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Thin Cats, Fat Dogs

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EDITORIAL

Known to generations of schoolchildren for his theorem on right-angled triangles, Pythagoras was much more than a mere mathematician. He actively pursued various interests, including astronomy, philosophy and religion, and it is generally agreed that he was an enthusiastic supporter of the notion that nature could be regarded as a system of opposing forces – so that good contrasted with evil, light opposed dark, left counteracted right, and so on. This fitted well with the ancient Greek belief that balance and equilibrium in the human body was essential for health, a concept which was eventually embraced by many cultures and is best exemplified by what became known as humoral medicine. This was based on the idea that a healthy body depended on a balance between four humors, or fluids: blood, black bile, yellow bile, and phlegm. A deficiency or excess in one or more of the humors, so that they were out of balance, was considered to cause disease. This view enjoyed a surprisingly long and popular following, and became the dominant theory in medical circles for many centuries, remaining a major influence on clinical practice and teaching until well into the 1800s. when a more rational approach to health and disease started to emerge.

But is there a link between Pythagoras and the chosen topic for this issue of *Veterinary Focus*? One can argue that fat dogs and thin cats, and the causes thereof, are not the same at all, but the Greek polymath may have approved, in that they can be said to be opposite ends of the same spectrum. Not only that, but the different articles in this issue consider the matter in various ways yet, as a whole, they offer a balance; and there is the overall emphasis on a concept that is both very modern and very ancient – namely, that a holistic approach to the patient is best. Pythagoras could have said that tackling the subject from various angles is indeed a formula that adds up to a whole.

Ewan McNeill – Editor-in-chief

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Editor-in-chief: Ewan McNeill, BVMS, Cert VR, MRCVS
Editorial secretary
- Laurent Cathalan
lcathalan@buena-media.fr

Artwork
- Pierre Ménard

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HOW I APPROACH...

The thin cat with chronic kidney disease

Jessica M. Quimby, DVM, PhD, Dipl. ACVIM
The Ohio State University, Columbus, OH, USA

Dr Quimby received her veterinary degree from the University of Wisconsin-Madison in 2003 and then completed a small animal rotating internship in California. She subsequently spent two years in feline-exclusive private practice before pursuing a combined small animal internal medicine residency and PhD program at Colorado State University. She has a particular interest in feline renal disease and has recently been appointed Associate Professor at the Veterinary Medical Center of The Ohio State University.

Assessment of the elderly thin cat with chronic kidney disease (CKD) is a common scenario in veterinary medicine. It has been demonstrated that cats have frequently lost weight before the CKD diagnosis, with further weight loss commonly noted as the disease progresses (1). Clinical signs of vomiting and dysrexia are often evident in feline CKD patients, and in a recent survey of owners of cats with CKD, 43% of respondents reported abnormal appetite in their pet, necessitating owners to coax the animal to eat more than 50% of the time (2). Weight loss and loss of lean body mass in these patients is therefore likely attributable to changes in appetite as well as processes such as cachexia and sarcopenia.

KEY POINTS

- **Weight loss and poor body condition are common in cats with chronic kidney disease (CKD) and are correlated with a poorer prognosis.**
- **Identification and management of factors contributing to weight loss is important, and every CKD patient should receive a full clinical work-up.**
- **Repeated nutritional assessment and determination of sufficient caloric intake is crucial.**
- **A key therapeutic target for feline CKD patients should be addressing appetite and food intake to meet caloric goals, with the subsequent outcome of maintaining body condition and muscle mass.**

What do I need to know about poor body condition?

The terms cachexia and sarcopenia are frequently used to describe thin patients, but it is important to understand the appropriate usage and implications of both words.

- **Cachexia** refers to a syndrome commonly associated with cancer and chronic illnesses such as kidney, heart and respiratory disease. The condition is characterized by weight loss, loss of muscle mass without loss of adipose tissue mass, and systemic inflammation. Cachexia is often associated with hyporexia and inadequate nutrition (3,4) and is differentiated from conditions such as sarcopenia, starvation, hyperthyroidism, and malabsorption. In cachexia, the negative protein energy balance that develops as a result of inadequate nutrition is combined with an abnormally increased metabolic state, resulting in loss of muscle mass and body condition. In addition, the systemic inflammatory state leads to catabolism of muscle via both protein and fat degradation, as well as impaired ability of amino acids to stimulate protein synthesis (4). Cachexia is a clinically important condition to recognize, as it has been linked to increased morbidity and mortality.
- **Sarcopenia** refers to the degeneration of skeletal muscle mass, quality and strength as part of normal aging, *i.e,* not associated with a disease process. Contributing factors include age-related decrease in nerve impulses from the brain to muscle, decreased physical activity, and decreased ability to synthesize muscle proteins, combined with inadequate protein nutrition to sustain muscle mass (3,4). Sarcopenia may be difficult to identify because total weight may not change; loss of lean body mass may be accompanied by an increase in adipose tissue mass. Little information exists for cats on changes in body condition with age.

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Both cachexia and sarcopenia involve muscle dysfunction which contributes to overall frailty, and can clinically manifest as weakness, hyporexia, and perceived poor quality of life. These syndromes can therefore have important clinical implications and should whenever possible be identified and addressed.

Poor body condition in CKD has been shown to be correlated to a poorer prognosis in several species, including dogs and cats ***(Figure 1),*** and a recent study demonstrated that weight loss is associated with a shortened survival time in CKD cats (1). In addition to complications such as hypertension, dehydration, anemia and hypokalemia, which can all negatively affect appetite, dysregulation of appetite stimulation is also described in CKD and may contribute to dysrexia (5). In humans, many additional factors are described that may contribute to CKD cachexia, including hypermetabolism, uremic toxins, metabolic acidosis, inflammation, and comorbidities (6,7). There is evidence that CKD results in an increased metabolic state, making adequate nutrition even more of a challenge (7). In fact, the phenomenon of the "obesity paradox" has been described in human medicine, where individuals with a higher body condition score (BCS) have a better clinical outcome; protein energy wasting and low BCS are associated with decreased survival, even in patients on dialysis (6).

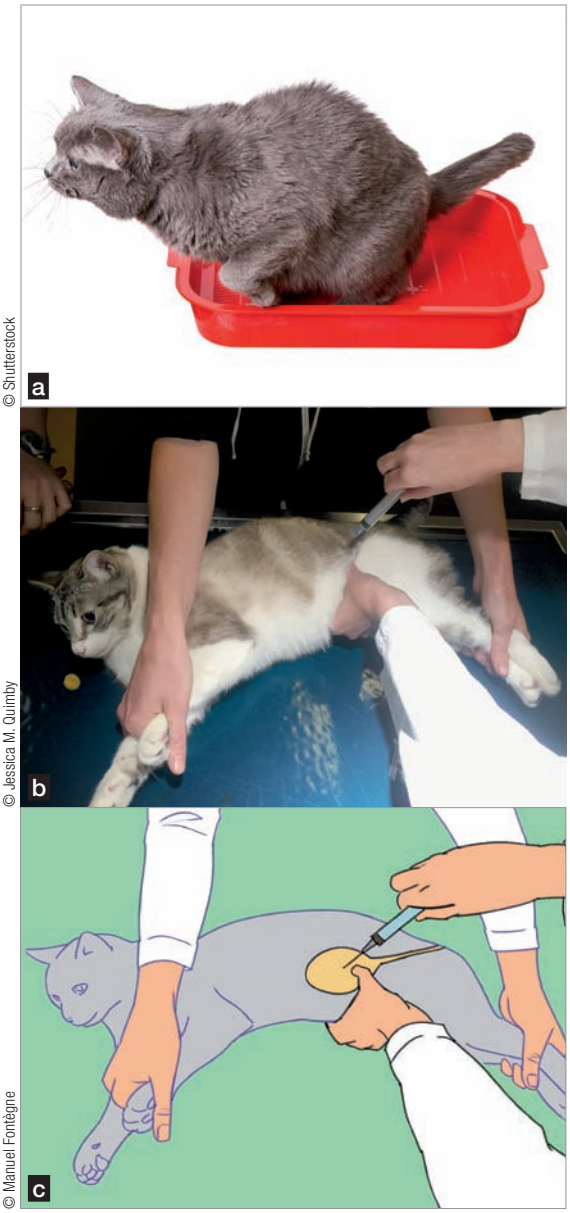
What diagnostic work-up should I consider for the thin CKD cat?

When approaching these patients, it is important to identify as many of the factors that contribute to poor body condition as possible. In both the newly diagnosed and long-term CKD, case identification and management of complications of kidney disease that may affect appetite (*e.g.,* dehydration, nausea, anemia, hypertension, and electrolyte imbalance) is key. Additionally, as comorbidities are common in the elderly feline patient population, determining other possible causes of weight loss is critical and a full diagnostic workup is needed. Therefore, the thin CKD cat should ideally undergo serum biochemistry with electrolytes, complete blood count, cystocentesis for urinalysis ***(Figure 2)*** (to include urine culture and protein:creatinine ratio), blood pressure ***(Figure 3)*** and total T4. Additionally, diagnostic imaging such as abdominal ultrasound and radiographs are valuable to assess for uroliths and/or ureteral obstruction, particularly in a recently diagnosed CKD cat or one that has newly elevated kidney values. It must be determined that the azotemia is from CKD and not processes such as ureteral obstruction or infection. In addition to the diagnostic work-up and identification of contributing diseases, recognizing the processes driving weight loss and muscle wasting in aging and disease will heighten awareness of how to address the needs of these patients.

What is unique about the elderly feline patient?

The potential to develop cachexia or sarcopenia in the feline patient is likely intensified by several unique characteristics of the species. Cats have a higher requirement for protein and amino acids than other animals. When nutrition is inadequate, energy is derived from mobilization of amino acids from muscle stores as opposed to fat. Elderly cats are also unique in comparison to other species as they have a stable or increased level of metabolism, rather than a decreased metabolism, and disease may further result in a hypermetabolic state (8). Exacerbating matters further, a reduced ability to digest protein and fat has been documented in elderly cats, hence they are even more susceptible to the development of cachexia and/or sarcopenia (9). These combined

Figure 1. Poor body condition and muscle mass, as evident here, is correlated with a worse prognosis in feline CKD and should be identified and actively addressed.



Urinalysis is an essential part of assessing any cat with CKD, but obtaining a urine sample can be problematic. Owners may be willing to attempt sample collection at home, but many cats dislike using an empty litter tray or a tray with a non-absorbent substrate in it **(a).** In addition, urine collected in this way can often be of poor quality. Urine collection via catheterization of the bladder is not recommended in a cat with CKD, as this introduces bacteria into a compromised urinary system.

The preferred method for collecting a urine sample is via cystocentesis; this allows a good quality urine sample to be collected from most cats without the need for sedation or general anesthesia. Whilst one person can hold a calm cat for cystocentesis if they support the cat's back against their abdomen **(b,c),** two assistants are usually required, one at each end of the cat. The cat should lie on its right side (assuming the clinician is right-handed), with the cat's back supported or braced, otherwise it tends to slide away from the operator. The rear legs should be pulled gently caudally and ventrally, taking care to not over-extend, which may cause the patient to struggle more.

The clinician palpates the abdomen with the left hand and isolates the bladder cranial to the thumb; light pressure applied between the thumb and the rest of the hand will immobilize the bladder and increase intravesicular pressure, improving success in a small bladder. The needle is held in the right hand at a slight angle directed towards the visualized center of the bladder **(c).** For maximum efficiency and safety, the hand should hold the syringe such that it does not require repositioning half way through the draw.

Urine collection can also be performed via ultrasound-guided cystocentesis (see video link below). This method provides an advantage in patients that still have significant belly fat and also for patients who have a painful bladder and urinate immediately upon palpation before a sample can be obtained.

https://idexxlearningcenter.idexx.com/idexx/resources/ondemand/sn/REST_Kaltura_Player.html?id=1_v9y3n8kw&survey=SN-CAG-IHD-UA-018

Figure 2. Cystocentesis allows collection of a good quality urine sample for analysis.

factors make special attention to nutrition and the utilization of high-quality, easily digestible food products critical for such animals, regardless of their disease state (8). A recent study assessing quality of life parameters in CKD cats revealed that these animals scored significantly lower than healthy young or geriatric cats in the categories of "appetite" and "liking food" (10). Thus, poor appetite is perceived as a significant quality of life concern and can cause significant emotional distress to owners, which may lead to a decision for humane euthanasia.

What is known about the pathophysiology of dysrexia in CKD?

The etiology of dysrexia in CKD is typically attributed to uremic effects on the intestinal tract, such as hyperacidity, uremic gastritis and ulceration, but our understanding of this pathophysiology in cats and dogs is incomplete.

Cats with CKD have been shown to have elevated concentrations of gastrin that increase with the severity of kidney disease (11), but the relationship between gastrin, gastric acid secretion, and gastric pathology has not been fully described. Gastrin is excreted by the kidneys, and it is hypothesized that as renal function declines, hypergastrinemia develops, resulting in gastric hyperacidity (11). However, cats that have gastrin-secreting tumors with levels of hypergastrinemia similar to those found in cats with CKD have significant gastric pathology, but similar findings have not been demonstrated in cats with CKD (12). In human CKD, the development of gastric hyperacidity appears to be inconsistent and may be more related to the presence of *Helicobacter spp.* infection. In a recent study evaluating the type and prevalence of gastric histopathologic lesions in cats with CKD, stomach fibrosis and mineralization were the main changes found, rather than the uremic gastropathy lesions previously described (uremic gastritis, ulceration, vascular injury, edema) in dogs and humans (12).

Uremic toxins are sensed by the chemoreceptor trigger zone (CRTZ) of the area postrema in the brain, which subsequently stimulates emesis by the vomiting center; research has shown that inhibition of this area can halt uremic vomiting in dogs (13), and therefore medications that target receptors in the CRTZ (*i.e.,* $5HT_3$ and NK_1) may be useful when managing nutrition in CKD cases.

In addition to the buildup of uremic toxins, the basic physiology of appetite regulation may be significantly abnormal in cats with CKD. Appetite regulation is comprised of orexigenic substances (*e.g.,* ghrelin) that activate the hunger center, and anorexigenic substances (*i.e.,* leptin, cholecystokinin, obestatin, des-acyl ghrelin) that activate the satiety center of the brain (5). In humans, CKD is associated with an increased accumulation of anorexigenic substances secondary to the decreased glomerular filtration rate without a concomitant increase in orexigenic substances. Additionally, anorexigenic substances have been demonstrated to be significantly higher in CKD patients with poor body condition than those with a normal BCS (5).

How do I assess nutrition in the thin CKD cat?

Serial evaluations of nutritional status are a key part of CKD patient management and a nutritional plan should be performed for every patient. Awareness of these parameters and tools for assessment have been made available by the WSAVA global nutritional initiative*.

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Figure 3. Hypertension is a common concurrent condition in CKD and blood pressure should be measured in all patients with CKD. It can be beneficial to measure blood pressure in the exam room after the cat has acclimatized to its surroundings and before other manipulations are done; keeping the cat in its carrier can help reduce stress levels during the procedure.

A nutritional assessment should include body weight, BCS, muscle mass score, a complete dietary history (including pet food, treats, supplements and items used to give medications), and a judgment on the overall caloric intake (which should include open-ended questions to the owner about how the cat is eating).

Although BCS is an invaluable tool for assessing animals, in obese patients with inadequate muscle mass, BCS by itself often does not adequately describe muscle loss. Assessment of muscle mass is particularly important in CKD patients, as it can have a profound effect on serum creatinine and affect the interpretation of the disease severity, as well as having notable implications for the patient's nutritional status. Muscle mass can be scored ***(page 6)*** based on assessment of epaxial, skull, scapular and iliac musculature, and documented in the medical record at each visit.

How do I manage nutrition in these patients?

Identification of nutritional inadequacy is a huge step in the right direction. Management of acute and chronic dysrexia should not be overlooked in the treatment of feline CKD patients, particularly given their unique nutritional needs. Assisted intervention should be instituted in acutely ill CKD cats that have had inadequate food intake for more than 3-5 days (including the time before presentation to the veterinary clinic). Adequate caloric

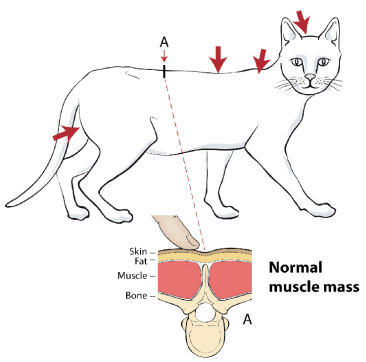
*www.wsava.org/nutrition-toolkit

WSAVA
Global Nutrition
Committee

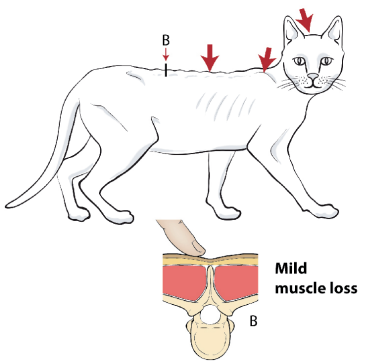
Muscle Condition Score

Muscle condition score is assessed by visualization and palpation of the spine, scapulae, skull, and wings of the ilia. Muscle loss is typically first noted in the epaxial muscles on each side of the spine; muscle loss at other sites can be more variable. Muscle condition score is graded as normal, mild loss, moderate loss, or severe loss. Note that animals can have significant muscle loss even if they are overweight (body condition score > 5/9). Conversely, animals can have a low body condition score (< 4/9) but have minimal muscle loss. Therefore, assessing both body condition score and muscle condition score on every animal at every visit is important. Palpation is especially important with mild muscle loss and in animals that are overweight. An example of each score is shown below.

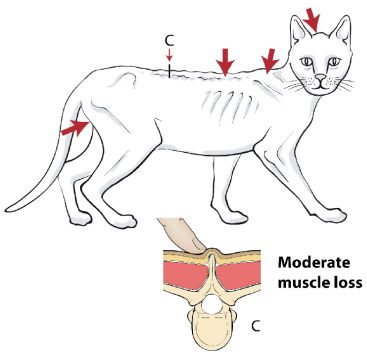
Normal muscle mass



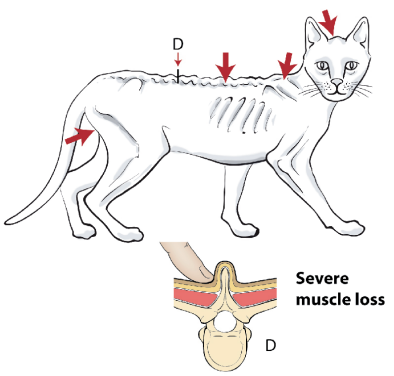
Mild muscle loss



Moderate muscle loss



Severe muscle loss



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support is crucial for effective recovery during any uremic crisis and to prevent significant overall decline in body condition, and a temporary nasoesophageal feeding tube can be a useful non-invasive option for the CKD cat in this scenario ***(Figure 4).***

In chronically ill CKD cats, specially formulated renal diets have been shown to be beneficial (14), but may not have the desired effect if inadequate calories are consumed. Therefore, a key therapeutic target for these patients should be ascertaining caloric goals and addressing appetite and food intake to meet those goals, with the subsequent outcome of maintenance of body condition and muscle mass. As stated above, identifying and managing complications of the kidney disease process that may affect appetite is important. In addition, using tools such as altering food type and temperature, feeding location, food enhancers and (ultimately) appetite stimulants are useful in nutritional management (3). If caloric intake is still inadequate then esophageal feeding tubes should be considered for long-term management ***(Figure 5)*** (15). Regular exercise is also crucial for maintaining muscle mass and strength.

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Figure 4. Nasoesophageal feeding tubes are a helpful non-invasive temporary option for the CKD cat in uremic crisis.

What medications are available to address dysrexia and nausea in CKD cats?

Although it is difficult to determine when CKD patients that are not actively vomiting may experience a sensation of nausea, it is still considered a worthwhile area to target therapeutically. Several anti-emetic/anti-nausea therapies are available that may be helpful in amelioration of nausea and vomiting associated with CKD. These include the NK_1 receptor antagonist maropitant citrate and the $5HT_3$ receptor antagonists ondansetron, dolasetron and mirtazapine. These drugs target the receptors in the CRTZ and vomiting center in the brain where uremic toxins are sensed, as well as at receptors in the GI tract. Maropitant is commonly prescribed short term for acute vomiting, although one study indicated that longer-term usage in cats appears safe and it is often employed for prolonged therapy in chronically ill patients (16). When given daily for two weeks at a dose of 4 mg/cat, maropitant was shown to palliate vomiting in cats with Stage 2 and 3 CKD (17). Due to the placebo-controlled design of the study, Stage 4 CKD cats were not assessed, but it should be noted that these are more typically the patients to which the drug would be prescribed.

Ondansetron and dolasetron are frequently used as anti-emetics, but recent studies have called into question

Figure 5. Esophageal feeding tubes can be very beneficial for long-term management of nutrition in CKD cats and also allow water and medications to be administered in a non-stressful manner.

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their efficacy at currently recommended doses in cats, as the oral bioavailability of ondansetron is poor (~35%) and the half-life is very short (~1 hour), making it most appropriately a Q8 h medication (18). Subcutaneous ondansetron had a slightly longer half-life of 3 hours. Ondansetron is also not appropriate as a transdermal medication; a recent abstract assessing transdermal absorption in cats demonstrated no clinically relevant blood levels after administration (19). Dolasetron has traditionally been recommended at 0.5-1 mg/kg Q24hr, but a recent abstract demonstrated that after a 0.8 mg/kg subcutaneous dose the drug is not detectable in the serum at 12 hours, and it does not palliate xylazine-induced emesis (20).

Although more commonly used as an appetite stimulant, mirtazapine also demonstrates anti-emetic properties, acting at the $5HT_3$ receptor. Several studies have described successful palliation of nausea and vomiting in humans, particularly cancer patients undergoing chemotherapy (21), and in cats the drug has been shown to significantly reduce vomiting associated with CKD (22).

In addition to addressing uremic nausea and vomiting, appetite stimulants can be used to encourage food intake, particularly if an owner is reluctant to use a feeding tube. It is ideal to start such drugs as soon as deficiencies in appetite and muscle mass are noted and caloric needs are not being met. Cyproheptadine has been used for some time and has anecdotal efficacy in many patients, but it has never been scientifically evaluated. Mirtazapine has become more commonly used as a potent appetite stimulant in cats and recent studies have provided information for more effective use (22-24), although higher doses are more commonly associated with side effects (hyperexcitability, vocalization, tremors) (25). Smaller, more frequent doses are therefore recommended to maintain efficacy but minimize side effects; the half-life is short enough that it can be administered daily in healthy cats (24), with a recommended dose of 1.88 mg per cat Q24h. Renal disease delays clearance, and hence alternate day administration (*i.e.,* 1.88 mg Q48 h) is recommended in CKD cats (23), with a recent clinical trial demonstrating that it was an effective appetite stimulant in cats with CKD at this dose, resulting in significantly increased appetite and weight gain (22) ***(Figure 6).*** Mirtazapine is also amenable to transdermal administration and has been shown to achieve both appropriate serum levels and appetite stimulation in healthy cats, and while clinical studies in CKD cats are forthcoming, starting doses for transdermal application

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Figure 6. Appetite stimulants such as mirtazapine have been demonstrated to result in increased appetite and weight gain in CKD cats.

in renal disease are anecdotally successful at 1.88-3.75 mg/0.1 mL gel every other day.

Future availability of the ghrelin agonist capromorelin may also provide additional opportunities to address appetite in CKD cats by targeting the pathophysiology of appetite regulation. There are reports that administration of ghrelin results in increased appetite and energy intake in CKD patients, and a recent study in cats showed that dosing with capromorelin resulted in increased food intake and weight gain (26).

Limiting gastric acidity with the use of H_2 blockers (*e.g.,* famotidine) or proton pump inhibitors (*e.g.,* omeprazole) anecdotally appears to palliate inappetence in some CKD patients. However, these medications have never been evaluated in a clinical trial, despite famotidine being one of the most commonly prescribed medications in CKD cats (2). Recent studies looking at the effect of omeprazole on the gastric pH in normal cats indicates that, at 1 mg/kg twice daily, it is superior to famotidine in its ability to inhibit acid production (27), but again no clinical trials have been performed in CKD cats, and as previously mentioned the degree to which cats with CKD experience hyperacidity is currently unknown. Problematically, proton pump inhibitors have recently been linked to an increased risk of kidney disease in humans (28); the applicability of this finding to veterinary patients is entirely unknown.

Conclusion

In conclusion, poor body condition and muscle mass in CKD cats has important clinical implications and should

be identified and actively managed. Detection of concurrent medical conditions contributing to poor body condition and appetite is a key part of management, and various medications to address appetite, as well as assisted feeding, may greatly benefit the outcome for these patients.

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Body condition scoring in dogs

Kazuya Otsuji, BSc, MSc, PhD (DAg)
Teikyo University of Science, Tokyo, Japan

Dr Otsuji received his PhD from the University of Kyoto (Japan) in 1995 and is currently professor of small animal nutrition in the Teikyo University of Science. His main interests are obesity control and nutritional assessment of small animals.

Akiko Koizumi, BSc
Teikyo University of Science, Tokyo, Japan

Akiko Koizumi received her degree from Teikyo University in 2011 and is currently assistant lecturer in the university's department of animal science. A qualified veterinary nurse, her main interest is developing weight loss programs for dogs using a body fat analyzer.

Body Condition Scoring (BCS)* has for many years been recognized by the American Animal Hospital Association (AAHA) as a preferred screening method for nutritional evaluation of animals (1), resulting in its adoption as a global model (2). BCS is intended to standardize assessment of whether animals are under-, over- or optimal weight, and is based on a scale between 1 to 5 or 1 to 9, where 1 is an emaciated animal and 5 or 9 is grossly overweight.

However, because the method requires observation and palpation of the animal, many veterinarians admit that assessing a patient's body condition score can be rather subjective. Effective and accurate scoring can assist a clinician when advising owners about their dog's nutritional status. This paper describes a simple model developed by the authors as a tool to improve accuracy when performing BCS.

The model is based on five "palpation compartments", as shown in ***Figure 1,*** with each compartment designed to correspond to one of the BCS categories. The most difficult problem encountered when developing the model was to accurately replicate the tactile sensation one has when palpating a dog, and various types and combinations of materials were evaluated to ensure the model was as realistic as possible. A cross section of the model can be seen in ***Figure 2;*** a base layer (using molded plastic to form artificial ribs) is overlaid with polychloroprene sponge and rubber sheeting (to mimic muscle and subcutaneous fat) and covered with artificial fur. Varying layers of sponge and rubber sheets are laminated to replicate different BCS categories.

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Figure 1. The BCS model.

The author's preferred method when assessing a dog for BCS is to start by palpating the animal's dorsal thorax. Next, touch each compartment of the model, and identify the compartment which best matches the perception of palpating the dog; this allows determination of the body condition score. Using a 9-point system, a score of 7, 8 or 9 is classified as obese, 6 is overweight, 3 is underweight and 1 or 2 is very thin; a score of 4 or 5 is ideal. With a 5-point system, 5 is obese, 4 is overweight, 3 is ideal, 2 is underweight and 1 is very thin.

A study was undertaken once the model had been developed to establish if it allowed more precise assessment of BCS (3). With the 5-point system, variability in estimating

*See the Body condition scoring charts in the middle of the issue.

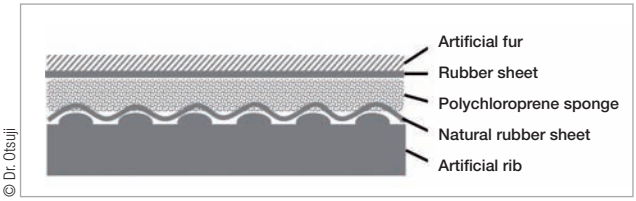

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Figure 2. A schematic cross-section of the model.

the body fat percentage in dogs with body condition scores of 3 and 4 using the model was found to be significantly lower ($P < 0.01$) than when the model was not used, as shown in ***Figure 3;*** the study concluded that the model supported more precise BCS assessment for dogs with scores of 3 and 4. Note that very thin and grossly overweight dogs (*i.e.,* with a score of 1 or 5) were not available for this study, and therefore precise correlation of dogs in these categories when compared to the BCS model requires further investigation.

A second study reviewed the efficacy of the BCS model when used in a clinical situation. 96% of veterinarians surveyed replied that the perceived feeling on palpation between dogs and the BCS model was consistent (4). Some owners can be unwilling to believe a veterinarian's opinion of their dog's nutritional status, but the survey showed that owners tended to accept the result when a clinician explained the dog's body condition score using the model ***(Figure 4).*** The survey also noted veterinarians believed that it was easier to explain a dog's nutritional status with the help of the BCS model, and that many owners liked the model, confirming that it helped them undertake their own assessment of their pet's body condition score.

The authors believe that the BCS model is a novel and supportive tool that can help evaluate and communicate a dog's nutritional status to both owner and veterinarian, and recommend that clinicians consider using a similar model in their animal hospitals.

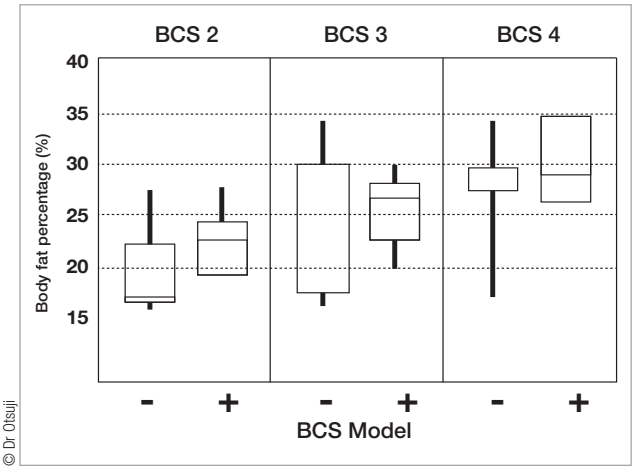

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Figure 3. A box and whiskers plot showing the relationship between estimated body fat percentage and actual body condition score in dogs; the variability in estimating the body fat percentage in dogs with a score of 3 or 4 using the model (+) was found to be significantly lower ($P < 0.01$) than when the same dogs were assessed without the model (-).


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Figure 4. Owners tend to accept the result when a clinician explains the nutritional status of their dog using the BCS model.

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Canine hypothyroidism

David Panciera, DVM, MS, Dipl. ACVIM (SAIM)
Virginia-Maryland College of Veterinary Medicine, USA

After graduating from Oklahoma State University in 1982, Dr Panciera obtained postgraduate training in small animal internal medicine at the University of Missouri and the University of Wisconsin. He has been on the faculty at three veterinary colleges, and was in private practice before joining the faculty at Virginia Tech in 1998, where he is currently Anne Hunter Professor of Small Animal Medicine in the Department of Small Animal Clinical Sciences. Board-certified in small animal internal medicine, his primary research interests are small animal endocrinology and canine hypothyroidism.

Hypothyroidism, the most common endocrine disorder diagnosed in the dog, results in numerous and diverse clinical abnormalities, but assessment of an overweight dog generally includes evaluation for a variety of diseases, including hypothyroidism. A careful history, physical examination, and interpretation of laboratory results are necessary to accurately diagnose this disorder.

Etiology

Primary hypothyroidism, resulting from autoimmune or idiopathic destruction of the thyroid gland, is responsible for the disease in the majority of affected dogs. Autoimmune thyroiditis is an immune-mediated disease that results in gradual destruction of the thyroid gland, leading to eventual hypothyroidism in some dogs (1). It has a genetic component in many breeds, where antibodies to thyroglobulin (the main protein in thyroid follicular colloid) are a marker of thyroiditis. Idiopathic atrophy of the thyroid gland is the other common cause of primary hypothyroidism, accounting for approximately 50% of cases. It is thought to represent an end-stage of autoimmune thyroiditis, but thyroglobulin autoantibodies are not present. The clinical manifestations of primary hypothyroidism are similar regardless of the etiology. Although most common in middle-aged, purebred dogs, the endocrinopathy can occur in any breed and at any age, and whilst the mean age at which dogs are diagnosed with hypothyroidism is 7 years, the disorder is likely present for a considerable time prior to diagnosis in many cases.

KEY POINTS

- **Hypothyroidism is the most common canine endocrine disorder, and any overweight dog should be evaluated for a variety of diseases including this endocrinopathy.**
- **Hypothyroidism can manifest in a myriad of ways, but most dogs have general metabolic and dermatologic abnormalities that are typical of the disease.**
- **Serum T4 concentration is the hormone most frequently used for initial testing of thyroid function; it has a high sensitivity but lower specificity and can be affected by numerous factors.**
- **Elevated T4 in a dog suspected of hypothyroidism should alert the veterinarian to the probability that the dog has autoimmune thyroiditis.**

Clinical findings

While hypothyroidism can manifest in a myriad of ways ***(Table 1),*** most dogs have general metabolic and dermatologic abnormalities that are typical of the disease (2,3). The reduced metabolic rate in hypothyroidism is responsible for the weight gain or obesity present in about 50% of affected dogs, but marked obesity rarely occurs. Lethargy and exercise intolerance are also manifestations of this change, although their gradual onset over a prolonged period often prevents owners from noting these changes until after treatment is instituted.

Dermatologic abnormalities are noted in more than 70% of affected dogs, with alopecia or hypotrichosis being the most common finding (2,3). While the distribution can be bilaterally symmetrical over the trunk – typical of endocrinopathies – hair loss usually begins in areas subject to friction, such as the tail, ventrum, caudal thighs, and dorsal aspect of the nose ***(Figure 1),*** but the pattern of alopecia varies considerably among dogs. Seborrhea is another common cutaneous manifestation of hypothyroidism, and can be dry or oily. Myxedema, or accumulation of intercellular mucin, resulting in non-pitting edema sometimes

Table 1. Clinical manifestations of canine hypothyroidism.

Common signs	Less common signs*
Lethargy	Neuromuscular signs
Weight gain	Female infertility
Alopecia	Myxedema
Pyoderma	Ocular disorders
Seborrhea	Cardiovascular disorders

* Other signs, including gastrointestinal disorders and behavioral problems, have been attributed to hypothyroidism, but these are generally found in poorly documented reports.

results in the appearance of thickened skin and a "tragic" facial expression ***(Figure 2).*** Hypothyroid dogs are predisposed to develop otitis externa, bacterial pyoderma, yeast infections, and generalized demodicosis.

Because thyroid hormones affect nearly all body systems, numerous other manifestations of hypothyroidism occur, albeit less commonly than those affecting the skin and general metabolism. Peripheral neuropathies, both generalized and localized, occur occasionally in hypothyroid dogs. Polyneuropathy in such cases is indistinguishable from that due to other causes, with weakness, ataxia, proprioceptive deficits, hyporeflexia, and muscle atrophy developing (4). Focal neuropathies have been reported to affect the facial and/or vestibulocochlear nerves, with subsequent loss of motor function to facial muscles, ataxia, nystagmus, head tilt, and circling. Megaesophagus, secondary to neuropathy or myopathy, is only rarely due to hypothyroidism. In addition to neuromuscular dysfunction, hypothyroidism can result in central nervous system dysfunction. The most common signs are due to lesions in the vestibulocerebellum or the myelencephalon, causing central vestibular signs, paresis, ataxia, hyperreflexia, and altered consciousness (4). Many dogs with central or peripheral neurologic complications do not have dermatologic abnormalities classically typical of hypothyroidism, and in these cases hypercholesterolemia can be an important finding that may trigger evaluation of thyroid function. Aggression and other behavioral changes have been reported to occur secondary to hypothyroidism, but there is little evidence that these problems are related.

Bradycardia, weak pulses, and reduced myocardial contractility are the cardiac manifestations of hypothyroidism (2,4). These abnormalities infrequently have clinically relevant consequences, but their presence can increase the suspicion of hypothyroidism. The reduced cardiac output due to hypothyroidism decreases the

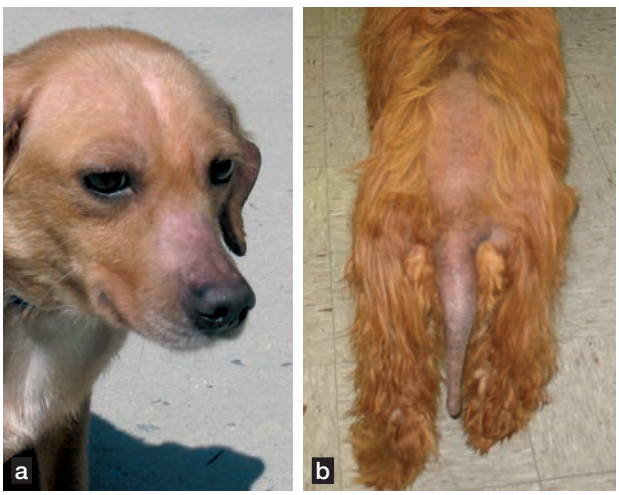

Figure 1. (a) Generalized hair loss and alopecia on the bridge of the nose in a dog with chronic hypothyroidism. **(b)** An 8-year-old Golden Retriever with alopecia of the tail and dorsum secondary to hypothyroidism.

Figure 2. A 5-year-old mixed breed dog with severe hypothyroidism and myxedema. Note the thickened, non-pitting edema of the facial tissues resulting in a "tragic" facial expression.

glomerular filtration rate by approximately 30%, but does not result in azotemia unless there is concurrent renal dysfunction (5).

The effects of hypothyroidism on reproduction are limited to affected females. Infertility, decreased body weight of pups, and increased periparturient mortality can occur in bitches if hypothyroidism has been present for more than six months (6). Ocular abnormalities, if present in hypothyroid dogs, are either secondary to hyperlipidemia or unrelated to the endocrinopathy. Insulin resistance is induced by hypothyroidism, so it should be considered a differential diagnosis in dogs with diabetes mellitus that are difficult to regulate or require high doses of insulin (7).

Myxedema stupor or coma is a rare manifestation of hypothyroidism and represents decompensation of prolonged, untreated hypothyroidism that is usually triggered by a precipitating event (8). In addition to classical signs of hypothyroidism, dogs with myxedema coma have altered mentation, hypothermia, bradycardia, hypoventilation, hypercholesterolemia, hypertriglyceridemia, and anemia. Treatment includes passive (rather than active) warming, judicious fluid replacement, a large loading dose of levothyroxine (administered IV if possible), and management of any concurrent illness such as infection or congestive heart failure.

Because of the systemic nature of hypothyroidism and the effects of non-thyroidal illness on thyroid function tests, it is recommended that all dogs suspected of hypothyroidism have a complete blood count, serum biochemistry and urinalysis evaluated. Findings on routine laboratory testing include hypercholesterolemia in about 75% of cases and mild non-regenerative anemia in 25% (2,3). Once clinical suspicion of hypothyroidism is determined, specific thyroid function testing should be performed.

Diagnosis of hypothyroidism

Evaluation of thyroid function should be reserved for dogs with clinical findings suggestive of hypothyroidism. Measurement of serum total thyroxine (T4) concentration when clinical signs of hypothyroidism are absent (such as during routine preventive health evaluations) is not recommended because of the limited specificity of the test. Many factors, including age, breed, body condition, body size, reproductive stage, exercise, non-thyroidal illness, and drugs can affect thyroid hormone concentrations. In addition, serum thyroid hormones fluctuate during the day, and may be randomly below their respective reference interval in euthyroid dogs during sampling ***(Figure 3).*** Thyroid function tests must therefore be interpreted in light of clinical findings.

Serum T4 concentration is the hormone most frequently used for initial testing of thyroid function because it is widely available, relatively inexpensive, and results can be obtained quickly (9). While it has a high sensitivity, the specificity is lower and can be affected by numerous factors commonly encountered in practice. A low serum T4 concentration is found in 90% of hypothyroid dogs, while the remaining 10% have low normal T4 (10, 11). This makes it a good screening test for hypothyroidism, since a serum T4 concentration that is well within the reference interval rules out a diagnosis of hypothyroidism when dogs with autoantibodies to T4 are excluded. The specificity of T4 is 75-80% when testing dogs suspected of hypothyroidism, but it is even lower when evaluating dogs with non-thyroidal illness or those receiving certain drugs, as discussed below (9).

Serum concentration of free T4 (fT4) is the most sensitive and specific single test for diagnosis of hypothyroidism. However, it is more costly, less available, and has a longer turn-around time than serum T4. In order for the advantages of measuring fT4 over T4 to occur, the hormone must be analyzed using an equilibrium dialysis assay (9). Thyroxine is highly protein-bound in the plasma, but only the portion that is unbound or “free” (approximately 0.1%) leaves the circulation to be transported into cells where its activity is asserted. Because some drugs, non-thyroidal illness, and thyroid hormone autoantibodies can affect protein binding and subsequently the measured concentrations of T4, accurate assessment of fT4 better reflects thyroid function than T4. These factors affect fT4 less frequently than T4, resulting in a sensitivity of 98% and a specificity of 93% (9,10). Another advantage of measuring fT4 using an equilibrium dialysis assay is that the test is not affected by autoantibodies to T4 (12).

Because hypothyroid dogs almost invariably have primary hypothyroidism, reduced thyroid hormone secretion results in less negative feedback on the pituitary gland, resulting in increased secretion of thyroid stimulating hormone (TSH). This reciprocal relationship can be exploited when a concurrent low T4 or fT4 and elevated TSH are found, as the sensitivity and specificity of this combination approaches 100%. Unfortunately, serum TSH concentration is elevated in only 65-75% of

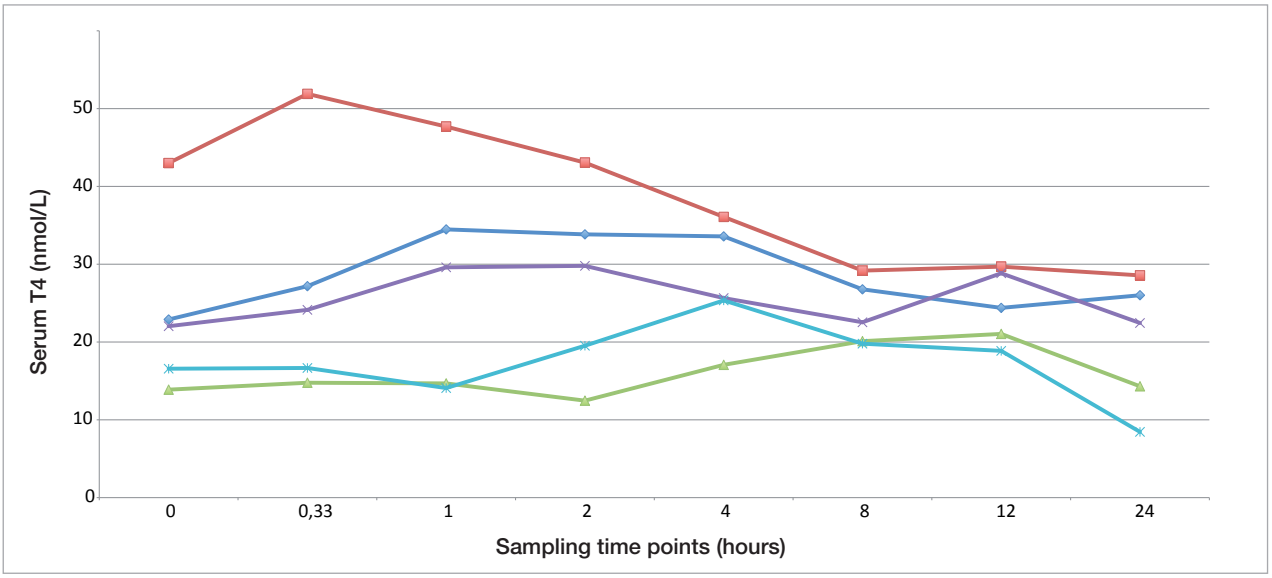

Figure 3. Serum T4 concentrations in five healthy, euthyroid dogs measured 8 times during a 24-hour period. Marked fluctuations occurred, with two dogs having T4 concentrations below the reference interval (13-55 nmol/L) at one or more sampling time points.

hypothyroid dogs, so the test has low sensitivity and it must be interpreted with a concurrent T4 or fT4 (9-11).

Recommendations for diagnosis of hypothyroidism are to measure either serum T4 or fT4 (by equilibrium dialysis) and interpret results in light of clinical findings. If there is a high degree of suspicion of hypothyroidism and no concurrent illness, a T4 or fT4 below the reference interval is sufficient to make a tentative diagnosis and initiate levothyroxine supplementation. Because fT4 is more accurate, any discrepancy between it and T4 should be interpreted using the fT4 concentration rather than the T4. Measuring TSH in addition to T4 and/or fT4 has the advantage that finding an elevated TSH with a concurrent low T4 or fT4 confirms the diagnosis. The accuracy of thyroid function tests is affected by the population being tested, so selection of patients with clinical findings supportive of hypothyroidism and without confounding factors is essential.

Interpretation of thyroid function tests becomes more complicated in numerous situations. The effect of breed on serum thyroid hormone concentrations is most important in sight hounds (13), where values are frequently below the reference interval established for other breeds; serum fT4 is similarly, but less frequently, affected. Concurrent measurement of serum fT4 and TSH concentrations is probably the best approach to assessing thyroid function in sight hounds. Other breeds may have specific reference ranges as well. Body condition can alter thyroid function tests, as obese dogs have higher serum T4 than lean dogs.

Non-thyroidal illness (NTI) is a syndrome whereby thyroid function tests are altered by illness not directly affecting the thyroid gland. More severe illness causes more substantial alterations of thyroid function tests compared with mild illness. In this setting, serum T4 concentration is frequently below the reference interval and it is not a reliable test of thyroid function (9,14). Serum fT4 is less frequently decreased, and serum TSH is rarely affected, so the combination of fT4 and TSH is suggested when it is critical to assess thyroid function during the illness. The most reliable – albeit sometimes impractical – way to ensure valid assessment of thyroid function is to test only after resolution of the illness. However, finding normal serum T4 or fT4 reliably rules out hypothyroidism.

Thyroid function tests can be altered by numerous drugs, including sulfonamides, glucocorticoids, phenobarbital, clomipramine, and some non-steroidal anti-inflammatory drugs (NSAIDs) (9,15). The most marked effect occurs with administration of trimethoprim-sulfonamide combinations, where biochemical hypothyroidism occurs after two weeks therapy, and clinical signs can result with more prolonged treatment. The effect is reversible within three weeks in most cases. Glucocorticoids, depending

on the dosage, duration, and specific drug, can suppress serum T4 (and in some cases fT4) concentrations. Phenobarbital causes a gradual reduction of thyroid hormones and can result in serum T4 and fT4 below and TSH above their respective reference intervals. Whether this biochemical hypothyroidism results in clinical signs has not been determined, but the effect is reversible after discontinuing phenobarbital. Clomipramine has a more modest effect, decreasing serum T4 and fT4 by about 30%, and the effect of NSAIDs is minor in most cases.

Occasionally, it is necessary to assess thyroid function in a dog that is currently receiving levothyroxine in order to confirm a diagnosis of hypothyroidism if it was not definitively diagnosed prior to treatment. Because hormone supplementation will suppress the hypothalamic-pituitary-thyroid axis, TSH and thyroid hormone secretion can be reduced for some period of time. It has recently been shown that thyroid function can be accurately assessed as early as one week after withdrawal of levothyroxine in euthyroid dogs treated for 16 weeks with once-daily supplementation (16).

Although not tests of thyroid function, markers of autoimmune thyroid disease can be useful when evaluating a dog for hypothyroidism. Dogs with autoimmune thyroiditis develop antibodies to thyroglobulin; whilst a positive thyroglobulin autoantibody test establishes a diagnosis of thyroiditis, these antibodies can be present in both euthyroid and hypothyroid dogs. However, euthyroid dogs with thyroglobulin autoantibodies are predisposed to develop hypothyroidism and should be monitored for clinical signs of hypothyroidism, with thyroid function tested at least annually. More importantly, some dogs with autoimmune thyroiditis develop antibodies against thyroid hormones; this interferes with laboratory measurement of serum T4 concentrations, leading to an increase in the reported T4 concentration, but without an *in vivo* effect on thyroid function. If the autoantibody titer is high, the serum T4 measurement will be above the reference range. Finding elevated T4 in a dog suspected of hypothyroidism should alert the veterinarian to the probability that the dog has autoimmune thyroiditis. If the antibody titer is lower, the measured T4 can be within the reference range, as has been reported in up to 10% of hypothyroid dogs, which can confound diagnostic testing (1,12). If autoantibodies to T4 are suspected, serum fT4 should be measured by equilibrium dialysis, as it is the only method that can determine if a patient with these antibodies is hypothyroid (12). Ultimately, diagnosis of hypothyroidism is confirmed by appropriate response to thyroid hormone supplementation. If treatment is not accompanied by the anticipated clinical improvement, the diagnosis should be reassessed.

Treatment recommendations

Hypothyroidism is managed by administration of synthetic levothyroxine, with a suggested initial dosage of 0.022 mg/kg orally once daily (17,18). Dosing should occur at least 30 minutes before feeding, as bioavailability is reduced by food. The initial dose should be reduced to approximately 0.005 mg/kg in dogs with diabetes mellitus, hypoadrenocorticism, renal failure, or heart failure. Clinical response, serum T4, and effects on concurrent illness should be assessed every two weeks, with an incremental increase in the dose when appropriate. Insulin resistance resolves rapidly after initiating supplementation, so assessment of glycemic control and adjustments in insulin dose may be necessary.

Increased metabolic rate occurs within the first few days of levothyroxine supplementation, and is reflected clinically in most dogs by an improvement in activity and mental alertness within the first 1-2 weeks of treatment. Weight loss also occurs early in treatment. When present, neurologic abnormalities generally improve within the first few days of initiating treatment, with resolution of most signs typically occurring within 4-6 weeks. The dermatologic abnormalities, particularly alopecia, may require several months to resolve.

Therapeutic monitoring is typically performed after 6-8 weeks of treatment to assess the clinical response and measure serum T4. The peak serum concentration of T4 occurs 4-6 hours after levothyroxine administration, with a recommended target peak serum T4 concentration at around 40-70 nmol/L. If a good clinical response to treatment is documented and the post-pill serum T4 is within the recommended range, treatment is continued and the dog should be rechecked in about 6 months, and annually thereafter. If the serum T4 concentration is above the recommended therapeutic range, the dose should be lowered, even if signs of hyperthyroidism are absent. If the clinical response is inadequate and the serum T4 is below the recommended concentration, the dosage should be increased by up to 25% and the dog rechecked in 2-4 weeks. If the post-treatment T4 is in the recommended range and the response is inadequate, one has to determine if compliance has been acceptable, if a concurrent illness is present, or the diagnosis of hypothyroidism was in error. A small proportion

of dogs will not respond completely to once-daily levothyroxine and require twice-daily administration for control of disease.

Hyperthyroidism is essentially the only potential side effect of treatment. Signs are similar to those found in hyperthyroid cats, with weight loss, polyuria, polydipsia, polyphagia, tachycardia, hyperactivity, panting, and vomiting being most common. Diagnosis is made based on clinical signs and an elevated serum T4 concentration. Most of the signs related to hyperthyroidism should resolve rapidly after discontinuing treatment for 2-3 days, and levothyroxine can then be reinstituted at a 25% reduction in dosage.

The prognosis is excellent, as complete recovery is anticipated with correct treatment, with the possible exception of residual head tilt in some dogs with neurologic complications. Because there is a heritable component to hypothyroidism, owners of affected dogs should be counseled prior to breeding.

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A SHORT GUIDE TO...

Radioactive iodine treatment for feline hyperthyroidism

Elsa Edery, DEDV, CEAV (IntMed), MSc (ClinOnc), MRCVS
Pride Veterinary Centre, Derby, UK

After qualifying from Maisons-Alfort Veterinary School, Dr Edery worked in first opinion practice, obtaining her French Certificate in Small Animal Internal Medicine in 2006. She was awarded a Master of Science in human clinical oncology in 2015 and is currently enrolled in an ECVIM-approved residency at a UK specialist veterinary center. She enjoys all aspect of internal medicine and has a particular interest in oncology.

Hyperthyroidism is the most common feline endocrinopathy, affecting around 10% of older cats. It is generally caused by benign changes (adenomatous hyperplasia or follicular cell adenoma) to one (30%) or both (70%) thyroid glands, although a small percentage (1-3%) of affected cats are diagnosed with a malignant thyroid carcinoma.

Following a diagnosis of hyperthyroidism, four treatment options are currently available to the clinician: administration of an anti-thyroid hormone drug; nutritional management with an iodine-deficient diet; surgical thyroidectomy; and radioactive iodine administration. Each method has both advantages and disadvantages, and these should be considered when discussing the best treatment option for a particular patient. Factors such as the animal's age, the presence of concurrent illness, the availability of treatment facilities, the owner's preferences and finances, and whether the cat lives indoors or outdoors can all greatly influence the choice of treatment. Radioactive iodine therapy is both simple and effective, curing 95-98% of hyperthyroid cats, and is currently considered by most authorities to be the treatment of choice for the condition.

Principles of radioactive iodine treatment

Like ordinary iodine, radioactive iodine concentrates in the thyroid glands, particularly in hyperplastic adenomatous tissue, causing destruction of the follicular cells. The remaining normal thyroid tissue absorbs less radioactive iodine as it tends to be atrophic and non-functional (due to reduced levels of TSH – thyroid-stimulating hormone – from the pituitary gland) and is spared destruction.

Indications for radioactive iodine treatment

Radioactive iodine is particularly indicated in cats with bilateral thyroid involvement or where there is ectopic intrathoracic thyroid tissue. Radioactive iodine is usually given subcutaneously; this enables easy dosing (being just as effective as oral or intravenous routes) and is safer for the personnel administering the therapy ***(Figure 1 and 2).*** In addition, it does not require general anesthesia, which may be contraindicated in elderly or frail patients. Because a cat will be radioactive for some time after treatment, special facilities for hospitalization, and strict adherence to local radiation protection regulations, are essential.

A recent cross-sectional study (1) in a large cohort of hyperthyroid cats treated medically showed that the prevalence of severe disease (defined as larger volume of thyroid nodules, multiple nodules, or suspected thyroid carcinoma) increased with the disease duration. This suggests that hyperthyroidism is a progressive condition which may not be controlled by reversible therapeutic modalities, with a possible increased risk of malignant transformation of adenomas in the long term. A definitive treatment modality (*i.e.,* thyroidectomy or radioactive iodine) is to be preferred when available, particularly in younger cats.

Treatment protocol

Pre-treatment screening should be performed to identify any concurrent illness which could preclude radioactive iodine treatment; patients with unstable cardiovascular, gastrointestinal, renal, endocrine or neurological disease may not be suitable candidates. During the hospitalization

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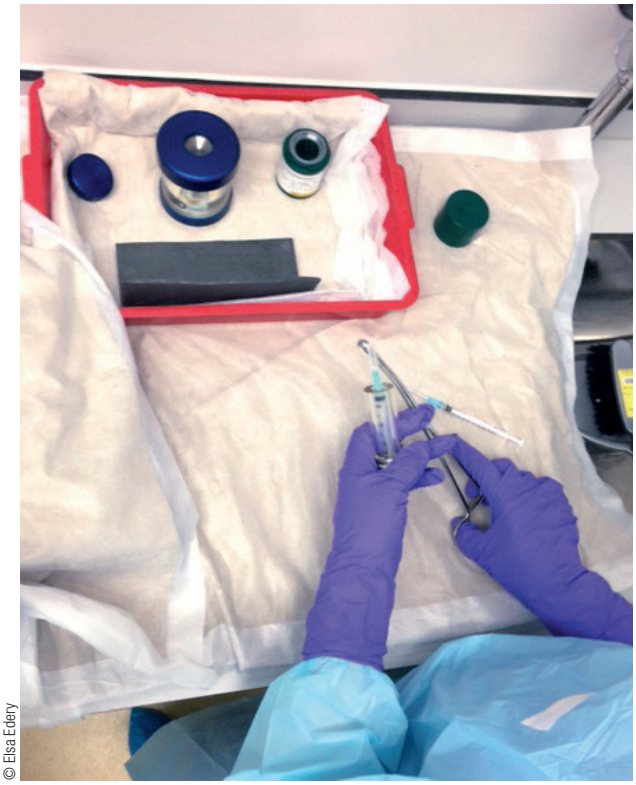

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Figure 1. Preparation of the radioactive iodine injection. Note the use of personnel protective equipment and the lead shields around the containers.

period, contact with the radioactive patient is reduced to the absolute minimum, and the cat cannot be moved out of isolation until radioactivity levels have fallen below a certain threshold. This precludes any therapeutic intervention for other illnesses during hospitalization.

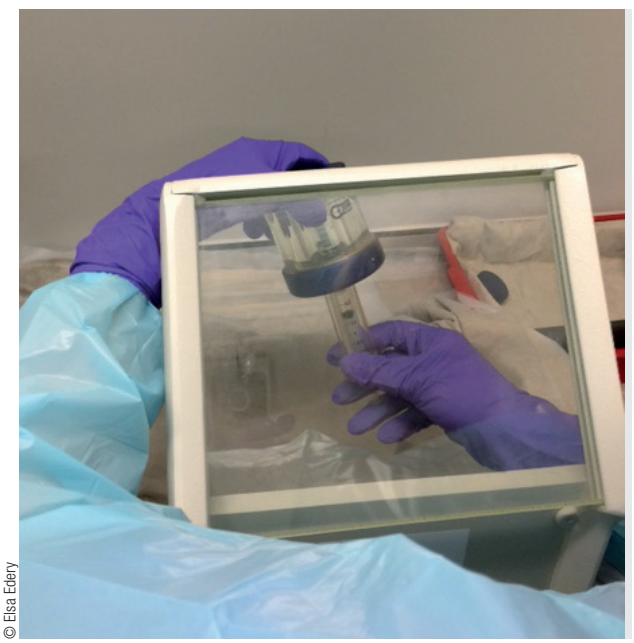
A minimal database is essential, including a complete blood count, serum biochemistry, urinalysis and blood pressure measurement. A pre-treatment total thyroxine (TT4) concentration is required to calculate the radioactive iodine dose. Thoracic radiography and cardiac ultrasonography are recommended if there is a strong suspicion of heart disease. Thyroid scintigraphy, although ideal, is not a pre-requisite before radioactive iodine treatment, although it may assist in deciding which treatment modality is most appropriate for a particular patient.

Most cats will have had either an anti-thyroid drug or an iodine-deficient diet prior to referral for radioactive iodine, and renal function should have been assessed once euthyroidism is achieved. In cats that have been treated with an anti-thyroid medication for more than two months, the drug should be stopped 1-2 weeks before radioactive iodine treatment and serum TT4 re-measured. This also allows the hyperthyroid state to re-establish, ensuring minimal uptake of radioactive iodine by normal thyroid tissue and better uptake by the hyperplastic cells.

If an iodine-deficient diet has been fed, there is a small risk that the thyroid tissue will be more sensitive to the radioactive iodine with a subsequent risk of iatrogenic hypothyroidism, and at present it is recommended that the diet is discontinued two weeks prior to radioactive iodine treatment.

The cat may be lightly sedated to ensure safety of personnel whilst administering the treatment. The aim is to administer enough radioactive iodine to restore euthyroidism without causing post-treatment hypothyroidism; whilst an optimal method to calculate the required dose is still controversial, many centers use a clinical scoring system which considers the severity of the clinical signs, the size of the thyroid goiter and the pre-treatment TT4 level. Note that, although radioactive iodine is the method of choice for malignant thyroid carcinoma, as it targets both the primary tumor and any potential metastases, significantly higher doses of radioactive iodine are generally necessary for successful treatment.

Figure 2. A protective lead screen allows the radioactive iodine to be prepared for injection before being injected into the sedated cat.



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The length of hospitalization varies according to the radioactive iodine dose administered and local radiation protection regulations. A cat will generally continue to excrete a small amount of radioactive iodine in body fluids (saliva, urine, feces) and will be slightly radioactive for 2-4 weeks following discharge ***(Figure 3).*** Regulations will dictate what precautions are required to reduce human exposure to radiation and can vary from country to country; however, general guidelines as to how the owner should care for their cat in the first few weeks post-discharge are included in ***Box 1.***

Treatment outcome and possible complications

A single radioactive iodine injection is successful in 85-95% of cats. If hyperthyroidism persists for more than 3 months post-treatment, this is usually due to an insufficient dose of radioactive iodine, which is more likely to occur in cats with severe hyperthyroidism (as defined above), large thyroid tumors, or where there is markedly elevated TT4. Occasionally, despite adequate dosing, poor iodine uptake or rapid iodine turnover can result in treatment failure, and in such cases a second injection at a higher dose is recommended.

Figure 3. A Geiger counter can be utilized to ensure radioactivity levels are low enough to allow a cat to be discharged home; it also allows monitoring of hospital personnel.

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Box 1. Precautions to follow post-discharge.

- The cat must be kept indoors.
- Contact time should be limited to a few minutes per person each day. Face to face contact, sitting on laps, and sleeping on the owner's bed is to be discouraged.
- Contact with pregnant women and children should be avoided.
- Urine and feces should be handled with gloves; the use of flushable litter or litter tray liners is recommended, with double bagging.
- Strict hand hygiene is recommended after handling urine and feces and after any contact with the cat.

Unmasking or worsening of pre-existing chronic kidney disease (CKD) occurs in 25% of treated cats, but this is not specific to the radioactive iodine therapy. As noted above, it is recommended that cats diagnosed with hyperthyroidism are first managed with a reversible modality (*i.e.,* drugs or a low iodine diet) with follow-up of biochemical parameters and urine analysis, as restoration of euthyroidism can lead to unmasking of CKD. Development of azotemia and overt clinical signs of renal failure require appropriate therapy, but do not necessarily preclude treatment of the hyperthyroidism.

Transient hypothyroidism commonly develops after treatment, but this resolves when pituitary TSH secretion resumes, although this may take a few months. Most cats regain the ability to secrete thyroid hormones, usually within 3 months of treatment, although some may take longer. Transient hypothyroidism does not require L-thyroxine supplementation unless azotemia develops or worsens.

Radioactive iodine treatment can cause destruction of normal thyroid tissue, and may in some cases result in permanent iatrogenic hypothyroidism (2). This has been reported to be as high as 79%, although most studies found the incidence to be less than 9%; the disparity may be due to differing timeframes for follow-up, as well as variation in the dose of radioactive iodine administered. Since iatrogenic hypothyroidism has been shown to contribute to the development of azotemia and reduced survival times (3), it is essential to calculate an individual dose of radio-active iodine for each patient to minimize this risk, and all cases of post-therapy hypothyroidism should be treated as necessary.

Long-term systemic effects of radioactive iodine therapy (carcinogenesis and genetic damage leading to occurrence of secondary cancers) are deemed negligible, as in human patients.

Follow-up

As normalization of TT4 occurs within four weeks of treatment in 85% of cats and 3 months in 95% of cats, clinical re-examination and routine bloodwork (including TT4) at one and three months post-discharge are recommended. This allows identification of patients with persistent hyperthyroidism or developing hypothyroidism. In the few cats where a longer recovery time is necessary, recheck at 6 months is recommended. Although recurrence of hyperthyroidism is rare (< 5% of cases), relapse times of 3 years or more have been reported (2), and annual measurement of serum T4 may be warranted in cats following treatment.

Acknowledgment: The author would like to thank Francis Boyer and Caroline Kisielewicz for sharing materials for this article.

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Further reading

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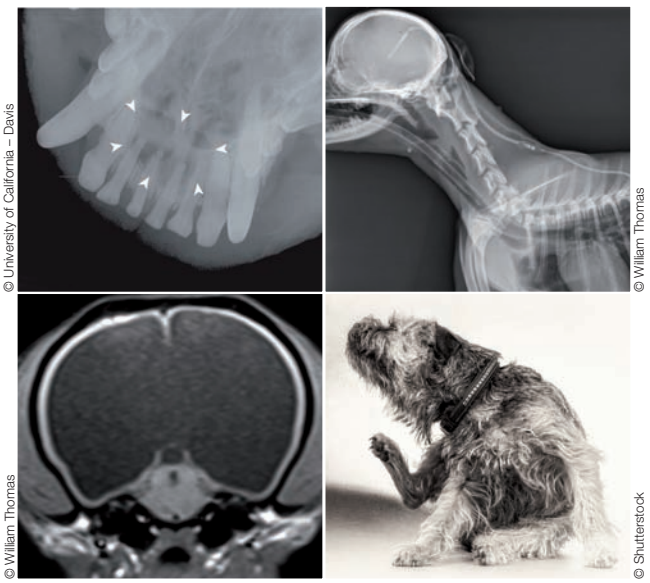
VETERINARY focus

The worldwide journal for the companion animal veterinarian



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IN OUR NEXT ISSUE...

In our next issue of *Veterinary Focus*, we will look at various problems commonly encountered in smaller dog breeds.

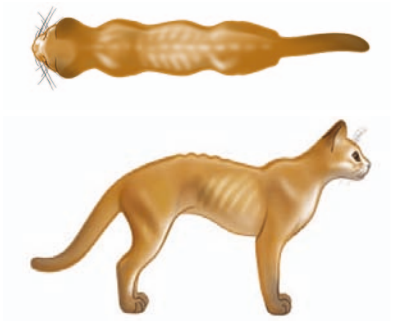
- **Perthes disease**
  Darryl Millis, USA
- **Ear, nose and throat problems**
  Gert ter Haar, Netherlands
- **The "Small Dog Trend": the impact of size on pet health**
  Jamie Freyer, USA
- **Canine hydrocephalus**
  William Thomas, USA
- **Syringomyelia in dogs**
  Ludovic Pelligand, UK
- **Using a texturometer to develop canned food**
  Hervé Réhault, France
- **Dental disease in small dogs**
  Jenna Winer and Frank Verstraete, USA
- **Paroxysmal dyskinesia in Border Terriers**
  Mark Lowrie, UK

ROYAL CANIN

CATS

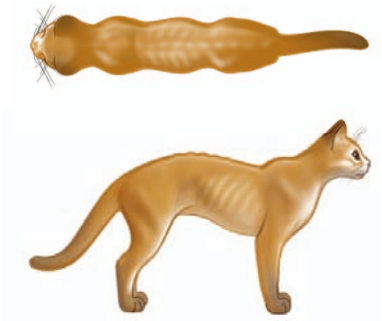
Body Condition Scoring

TOO THIN

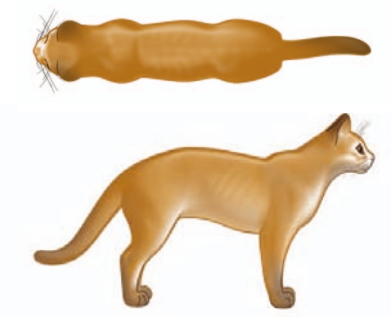


1

- Ribs, spine and pelvic bones easily visible on shorthaired cats
- Very narrow waist
- Small amount of muscle
- No palpable fat on the rib cage
- Severe abdominal tuck

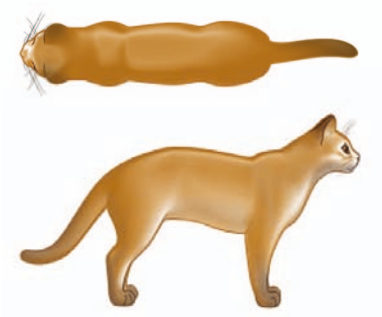


IDEAL

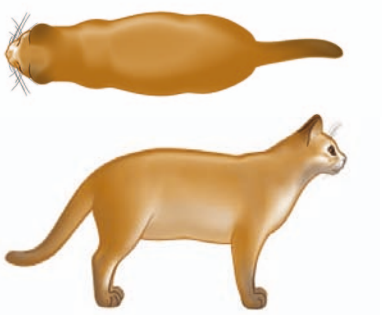


4

- Ribs not visible but are easily palpable
- Obvious waist
- Minimal amount of abdominal fat

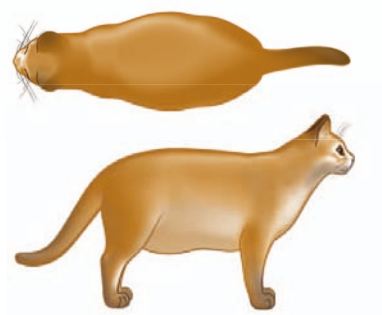


TOO HEAVY



7

- Ribs difficult to palpate under the fat
- Waist barely visible
- No abdominal tuck
- Rounding of abdomen with moderate abdominal pad



ROYAL CANIN®

2

- Ribs easily visible on shorthaired cats
- Very narrow waist
- Loss of muscle mass
- No palpable fat on the rib cage
- Very pronounced abdominal tuck

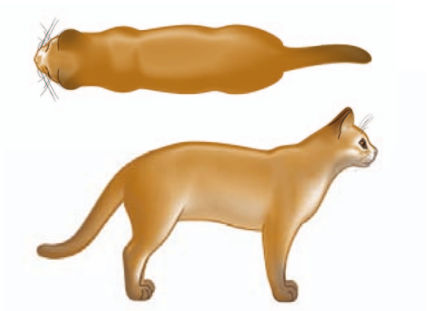
3

- Ribs visible on shorthaired cats
- Obvious waist
- Very small amount of abdominal fat
- Marked abdominal tuck

OVERWEIGHT

5

- Well proportioned
- Ribs not visible but are easily palpable
- Obvious waist
- Small amount of abdominal fat
- Slight abdominal tuck

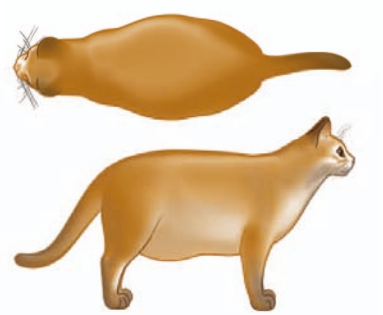


6

- Ribs not visible but palpable
- Waist not clearly defined when seen from above
- Very slight abdominal tuck

8

- Ribs not palpable under the fat
- Waist not visible
- Slight abdominal distension



9

- Ribs not palpable under a thick layer of fat
- Waist absent
- Obvious abdominal distension
- Extensive abdominal fat deposits

DOGS

Body Condition Scoring

TOO THIN

1

- Ribs, lumbar vertebrae, pelvic bones and all bony prominences evident from a distance
- No discernible body fat
- Obvious loss of muscle mass

IDEAL

4

- Ribs easily palpable with minimal fat covering
- Waist easily noted when viewed from above
- Abdominal tuck evident

TOO HEAVY

7

- Ribs palpable with difficulty, heavy fat cover
- Noticeable fat deposits over lumbar area and base of tail
- Waist absent or barely visible
- Abdominal tuck may be absent

ROYAL CANIN®

2

- Ribs, lumbar vertebrae, and pelvic bones easily visible
- No palpable fat
- Some bony prominences visible from a distance
- Minimal loss of muscle mass

3

- Ribs easily palpable and may be visible with no palpable fat
- Tops of lumbar vertebrae visible, pelvic bones becoming prominent
- Obvious waist and abdominal tuck

OVERWEIGHT

5

- Ribs palpable without excess fat covering
- Waist observed behind ribs when viewed from above
- Abdomen tucked up when viewed from side

6

- Ribs palpable with slight excess of fat covering
- Waist is discernible when viewed from above but is not prominent
- Abdominal tuck apparent

8

- Ribs not palpable under very heavy fat cover or palpable only with significant pressure
- Heavy fat deposits over lumbar area and base of tail
- Waist absent
- No abdominal tuck
- Obvious abdominal distension may be present

9

- Massive fat deposits over thorax, spine, and base of tail
- Waist and abdominal tuck absent
- Fat deposits on neck and limbs
- Obvious abdominal distension

 Adapted from Laflamme D.Development and validation of a body condition score system for dogs. *Canine practice* vol 22,n°4, 1997

Canine diabetes mellitus

Sara Corradini, DVM, PhD
Department of Veterinary Medical Sciences, University of Bologna, Italy

Dr Corradini graduated from the University of Bologna in 2009 and completed an endocrinology doctorate in 2014. She is currently a research associate at the University of Bologna and is following an ECVIM-CA residency in Internal Medicine.

Federico Fracassi, DVM, PhD, Dipl. ECVIM-CA
Department of Veterinary Medical Sciences, University of Bologna, Italy

Dr Fracassi graduated with honors from the University of Bologna and gained his research doctorate in 2005. He was awarded his ECVIM diploma in 2012 and is now Associate Professor at the University of Bologna. He is currently president of the Italian Society of Veterinary Internal Medicine and vice-president of the European Society of Veterinary Endocrinology. His main field of research is internal medicine, and in particular small animal endocrinology.

Introduction

It is almost a century since it was discovered that diabetes mellitus (DM) is a disease process secondary to insulin deficiency, with the original researchers successfully treating dogs that had undergone pancreatectomy using insulin derived from the pancreas of healthy dogs (1).

KEY POINTS

- **Diabetes mellitus (DM) in dogs is characterized by persistent fasting hyperglycemia secondary to hypoinsulinemia.**
- **The most common form in dogs is DM type 1, which requires lifelong administration of insulin.**
- **Contrary to the situation in humans and cats, DM type 2 is rare in dogs, and the relationship between canine DM and obesity is present but less evident.**
- **The objectives of therapy include remission of the clinical symptoms, good glycemic control, and avoidance of hypoglycemia and ketoacidosis.**
- **Measurement of blood glucose levels using a portable glucometer is essential for both short- and long-term monitoring of diabetic dogs; new technologies now offer the option of continuous monitoring.**

DM is now recognized as one of the most common endocrinopathies in dogs, with an estimated prevalence of between 0.3-1.33% of the canine population, although geographical variations exist (2,3).

There is currently no universally accepted classification for DM in veterinary medicine, although the terms insulin-dependent diabetes mellitus (IDDM) and non-insulin-dependent diabetes mellitus (NIDDM) have generally been replaced by diabetes type 1 and diabetes type 2, respectively.

Until a few years ago, canine DM was thought to result from destruction of the pancreatic beta cells by an immune-mediated mechanism. Recent studies have cast significant doubt on this theory, and it is now considered to have a multifactorial pathogenesis, with additional mechanisms involved in the development of the disease (4-7). It is clear that there is a genetic bias for canine DM, with certain breeds predisposed to the disease (including Yorkshire Terriers, Tibetan Terriers, Border Terriers, Cairn Terriers, Samoyeds, Keeshounds, Miniature Poodles and English Setters) and other breeds (*e.g.,* Boxers and German Shepherds) apparently particularly resistant. A familial trend for DM has also been described in several breeds (2,8,9).

DM type 1

The most common form in dogs is DM type 1. In human medicine, DM type 1 is usually diagnosed in youngsters, but in dogs the onset time is later, with middle-aged and older animals affected (10) and it has been hypothesized that canine diabetes is similar to the LADA (latent

autoimmune diabetes of adults) form in humans. Autoimmune mechanisms, in combination with genetic, environmental, pharmacological and insulin-resistance factors, are involved in the onset and progression of canine DM, with loss of beta cell functionality and consequent permanent hypoinsulinemia.

DM type 2

DM type 2 does not seem to occur in dogs (11) but it is the form most commonly found in humans (approximately 90% of cases). Diabetes mellitus in cats appears to be very similar to human DM type 2, which is linked to impaired insulin secretion or insulin resistance and is often associated with obesity. In human patients, injectable insulin is often not required; dietary modification, increased physical exercise and oral hypoglycemic drugs are usually sufficient to control the disease. However, if there is severe insulin resistance and/or significant damage to the beta cells, it may be necessary to resort to insulin.

DM and obesity in dogs

Unlike the situation in cats and humans, where a strict correlation has been demonstrated between obesity and the development of DM, in dogs this relationship seems less clear. Insulin resistance induced by obesity has been reported in dogs (12), but a hyperglycemic state is not usually observed in these animals ***(Figure 1)*** (13); this is presumably linked to the fact that the resulting over-secretion of insulin (hyperinsulinemia) successfully offsets insulin resistance, thus maintaining euglycemia (14). In humans, hormones produced by adipose tissue have been studied extensively and are used as biomarkers in an attempt to indicate the development of DM; in particular, reduced levels of adiponectin can predict the onset of DM type 2. In dogs, an increase in visceral fat deposits is not in itself sufficient to cause insulin resistance (15) and this supports the idea that accumulation of body fat does not seem to be the only factor in determining insulin resistance. Unlike the human situation, the levels of adiponectin in obese dogs are not lower than those found in lean dogs, and do not appear to be associated with reduced-insulin sensitivity (15,16). Although the incidence of canine obesity has increased within the last decade, studies that demonstrate a concomitant increase in the incidence of DM are lacking.

Other types of DM

Diestrus and pregnancy both lead to significant increases in progesteronemia. Elevated levels of endogenous progesterone (or the exogenous administration of progestins) stimulate a pronounced rise in the amount of growth hormone (GH) secreted from a bitch's mammary tissue. GH is strongly diabetogenic, inducing insulin resistance which may lead to, or predispose to, the development of DM (6). Progesterone-induced DM in dogs is one of the few forms where complete remission can be achieved, especially if it is recognized in the early stages and treated via sterilization of the bitch. The inability to correct insulin resistance often leads to a gradual loss of beta cells, with a greater probability of developing permanent DM. Animals that show remission of DM at the end of diestrus are very likely to develop the disease again during a subsequent cycle (17) and neutering should be promptly arranged for all unsterilized bitches that develop DM.

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Figure 1. An obese (BCS 9/9) six-year-old, unsterilized female Cocker Spaniel. Insulin resistance has been reported in such animals, but a hyperglycemic state is not usually observed.

Administration of diabetogenic drugs such as corticosteroids, which induce insulin resistance and promote gluconeogenesis, can also predispose to the development of DM. By the same mechanism, dogs with Cushing's syndrome may develop hyperglycemia, which can sometimes progress to full DM.

Pathophysiology

DM is a disease characterized by persistent fasting hyperglycemia (glycemia > 144 mg/dL or 8-12 mmol/L) resulting from absolute or relative insulin deficiency (6). Insulin deficiency therefore leads to reduced use of glucose, amino acids and fatty acids on a peripheral level and an increase in hepatic glycogenolysis and gluconeogenesis, with a resulting hyperglycemia. The increase in blood glucose levels above the threshold values for

reabsorption by the proximal convoluted tubule (180-220 mg/dL or 10-12 mmol/L) leads to glycosuria, which results in osmotic polyuria and a compensatory polydipsia. The lack of insulin also leads to reduced use of glucose by body tissues with subsequent weight loss, and the resulting energy deficiency may result in polyphagia.

Clinical signs and symptoms

Most dogs with DM are diagnosed between the ages of 5-15 years, with peak prevalence between 7-9 years. Females, castrated and crossbreed dogs, and animals weighing less than 22 kg (48 lbs) have a greater risk of developing DM (11). Practically all affected animals have a recent history of polyuria, polydipsia and weight loss; polyphagia may also be reported, and an owner may describe sensory impairment and asthenia. Such clinical signs traditionally accompany the compensated form of DM, but severe sensory impairment, vomiting, diarrhea and anorexia can appear if the condition decompensates, or with the onset of diabetic ketoacidosis (DKA). It is not uncommon for dogs to be presented with sudden onset visual impairment due to the development of diabetic cataracts. This is due to the accumulation of sorbitol within the lens, which draws water into the tissues via osmosis, resulting in edema, rupture of the fibers and opacification of the lens ***(Figure 2).***

Diagnosis

An early and accurate diagnosis of DM is essential to prevent complications, delay progression of the disease, and establish a suitable and effective therapeutic strategy. The presence of typical clinical signs, along with persistent fasting hyperglycemia and glycosuria, point to a diagnosis of DM. To date, however, and unlike the situation in human medicine, a distinct glycemic value has not been established above which a state of DM can be clearly defined (6).

The detection of ketone bodies in urine (acetoacetate) or plasma (3-beta-hydroxybutyrate or 3-HB) using test strips enable diabetic ketosis to be diagnosed; if the ketosis is associated with metabolic acidosis, this is referred to as DKA. Another useful parameter for diagnosis is to measure serum fructosamine levels; these are glycosylated proteins formed by an irreversible, non-enzymatic, non-insulin-dependent bond between glucose and plasma proteins. The degree of glycosylation is directly related to the animal's glycemia; the fructosamine value will reflect the average glycemia concentration over the previous 2-3 weeks, and will usually be high in diabetic subjects, except with recent-onset DM, where levels may be within the normal reference range (11). It is not essential to determine fructosamine concentrations to diagnose canine DM, but it may be useful to know the baseline value during therapeutic monitoring. When DM is diagnosed, it is essential to perform blood and urine culture tests to investigate potential concomitant illnesses, and a culture should be done to exclude urinary tract infection. Ultrasound examination of the abdomen and thoracic radiography may be indicated to highlight pathologies that could have triggered DM (*e.g.,* pancreatitis, Cushing's syndrome, malignancies) or initiated insulin resistance.

Therapy

The treatment objective is to resolve the clinical signs and to prevent the onset of DKA and the development of hypoglycemia, thus ensuring good quality of life. This usually applies when glycemic values are maintained between 90-250 mg/dL or 5-14 mmol/L. If given the correct insulin therapy, in combination with a suitable diet, the canine diabetic patient has a life prognosis comparable to a healthy subject. Effective treatment is only achieved when there is good communication and mutual trust between the owner and the veterinarian (11); good client education is essential, and the pet owner should be kept informed of the objectives and therapeutic progress on an ongoing basis. In particular, clinical examination and monitoring should be at set time points (see ***Table 1).***

The crucial points for DM therapy are administration of insulin (18), appropriate diet, physical exercise and close therapeutic monitoring. It is also essential to identify and

Figure 2. Bilateral cataracts in a dog with DM causing blindness.

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Table 1. Therapeutic and monitoring protocol for diabetic dogs.

INITIAL DIAGNOSIS AND TREATMENT

- Diagnosis: history, physical examination, hyperglycemia, glycosuria and increased fructosamine levels
- Diagnostic protocol
  - Complete blood count, blood chemistry profile, urinalysis with culture
  - Abdominal ultrasound and cPLI (canine pancreatic lipase immunoreactivity), if indicated
  - Suspension of any diabetogenic drugs
- Therapy: administration of intermediate- or long-acting insulin
- Treatment of concomitant diseases
- Diet
  - Quantity: standardize the amount of food fed every 12 hours at the same time as, or just before, insulin administration
  - Type: high-fiber, low-calorie commercial diets for diabetic dogs
  - Any other concomitant disease (chronic kidney disease, liver disease, allergies/intolerances, pancreatitis) from a dietary point of view should be considered priority with respect to DM
  - Emaciated dogs or those with BCS < 4/9 should be fed a balanced maintenance diet until an optimal BCS is achieved
  - Obese or overweight dogs: a program should be drawn up to reduce body weight by 1-2% per week
- Owner education: verbal instructions supported by demonstrating insulin administration and information leaflets
- All entire females should undergo ovariohysterectomy as soon as possible

CLINICAL RE-EVALUATION 1 WEEK AFTER DIAGNOSIS

- Clinical exam
- Perform a blood glucose curve (BGC), with food and insulin administered at the clinic after the first glycemia measurement. Alternatively, patients can be taken to the clinic immediately after the owner has administered the meal and insulin at home
- Determination of serum fructosamine concentration
- Therapy: insulin dosage adjustment by 10-15% if necessary

CLINICAL RE-EVALUATION 2-3 WEEKS AFTER DIAGNOSIS

- Clinical exam
- Perform a BGC
- Determination of serum fructosamine concentration
- Therapy: insulin dosage adjustment by 10-15% as necessary
- Introduction of home monitoring for glycemic control, with written instructions for the owner
- The owner should produce a BGC every 2 weeks and measure fasting glycemia approximately twice a week

CLINICAL RE-EVALUATION 6-8, 10-12 WEEKS AND EVERY 4 MONTHS AFTER DIAGNOSIS

- Clinical exam
- BGC and serum fructosamine, with dosage adjustment as necessary
- Evaluate the owner's insulin administration technique

control any concomitant disorders (*e.g.,* infections, malignancies, other endocrinopathies, and nephropathies) capable of causing insulin resistance.

Insulin

Various types of insulin can be used to treat canine DM, and insulin analogues (*e.g.,* glargine, detemir) may also be useful in managing the condition, as indicated in ***Table 2*** (19-21). It is important to ensure that the correct dose of insulin is given. Owners should be aware that the units/mL can vary with different types of insulin (see ***Table 2)*** and must use the correct syringes. Some owners may find that an insulin "pen" ***(Figure 3)*** helps improve administration of the insulin.

Diet

Diet is an extremely important aspect in the therapeutic protocol. Dogs with DM may be underweight or overweight;

Table 2. Types of insulin commonly used for the long-term therapeutic management of canine DM.

Insulin type	Origin	Concentration (IU/mL)	Duration (hours)	Frequency of administration	Starting dose (IU/kg/injection)
Lente	Swine	40	8-14	BID	0.25
NPH	RH	100	4-10	BID	0.25
PZI	RH	100	10-16	BID	0.25-0.5
Glargine	RH	100	8-16	BID (SID)	0.3
Detemir	RH	100	8-16	BID (SID)	0.1

NPH: neutral protamine Hagedorn. PZI: protamine zinc. RH: recombinant human

in both cases, the objective is to reach and maintain an ideal body weight. In an obese or overweight dog a reduced-calorie diet, rich in insoluble fiber and low in fat, should be provided. Reduction of body weight is essential; animals that present with a BCS > 5/9 at the time of diagnosis can easily gain weight after treatment commences due to the anabolic effect of exogenous insulin. Underweight or emaciated patients should be fed a balanced maintenance diet. As a rule, dogs with DM should be fed every 12 hours with a standardized amount of food, with insulin administered at the same time.

Physical exercise

Controlled physical exercise is recommended for all dogs with DM; this enables the glycemia concentrations to be reduced and better absorption and use of insulin by the muscles. Physical exercise should be consistent; this is particularly relevant for working dogs, as sudden exertion can predispose to hypoglycemia and should be avoided. If a dog has to perform substantial, non-routine physical exercise, the insulin dose on that day should be reduced by about 50%.

Glycemic monitoring

Glycemic curves

Serial glycemic curves are essential for both short- and long-term monitoring, and enable rational adjustment of the insulin dose as necessary ***(Table 1)*** (22,23). To measure glycemia, portable glucometers (PBGM) are commonly employed, requiring a small blood sample usually obtained from the dog's pinna ***(Figure 4).*** There are many PBGMs on the market, but most are designed for human use and can be inaccurate when used in dogs (24). PBGMs designed specifically for veterinary patients are preferred, as they require an extremely small sample volume (0.3 µL) and deliver more precise results.

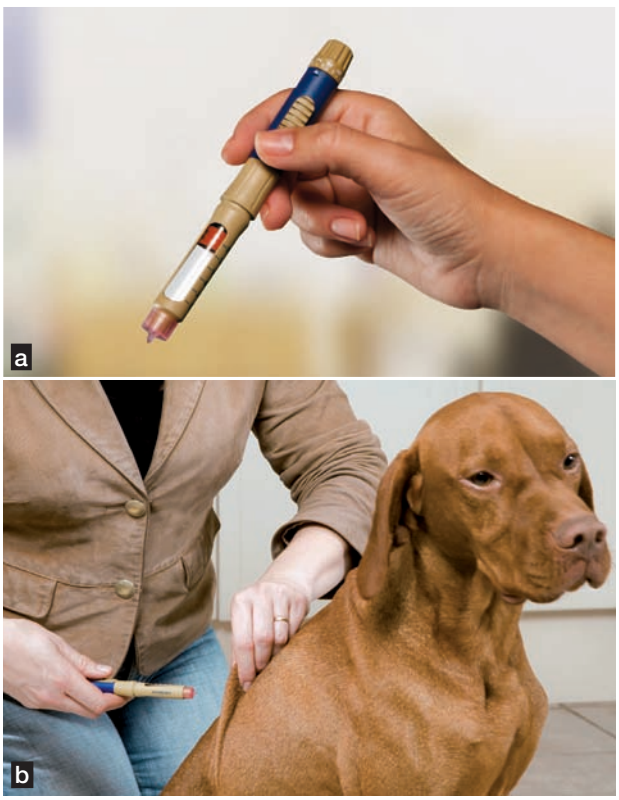

Figure 3. Some owners may find that using a special insulin "pen" **(a)** helps ensure accurate dosing of the insulin and easier injection **(b).**

Blood glucose curves (BGC) can be produced in the clinic or at home by the owner. The test requires glucose levels to be monitored every 2 hours, starting immediately before a meal and insulin administration and continuing for about 8-10 hours throughout the day. The trend of the glycemic curve is used to verify insulin effectiveness, identify the glycemic nadir (ideally between 90-150 mg/dL or 5-8 mmol/L), and determine the time necessary to reach

peak glycemia, as well as the degree of fluctuation in glycemia levels. The BGC is considered optimal when most of the glycemic values fall between 90-250 mg/dL or 5-14 mmol/L. If poor glycemic control is noted during a clinical exam, it is essential that the cause is identified and rectified as soon as possible ***(Table 3).*** Various possible scenarios can lead to poor glycemic control; the more frequent reasons include owner error, such as using a 100 IU/mL syringe with 40 IU/mL insulin – or vice versa

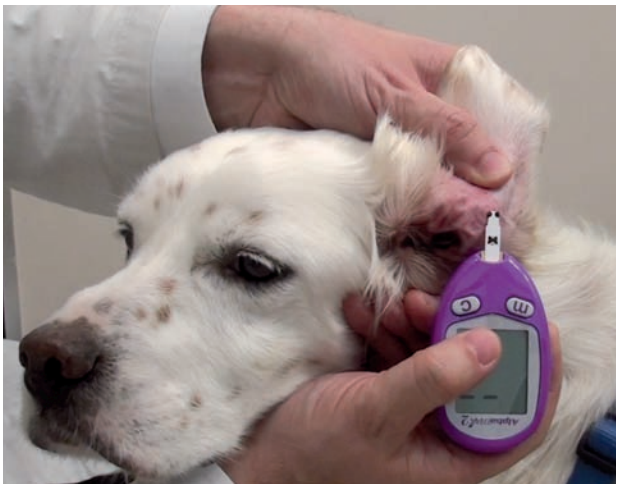

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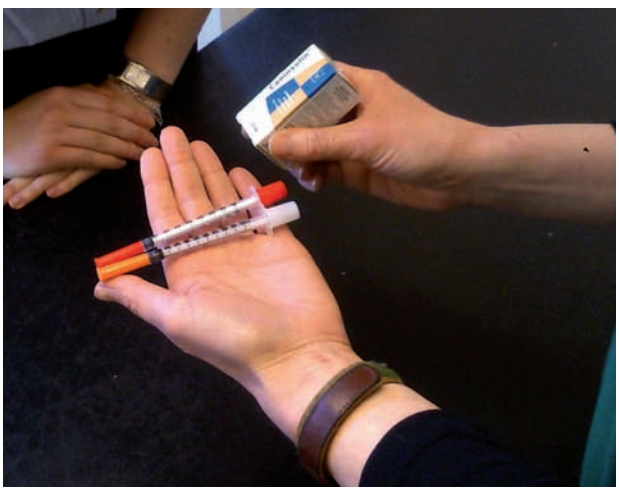
Figure 4. An owner can employ a portable glucometer (PBGM) to measure their dog's glycemia but note that many PBGMs on the market are designed for human use and can be inaccurate when used in animals. An instrument calibrated for dogs, as shown in the photo, is preferred.

Figure 5. Good owner education is central to ensuring a diabetic dog is well controlled; for example, the clinician should take time to explain the importance of using the correct syringe for the type of insulin chosen.



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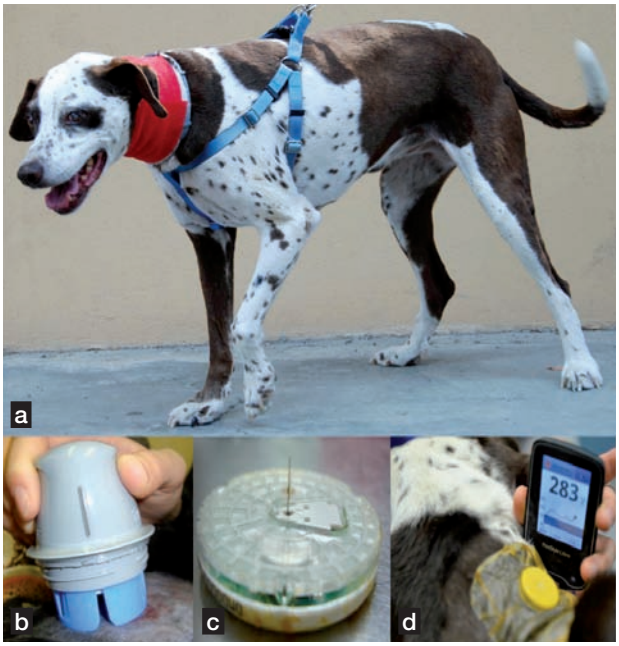

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Figure 6. A continuous monitoring system in a diabetic dog. **(a)** A bandage on the patient's neck protects the sensor. **(b)** The sensor is applied to the skin using an applicator after clipping the hair. **(c)** The sensor filament is capable of measuring interstitial glucose levels. **(d)** When scanned, the sensor transmits the glucose value directly onto the screen of the reader.

Table 3. Main causes of poor therapeutic control in dogs with DM.

- Owner error when administering insulin
  - Inappropriate administration method
  - Inappropriate syringe (*e.g.,* using 100 U/mL syringes with 40 U/mL insulin)
- Incorrect insulin storage or preparation
  - Frozen insulin
  - Inadequate mixing of insulin (*e.g.,* with zinc suspension insulin and NPH)
- Insulin underdose
- Somogyi Effect (hypoglycemia and resulting hyperglycemia secondary to compensatory mechanisms due to the action of hormones that increase blood sugar levels)
- Short-acting insulin
- Inadequate insulin absorption (*e.g.,* chronic skin inflammation, allergic reactions)
- Concomitant pathologies leading to insulin resistance (*e.g.,* inflammatory, infective, neoplastic or immune-mediated processes, and the use of diabetogenic drugs)

(Figure 5) – poor injection technique, or inadequate storage and handling of the insulin. ***Table 3*** outlines the most common reasons for poor therapeutic control.

Continuous glucose monitoring

More recently, continuous glucose-monitoring systems (CGMS) have been introduced which enable glycemic trends to be evaluated without the need for serial blood samples. These systems actually monitor interstitial glucose concentrations, which reflect serum glucose concentrations. Some CGMS are capable of recording a patient's glycemic trends by means of wireless technology, whereby a flexible sensor inserted subcutaneously transmits data to a monitor up to 3 meters distant. However, this type of instrument needs to be calibrated 2-3 times daily. Newer technology enables real-time monitoring of glycemia values (25); a small sensor applied to the neck area delivers instant read-outs to a monitor when scanned ***(Figure 6),*** with the added advantage that a sensor lasts for 14 days and does not require calibration.

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Canine obesity – genetics and physiology in action

Eleanor Raffan, BVM&S, PhD, CertSAM, Dipl. ECVIM-CA, MRCVS
Institute of Metabolic Science, Addenbrooke's Hospital, Cambridge, UK

Dr Raffan is an Edinburgh graduate who spent time in general practice and did internship and residency training in small animal medicine at Liverpool and Cambridge vet schools before doing a PhD in genetics and biochemistry. She now combines research with clinical work and teaching at the University of Cambridge.

Obesity is a big problem. The toll human obesity exerts on both people's health and healthcare budgets means that it regularly features in media stories, but animal obesity is also a real and growing problem. In recent years, the increasing incidence of obesity in companion animal species, particularly dogs, cats and horses, has also attracted headlines, whilst the impact of obesity in reducing productivity and fertility in farm animals has long been recognized. The coverage in both the popular and veterinary press has, almost universally, been rather pejorative in tone, blaming poor owner management for this increase. However, the scientific literature is increasingly focused on the biology underlying obesity development, and there is now a great weight of evidence that obesity is perhaps best viewed as a complex homeostatic mechanism gone awry, influenced by genetics. This article reviews the impact of obesity in dogs and our current understanding of obesity biology. It aims to introduce readers to some of the nuances of energy homeostasis, and perhaps reframe the notion that the owner is invariably to blame for pet obesity, such that clinical approaches can take the variation in an individual's biology into account and improve outcomes for obese and at-risk patients.

KEY POINTS

- **Obesity is an increasingly significant problem in dogs; between 40-59% of pet dogs are overweight or obese.**
- **Obesity is perhaps best viewed as a complex homeostatic mechanism gone awry, influenced by genetics.**
- **Recent research has discovered a genetic mutation in Labrador Retrievers which is associated with food motivation.**
- **Understanding the complexity of energy homeostasis and how genetic differences predispose individual dogs to obesity will allow clinicians to better manage obesity in their patients.**

Should we care about obesity?

Between 40-59% of pet dogs are overweight or obese, and similar statistics apply to pet cats. These figures have increased in recent years and veterinarians in practice certainly corroborate that obesity is an increasingly significant problem in both their canine and feline patients. The increased incidence of metabolic, endocrine, respiratory, orthopedic, dermatological and other diseases associated with obesity means that clinicians should be concerned with its prevention and treatment. At the most simplistic level, obesity shortens lives: in a longitudinal study of Labradors, dogs which were kept at a lean (healthy) weight lived on average two years longer than a matched, similarly managed group of dogs which were allowed to eat more and became overweight (1).

That obesity is an increasing problem, and that it exerts a toll on canine welfare due to its associated morbidities, is not controversial. But it is necessary to next address the causes of obesity, as it can be argued that clinicians should understand more than just lifestyle and diet risks if they are to help obese patients.

What causes obesity?

There is an easy answer to this question; an individual gains weight if they eat more calories in food than they burn off each day. This is reflected in the literature, which has mainly focused on how owners manage their dogs,

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Figure 1. The majority of evolution has occurred in a resource-poor environment, and there is a physiological drive to promote eating in excess of energy requirements in order to lay down fat during times of plenty in preparation for periods of fast to come. Wild canids still epitomize this behavior, with their "feast and fast" lifestyle.

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Figure 2. Most dogs now live indoors and require less energy to keep warm; however, dogs kept outdoors use up to a third of their energy intake for thermogenesis.

and the list of obesity risk factors will come as no surprise to veterinary professionals; dogs that are offered human food, or a calorie-dense diet, or fed more frequently, are prone to obesity, as are those which get less exercise because their owners take them for shorter or fewer walks. But breed, age and gender also are on the list, and remain there even when management factors are evened out in mathematical modeling, meaning their effects are physiological and independent of owners.

So while the physics of the energy balance equation between intake and expenditure is of course true, and owners should be able to control their dogs' food and exercise to keep them lean, it is disingenuous to dismiss the role of physiology in obesity development.

When getting fat was a good thing

If sustained, obesity has adverse consequences, but it is worth remembering that there are good physiological reasons for storing excess energy as fat – namely, to build up energy reserves in case of future food scarcity. Given that the majority of evolution has occurred in a resource-poor environment, it makes sense that there is a physiological drive to promote eating in excess of energy requirements in order to lay down fat during times of plenty in preparation for periods of fast to come ***(Figure 1).***

In recent years, the lifestyle of pet dogs (and humans) has changed, such that most now live relatively inactive lives and have ongoing access to calorie-dense food. Most dogs also now live indoors, and use less energy to keep warm ***(Figure 2)*** – whereas outdoor dogs use up to a third of their energy intake for thermogenesis.

It is those rapid environmental changes, based on an unchanged genetic background that has evolved to cope with scarce food resources, which underlie the obesity epidemic in today's pets. The consistent message of "feed less, exercise more" to control pet obesity clearly does not always work, and a more comprehensive understanding of the biology which drives obesity may help veterinary professionals to manage their patients' obesity better.

The biology of energy homeostasis

Figure 3 summarizes the homeostatic mechanisms that regulate eating behavior and energy expenditure. It shows how messages from the gut and circulation concerning

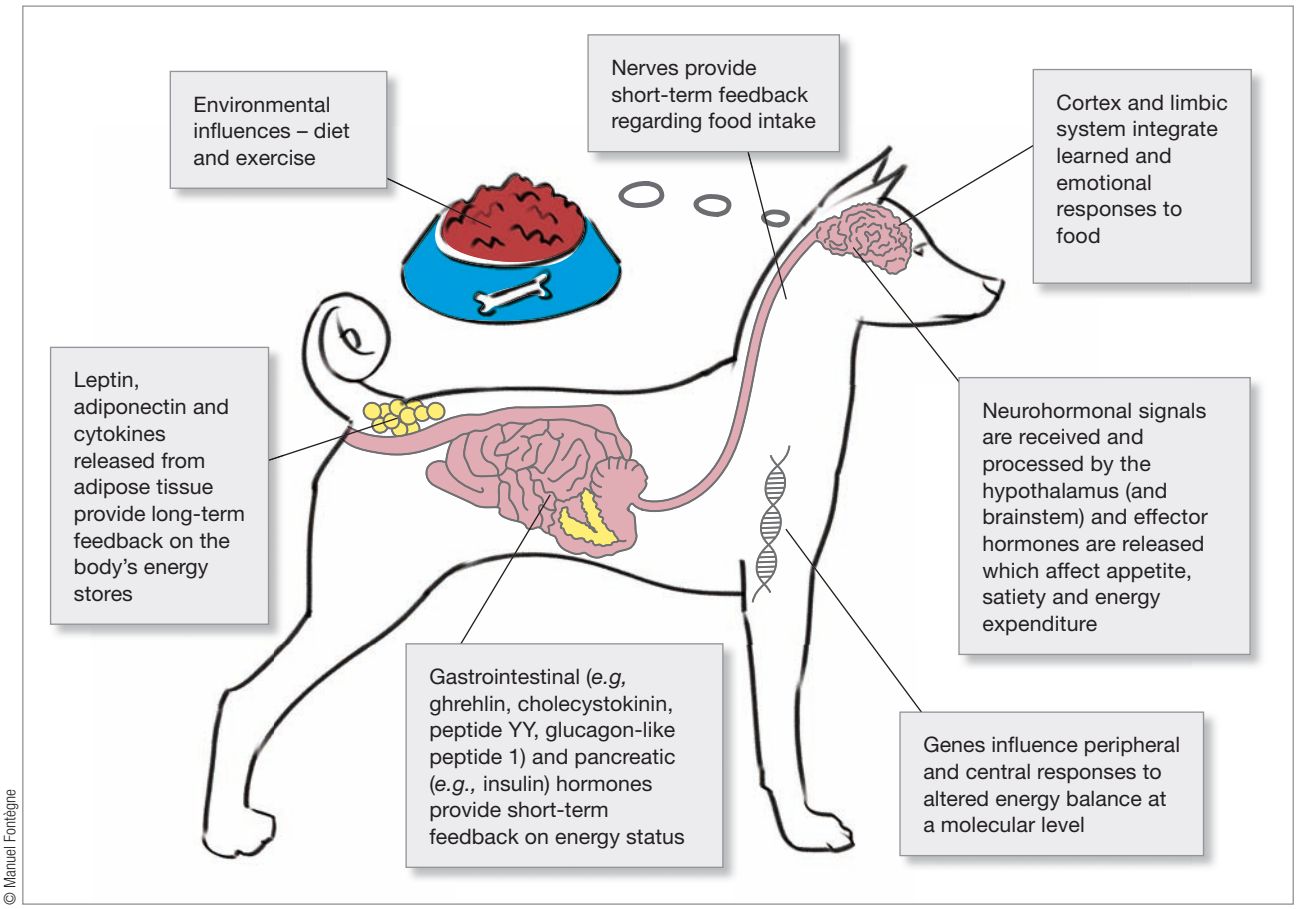


Figure 3. Physiological control of energy homeostasis (5).

short-term energy flux, and longer-term messages from adipose tissue regarding energy stores, are sent via endocrine or neurological signaling pathways to the hypothalamus. There they are integrated to produce conscious sensations (hunger, satiety) and behavioral outcomes (food seeking, food choice, eating).

Long-term signals – fat as an endocrine organ

Fat is more than an inert energy storage depot; it is better regarded as an endocrine organ. Adipose tissue secretes hormones (including leptin and adiponectin) and cytokines which travel in the blood to exert distant effects. The amounts and types of these "adipokines" released will alter depending on fat mass, so they act as distant signals of the body's energy reserves.

Key in the control of obesity is leptin, which is released in greater amounts from lipid-rich adipocytes. Its major site of action is in the leptin-melanocortin signaling pathway in the hypothalamus, where it acts to "turn off" background hunger; this means that when the body's energy reserves are replete, food seeking becomes less of a priority ***(Figure 4).*** As such, it can be considered a "dimmer switch", altering the background hunger in response to the body's need.

Short-term signals of satiety and energy balance

If leptin is the dimmer switch, the gut and pancreatic peptides are the "spotlights" highlighting short-term fluxes in energy and controlling meal-to-meal eating behavior. The key "hunger hormone" is ghrehlin, which increases to a maximum just before a meal, then drops after feeding. In contrast, a number of other hormones (*e.g.,* glucagon-like peptide 1, peptide YY, oxyntomodulin, cholecystokinin) are responsible for detecting nutrients in the gut lumen, or after absorption in circulation, and promote satiety.

In reality, leptin, gut peptides and other hormones (including insulin, glucagon and thyroxine) work together to send a complex mix of signals which are integrated by the hypothalamus and translated into observable behavioral

outcomes. Cortical (conscious) control also plays a part in decision making, planning and executing food seeking and eating, but it does so against a background of those physiological drives to hunger, satiety and desire for particular nutrients.

Importance of genetics

In this context, it is easy to understand that genetic variation might play a part in altering energy homeostasis. This is borne out both in dogs, where breed predispositions to obesity clearly implicate genetics, and humans, where 40-70% of a person's tendency to become obese is a consequence of their genetic make-up (2).

Monogenic obesity syndromes occur in humans, but these are uncommon. The genes involved have been investigated in depth, and most causative mutations affect the hypothalamic pathways governing eating behavior. "Common" obesity is polygenic – with tens to hundreds of genetic variants each contributing incrementally to increase or decrease a person's tendency to gain weight. The genes responsible are less well defined; where they are identified, they often influence eating behavior, but many are poorly understood at a mechanistic level (2). It is likely that genetics can influence metabolic rate too, but to date the expected major modifiers have not been identified in humans.

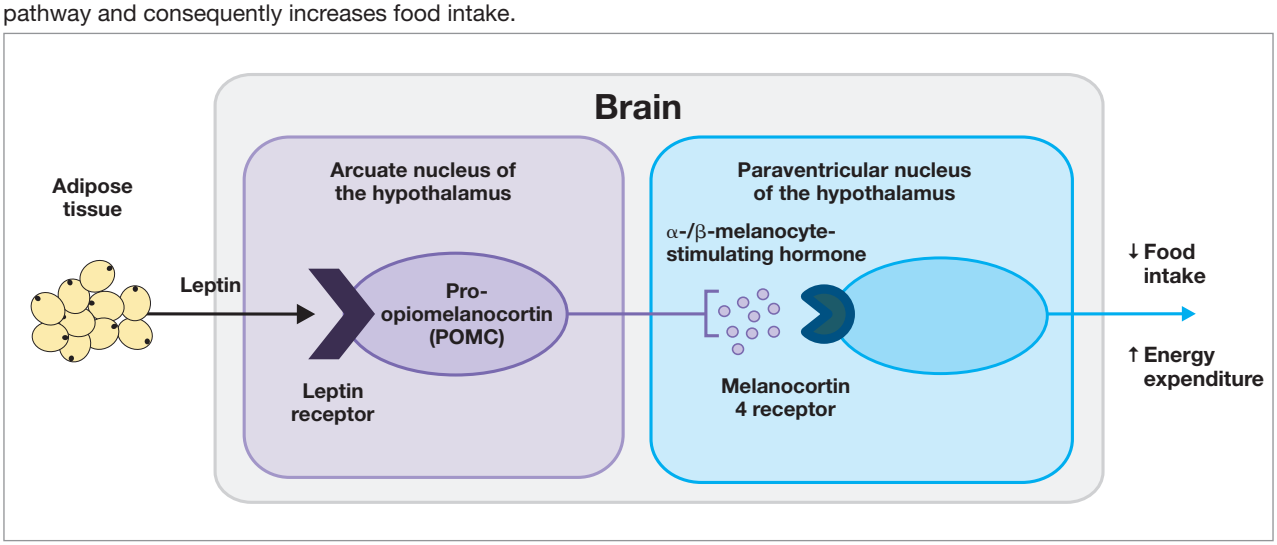
In dogs, some breeds (*e.g.,* Labrador Retrievers) are at high risk of obesity, whilst others (*e.g.,* Yorkshire Terriers) are relatively resistant. This is sometimes also blamed on owners – but it is difficult to believe that all Yorkshire Terriers are kept lean by owners who never give them tidbits and take them for long walks every day, whereas Labrador owners are much more indulgent and exercise-averse. Rather, the prominence of breed as a risk factor for obesity clearly implies genetics are playing a role in its development.

The author has recently reported (3) a mutation in Labrador Retrievers which is associated with food motivation and weight ***(Box 1).*** A mutation in the POMC gene is carried by around a quarter of the pet Labrador population. POMC is a pro-peptide produced by neurons in the hypothalamus and broken down to neuroactive ligands before being released. The Labrador mutation disrupts production of β-MSH which usually acts to promote satiety and increase energy expenditure ***(Figure 4).*** This is the first time a major genetic modifier of weight and eating behavior has been pinpointed in dogs, but there is still much more to discover. Assuming dogs follow the patterns seen in humans and other species, it is likely that the largest genetic influences will be to do with how the brain controls food intake.

Clinically applying knowledge of obesity biology

From a practical point of view, veterinarians can apply this understanding to clinical obesity management by acknowledging that some dogs really are "hungrier" than others. From an owner's perspective, it means that some dogs not only pester them for food more often,

Figure 4. The leptin-melanocortin signaling pathway. Increased production of melanocyte-stimulating hormone (MSH) promotes satiety and increases energy expenditure. The Labrador POMC mutation disrupts signaling through this pathway and consequently increases food intake.



but are also more grateful when they get it – so there are both negative and positive reasons to feed dogs that exhibit this sort of behavior ***(Figure 5).*** These same dogs are likely to find illicit food by scavenging wherever they can, which adds to their likelihood of gaining weight.

It therefore follows that, rather than dismissing an owner of a fat dog as being careless about feeding, the clinician can acknowledge that they are responding to their dog's behavior and are likely to have to make more of an effort to control their pet's food intake than owners of less food-motivated breeds. Indeed, data suggest that an owner of a highly food-motivated, overweight dog will often work harder to restrict their pet's food intake (*e.g.,* by restricting tidbits, measuring meal sizes etc.) than owners of slim dogs – they just do not succeed (4).

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Figure 5. Some dogs will pester their owner for food at every opportunity – and may demonstrate gratitude when they get it. This can result in reinforcement behavior for both pet and owner.

Box 1. An obesity-causing mutation in Labrador Retrievers (3).

Labradors repeatedly top the table of obese-prone breeds, and recent research has gone some way to explain why: around a quarter of pet Labradors carry a mutation in the gene POMC, which plays a key role in the hypothalamic control of appetite. In the leptin melanocortin signaling pathway (see **Figure 4),** the POMC mutation reduces the signal at the melanocortin 4 receptor (MC4R), such that the dog is less likely to feel satiated.

The mutation is associated with body condition score, weight and food-related behavior; for each additional mutant allele dogs are, on average, 0.5 of a body condition score point greater (on a 9-point scale) or 2 kg heavier.

The mutation has not been found in other breeds, other than the closely related Flat-coated Retriever, where the effect on weight and food motivation was similar to Labradors. Analysis showed the mutation came from a common ancestor (likely the St John's Water dog) and it can be speculated that a genetic predisposition to hunger was no bad thing for these dogs, as they worked for fishermen in the icy waters of Newfoundland, and may even have made them more willing to work hard for food rewards.

The mutation is noticeably much more common in assistance dogs. It is speculated that the mutation could have been beneficial in animals bred as working dogs, and it is possible that the gene may have even been inadvertently selected for in the assistance dog population, but it now predisposes modern pet Labradors to obesity.

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Labradors are more prone to obesity than any other breed of dog.

The POMC mutation is commonly found in assistance dogs (*e.g.,* dogs that work for people with impaired vision or poor hearing).

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The gene mutation is thought to have arisen in the St John's Water dog, a breed once used as a working dog in Canada but now extinct.

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Figure 6. Using trickle feeding (*e.g.,* with a puzzle feeder) may help with weight management programs.

From a dog's perspective, those which have a genetic drive to eat (such as POMC mutation-bearing Labradors) do genuinely feel "hungrier" than others. There is a real tension here to do with two of the "five freedoms" of animal welfare: freedom from hunger and freedom from disease. If a dog that is genetically hard-wired to be hungry is kept slim and healthy by restricting its food, is that always right? A rational response is that food restriction should be done in a way that tries to make the body believe it is getting more food than it really is.

To engender maximum possible satiety from lower energy rations, feeding practices and foods that trigger prolonged gut satiety hormone release may be of use. There is a good evidence that high-fiber, high-protein diets designed for weight loss promote satiety and their use therefore seems sensible in highly food-motivated dogs. Replacing twice-daily meals with trickle feeding (*e.g.,* using puzzle feeders) may also reduce hunger, and could improve welfare too by providing repeated, albeit small, food rewards to particularly food-motivated dogs ***(Figure 6).*** Both approaches are worth trying with problem obesity patients, or as strategies to prevent obesity developing in at-risk dogs.

Conclusion

Acknowledging the complexity of energy homeostasis and how genetic differences predispose individual dogs to obesity is valuable in allowing veterinarians and owners to understand and manage obesity in dogs both effectively and sympathetically. The article on page 40 addresses the human factors involved in pet obesity and how understanding those can also help with effective management.

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HOW I APPROACH...

Owners with obese pets

Cornelia Ewering, Dr. med. vet., PhD
Mars Petcare, Verden, Germany

Dr Ewering studied veterinary medicine in Hanover and completed her doctorate in 1993 for studying body measurements in breeding sows. She has worked in both small and large animal practices and since 1998 has held the post of pet nutrition expert at Mars Petcare in Germany. She has a particular interest in improving communication between veterinarians and pet owners.

Introduction

Cats lie on the sofa with their owners, dogs are driven to the park in a car, or ride with the family in a bike trailer… animals are expending less energy than they have in past decades, and yet their love of food is as strong as ever ***(Figure 1).*** Consequently, obesity is a growing concern in companion animals, just as it is in humans (1). Despite the fact that statistics estimate up to 40% of dogs and cats in developed countries are overweight or obese, many owners do not recognize when this is true of their own pet. And other factors can make this scenario worse: an indulgent pet owner who owns a Labrador finds it difficult to cope with an effective weight management program – at least when compared to the owner of a dog that is less food-motivated. Interest in food is linked to a dog's genetic make-up, and for many animals the lack of satiety drives them to be continually looking for food.

It is increasingly recognized that there is genuine biological variability between different dog breeds in their drive to seek food. As a consequence, some owners need to be able to adapt both how they feed their dog, and how their dog behaves towards food, to ensure that their pet's weight remains optimal.

With the observation that indulgent owners often choose dog breeds well known for their trainability (which is frequently done using treats) or their companionship with children (who also tend to indulge their pets), it becomes obvious how some categories of pet owner, and the genetics of some dog breeds, can combine to encourage the animal to become obese. This paper considers the different types of pet owner and possible entry points which will improve communication with owners, with the aim of improving weight control.

KEY POINTS

- **The tendency of some pet owners to indulge their pets, combined with the genetics of some breeds of dog, can increase the risk of obesity.**
- **Although statistics estimate that up to 40% of dogs and cats in developed countries are overweight or obese, many owners do not recognize when this is true of their own pet. Weight management can therefore be a sensitive topic in veterinary practice.**
- **An understanding of pet ownership styles, and their potential role in the current pet obesity epidemic, may help the clinician to better design and address weight management strategies and programs.**
- **Adopting standardized protocols for diagnosis and recommendations will help to improve the communication process between the healthcare team and pet owners and ensure that clients receive consistent information.**

Why should communication on weight management be improved?

Veterinarians can be a trusted source of information on feeding and nutrition, but a recent study found that weight management is discussed only once in every hundred veterinary consultations (2). Participant responses indicated that a pet's weight was a difficult topic to address in direct conversation; this may be partly due to the worry that it will be embarrassing and could even potentially lose a client.

Why does "feed less and move more" not always work?

Recent trends in pet-keeping are sometimes summarized as the progression of pets from "outside in the

yard" to "inside the home" to "on the bed". This closer co-habitation has facilitated the development of strong, emotionally rewarding relationships between people and pets (3). However, each relationship is unique, and individual owners will vary in how they interact with their pet, including the way they use food, both as a point of contact and to express their attachment to their pet (4). An understanding of different ownership styles, and their potential role in pet obesity, may help improve communication with owners, such that better pet nutrition can be achieved and weight management strategies and programs are more likely to succeed.

What are the parallels between parenting and pet ownership styles?

Just as parents' awareness and understanding of what is healthy for their children is important (5-9), it is also vital for pet owners to understand how to provide optimal nutrition for their pets, in terms of how, what and when they are fed. Given the familial status of many pets, it is logical to assume that the family food environment also affects attitudes toward pet feeding practices.

The model put forward by MacCoby and Martin (10) on different parenting styles is currently considered to have the strongest scientific foundation. Distinguishing between Authoritative, Authoritarian, Indulgent and Uninvolved parenting styles, the model could potentially be applicable to pet owners ***(Figure 2).*** However, because pets will always depend on their owners, will never mature in the way children do, and cannot be expected to control the amount of food they consume, the model must be adapted for pet ownership styles (11).

Figure 1. Animals are expending less energy than they have in past decades; changes in human lifestyle impacts on how we treat our pets.

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How do I recognize an authoritarian or an authoritative owner?

The Authoritarian pet owner – who may have a working breed of dog – can be regarded as someone who is as assertive as they perceive is necessary. He or she is driven by facts rather than by emotions, and is interested in evidence from studies, statistics, etc. This person can deal well with objective systems (*e.g.,* kilocalories) and rules, and appreciates a strictly methodological approach; although dutiful, he or she may need advice on how to get support of all those involved (including the pet) when starting a weight control program for their animal.

The Authoritative owner has been described as being as rational as an Authoritarian type, but with less need to control the pet and with a greater orientation towards the pet's needs.

When preparing for a consultation with an Authoritarian or Authoritative client, the following questions might be helpful for the veterinary team prior to the appointment:

- What are the observable and verifiable facts?
- What are the logical consequences of the facts?
- What are the resulting options for action?
- Why should the owner care about dieting their pet?
- What previous experiences can the team draw on?
- What is the overall goal?

The clinician may then choose an appropriate approach when discussing the need for weight management, as shown in ***Figure 3.***

How do I recognize an indulgent or uninvolved owner?

The Indulgent owner enjoys helping and sharing. He or she is emotionally sensitive and geared toward partnership, and all parties contribute to the solution for a problem. He or she needs advice on a few essential rules, and requires understanding in order to adhere to guidelines and to alter factors that need to be changed. A person in this category needs to understand the benefits for his/her family, including the pet. Expert advice, as well as fun and joy, are important.

The Uninvolved owner appreciates flexibility and freedom; feeding and bonding are simply not a high priority. The animal is part of the family or team, but feeding is just not as important as time spent doing other things (*e.g.,* working, recreation). This pet owner prefers freedom of choice and positive affirmation.

Figure 2. The Authoritarian, Authoritative, Indulgent and Uninvolved parenting styles, as they apply to pets at meal times, and how each type of owner may react to a pet begging for food at the dinner table.

	IN GENERAL		AN EXAMPLE: DINNER TIME AT HOME
AUTHORITARIAN		The owners are very consistent; they appropriately take care of — or rather control — the pet's nutrition, and they have clear rules regarding what the pet can and cannot do.	The owner sends the pet to his/her place. Later, the owner puts petfood in the bowl, and then insists the animal waits until he/she is allowed to eat.
AUTHORITATIVE		The owners are very consistent; for example, they never provide scraps from the table while they are eating, and they feed their pet at the same time each day.	The owner sends the pet to his/her place with clear but friendly words. After a while, petfood is offered in the food bowl.
INDULGENT		Feeding is determined by the pet. The owner always selects the "best" petfood and apply few or no limits; they love to indulge their pet.	The owner tells the pet that he/she is "such a good pet" and that he/she "loves him/her". He/she offers the animal the best pieces of meat from his/her own plate.
UNINVOLVED		The owner feeds the pet at different times each day, and can sometimes forget to feed the animal or to buy petfood. He/she feeds different amounts of food depending on what is available.	The owner ignores the pet during dinner. Later, he/she feeds whatever food is available.

Figure 3. When dealing with an obese pet, the clinician should vary the approach depending on the type of owner.

AUTHORITARIAN	AUTHORITATIVE	INDULGENT	UNINVOLVED
• "Studies show…" • "You can avoid costly treatments if you now…" • " I have had good experiences with this program in my practice, when…" • "Why don't you try this program for two weeks, then, if necessary, we can make any needed changes…" • Praise the pet • Avoid being too critical when things do not go according to plans	• "Studies show…" • "You can avoid costly treatments if you had now…" • " I have made good experiences with this program in my practice, when..." • "Why don't you try this program for two weeks, then, if necessary, we can make any needed changes…"	• "If we tackle this together, you and your pet can have a long, fun, happy life together." • "What makes your pet the happiest?" • "You are a great team. Let's think about how we can work together to…" • "Look at those beautiful eyes. I can understand why it is difficult to look at him/her and not feed him/her whatever he/she wants. Many of my clients have had similar experiences with their pets, and this tip has worked well…"	• "If we tackle this together, you and your pet can have a long, fun, happy life together." • "With a few steps, we can achieve a lot."

When preparing for a consultation with an Indulgent or Uninvolved owner, asking the following questions might be helpful for the veterinary team prior to the appointment:

- Are there exceptions among individual family members (*e.g.,* children) that must be considered?
- What emotional values are important to the owner in relation to the pet?
- What are the likely reactions of those affected?
- What does the effort for the owner and the group (which includes the animal) mean?
- Can the team provide reports from other owners as to how compliance with recommendations has improved these relationships?

Again the clinician may then choose how to discuss weight management accordingly, as suggested in ***Figure 3.***

How can I support the establishment of new feeding regimes?

It is important to understand if a pet owner is aware of the needs of his/her pet in terms of energy intake and expenditure. If more than one person is involved with feeding, it is essential to establish who takes overall control of the amount of food offered daily. It is also important to appreciate that an Indulgent owner will look at feeding differently compared to an Authoritarian type; for example, an indulgent owner will take the behavior of a "greedy" dog (as driven by its genetic make-up) as the perfect excuse to offer more food. When discussing a new feeding regime, different approaches, tailored to the type of owner, can be very helpful, as illustrated in ***Figure 4.***

How can I support increasing physical activity?

Increasing physical activity is an important aspect of any weight management program. In dogs, this can be achieved by walking longer distances, walking more frequently, and/or increasing play. It is important that the veterinarian advises the owner to increase physical activity gradually, in a step-wise progression. For cats, playing and providing games and toys that require them to work for food rewards (*e.g.,* hiding food so that the cat has to find it) are good ways to increase activity. Again certain approaches may suit different owner types, as shown in ***Figure 5.***

What is the role of the veterinary team?

Owner compliance depends both on their own behavior and the response of the veterinary team. Pet owner's follow-through, the staff's attitude and behavior, and an aligned understanding of treatments, screenings and procedures, all contribute to compliance (12). Problems

Figure 4. When establishing a new feeding regime the approach may need to vary depending on the type of owner.

AUTHORITARIAN / AUTHORITATIVE	INDULGENT	UNINVOLVED
• Calculate the recommended energy intake • Calculate the recommended amount of food based on the manufacturer's nutritional information • Calculate the proportion of dry food that can be used for praise or during play • Calculate the energy intake for snacks per day/per week that fit within the program	• The calculated amount of food is weighed in the practice • The recommended amount of food is marked on a measuring cup on the pet's bowl • If required, recommend a smaller bowl • Fill a container with the recommended amount of treats and extras for one week. Explain that the container should only be refilled once per week.	• The practice calculates a balanced amount of the food most frequently provided • Recommend the amount of food that should be purchased weekly or monthly
• Provide and explain the use of a feeding diary and weighing card • Build regular check-ups into the program	• Provide regular check-in calls • Provide incentives • Offer opportunities for owners following pet weight management protocols to meet and provide each other with support	• Suggest a range of measuring cups or containers that can be used with a kitchen scale • Provide monthly check-ups and reminders • Provide incentives

Figure 5. When a clinician offers owner support to increase their pet's physical activity, different methods may appeal to certain owner types.

AUTHORITARIAN / AUTHORITATIVE	INDULGENT / UNINVOLVED
• Clarify preferences/habits • Specific recommendations on training intensity with regard to the pet's age, breed and size • Recommendations for how and when to schedule exercise into the day • Set objectives, *e.g.,* what should the pet have learned/accomplished after four weeks?	• Clarify preferences • Recommend games and fun approaches to increasing the pet's physical activity • Provide concrete ideas for increasing physical activity, *e.g.,* a fact sheet • Demonstrate a variety of toys, and let the owner try them out
• Partner with local dog training schools to perhaps provide badges, certificates • Partner with local pet stores • Provide checklists that include ideas for active play, exercises for different breeds/sizes • Provide tips on places to walk/run and distances	• Offer a notice board (or online group) for participating clients to meet and perhaps walk together, or provide each other with support. Include information about local dog walking services

with communication between owners and veterinary staff can threaten compliance; for example, if there was not enough time to explain something during a consultation, if the advice given was too vague, or if no clear instructions were offered. Information overload or contradictory messages from different team members can also compromise compliance. Particular attention should be paid to the clarity of communications, and it is essential to avoid mixed messages, which can occur if a staff member is asked to give advice and recommendations that run contrary to their own beliefs.

How can team compliance be increased?

Veterinary practices can benefit from having protocols that will help deliver healthy weight management for their patients (12). Agreed protocols should allow standardized diagnosis and recommendations, and will help to improve the communication process between the healthcare team and pet owners so that consistent information is delivered. Internal compliance (*i.e.,* ensuring the approved systematic application of the protocols by all members of the team is actually followed) is an active, ongoing process, and regular review of the protocols should ensure that they are as simple and as practical as possible. It is helpful to design a monitoring scheme to enable early detection of compliance problems and application of corrective measures; measuring and recording compliance is a key feature of this process.

Practical applications

Each owner and each pet is unique, and weight loss programs that take individual differences and circumstances into account will have a greater chance of achieving adoption and compliance. An owner will be more likely to buy-in and commit to a plan that they have been involved in creating. Because excess body weight tends to develop slowly over time, measuring a pet's weight regularly is a key step for successful weight management, and should be considered as part of any protocol. Practices may consider offering free weight checks at chosen intervals, possibly using the nurse or technician to assist in management protocols by weighing patients and supporting clients during these brief visits. This arrangement has several advantages. By visiting the practice and speaking to the nurse, owner loyalty to the practice may be enhanced. Advice can be given and noted in a "weight diary", and by plotting one or more graphs of data points (*e.g.,* the pet's weight, and perhaps the time spent engaged in physical activity) over time, owners can see that their efforts are successful.

Figure 6. Posters and client brochures can be very useful when promoting weight control for pets. These can target various aspects of feeding and ownership style, for example;

"It's not about dieting, it's about establishing new feeding habits"

- Encourage owners to seek advice from the clinical team about what is required for a balanced diet.
- Make a mark on the pet's bowl to ensure the same amount of food is always given.
- Invest in a smaller food bowl to create the illusion of more food; this is particularly effective with owners who worry that their pet is getting too little to eat. This works well for cats that like several small, fresh meals.
- Replace at least half of the pet's normal treats with pieces of apple, pear or carrot, or cooked chicken; when mixed in a bag with standard treats, they take on the aroma of the usual treats. This trick works particularly well for dogs.

"Turn meal times into an event, rather than focusing on the food itself"

- Make the act of eating exciting in itself, *e.g.,* put the reduced ration of food into an interactive toy. The more compartments the pet has to push open, turn and pull up, the longer meal times will take, and the animal will not notice the smaller ration.

"Less focus on food, more focus on adventure"

- This works especially well with dogs, and can include encouraging the owner to take the dog out more often – even if simply posting a letter or going to a nearby shop. Another option is to offer new stimuli, for example occasionally going to a local zoo rather than the park.

"Introduce more structure to daily meals"

- Encourage an owner to list everything the pet gets to eat during the day – including snacks from grandparents, biscuits from children, and the food the pet might have vacuumed up from the floor or stolen from the table.

Giving this kind of support will provide encouragement, especially for the authoritarian type of owner, and hopefully will be reflected in continued compliance.

When it is not possible for the owner to visit the practice (*e.g.,* for an animal who finds travelling stressful) telephone

check-ins may be an alternative. Owners can be taught how to weigh their pet at home, preferably on the same scales each time, and can share this information and receive support over the phone. If an owner fails to attend an appointment, it can sometimes help to ring them and enquire about their absence. If an owner is experiencing problems with the weight loss program, these may be addressed during the call. Practices may also wish to consider utilizing their waiting rooms to deliver and reinforce messages about healthy weight management, employing posters and leaflets to provide useful information ***(Figure 6).***

Conclusions

There can be few veterinarians – and indeed few owners – who think that dieting a pet is easy. It is important to appreciate that a "one size fits all" approach is unlikely to work, and a wise clinician will involve the entire hospital team in devising strategies that will maximize the chances of weight control programs being successful. With a planned approach, consistent application of protocols, and good follow-through, the rewards, both in terms of slimmer, healthier pets and happier owners, can be immense.

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Further reading

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Comorbidities in underweight cats

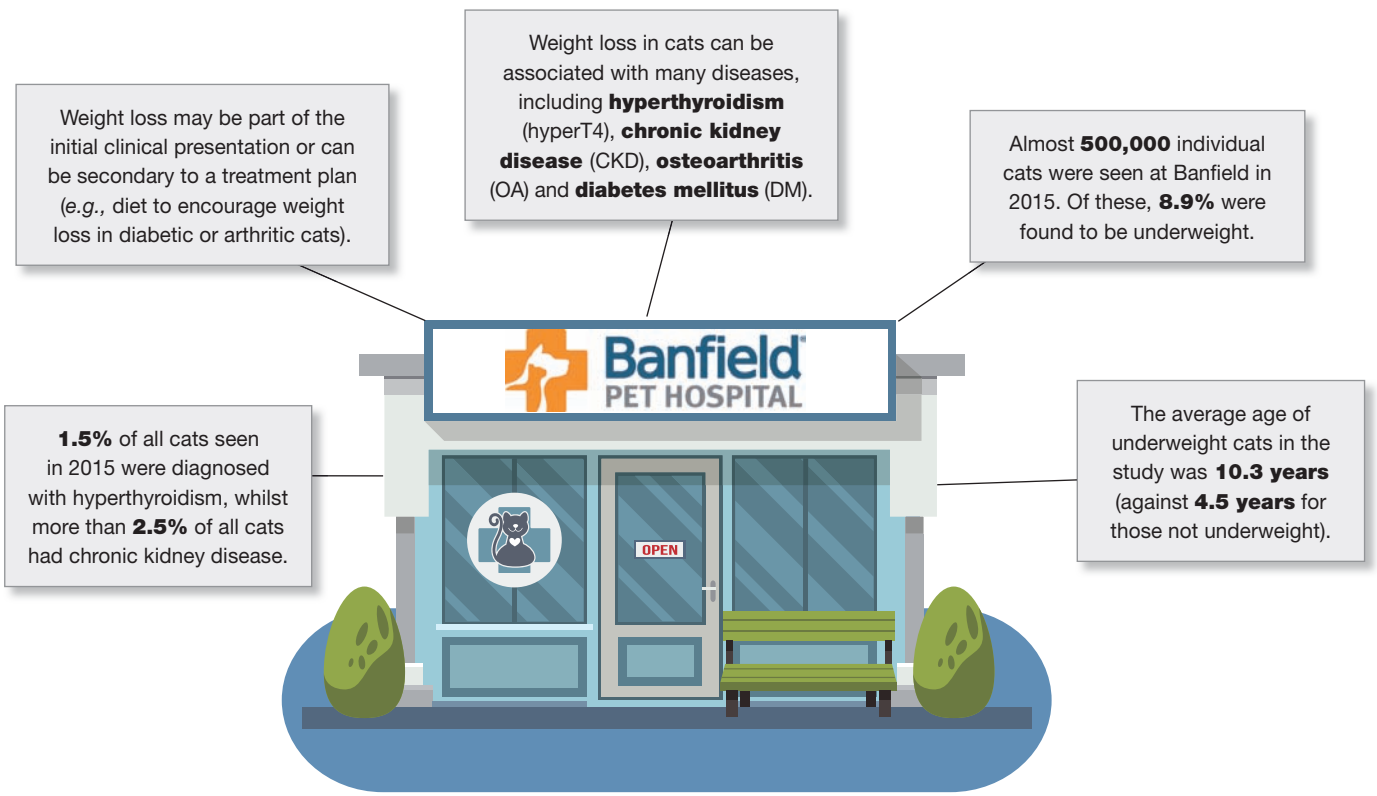
Emi Saito, VMD, MSPH, MBA, Dipl. ACVPM (Epidemiology)

Dr Saito qualified from the Veterinary Faculty at the University of Pennsylvania in 1997 and joined Banfield's Applied Research and Knowledge (BARK) team in 2013 after several years working for US Government departments as an epidemiologist.

Silke Kleinhenz, BA

Silke Kleinhenz has degrees in Marketing and Advertising and worked as a report writer for the anesthesiology department at Oregon Health Sciences University before joining Banfield in 2013; she now works as part of the BARK team as a senior data analyst.

Weight loss in cats may be due to a variety of reasons including – among others – gastrointestinal disease, sarcopenia, pain, neoplasia and endocrinopathies (1). A recent search of peer-review literature failed to identify studies that had reviewed the most common diseases diagnosed when a cat was noted to have weight loss; this short paper examines prevalence and relative risk of a few of the common feline medical conditions that may be associated with being thin. The health records of all cats examined by a veterinarian at Banfield Pet Hospital in 2015 were screened to identify those that had a diagnosis of underweight or emaciated (hereafter referred to as underweight), chronic kidney disease (CKD), hyperthyroidism (hyperT4), diabetes mellitus (DM) and osteoarthritis (OA). Some key facts emerged and are shown below.



Underweight cats were at least **11 times** more likely to be diagnosed with CKD than cats that were not underweight.

Weight loss is a risk factor for developing CKD (2) and continued weight loss after CKD diagnosis can affect cat survival (3)

Cats with CKD were **6.9 times** more likely to be underweight than those without CKD.

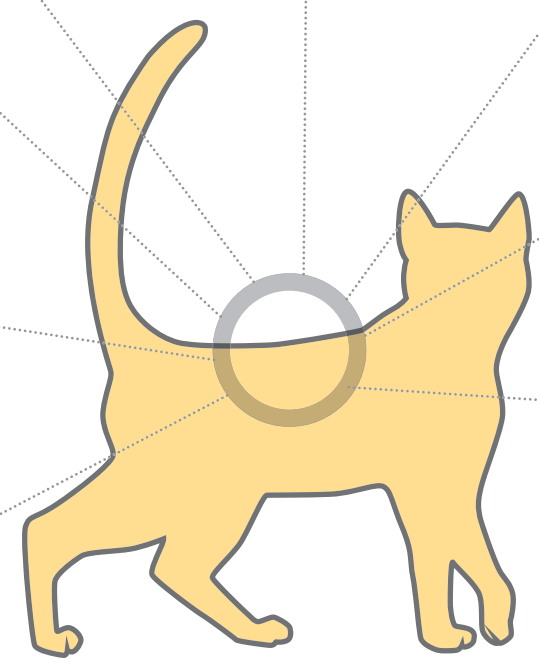
Hyperthyroid cats were **8.3 times** more likely to be underweight.

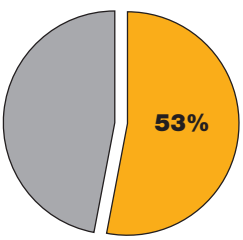
Cats with OA were **3.7 times** more likely to be underweight.

Underweight cats were **20 times** more likely to be diagnosed with hyperthyroidism than cats that were not underweight.

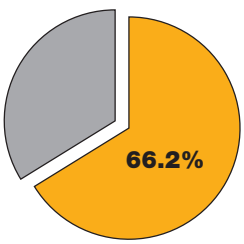
Cats with DM were **3.1 times** more likely to be underweight.

11.1% of underweight cats were diagnosed with hyperT4.

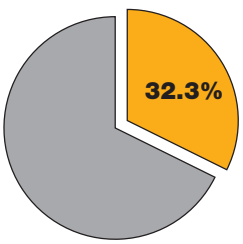




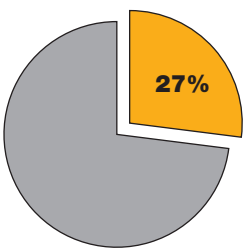
53% of cats with **CKD** were **underweight**



66.2% of cats with **hyperT4** were **underweight**



32.3% of cats with **OA** were **underweight**



27% of cats with **DM** were **underweight**

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