Obesity and Cancer in Dogs & Cats

Dr. Catherine Chan[1] BVSc (Hons I) FANZCVS (Veterinary Oncology) DACVIM (Oncology)

Registered Specialist in Veterinary Oncology

[1]**The Pet Oncologist Pty Ltd**, info@thepetoncologist.com

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Introduction

Cancer is common in both dogs and cats. One in four dogs and one in five cats will develop cancer [1]. The prevalence increases significantly with age: about 50% of dogs and one in three cats over ten years develop cancer [2,3].

Cancer is a leading cause of death in the ageing pet population, with nearly half of dogs and one in three cats over ten years dying from cancer [4–9]. Whilst cancer is less common in cats, they are more likely to have malignant tumours, with 79% of feline cancers being malignant compared to approximately 46% in dogs [3,5]. Early detection and regular screening for cancer are critical, especially in older pets.

Obesity has emerged as a significant health concern in companion animals, with growing evidence linking it to increased cancer risk, progression, and poorer outcomes [10,11]. This presentation explores the complex relationship between obesity and cancer in dogs and cats, with a focus on prevention and clinical management.

Key Questions Addressed in This Presentation

In response to the most common and important questions raised by veterinary professionals regarding obesity and cancer in companion animals, this presentation aims to address the following:

1. Which cancers are more prevalent in dogs and cats with obesity?
2. Are there any positive effects of obesity for cancer patients in dogs and cats?
3. What are the negative effects of obesity in dogs and cats with cancer?
4. What does the literature say about obesity and specific cancers?
5. What are the best dietary management strategies for obesity and cancer patients?
6. Do ketogenic or carbohydrate-restricted diets have the same effects in dogs and cats as in humans with cancer?
7. What is the role of adiponectins in cancer?

8. How should chemotherapy dosing be approached in obese patients?
9. What are the facts about obesity and cancer nutrition?
10. Are there concerns regarding drugs that deposit in fat in obese patients?
11. Is it better for cancer patients to be overweight or obese?
12. How should obesity and overweight be managed during chemotherapy?
13. What is the relationship between fat dogs and lipoma development?
14. What is the association between obesity and mast cell tumour development in dogs and cats?
15. How does obesity relate to other cancers and such as insulinoma and colon cancer?

1. Epidemiology of Obesity and Cancer in Pets

1.1 Current Prevalence Data

Obesity is increasingly recognised in companion animals and mirrors trends observed in human populations [12–15]. In Australia, a significant proportion of dogs and cats are classified as overweight or obese [13,15,16]. In my clinical experience, many pet owners do not recognise when their pets are overweight. This represents a critical gap in preventive veterinary care and highlights the importance of professional body condition assessment during routine examinations.

1.2 Risk Factors for Obesity Development

Several key factors predispose companion animals to obesity.

Neutering: The literature strongly supports that neutering significantly increases obesity risk in both dogs and cats, primarily through mechanisms such as reduced metabolic rate and increased appetite [12,14,17]. Early-age desexing particularly amplifies this risk, with cats desexed before six months showing higher obesity rates than those desexed later [14].

Breed predisposition also plays a major role, with certain breeds—including Labrador Retrievers, Cocker Spaniels, and Cavalier King Charles Spaniels—genetically more susceptible to obesity due to mutations affecting appetite regulation and metabolism [12,14].

Advancing age contributes to obesity risk, as older animals experience a decline in lean body mass and activity levels, which in the absence of appropriate dietary modifications, is conducive to weight gain [12,14].

2. Mechanisms Linking Obesity and Cancer

Obesity promotes a chronic, low-grade inflammatory state, which is now recognised as a key driver of carcinogenesis [10,11].

2.1 Adipose Tissue as an Endocrine Organ

White adipose tissue acts as a metabolically active endocrine organ, secreting adipokines, hormones (such as oestrogens), inflammatory cytokines, and growth factors that can influence tumour development and progression [18]. In obesity, adipose tissue undergoes pathological remodelling, leading to dysregulated production of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and monocyte chemoattractant protein-1 (MCP-1) [11]. This chronic low-grade inflammatory state fosters carcinogenesis through DNA damage, altered immune function, and disrupted cellular signalling [10,11]. The inflammatory microenvironment in obesity shares similarities with the tumour microenvironment that favours tumour growth, angiogenesis, and metastasis [10,11].

2.2 Adipokine Dysregulation

2.2.1 Leptin

Leptin, produced by white adipocytes, becomes dysregulated in obesity, leading to hyperleptinaemia and leptin resistance [11]. Elevated leptin levels have been linked with various cancers in humans via JAK/STAT3 and MAPK signalling. In dogs, obesity induces excessive leptin production, with levels decreasing after weight loss [11].

2.2.2 Adiponectin

Adiponectin demonstrates anti-inflammatory and potentially anti-cancer properties. Obese animals tend to have lower adiponectin levels compared to lean counterparts, which may compromise protective mechanisms against cancer development. Studies in dogs consistently show inverse relationships between body condition and adiponectin concentrations [11].

2.2.3 Obesity-Related Insulin Resistance

Obesity-related insulin resistance and increased levels of insulin-like growth factors (IGF-1 and IGF-2) may promote tumorigenesis in dogs by enhancing cell proliferation and inhibiting apoptosis [10, 19, 20].

Insulin resistance and hyperinsulinaemia are common in obese animals and can directly or indirectly stimulate tumour cell growth through multiple pathways, including increased bioavailability of IGFs and chronic low-grade inflammation [10, 18].

In dogs, pancreatic islet cell tumours (insulinomas) cause paraneoplastic hypoglycaemia primarily by secreting insulin. Rarely, some pancreatic neuroendocrine tumours may secrete IGF-2, but this is uncommon [21-23]. Conversely, non-islet cell tumours causing hypoglycaemia typically do so via ectopic production of IGF-2 or its high-molecular-weight precursor (big-IGF-2) rather than insulin itself [18,24]. Tumour types reported to cause IGF-2-mediated hypoglycaemia include hepatocellular carcinoma, hepatoma, leiomyoma, leiomyosarcoma, fibrosarcoma, mammary gland carcinoma, pulmonary carcinoma, renal carcinoma, nephroblastoma, haemangiosarcoma, melanoma and

osteosarcoma [25–28]. These tumours induce non-islet cell tumour hypoglycaemia (NICTH) by secreting IGF-2, which mimics insulin's effects, leading to hypoglycaemia despite low circulating insulin levels [22,24].

In cats, published reports of IGF-secreting tumours are extremely rare and less well documented than in dogs [22,28,30]. Most feline cases of paraneoplastic hypoglycaemia are due to insulinomas, with only sporadic reports of non-islet cell tumour hypoglycaemia, and almost no confirmed cases of IGF-2-secreting tumours [27-29].

Resistin, secreted by white adipocytes, promotes insulin resistance and inflammatory responses. Elevated resistin levels in obese patients may contribute to metabolic dysfunction and potentially influence cancer risk through insulin resistance pathways. While the literature is not as extensive as in human medicine, resistin is now recognised as an important link between obesity, inflammation, and cancer in dogs—especially mammary carcinoma [35]. For other cancers (e.g. lymphoma, osteosarcoma), the relationship is less clear and not yet confirmed.

3. Specific Cancers Associated with Obesity

Dogs are at increased risk of mammary tumours, transitional cell carcinoma (TCC), and mast cell tumours (MCTs). In cats, mammary cancer is of particular concern, with obesity suspected to play a similar role as in human breast cancer, though more research is needed [11].

3.1 Canine Mammary Carcinoma

The strongest evidence for obesity–cancer associations in veterinary medicine involves canine mammary carcinoma. Multiple studies demonstrate that overweight and obese female dogs develop mammary tumours at younger ages with more aggressive characteristics [37,38]. Key findings include earlier age of tumour development in overweight or obese dogs, higher histological grades and increased lymphatic invasion, greater tumour-associated macrophage infiltration and angiogenesis, reduced adiponectin expression correlating with poor prognosis markers, and shorter cancer-specific survival times in overweight or obese patients [31,32]. Obesity influences mammary carcinoma through increased aromatase expression, with 80% of aromatase-positive tumours occurring in overweight or obese dogs [32].

3.2 Feline Mammary Carcinoma

Obesity is suspected to play a similar role in feline mammary cancer as in human breast cancer, but direct evidence in cats is limited and more species-specific studies are needed [11].

3.3 Transitional Cell Carcinoma (TCC)

Obesity is a risk factor for canine TCC, with obese dogs showing increased bladder cancer risk regardless of chemical exposure [34]. This cancer type demonstrates strong breed predisposition and is associated with female sex and older age [36].

3.4 Mast Cell Tumours (MCTs)

Weight is a significant risk factor for mast cell tumour (MCT) development in dogs, with studies showing heavier dogs having increased odds of developing MCTs compared to those weighing ≤10 kg [37,38].

3.5 Lipomas

While benign, lipomas show strong associations with body weight. In dogs, the odds of developing lipomas increase progressively with bodyweight. Purebreds such as Weimaraners, Dobermann Pinschers, German Pointers, Springer Spaniels, and Labrador Retrievers have higher prevalence rates compared to crossbreds. Neutered dogs have higher odds of developing lipomas. Advanced age is also a major risk factor [39].

3.6 Other Cancer Types

Limited evidence suggests potential associations between obesity and other cancer types, though data remain sparse. Osteosarcoma risk increases with body weight and height, though the relationship with obesity specifically (as opposed to large size) requires further investigation [40]. The relationship between obesity and lymphoma risk in pets is less clear. Some studies in dogs suggest a possible association, but evidence remains limited [11].

4. Body Condition and Obesity: Impact on Cancer Prognosis and Survival

4.1 The Obesity Paradox

Some studies reveal an "obesity paradox" in cancer patients. While obesity increases cancer risk, being underweight at diagnosis consistently predicts worse outcomes [41,42].

In dogs with lymphoma, underweight patients have significantly shorter survival times compared to ideal-weight or overweight dogs [41].

For feline cancer patients, cats with body condition scores (BCS) <5 had reduced median survival times compared to cats with scores ≥5 [42]. This is particularly evident in cats with lymphoma, where lymphoma-specific survival times were significantly shorter in cats that lose ≥5% of their body weight within the first month of treatment [42]. Other studies show both low body weight and BCS are associated with significantly shorter survival times in cats with lymphoma and other cancers [41]. These findings underscore the importance of maintaining adequate body condition during cancer treatment.

4.2 Weight Changes During Treatment

Dogs gaining more than 10% body weight during cancer treatment demonstrated longer median survival times compared to those losing weight or maintaining stable weight [41].

These findings support the importance of maintaining adequate body condition and nutritional support during cancer therapy.

5. Clinical Management Considerations

5.1 Chemotherapy Dosing in Obese Patients

Chemotherapy dosing in obese patients remains controversial.

Human oncology guidelines recommend using actual body weight rather than ideal body weight to avoid underdosing. Studies show that obese human patients dosed by actual body weight experience similar or lower toxicity rates compared to normal-weight patients [43,44].

In veterinary oncology, most oncologists—including myself—dose according to actual body weight, as empirical dose reductions may compromise treatment efficacy and survival outcomes [45].

Whilst research shows that smaller dogs are more susceptible to chemotherapy toxicity, with dogs ≤10 kg having a higher risk of severe neutropenia with certain drugs [46], in my clinical experience, careful dose selection and monitoring are key, especially in smaller patients. For example, I often use the lower end of the recommended dosing range (such as 1 mg/kg for dogs under 15 kg receiving doxorubicin, rather than 30 mg/m^2), which helps to minimise toxicity while maintaining efficacy. Dose adjustments should be made only in response to observed toxicity, not pre-emptively.

Additionally, although the use of prophylactic antibiotics with myelosuppressive chemotherapy agents remains debated, I strongly advocate for their use when starting a new potentially myelosuppressive chemotherapy drug. A double-blinded, placebo-controlled study demonstrated that prophylactic trimethoprim-sulfadiazine significantly reduced rates of hospitalisation and non-haematological toxicities, including gastrointestinal toxicity, compared to dogs who did not receive prophylactic antibiotics (placebo) [47].

5.2 Nutritional Management of Cancer Patients

Nutritional management of obese cancer patients requires individualised approaches addressing both obesity and cancer-related cachexia [48–50]. This involves balancing the need for controlled weight loss with the prevention of malnutrition and muscle wasting, which are common in cancer patients [49-51]. Key principles include:

- High-protein diets to support lean body mass.
- Moderate carbohydrate intake has not been shown to cause metabolic syndrome or worsen cancer outcomes in dogs and cats. In fact, total carbohydrate restriction may be harmful. While ketogenic diets show promise in human oncology, they may not reliably induce or sustain ketosis in dogs and cats due to species differences [52-55].
- Commercial diets that are complete and balanced, provide optimal nutrition for cancer patients [41].
- Gradual weight reduction in obese cancer patients to avoid exacerbating muscle loss of malnutrition [49].
- Supplementation with omega-3 fatty acids (EPA and DHA) has shown positive effects in dogs with cancer [56].

- Vitamin E is an antioxidant that may help protect against free radical damage and support immune function, but there is no established dose for supplementation in veterinary cancer patients, and more research is needed [50].

Summary:
Nutritional management in obese cancer patients should prioritise high-protein, moderate-carbohydrate, and energy- or calorie-dense diets, with careful weight loss protocols and consideration of omega-3 supplementation.

The main aim when pets have a diagnosis of cancer is to maintain an optimal body weight and condition that preserves lean body mass and avoids both excessive weight loss and anorexia. Feeding a cancer diet during therapy has not been shown to improve survival outcomes, but it can help improve quality of life. An adequate body condition can help improve tolerance to anti-cancer therapies. The best cancer diet is a complete and balanced commercial diet, that the patient enjoys. If there is cachexia or weight loss, a calorie-dense diet is best. However, it is important to avoid snacks and high-fat foods that can lead to gastrointestinal signs and pancreatitis.

Click this link if you'd like to learn more about nutrition in dogs and cats with cancer: https://www.thepetoncologist.com/blog/category/Nutrition

5.3 Ketogenic Diets and Carbohydrate Restriction in Veterinary Oncology

Interest in ketogenic diets (KDs) and carbohydrate restriction for cancer management in pets has grown, largely extrapolated from human and rodent oncology research. The rationale is based on the 'Warburg effect', which describes how many cancer cells preferentially utilise glucose via anaerobic glycolysis. This suggests that restricting dietary carbohydrates could theoretically "starve" cancer cells and slow tumour progression. However, robust evidence supporting the use of KDs in veterinary oncology is lacking. Most studies are limited to case reports or small series, and controlled trials have not demonstrated a survival benefit [52,57]. For example, the KetoPet Sanctuary project reported anecdotal benefits in dogs with cancer fed a ketogenic diet, but these findings lack robust scientific controls and statistical analysis [57]. A controlled study in dogs with lymphoma found no significant difference in remission times or energy requirements between high- and low-carbohydrate diets [56].

It is important to recognise significant species differences in response to ketogenic diets limit the applicability of ketogenic diets in veterinary patients. In humans, KDs can reliably induce and sustain ketosis, but dogs metabolise ketone bodies more rapidly and typically achieve much lower ketone levels, making sustained ketosis difficult [52]. In cats, which are obligate carnivores, inducing ketosis is even more challenging and may increase the risk of hepatic lipidosis [54]. Extreme carbohydrate restriction in pets can also risk nutritional deficiencies and worsen cachexia, particularly in those with poor appetite or muscle loss. Therefore, while KDs are established in human oncology, their application in dogs and cats remains unproven and should be approached with caution [52,55,54].

6. Practice Recommendations for General Practitioners

6.1 Prevention Strategies

Body condition assessment: Perform regular BCS evaluations during routine examinations. Maintaining an ideal BCS helps reduce cancer risk and improves patient outcomes

Client education: Actively address the significant awareness gap regarding pet obesity, where most owners do not recognise when their pet is overweight

Spay or neuter timing: Discuss the potential impact of desexing on obesity risk when advising clients about optimal timing. However, note that the timing of neutering remains a highly controversial and debated topic.

Breed-specific counselling: Provide enhanced monitoring and tailored advice for breeds known to be predisposed to obesity.

Weight management strategies: Encourage appropriate diet, regular exercise, and ongoing monitoring to maintain optimal body condition in all pets.

6.2 Cancer Screening in Obese Patients

Enhanced surveillance: Schedule more frequent examinations for pets at increased risk of certain cancer types.

Mammary gland monitoring: Pay particular attention to intact or late-spayed female dogs, as they are at higher risk for mammary tumours.

Urinary monitoring: Maintain vigilance for urinary symptoms in obese dogs, which may be at increased risk for urothelial carcinoma.

Mass evaluation: Promptly investigate any new masses, especially in anatomical areas prone to specific tumour types.

6.3 Treatment Considerations

Avoid severe underweight: Strive to maintain adequate body condition throughout cancer treatment, as underweight status can negatively affect outcomes.

Coordinate care: Close collaboration with veterinary oncologists when managing complex cancer cases to optimise patient care.

Nutritional support: Implement appropriate dietary management protocols to support the nutritional needs of cancer patients.

Monitor comorbidities: Address obesity-related conditions (e.g. diabetes, osteoarthritis) that may complicate cancer treatment and recovery.

7. Knowledge Gaps and Future Directions

7.1 Species Differences

Current research demonstrates significant knowledge gaps regarding obesity–cancer relationships in cats compared to dogs [10]. While mechanisms appear similar across species, specific cancer associations in felines require further investigation [11].

7.2 Therapeutic Implications

More research is needed to establish optimal chemotherapy dosing strategies in obese veterinary patients. The balance between avoiding toxicity and maintaining therapeutic efficacy requires species-specific investigation [43].

Conclusion

Obesity represents a significant and modifiable risk factor for cancer development in companion animals, particularly for canine mammary carcinoma, transitional cell carcinoma, and mast cell tumours. The relationship between obesity and cancer involves complex mechanisms, including chronic inflammation, adipokine dysregulation, and hormonal alterations. General practitioners play a crucial role in both the prevention of obesity and the early intervention for at-risk patients.

Early intervention and client education are paramount, as obesity significantly increases cancer risk through multiple mechanisms. However, it is equally as important to avoid severe underweight in cancer patients while also addressing obesity, as both extremes can negatively impact patient outcomes. Collaboration with veterinary oncologists is recommended to ensure optimal management and support for complex cases, and enhanced surveillance for cancer development in obese patients is advised.

By understanding these relationships and implementing evidence-based management strategies, veterinarians can have a meaningful impact on both cancer prevention and treatment outcomes in companion animals.

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