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The Growing Problem of Obesity in Dogs and Cats[1–3]

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ABSTRACT Obesity is defined as an accumulation of excessive amounts of adipose tissue in the body, and is the most common nutritional disorder in companion animals. Obesity is usually the result of either excessive dietary intake or inadequate energy utilization, which causes a state of positive energy balance. Numerous factors may predispose an individual to obesity including genetics, the amount of physical activity, and the energy content of the diet. The main medical concern of obesity relates to the many disease associations that accompany the adiposity. Numerous studies demonstrated that obesity can have detrimental effects on the health and longevity of dogs and cats. The problems to which obese companion animals may be predisposed include orthopedic disease, diabetes mellitus, abnormalities in circulating lipid profiles, cardiorespiratory disease, urinary disorders, reproductive disorders, neoplasia (mammary tumors, transitional cell carcinoma), dermatological diseases, and anesthetic complications. The main therapeutic options for obesity in companion animals include dietary management and increasing physical activity. Although no pharmaceutical compounds are yet licensed for weight loss in dogs and cats, it is envisaged that such agents will be available in the future. Dietary therapy forms the cornerstone of weight management in dogs and cats, but increasing exercise and behavioral management form useful adjuncts. There is a need to increase the awareness of companion animal obesity as a serious medical concern within the veterinary profession. J. Nutr. 136: 1940S–1946S, 2006.

KEY WORDS: • *overweight* • *nutrition* • *canine* • *feline* • *adipose tissue*

Obesity is defined as an accumulation of excessive amounts of adipose tissue in the body (1). In humans, the application of this definition is based upon epidemiologic data, which demonstrate increased morbidity and mortality risk with increasing body fat mass. Criteria have been established for what constitutes "overweight" and what constitutes "obesity"; such criteria are usually based on measures of adiposity such as the BMI [weight (kg) divided by height^2 (m)]; Caucasians, for example, are defined as overweight when BMI is >25 kg/m^2, and obese when BMI exceeds 30. In contrast, one report classified cats and dogs as overweight when their body weight is $>15\%$ above their "optimal body weight," and as obese when their body weight exceeds 30% of optimal (1). However, these criteria have not been confirmed with rigorous epidemiologic studies, and limited data exist on the nature of an optimal body weight.

Obesity is an escalating global problem in humans (2), and current estimates suggest that almost two-thirds of adults in the United States are overweight or obese (3). Studies from various parts of the world have estimated the incidence of obesity in the dog population to be between 22 and 40% (4). The most recently published data come from a large study in Australia in which 33.5% of dogs were classed as overweight, whereas 7.6% were judged to be obese (4). The incidence of feline obesity is similar (1,5,6). Most investigators agree that, as in humans, the incidence in the pet population is increasing.

Measurement of obesity in companion animals

All measures of adiposity involve defining body composition, or the "relative amounts of the various biological components of the body." The main conceptual division of importance is between fat mass (FM,[5] the triglyceride component in adipose tissue) and lean body mass (LBM) (7). Various techniques are available to measure body composition (**Table 1**), and these differ in applicability to research, referral veterinary practice, and first-opinion practice. Whatever method is used, investigators should be aware of both its precision and accuracy. The accuracy of a test is defined as the closeness with which

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[5] Abbreviations used: CLA; conjugated linoleic acid; cTSH, canine thyroid-stimulating hormone; DM, diabetes mellitus; DXA, dual-energy X-ray absorptiometry; FM, fat mass; HDL-C, HDL cholesterol; LBM, lean body mass; LIM, limb index measurement; T3, triiodothyronine; T4, thyroxine; USMI, urethral sphincter mechanism incompetence.

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TABLE 1

Methods for body composition analysis in dogs and cats

Common research techniques
Chemical analysis
Densitometry
Total body water
Isotope dilution
Deuterium
Tritium
Total body potassium
Absorptiometry
Photon absorptiometry
Single-photon absorptiometry (SPA)
Dual-photon absorptiometry (DPA)
X-ray absorptiometry
Single-energy X-ray absorptiometry (SXA)
Dual-energy X-ray absorptiometry (DXA or DEXA)
Ultrasound
Subcutaneous
Visceral
Electrical impedance
Common clinical methods
Body weight
Morphometric methods (zoometry)
Body condition score
9-Point scale
5-Point scale
6-Point scale
Tape measurements
BMI
Other methods
Muscle metabolite markers
Neutron activation analysis
Electrical conductance (bioelectrical impedance)
Near infrared interactance (NIRI)
Computed tomography
MRI

a measurement of the variable represents its true value, whereas precision is the ability to yield the same estimated result on repeated analysis (irrespective of accuracy). Ideally, a test that is both accurate and precise should be used; however, many tests for body composition are precise but not accurate, whereas some lack both precision and accuracy. Other important aspects of a test are cost, ease of use, acceptance by veterinarians and clients, and invasiveness. Currently, there is no method that cannot be criticized; therefore, the perfect tool for analysis does not yet exist.

Potential research techniques include chemical analysis, densitometry, total body water measurement, absorptiometry [including dual-energy X-ray absorptiometry (DXA)], ultrasonography, electrical conductance, and advanced imaging techniques (computed tomography and MRI; Table 1). In the clinical setting, there is a need for quick, inexpensive, and noninvasive methods of body composition measurement. The most widely adopted quantitative procedures include measurement of body weight and morphometry.

Morphometry. This is defined as the measurement of "form"; in relation to body composition analysis, it refers to a variety of measured parameters that are used to estimate body composition. The 3 main approaches are measurement of skinfold thickness, dimensional evaluations (in which various measures of stature are combined with weight), and body condition scores.

Dimensional evaluations. Such evaluations are usually performed by tape measure, and a number were reported in dogs and cats. Measurements of "length" (e.g., head, thorax, and limb) are correlated with lean body components (8), whereas measurements of girth were shown to correlate with both LBM (8) and FM (9). Segmental limb measures and (likely) truncal length are thought to be better measures of stature and thus correlate best with LBM. By combining >1 measure (usually 1 that correlates with FM, and 1 correlating with LBM), equations can be generated to predict different body components. The best example of such a measure is the feline BMI (9), where:

$$\text{Body fat}(\%) = \{[(\text{Ribcage}/0.7067) - \text{LIM}]/0.9156\} - \text{LIM} \quad [1].$$

Here, the ribcage measurement is the circumference measured at the 9th rib, and LIM stands for the "limb index measurement," which is the distance between the patella and calcaneus of the left hindlimb. All measurements are made in centimeters, and measurements are made with the cat in a standing position, with the legs perpendicular to the ground and the head upright.

Such techniques do provide a more objective measure of body composition than body condition scoring (see below), but problems exist when similar schemes are extrapolated to the many breeds of dog. Despite this, a BMI has been suggested for dogs (10).

Body condition scoring. This is a subjective, semiquantitative method of evaluating body composition. A number of schemes were devised, with a 9-point scheme being the most widely accepted (11,12). All systems assess visual and palpable characteristics that correlate subcutaneous fat, abdominal fat, and superficial musculature (e.g., ribcage, dorsal spinous processes, and waist). A new 7-point algorithm-based approach, specifically designed to be used by owners to assess their own pets, was developed recently. A recent study demonstrated good correlation between the system and body fat measurements made by DXA and excellent agreement among experienced operators (13). Most importantly, good agreement was found between measurements by the experienced operators and the owners, suggesting that the method is reliable when used without prior training.

Causes of obesity

Although some diseases (e.g., hypothyroidism and hyperadrenocortism in dogs), pharmaceuticals (e.g., drug-induced polyphagia caused by glucocorticoids and anticonvulsant drugs), and rare genetic defects (in humans) can cause obesity, the main reason for the development of obesity is having a positive mismatch between energy intake and energy expenditure. Therefore, either excessive dietary intake or inadequate energy utilization can lead to a state of positive energy balance; numerous factors may be involved, including genetics, the amount of physical activity, and the energy content of the diet (1).

The effect of genetics is illustrated by recognized breed associations in both dogs (e.g., Labrador Retriever, Cairn Terrier, Cavalier King Charles Spaniel, Scottish Terrier, Cocker Spaniel) and cats (e.g., Domestic Shorthair) (14,15).

Neutering is an important risk factor for obesity in both species; many studies suggested that this is due to a decrease in metabolic rate after neutering (16–19). However, increased FM is usually present in neutered animals; when energy expenditure is expressed on a lean mass basis, no difference in metabolic rate is noted between neutered and entire individuals (20–23). Alternative explanations for the effect of neutering on obesity is an alteration in feeding behavior leading to increased food intake (17,18,21–25), and decreased activity without a corresponding decrease in energy intake (26,27). Gender itself is also a predisposing factor in some canine studies, with females overrepresented (14,28). Other recognized

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associations in dogs include indoor lifestyle and middle age (4,14,15). In cats, middle age and apartment dwelling are possible risk factors (6).

Dietary factors can also lead to the development of obesity in both species. For instance, obesity in dogs is associated with the number of meals and snacks fed, the feeding of table scraps, and the dog's presence when its owners prepared or ate their own meal (29). Interestingly, the type of diet fed (prepared pet food vs. homemade) does not appear to predispose to obesity (14,15,29). However, the price of the pet food does have a notable effect, i.e., obese dogs are more likely to have been fed inexpensive rather than more expensive foods. Further, obese cats more commonly have a free choice of food intake (30).

Behavioral factors also play a part in the development of obesity. For cats, possible factors involved in the development of obesity include anxiety, depression, failure to establish a normal feeding behavior, and failure to develop control of satiety (31). The human-animal relationship is also of importance and was shown to be more intense in the owners of obese cats (30). Further, misinterpretation of feline behavior on the part of the owner is also of importance; in this regard, many owners misread signals about the behavior of their cat associated wth eating. In contrast to humans and dogs for whom eating is a social function, cats do not have any inherent need for social interaction during feeding times. When the cat initiates contact, owners often assume that they are hungry and are asking for food when they are not (31). Nevertheless, if food is provided at such times, the cat soon learns that initiating contact results in a food reward. For dogs, owner factors that are of importance include the duration that the owner observed the dog eating (more likely to be longer in obese dogs), interest in pet nutrition, obesity of the owner, health consciousness of the owner (both for their pet and themselves), and lower income (29).

The pathological importance of obesity

In humans, obesity is important because it increases mortality risk and can predispose to a variety of diseases. Obese humans, on average, do not live as long, and are more likely to suffer from diseases such as type II diabetes mellitus (DM), hypertension, coronary heart disease, certain cancers (e.g., breast, ovarian, prostate), osteoarthritis, respiratory disease, and reproductive disorders. Similarly, obesity has detrimental effects on the health and longevity of dogs and cats (**Table 2**), although data are more limited. Problems to which obese companion animals may be predisposed include orthopedic disease, DM, abnormalities in circulating lipid profiles, cardiorespiratory disease, urinary disorders, reproductive disorders, neoplasia (mammary tumors, transitional cell carcinoma), dermatological diseases, and anesthetic complications. Human obesity is associated with an increased risk of type II DM, cancer, cardiac disease, hypertension, and decreased longevity (32). Some studies do suggest an increase in morbidity in sick patients with poorer body condition (33,34).

Clinical evaluation, physiology and anesthesia. Overall, obesity makes clinical evaluation more difficult; techniques that are more problematic in obese patients include physical examination, thoracic auscultation, palpation and aspiration of peripheral lymph nodes, abdominal palpation, blood sampling, cystocentesis, and diagnostic imaging (especially ultrasonography). Anesthetic risk is reportedly increased in obese companion animals, most likely due to recognized problems with estimation of anesthetic dose, catheter placement, and prolonged operating time (35,36). Finally, decreased heat tolerance and stamina were also reported in obese animals (1).

TABLE 2

Diseases reported to be associated with obesity in companion animals

Metabolic abnormalities
Hyperlipidemia/dyslipidemia
Insulin resistance
Glucose intolerance
Metabolic syndrome
Hepatic lipidosis (cat)
Endocrinopathies
Hyperadrenocorticism
Hypothyroidism
Diabetes mellitus
Insulinoma
Hypopituitarism
Hypothalamic lesions
Orthopedic disorders
Osteoarthritis
Humeral condylar fractures
Cranial cruciate ligament rupture
Intervertebral disk disease
Cardiorespiratory disease
Tracheal collapse
Brachycephalic airway obstruction syndrome
Laryngeal paralysis
Urogenital system
Urethral sphincter mechanism incompetence
Urolithiasis (calcium oxalate)
Transitional cell carcinoma
Dystocia
Neoplasia
Mammary
Transitional cell carcinoma
Functional alterations
Joint disorders
Respiratory compromise, e.g., dyspnea
Hypertension
Dystocia
Exercise intolerance
Heat intolerance/heat stroke
Decreased immune functions
Increased anesthetic risk
Decreased lifespan

Longevity. Dietary restriction can increase longevity in other species (37–39), and a recent prospective study confirmed a similar effect in dogs (40–45). Labrador retrievers (24 pairs, 48 in total) participated in the study, and 1 dog in each pair was randomly assigned to 1 of 2 groups (43). The dogs in one group consumed food ad libitum, whereas the dogs in the other group were fed 75% of the amount consumed by their counterparts. In the energy-restricted group, the body condition score was closer to "optimal" (e.g., group mean 4.5/9) than in the ad libitum feeding group (e.g., group mean 6.8/9). Although causes of death did not differ between the 2 groups, the lifespan was increased in the energy-restricted group (e.g., median 13 y with energy restriction vs. 11.2 y with ad libitum consumption) (45). Additional beneficial effects of feed restriction (and thus maintenance of body condition) included a reduced risk of hip dysplasia and osteoarthritis, and improved glucose tolerance (40–45).

Diseases associated with obesity

Endocrine and metabolic diseases. Hormonal diseases with a reported association with obesity include DM, hypothyroidism, hyperadrenocorticism, and insulinoma (1). Some conditions predispose to obesity, whereas others arise more commonly

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in animals that are obese. Acromegaly can lead to a generalized increase in tissue mass, and is thus a differential diagnosis for obesity. However, in this condition, lean tissue and bone mineral are likely to be deposited in addition to adipose tissue.

Insulin resistance, DM, and the metabolic syndrome. Insulin secreted by pancreatic β cells controls the uptake and use of glucose in peripheral tissues. In humans, tissues become less sensitive to insulin (i.e., become "insulin resistant") with excessive energy intake (46), and plasma concentrations of insulin increase in direct proportion to increasing BMI in both men and women (47). Thus, obesity, particularly abdominal obesity, is a major determinant of insulin resistance and hyperinsulinemia (48). Cats most often suffer from DM, which resembles "type II" DM in humans; therefore, obesity is a major risk factor in this species (49). Indeed, it was proven experimentally that diabetic cats have significantly lower sensitivity to insulin than cats without DM (50). In contrast, dogs more commonly suffer from DM resembling human type I DM. Obesity causes insulin resistance (45), and obesity is a risk factor for DM in this species (51). However, because type II DM is uncommon in dogs, obesity rarely leads to overt clinical signs of DM (52).

In humans, the metabolic syndrome was originally termed "syndrome of insulin resistance"; in fact, it is a group of risk factors associated with both insulin resistance and cardiovascular disease (53). The main characteristics of metabolic syndrome are as follows: *1*) fasting plasma glucose $>$ 110 mg/dL (6.10 mmol/L); *2*) visceral obesity (e.g., waist circumference $>$ 90 cm in women and $>$ 102 cm in men; *3*) Hypertension e.g., blood pressure $>$ 130/85 mm Hg; and *4*) low concentrations of HDL cholesterol (HDL-C; $<$40 mg/dL in men, $<$ 50 mg/dL in women).

Additional features may include systemic inflammation, prothrombotic state, and increased oxidant stress (54). Further, in ~20% of cases of metabolic syndrome, there is concurrent pancreatic β-cell dysfunction leading to DM (53). Some of these criteria were applied to dogs, and this species is often used as a model for human metabolic syndrome (55).

Hypothyroidism and thyroid function. Although hypothyroidism is commonly cited as an underlying cause for obesity, such cases are the exception rather than the rule. The prevalence of hypothyroidism in dogs is estimated at 0.2%, with less than half of these dogs reported to be obese (56). In contrast, the proportion of dogs that are obese is much greater (25–40%) (4). Hypothyroidism is extremely rare in cats. Thus, although hypothyroidism should always be considered, it is rarely the reason for obesity. Obesity itself has a subtle, but likely clinically insignificant effect on thyroid function (57); obese dogs had higher concentrations of both total thyroxine (T4) and total triiodothyronine (T3) than nonobese controls, although such concentrations remained within the reference range and other parameters [e.g., free T4, canine thyroid-stimulating hormone (cTSH), TSH stimulation test] did not differ. Further, weight loss caused significant decreases in total T3 and cTSH. Thus, although obesity and subsequent weight restriction may have some effects on energy balance and thyroid homeostasis, such changes are unlikely to affect the interpretation of thyroid function tests.

Hyperlipidemia and dyslipidemia. Limited data exist for dogs with naturally occurring obesity, and most information was derived from experimental studies. Published data suggest that lipid alterations can occur in obese dogs, with increases in cholesterol, triglycerides, and phospholipids all noted, albeit often not exceeding the upper limit of the reference range (58–60). Making laboratory dogs obese by feeding a hyperenergetic diet was shown to increase plasma nonesterified fatty acid and triglyceride concentrations by increasing concentrations of VLDL and HDL, while decreasing those of HDL-C (59). Such changes were associated with insulin resistance and, interestingly, were also described in insulin-resistant humans. Whether lipid alterations account for the increased incidence of pancreatitis in obese dogs requires further studies (61). Thus, additional work is warranted to assess further the significance of lipid abnormalities in dogs.

Orthopedic disorders. Obesity is a major risk factor for orthopedic diseases in companion animals, especially dogs. An increased incidence of both traumatic and degenerative orthopedic disorders was reported (14,62). One study reported body weight to be a predisposing factor in humeral condylar fractures, cranial cruciate ligament rupture, and intervertebral disc disease in cocker spaniels (63). A recent study in boxers reported a link between neutering and hip dysplasia (64); although the effect of obesity was not assessed directly in that study, this association was attributed to an increased incidence of obesity in neutered dogs. Further, a number of studies highlighted the association between obesity and the development of osteoarthritis (41,42), whereas weight reduction can lead to a substantial improvement in the degree of lameness in dogs with hip osteoarthritis (65).

Cardiorespiratory disease and hypertension. Obesity can have a profound effect on respiratory system function. Most notably, obesity is an important risk factor for the development of tracheal collapse in small dogs (66). Obesity can exacerbate heatstroke in dogs; other respiratory diseases that can be exacerbated by obesity include laryngeal paralysis and brachycephalic airway obstruction syndrome. Obesity can also affect cardiac function; increased body weight can result in effects on cardiac rhythm and increased left ventricular volume, blood pressure, and plasma volume. The effect of obesity on hypertension is controversial in dogs. One study suggested that obesity was significantly associated with hypertension, but its effect was only minor (67). In contrast, many experimental studies utilized the obese dog as a model for the pathogenesis of hypertension and insulin resistance (68). Obesity may also be associated with portal vein thrombosis (69) and myocardial hypoxia (70).

Urinary tract and reproductive disorders. There is evidence from experimental dogs that the onset of obesity is associated with histologic changes in the kidney, most notably an increase in Bowman's space (as a result of expansion of the Bowman's capsule), increased mesangial matrix, thickening of glomerular and tubular basement membranes, and an increased number of dividing cells per glomerulus (71). Functional changes were noted in the same study and included increases in plasma renin concentrations, insulin concentrations, mean arterial pressure, and plasma renal flow. As a consequence, the authors speculated that these changes, if prolonged, could predispose to more severe glomerular and renal injury. An association between obesity and some cases of urethral sphincter mechanism incompetence (USMI) was reported. Obesity is not the only risk factor, with ovariohysterectomy (and consequent lack of sex hormones) itself also playing a major role. Nevertheless, the effect of obesity is clear in some dogs that become incontinent only when they become obese. Further, weight reduction in overweight dogs with USMI can often be all that is required for continence to be restored. The mechanisms that predispose obese animals to USMI are not known, although it was suggested that the effect is purely mechanical, e.g., increased retroperitoneal fat leading to caudal displacement of the bladder (72). The risk of developing calcium oxalate urolithiasis is also reported to be increased in obese dogs (73). Finally, obese animals are reported to suffer

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from an increased risk of dystocia, likely related to excess adipose tissue in and around the birth canal (14,74,75).

Neoplasia. In humans, obesity predisposes to a number of different types of cancer; the International Agency for Research on Cancer found a significant link between obesity and cancers of the female breast (postmenopausal), colon/rectum, kidney (renal cell), and esophagus (47). It is estimated that, if this link is entirely causal, 1 in 7 cancer deaths in both men and women in the United States might be the direct result of being overweight or obese (47). Breast cancer is the most common form of cancer among women (76), and obesity was shown consistently to increase rates of breast cancer in postmenopausal women by 30–50% (48). An association between mammary carcinoma and obesity was also reported in some (74) but not all (77,78) canine reports. Overweight dogs were also reported to have an increased risk of developing transitional cell carcinoma of the bladder (79).

Miscellaneous disorders. Obese animals were reported to be at increased risk of certain dermatologic disorders. Diffuse scale is commonly observed (especially in cats), most likely due to a reduced ability to groom efficiently. Animals that are severely obese can develop pressure sores. Decreased immune function has also been documented, with obese dogs showing less resistance to the development of infections (80,81).

Treatment of obesity

In humans, current therapeutic options for obesity include dietary management, exercise, psychological and behavioral modification, drug therapy, and surgery. Many of these options are available for companion animals, although it is not ethically justifiable to consider surgical approaches. Further, to date, there are no pharmaceutical compounds licensed for weight loss in dogs and cats. Dietary therapy forms the cornerstone to weight management in dogs and cats, but increasing exercise and behavioral management comprise useful adjuncts.

Dietary management. It is recommended that the weight reduction protocol be tailored toward the individual patient. Although total energy restriction (starvation) successfully leads to weight loss, it has the disadvantages of causing excessive protein (and thus lean body mass) loss and requiring hospitalization for proper monitoring (1). Therefore, it is preferable to use purpose-formulated weight reduction diets, which generally are restricted in fat and energy, while being supplemented in protein and micronutrients. Protein supplementation is important because the amount of lean tissue lost is minimized even though the weight loss is not more rapid (82,83). Supplementation of micronutrients ensures that deficiency states do not arise (84,85).

Additional dietary factors that may be of benefit for weight loss include L-carnitine supplementation (to maintain lean mass), conjugated linoleic acid (CLA), and the use of high-fiber diets (to provide satiety).

L-Carnitine is an amino acid that is synthesized de novo in the liver and kidneys from lysine and methionine in the presence of ascorbate. Dietary supplementation of L-carnitine improves nitrogen retention, increasing lean mass and reducing fat mass (86). Incorporation of L-carnitine at a level of 50–300 ppm, in weight reduction diets, was shown to reduce lean tissue loss during weight loss (86,87). Possible mechanisms for this protective effect on lean tissue include enhancing fatty acid oxidation and energy availability for protein synthesis during times of need.

CLA is a family of fatty acid isomers derived from linoleic acid. Various studies in experimental animals suggested that it has an antiadipogenic effect; proposed mechanisms include inhibition of stearoyl-CoA desaturase activity, which limits the synthesis of monounsaturated fatty acids for triglyceride synthesis, and suppression of elongation and desaturation of fatty acids into long-chain fatty acids (86). At present, data on the use of CLA as an antiobesity agent in humans and cats are conflicting, with the most recent data suggesting the lack of a significant effect (88,89). Therefore, more information is required before its use can be recommended. There is also controversy concerning the effect of fiber satiety; some reports suggested that feeding up to 12–16% of dry matter as dietary fiber has no effect (90–92), whereas other work demonstrated appetite suppression when 21% of the diet was consumed as dietary fiber (93).

Lifestyle management. Increasing physical activity is a useful adjunct to dietary therapy; when used in combination with dietary therapy, it promotes fat loss (94) and may assist in lean tissue preservation (95). There is also some evidence that exercise may help prevent the rapid regain in weight that can occur after successful weight loss (94). The exact program must be tailored to the individual and take into account any concurrent medical concerns. Suitable exercise strategies in dogs include lead walking, swimming, hydrotherapy, and treadmills. Exercise in cats can be encouraged by increasing play activity, using cat toys (e.g., fishing rod toys), motorized units, and feeding toys. Cats can also be encouraged to "work" for their food by moving the food bowl between rooms before feeding, or by the use of feeding toys.

Monitoring of weight loss. In addition to the above strategies, it is essential that the whole weight reduction regimen be supervised. This is labor intensive, requires some degree of expertise and training in owner counseling, and often requires a dedicated member of staff. Nevertheless, in the author's opinion, correct monitoring is the single most important component of the weight loss strategy. A recent study demonstrated that weight loss is more successful if an organized strategy is followed with regular weigh-in sessions (96). It is essential to continue to monitor body weight after the ideal weight has been achieved to ensure that weight that was lost is not regained; as with humans, a rebound effect was demonstrated after weight loss in dogs (97).

Summary

Obesity is a growing concern in companion animals, and the increasing incidence appears to be mirroring the trend observed in humans. The main medical concern of obesity relates to the many disease associations that accompany the adiposity. There is a need to increase awareness within the veterinary profession that obesity in companion animals is a serious medical concern.

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