weight gain — expected consequences rather than true adverse effects, but the ones owners notice. Longer courses risk **iatrogenic hyperadrenocorticism** — steroid-induced **Cushing's syndrome** — gastric ulceration, muscle wasting, alopecia, calcinosis cutis, hypertension and precipitation of diabetes mellitus.

When to refer

- **Physiological** — prednisolone 0.2–0.4 mg/kg/day.
- **Anti-inflammatory** — 0.5–1 mg/kg q24h; **max 40 mg/dog** in large breeds.
- **Immunosuppressive** — 2–4 mg/kg q24h; **50–60 mg/m²** if over 25 kg.
- **Consensus: no extra benefit above 2 mg/kg/day** — add an agent, do not climb.
- **Potency** — hydrocortisone 1, prednisolone 4, dexamethasone 30.
- **Long-acting = no mineralocorticoid, +++ HPA suppression, no alternate-day.**
- **Prednisolone, not prednisone** — especially in liver disease and cats.
- **Suppression by 2 weeks**; recovery about 2 weeks after stopping.
- **Never with an NSAID.** Wash out first.
- **Sample before you treat** if lymphoma is possible.

Sources

1. Riviere JE, Papich MG (eds). *Veterinary Pharmacology and Therapeutics*, 10th ed. Wiley-Blackwell — Chapter 29. Source of the potency/duration/HPA table (Table 29.3), the structure–activity relationships, the HPA-suppression studies (Chastain & Graham 1979; Moore & Hoenig 1992; Crager et al. 1994; Pey et al. 2012), the gastric-lesion data (Boston et al. 2003; Chung et al. 1978) the NSAID interaction (Dow et al. 1990; Narita et al. 2007) and the "soft steroid" description of budesonide.
2. *Plumb's Veterinary Drug Handbook*, 10th ed. — source of the canine dose bands, the 40 mg-per-dog anti-inflammatory ceiling in large breeds, the 50–60 mg/m² rule for dogs over 25 kg, the dexamethasone doses, the methylprednisolone label (dosed in mg per dog, not mg/kg), the budesonide prescriber highlights, and the ACTH-response-test cross-reactivity notes.
3. Merck Veterinary Manual — Corticosteroids in Animals — open-access overview of mechanism, relative potencies and adverse effects.
4. Clinician's Brief — Prescribing & Dosing Glucocorticoids — practitioner-facing summary of glucocorticoid selection and dosing.

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