Vet eBooks
VET-EBOOKS.COM

FREE · NO SIGN-UP

PHYSIOLOGY · STUDY GUIDE

Cancer in Animals

How Normal Cells Turn Cancerous, Grow and Spread

Cancer is cellular homeostasis lost — a single body cell escapes the controls on when it grows, specialises and dies.

PAGES	SECTIONS	FIGURES	TABLES	SELF-TEST
11	11	5	1	16 questions

INSIDE THIS GUIDE

01 What is cancer? Homeostasis, lost

02 The genetic engine: oncogenes, suppressors & the cell cycle

03 Apoptosis: why cancer cells refuse to die

04 What causes cancer in animals, and why it takes time

05 The 10 hallmarks of cancer

06 Metastasis — how cancer spreads

07 Cancer in dogs & cats

08 Paraneoplastic syndromes — when the signs are far from the tumour

09 Clinical — diagnosis, grading, staging & targeting the hallmarks

10 Quiz: test yourself

11 Cancer in animals — frequently asked questions

EDITION
November 2026

READ ONLINE
vet-ebooks.com/articles/cancer-in-animals

Contents

The 11 sections of this guide, in order.

01 What is cancer? Homeostasis, lost

02 The genetic engine: oncogenes, suppressors & the cell cycle

03 Apoptosis: why cancer cells refuse to die

04 What causes cancer in animals, and why it takes time

05 The 10 hallmarks of cancer

06 Metastasis — how cancer spreads

07 Cancer in dogs & cats

08 Paraneoplastic syndromes — when the signs are far from the tumour

09 Clinical — diagnosis, grading, staging & targeting the hallmarks

10 Quiz: test yourself

11 Cancer in animals — frequently asked questions

IN SHORT

Cancer is **cellular homeostasis lost** — a single body cell escapes the controls on when it grows, specialises and dies. In 2000, Hanahan and Weinberg distilled every cancer into a set of **hallmarks**: capabilities a cell must acquire to become malignant, from driving its own growth to evading death, building a blood supply and spreading. The same hallmarks underlie the lymphomas, mast-cell tumours, osteosarcomas and mammary cancers vets see every week — and they are exactly what modern oncology aims to switch off.

KEY POINTS

1. Cancer is the **breakdown of cellular homeostasis**: a single cell escapes the rules that govern when it divides, specialises and dies.
2. It is a **genetic disease of body (somatic) cells**, driven by two opposing gene classes — **oncogenes** (accelerator) and **tumour-suppressor genes** (brakes).
3. Cancer is **multistep** — several mutations must accumulate, which is why risk rises steeply with age.
4. The **10 hallmarks** (Hanahan & Weinberg) are the capabilities a normal cell must acquire to become malignant.
5. **Metastasis** — invasion and spread to distant sites — is what makes cancer lethal, and follows a defined cascade.
6. Animals acquire the same hallmarks: **dogs skew to sarcomas** (osteosarcoma, haemangiosarcoma) plus lymphoma, mast-cell and mammary tumours.
7. Every hallmark is a **drug target** — which is why this biology underpins modern veterinary oncology.

01 What is cancer? Homeostasis, lost

Every healthy cell lives under tight rules: it divides only when told to, stops when it should, specialises for a job, and — when it is damaged or no longer needed — quietly kills itself. That balance is cellular homeostasis. **Cancer is what happens when a single cell breaks those rules** and grows on its own terms, deaf to the signals that normally govern it.

The growth itself is a **neoplasm** ("new growth"), or tumour — spelled *tumor* in American usage. **Neoplasia** is the umbrella term for the process rather than the lump, and it is the word you will meet on most pathology reports. Pathologists read two features to judge it: how well the cells still resemble their tissue of origin (**differentiation**) and how they behave. A **benign** tumour is well-differentiated, slow, encapsulated and stays put — a lipoma is the classic example. A **malignant** tumour is poorly differentiated (*anaplastic*), grows fast, and — the decisive trait — it **invades** the tissue around it and can **metastasise** to distant sites. That single capability, to invade and spread, is the line between a lump you excise and a disease that kills.

Cancer is also fundamentally a disease of **one lineage**: it begins in a single somatic cell whose descendants inherit its faults and add more. Everything that follows — the genetics, the hallmarks, the spread — is the story of how one cell's homeostatic brakes come off, one mutation at a time.

For vets Not every lump is cancer — abscesses, cysts, granulomas, haematomas (hematomas) and reactive lymph nodes all mimic tumours. That is precisely why a mass is *sampled* (aspirate or biopsy), never diagnosed by eye or left to "watch and wait".

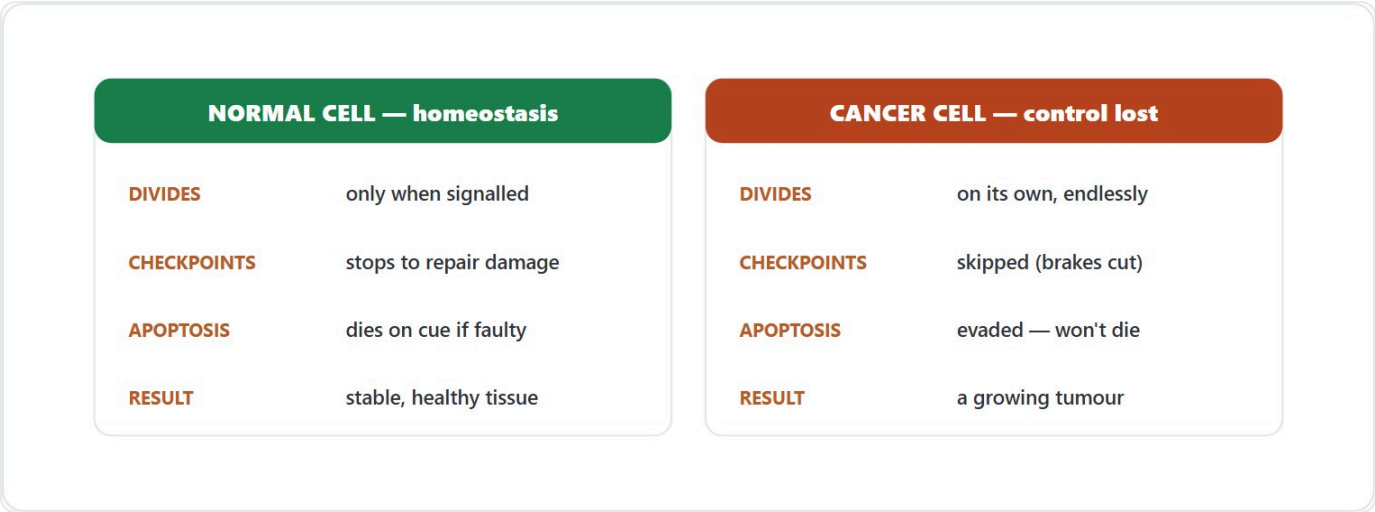


Fig 1 — A normal cell obeys the controls of homeostasis; a cancer cell has lost every one of them.

Diagram — Vet-eBooks.com, built for this guide. The contrast it draws between a controlled cell and a cancer cell follows the hallmarks framework of Hanahan and Weinberg.

02 The genetic engine: oncogenes, suppressors & the cell cycle

Cancer is, at root, a **genetic disease of somatic cells** — caused by mutations in the genes that run the cell cycle. Those genes fall into two opposing classes, and the car analogy is exact.

Oncogenes are a stuck accelerator. Normal growth-promoting genes — *proto-oncogenes* — carry the "grow" signal from the cell surface to the nucleus. The canonical route: a growth factor binds a **receptor tyrosine kinase**, which switches on **Ras** (a small G-protein flipping from GDP to GTP), which fires the **MAP-kinase cascade** (Mek → Erk) and drives transcription factors such as **Myc** to make cyclins. A gain-of-function mutation jams any step "on", so the cell hears a constant grow command. Both *ras* and *myc* are mutated in a high proportion of tumours, and because the accelerator only has to stick once, **a single mutated copy is enough**.

Tumour-suppressor genes are the brakes. Deep inside the cell, **cyclin-dependent kinases (CDKs)** are the engines that drive the cycle from G1 → S → G2 → M, and **checkpoints** (the first sits just before S phase) stop a cell that is damaged or unready. The two great gatekeepers are **Rb** (retinoblastoma protein), which holds the cell in G1 by binding the E2F transcription factor, and **p53**, the "guardian of the genome", which halts the cycle to allow repair or, if the damage is too great, orders apoptosis. Cancer needs these brakes cut — and because there are two gene copies, **both must be lost** (Knudson's "two-hit" rule). That is why *p53* is the single most commonly mutated gene in cancer.

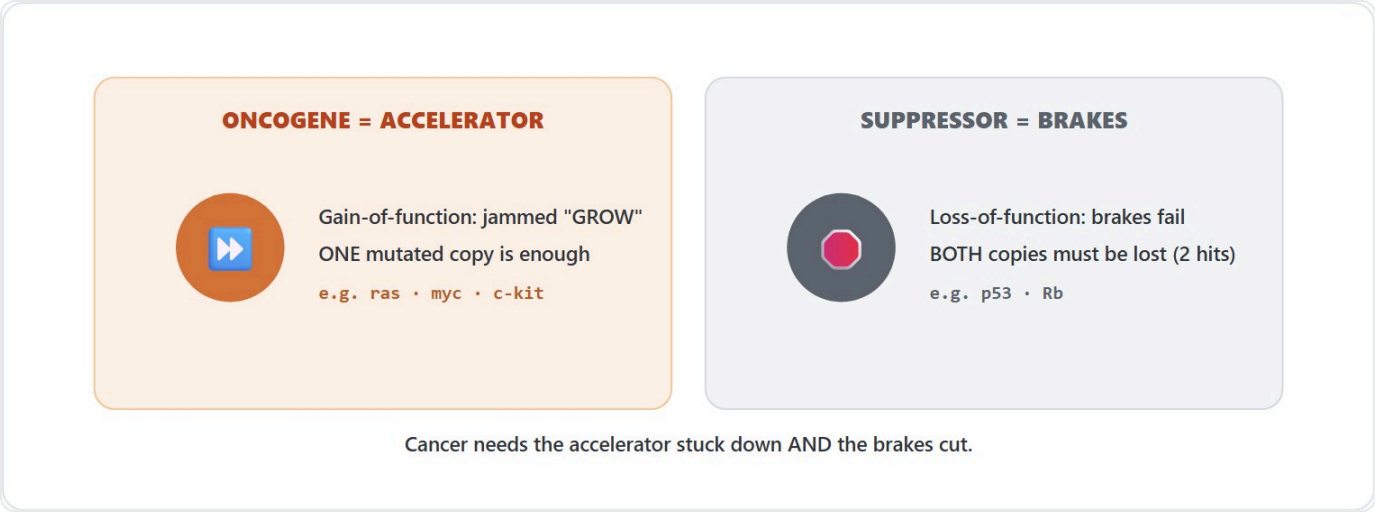


Fig 2 — Two gene classes, opposite logic: oncogenes need one activating hit; tumour suppressors need both copies lost.

Diagram — Vet-eBooks.com, built for this guide. The accelerator-and-brake contrast between oncogenes and tumour-suppressor genes is the standard teaching model set out in the sources below.

For students Growth factor → RTK → **Ras** → Mek → Erk → **Myc** → cyclins → CDKs → division. Learn that one line and you understand where most oncogenes act — and where the drugs aim.

03 Apoptosis: why cancer cells refuse to die

A normal cell that is damaged, infected or surplus commits **apoptosis** — programmed cell suicide, a core homeostatic tool. The executioners are **caspases**, proteases that cleave hundreds of target proteins and dismantle the cell cleanly, without the inflammation of necrosis. Two switches turn them on: the **extrinsic pathway**, triggered when a "death-receptor" on the surface is engaged (e.g. by immune cells), and the more complex **intrinsic (mitochondrial) pathway**, governed by the **Bcl-2 family** — a tug-of-war between pro-death and pro-survival members that decides whether the mitochondria release the signals that activate caspases.

Resisting apoptosis is one of cancer's defining hallmarks, and it is reached in predictable ways: over-expressing pro-survival **Bcl-2** (classic in lymphoma), or losing **p53**, which normally links DNA damage to the death programme. A cell that cannot die accumulates the very mutations it should have been eliminated for — so evading apoptosis both keeps the tumour alive and speeds its evolution. It is also why many chemotherapy drugs work by *restoring* the apoptotic trigger: they damage DNA or microtubules until even a resistant cell tips over the edge.

04 What causes cancer in animals, and why it takes time

A normal cell does not turn malignant in a single jump. It must accumulate **several mutations** — usually in both oncogenes and tumour suppressors — each giving its descendants a small growth advantage that natural selection then amplifies within the tissue. This stepwise, Darwinian process (the modern form of Knudson's hypothesis) is why **cancer risk climbs steeply with age**, why DNA-damaging carcinogens (UV light, tobacco smoke, some viruses) raise risk, and why **genome instability** — a sloppy DNA-repair system — accelerates everything by raising the mutation rate. Roughly **one in four dogs** develops cancer in its lifetime, and it accounts for **nearly half of deaths in dogs over ten** — the arithmetic of accumulated mutation over a lifespan.

What actually triggers those mutations? Several forces, and many are distinctly veterinary. **Inherited / breed predisposition** loads the dice — golden retrievers and Bernese mountain dogs for haemangiosarcoma and histiocytic sarcoma, boxers and pugs for mast-cell tumours, and large and giant breeds for osteosarcoma. **Physical and chemical carcinogens** damage DNA directly: ultraviolet light drives squamous cell carcinoma on the thinly-haired, non-pigmented skin of white cats (ear tips, nose) and dogs. Uniquely in animals, several **oncogenic viruses** cause cancer outright — **feline leukaemia virus (FeLV)** → feline lymphoma, **papillomaviruses** → warts and some squamous cell carcinomas, **bovine leukaemia virus** → enzootic bovine leucosis, and Marek's disease herpesvirus → lymphoma in poultry. **Chronic inflammation** and **hormones** (the mammary-tumour link) complete the picture. The practical pay-off: because some causes are known, some cancers are *preventable* — FeLV vaccination, early spaying, and keeping white-coated pets out of strong sun all measurably lower risk.

05 The 10 hallmarks of cancer

In 2000, Douglas Hanahan and Robert Weinberg distilled the bewildering variety of cancers into a short list of shared **hallmarks** — the capabilities a normal cell *must* acquire to become malignant. The original six were joined in the 2011 update by two **enabling characteristics** (genome instability and tumour-promoting inflammation) and two **emerging hallmarks** (reprogrammed energetics and immune evasion). It is the single most useful framework in oncology, human and veterinary alike, because it turns "cancer" from a mystery into a checklist of ten breakable systems.

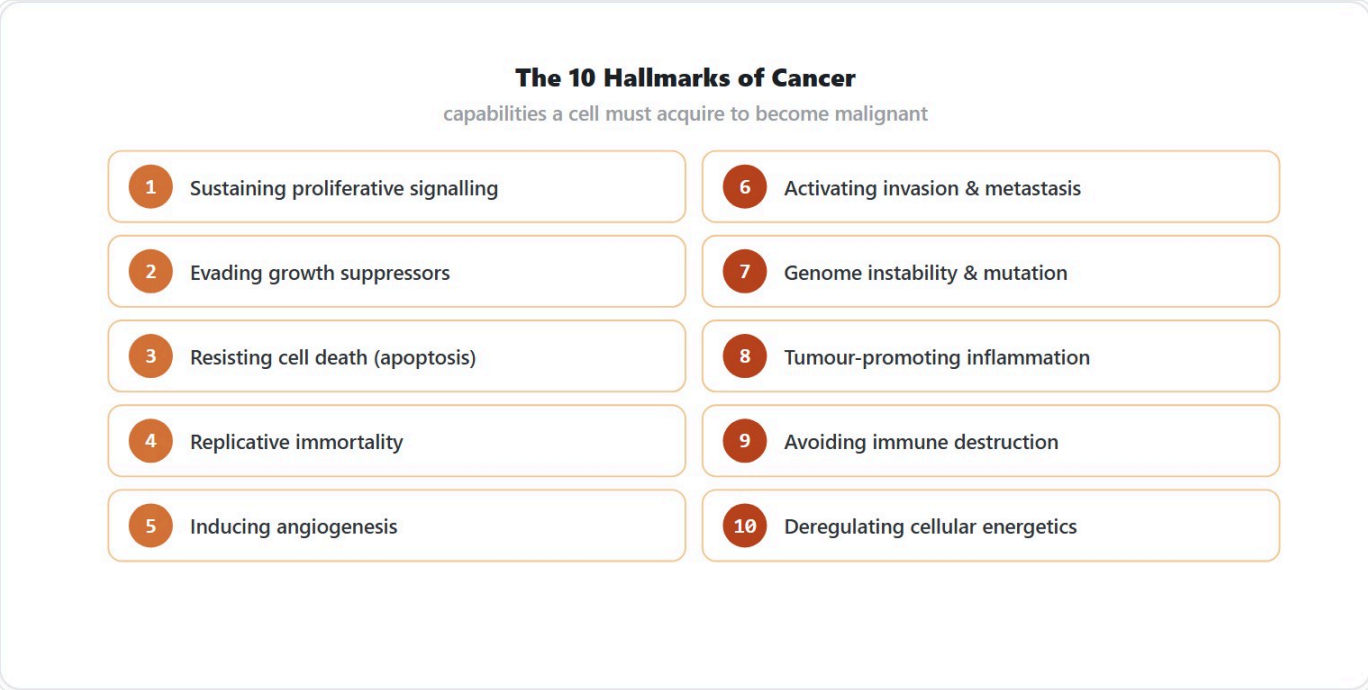


Fig 3 — The ten hallmarks: six core capabilities plus four enabling/emerging ones.

Diagram — Vet-eBooks.com, built for this guide. The six core capabilities plus the four enabling and emerging ones are those of Hanahan and Weinberg's hallmarks of cancer.

HALLMARK	WHAT THE CELL GAINS	ANIMAL EXAMPLE
Sustaining proliferative signalling	Makes its own "grow" signals	*c-kit* mutation in canine mast-cell tumours
Evading growth suppressors	Cuts the brakes (p53, Rb)	p53 loss across many tumours
Resisting cell death	Dodges apoptosis	Bcl-2 over-expression in lymphoma
Replicative immortality	Re-activates telomerase	Most malignant tumours
Inducing angiogenesis	Builds its own blood supply (VEGF)	Many solid tumours
Invasion & metastasis	Spreads to distant organs	Osteosarcoma → lungs
Genome instability	Mutates faster (enabling)	Defective DNA repair
Tumour-promoting inflammation	Hijacks immune cells to help it (enabling)	Feline injection-site sarcoma
Avoiding immune destruction	Hides from T-cells (emerging)	Target of new immunotherapies
Deregulating energetics	Aerobic glycolysis, the Warburg effect (emerging)	Most cancers

06 Metastasis — how cancer spreads

Metastasis is the hallmark that kills, and it is not random — cells follow a defined **cascade**. First the cells loosen their grip on their neighbours (an **epithelial-to-mesenchymal transition**) and secrete enzymes (matrix metalloproteinases) that dissolve the **basement membrane**, letting them **invade** local tissue. They then break into a blood or lymphatic vessel (**intravasation**), survive the shear and immune assault of the **circulation**, lodge and squeeze out at a distant site (**extravasation**), and finally **colonise** that organ to grow a secondary tumour. Where they land is not pure chance — the "seed and soil" principle means certain tumours favour certain organs.

Each step is inefficient — the vast majority of cells die in transit — but a tumour sheds millions, so a few survivors are enough. In dogs, **osteosarcoma** seeding the lungs is the textbook case: the affected leg can be amputated, yet micrometastases already in the lungs at diagnosis decide survival. That is why chemotherapy follows amputation even when the primary is gone.

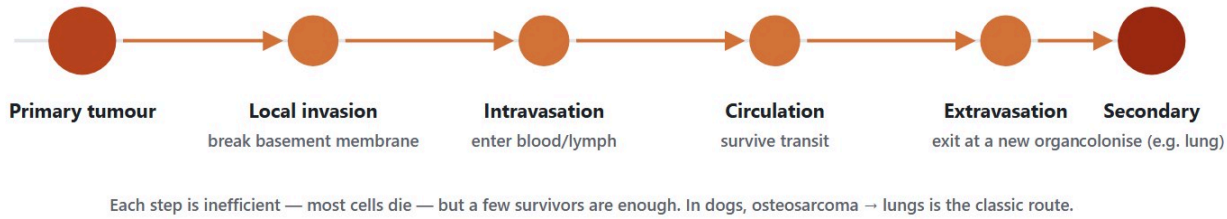


Fig 4 — The metastatic cascade: invade → intravasate → circulate → extravasate → colonise.

Diagram — Vet-eBooks.com, built for this guide. The steps of the cascade — local invasion, intravasation, survival in transit, extravasation and colonisation — follow the sources below.

> Metastasis is the main reason cancer is lethal — once a tumour has spread, local surgery alone can rarely cure it. That is why *staging* (finding spread) drives the whole treatment plan.

07 Cancer in dogs & cats

Companion animals develop cancers with the same hallmarks as people — which is exactly why dogs are prized in **comparative oncology**. But the spectrum differs: where carcinomas (epithelial) dominate in humans, **dogs develop proportionally more sarcomas** (mesenchymal). The ones you will meet most often are worth knowing on sight.

In dogs: **lymphoma** — often multicentric (big, non-painful lymph nodes) and highly chemo-responsive; **mast-cell tumours** — the "great pretender" skin lump, driven by *c-kit* and graded for aggressiveness; **osteosarcoma** — large and giant breeds, lameness/pathological fracture, early lung spread; **haemangiosarcoma** (hemangiosarcoma) — spleen or right atrium, presenting as sudden collapse from internal bleeding; and **mammary tumours** — roughly half malignant in the bitch, and strongly hormone-driven.

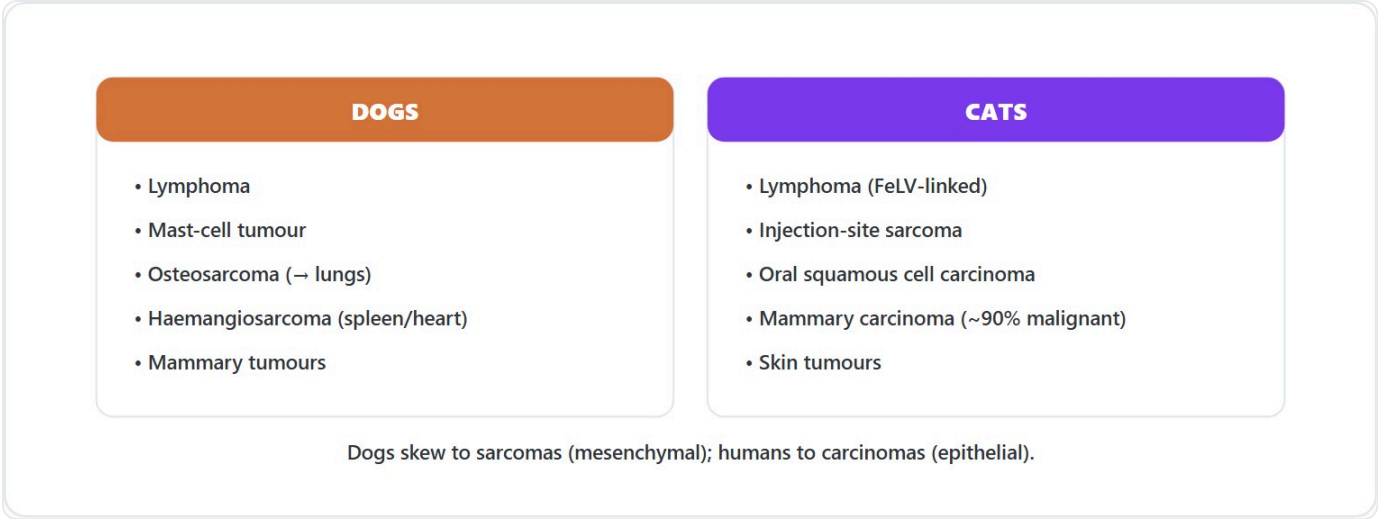


Fig 5 — The common cancers of dogs and cats. Note the species shift toward sarcomas in the dog.

Diagram — Vet-eBooks.com, built for this guide. The tumour types and their relative frequency in the dog and cat are drawn from the sources below.

Cancer in cats shifts the pattern again: **lymphoma** (historically FeLV-associated), **injection-site sarcoma** (a firm mass weeks-to-months after a vaccine or injection), oral **squamous cell carcinoma**, and **mammary carcinoma** — which, unlike the dog, is malignant in roughly 90% of cats and warrants aggressive treatment.

Outside the dog and cat the same biology produces a different cast: **equine sarcoids** and grey-horse **melanoma**, **bovine ocular squamous cell carcinoma** ("cancer eye"), enzootic **bovine leukosis**, and the **transmissible venereal tumour** of dogs — one of the few naturally transmissible cancers, in which the tumour cells themselves are the infectious agent, passed intact between animals.

08 Paraneoplastic syndromes — when the signs are far from the tumour

Some tumours cause illness at a distance, through what they secrete rather than the space they occupy. These **paraneoplastic syndromes** matter because they are so often the *presenting* problem: the animal is investigated for something else entirely and the cancer is found second.

The ones worth recognising on sight: **hypercalcaemia of malignancy** (hypercalcemia), classically from lymphoma, anal-sac apocrine gland adenocarcinoma, thymoma or multiple myeloma, presenting as polyuria, polydipsia and weakness; **hypoglycaemia (hypoglycemia)** from an insulinoma or a large hepatic or smooth-muscle tumour, presenting as collapse or seizures; **anaemia** (anemia) of chronic disease, blood loss or marrow crowding; **hypertrophic osteopathy**, a painful periosteal reaction along the long bones secondary to an intrathoracic mass; and **cancer cachexia**, the loss of lean body mass that continues even while the animal is still eating.

For vetsA persistently raised ionised calcium with no obvious cause is a hunt for a tumour until proven otherwise. Start with a rectal examination for the anal sacs, then image the thorax and the abdominal lymph nodes.

09 Clinical — diagnosis, grading, staging & targeting the hallmarks

Diagnosis means getting cells or tissue: a fine-needle aspirate for **cytology** (fast, cheap, often enough for lymphoma or mast-cell tumours), or a **biopsy** for **histopathology** when architecture matters. The pathologist then **grades** the tumour — how aggressive the cells look, judged on differentiation and mitotic count — and the clinician **stages** it — how far it has spread, using the TNM scheme (Tumour, Nodes, Metastasis) with imaging and node sampling. Grade and stage together set prognosis and plan.

Why the hallmarks matter at the cage side: each one is a drug target. Surgery and radiation remove or destroy the local mass; cytotoxic **chemotherapy** (e.g. doxorubicin/vincristine-based **CHOP** for lymphoma, carboplatin after amputation for osteosarcoma) tips proliferating cells into apoptosis. Newer **targeted** drugs hit specific hallmarks — the tyrosine-kinase inhibitor **toceranib (Palladia)** blocks the proliferative-signalling and angiogenesis hallmarks in *c-kit*-driven mast-cell tumours; the canine **melanoma vaccine** attacks the immune-evasion hallmark. And prevention follows the biology too: **spaying before the first seasons** sharply cuts mammary-tumour risk, and vaccinating against FeLV lowers feline lymphoma.

Prognosis is the owner's first question, and honest numbers depend on grade and stage rather than the word "cancer" alone: many canine **lymphomas** reach remission for around a year on a CHOP protocol, a completely excised low-grade **mast-cell tumour** is often cured,

whereas **osteosarcoma** already in the lungs is measured in months. Framing that honestly — cure, control, or comfort — is as much a part of oncology as the drugs.

> **Red flag**
>
> Any mass that is growing, ulcerated, fixed to deeper tissue, or recurs after removal — especially a new "lump" in an older animal — warrants an aspirate or biopsy, not "watch and wait". Early diagnosis is the single biggest lever on outcome.

10 Quiz: test yourself

Sixteen questions across the genetic engine, apoptosis, the ten hallmarks, metastasis and the species patterns you will actually meet. Answers are beneath each question — cover them and work down.

1. In one sentence, what is cancer?**Answer:** A single cell breaking the rules and **growing on its own terms**, deaf to the signals that normally govern it. Homeostasis, lost.2. What is a proto-oncogene, and what turns it into an oncogene?**Answer:** A normal **growth-promoting** gene carrying the "grow" signal from the cell surface to the nucleus. A **gain-of-function** mutation jams a step permanently on, so the cell hears a constant grow command.3. What is the canonical proliferative signalling route?**Answer:** Growth factor → **receptor tyrosine kinase** → **Ras** (a small G-protein flipping GDP to GTP) → the **MAP-kinase cascade** (Mek → Erk) → transcription factors such as **Myc** → cyclins.4. What drives the cell cycle, and where does the first checkpoint sit?**Answer: Cyclin-dependent kinases (CDKs)** drive G1 → S → G2 → M. The first checkpoint sits **just before S phase**, stopping a cell that is damaged or unready.5. What are the two pathways to apoptosis?**Answer:** The **extrinsic** pathway, triggered when a death receptor on the surface is engaged (for example by immune cells), and the **intrinsic (mitochondrial)** pathway, governed by the **Bcl-2 family** — a tug-of-war between pro-death and pro-survival members.6. What are two ways a tumour resists apoptosis, with an example of each?**Answer: Over-expressing pro-survival Bcl-2** — classic in lymphoma — or **losing p53**, which normally links DNA damage to the death programme.7. Who described the hallmarks of cancer, and how did the list grow?**Answer: Hanahan and Weinberg**, in **2000**. The original six gained, in the **2011** update, two enabling characteristics (genome instability, tumour-promoting inflammation) and two emerging hallmarks (reprogrammed energetics, immune evasion) — ten in all.8. Which hallmark does a *c-kit* mutation in a canine mast-cell tumour represent?**Answer: Sustaining proliferative signalling** — the cell makes its own grow signal.9. Which hallmark does re-activating telomerase represent, and how common is it?**Answer: Replicative immortality**, and it is reached by **most malignant tumours**.10. Which hallmark does VEGF secretion serve?**Answer: Inducing angiogenesis** — the tumour builds its own blood supply.11. Why does cancer take time to develop?**Answer:** Because **no single mutation is enough**. The hallmarks are acquired one at a time, which is why incidence climbs with age.12. Which cancers are common in the cat?**Answer: Lymphoma** (historically FeLV-associated), **injection-site sarcoma** (a firm mass weeks to months after a vaccine or injection), **oral squamous cell carcinoma** and **mammary carcinoma**.13. How does feline mammary carcinoma differ from canine mammary tumour?**Answer:** In the cat it is **malignant in the great majority of cases**, where the dog's is far more often benign. The species changes the prognosis, not just the patient.14. What is a paraneoplastic syndrome?**Answer:** Illness caused **at a distance** from the tumour — through what it **secretes** rather than the space it occupies.15. What is the difference between grading and staging?**Answer: Grade** is how aggressive the cells look, judged on differentiation and mitotic count, by the pathologist. **Stage** is how far it has spread, by the clinician, using **TNM** — Tumour, Nodes, Metastasis. Together they set prognosis and plan.16. Which hallmarks does toceranib (Palladia) target, and in what tumour?**Answer: Proliferative signalling and angiogenesis**, in *c-kit*-driven canine **mast-cell tumours**. It is a tyrosine-kinase inhibitor — a hallmark targeted directly.

11 Cancer in animals — frequently asked questions

What actually is cancer, in simple terms?

A cell that has escaped the body's controls on growth and death and multiplies on its own — the breakdown of normal cellular homeostasis. When that growth can invade and spread, it is malignant: cancer.

What are the hallmarks of cancer?

Ten shared capabilities every cancer acquires: sustaining its own growth signals, evading growth suppressors, resisting cell death, becoming immortal, inducing a blood supply, invading and metastasising, genome instability, tumour-promoting inflammation, immune evasion, and deregulated energetics.

Are cancers in dogs and cats like human cancers?

Biologically, yes — they share the same hallmarks, which is why dogs are used as natural models in comparative oncology. The mix differs: dogs get relatively more sarcomas, humans more carcinomas.

What causes cancer in dogs?

Not one thing. Cancer is the end point of accumulated **DNA damage** in a single cell line, and the contributors stack over a lifetime: inherited breed predisposition (boxers and golden retrievers; large and giant breeds for osteosarcoma), hormonal drive (entire bitches and mammary tumours), viruses (FeLV in cats, papillomavirus), ultraviolet light on unpigmented skin, chemical carcinogens, chronic inflammation, and simple age — every cell division is another chance for an uncorrected error. That is why cancer is largely a disease of older animals: it takes time to collect enough mutations.

What are the first signs of cancer in dogs?

There is no single sign, which is exactly the problem. The ones that should trigger investigation rather than watchful waiting: a lump that is **growing, ulcerated or fixed** to deeper tissue; unexplained **weight loss** despite a normal appetite; **lameness** in a large-breed dog that does not resolve; enlarged but non-painful **lymph nodes**; sudden **collapse** with pale gums, which suggests a bleeding splenic haemangiosarcoma; an oral mass or a wound that will not heal; or new polyuria and polydipsia, which may be paraneoplastic hypercalcaemia. A mass old enough to have been noticed is old enough to be sampled.

Is cancer in dogs curable?

Sometimes genuinely cured, more often controlled, and the honest answer turns on **grade and stage** rather than on the word "cancer" itself. A completely excised low-grade mast-cell tumour is frequently cured outright. Canine lymphoma is usually *controlled* — most dogs reach remission on a CHOP protocol for roughly a year, but the disease is not eradicated. Osteosarcoma that has already seeded the lungs is measured in months. The useful framing for an owner is cure, control, or comfort — and which of the three applies is knowable only after grading and staging.

Can I download these cancer study notes as a free PDF?

Yes — these are complete cancer-biology study notes, free to download as a PDF with no signup. For more, browse our veterinary physiology study notes.

When to refer

- Any rapidly growing, ulcerated, or deeply fixed mass — aspirate/biopsy promptly; don't 'watch and wait'.
- Confirmed malignancy — stage it (imaging ± lymph-node/organ sampling) before surgery, and refer to oncology for chemo/targeted options.
- Pathological fracture or bone pain in a large-breed dog — suspect **osteosarcoma**; image and refer.
- Cat with a firm mass at a previous injection site — suspect **injection-site sarcoma**; plan a wide biopsy + referral.

Sources

1. Cunningham JG, Klein BG. Cunningham's Textbook of Veterinary Physiology. 6th ed. St Louis: Elsevier; 2020. Chapter 2 — Cancer (pp 41–52): cell cycle, the ras/MAP-kinase pathway, Rb/p53 gatekeepers, apoptosis.
2. Sjaastad ØV, Sand O, Hove K. Physiology of Domestic Animals. 3rd ed. Oslo: Scandinavian Veterinary Press; 2016. Chapter 2 — Cells & Chapter 21 — Bioenergetics and Growth (cell structure, division and growth control).
3. Hanahan D, Weinberg RA. Hallmarks of Cancer: The Next Generation. Cell. 2011;144(5):646–674.
4. Withrow SJ, Vail DM, Page RL. Withrow & MacEwen's Small Animal Clinical Oncology. 5th ed. St Louis: Elsevier; 2013 — canine/feline tumour spectrum, grading/staging and hallmark-targeted therapy.

Vet-eBooks.com · free veterinary study guides · the full illustrated article, with every figure at full size, is at vet-ebooks.com/articles/cancer-in-animals/

Vet eBooks
VET-EBOOKS.COM

MORE FREE STUDY GUIDES · RESOURCES

More free veterinary study guides

This is one of a growing library of free veterinary study guides on vet-ebooks.com — written for practising vets and students, sourced from the standard veterinary texts, and every one free with a downloadable PDF and no sign-up.

3,400+
veterinary books

400+
drug profiles

Study guides
a free PDF with each

All free
no sign-up needed

www.vet-ebooks.com

For educational use. Always confirm doses and clinical decisions against current authoritative texts and your own clinical judgment. © vet-ebooks.com — share freely, do not modify.