Controversies in Pet Obesity Management

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Abstract
Controversy occurs in science when there are gaps in our understanding, and there are a surprising number of gaps in our understanding of obesity in cats and dogs. This talk will discuss several of these gaps, which include nebulous definitions, illusions of accuracy, inadequate evidence, and a basic lack of knowledge.

Glossary of Abbreviations
AAFCO: Association of American Feed Control Officials
BCS: Body Condition Score
BFM: Body Fat Mass
BMI: Body Mass Index
BW: Body Weight
CCK: Cholecystokinin
FEDIAF: European Pet Food Industry Federation
LBM: Lean Body Mass
MER: Maintenance Energy Requirement

Introduction
Controversy and uncertainty are neither synonyms nor inevitable bedfellows. Scientific method demands that when there is uncertainty, we pose hypotheses, then attempt to disprove them. When we have failed sufficiently to disprove a hypothesis, we believe it is true. Controversy can only exist when we are shrouded in the fog of ignorance and emotionally attach ourselves to a hypothesis. Controversy can motivate science, but it is neither required nor necessarily helpful. Controversies in obesity management are, therefore, positions of ignorance in which members of the community hold unfounded or unproven positions. Obesity is easy to diagnose, is completely preventable, and, in theory, easily treatable. Despite that, the prevalence appears to be increasing, and our success in prevention and management on a population level is dubious. It appears that our understanding of the true impediments to obesity prevention and management is incomplete. Sadly, in veterinary science our body of knowledge generally resembles less of a wall of carefully accreted bricks of understanding and more of a disappointing Swiss cheese dominated by holes in places. This talk examines some of these holes.

Defining Obesity
Obesity is defined by the Oxford English dictionary as "the condition of being extremely fat or overweight." In the veterinary literature, it is defined as an "accumulation of excess body fat"[1] Although such lexical definitions may be suitable for lay use, they do not discriminate between degrees of excess, justify the basis of excess, or suggest a biological difference between obese and mildly overweight other than the mass of fat.

Standardization of the terms "overweight" and "obese" can only occur through arbitrary consensus or preferably through validation by observation of the association of objective disease phenomena and certain body fat mass (BFM) percentages. Although the incidences of certain diseases are strongly associated with "obesity" in dogs and cats, not all animals at a given fat percent will develop a given disease. Thus, individual and environmental factors are involved. It has been strongly argued that human obesity should be defined as a disease that "is an impairment of the normal state of the human organism and modifies vital bodily functions."[2] Even with this definition, there is a clear difference between the effects of visceral adiposity and subcutaneous adiposity in disease risk, such as cardiovascular disease, and thus BFM% is a blunt measure of disease risk.[3] Thus, an objective and physiologically meaningful definition of obesity in dogs and cats remains elusive until the BFM percentage at which the normal state is impaired has been established. In the absence of a validated definition of the BFM threshold that marks obesity, obesity can still be defined in general terms. However, those general terms should include a component of disease. A suggestion is to define obesity as "an excess of body fat that is associated with disease or the increased risk of disease," but an explicit caveat that the exact BFM percentage at which a given animal would be termed obese is variable and significantly influenced by the individual.

Trajectory to Obesity
Obesity is a completely preventable disease that is frustratingly difficult to manage in client-owned animals. Thus, there has been a historical overemphasis on research and education in understanding and managing the obese state and inadequate attention given to the prevention of obesity. Sadly, the risk factors for obesity are still not well defined, and many may be difficult to address (e.g., activity), or impractical to prevent (e.g., neutering). However, there may be key periods during growth or adulthood during which intervention could be targeted to reduce the incidence of obesity. More effort

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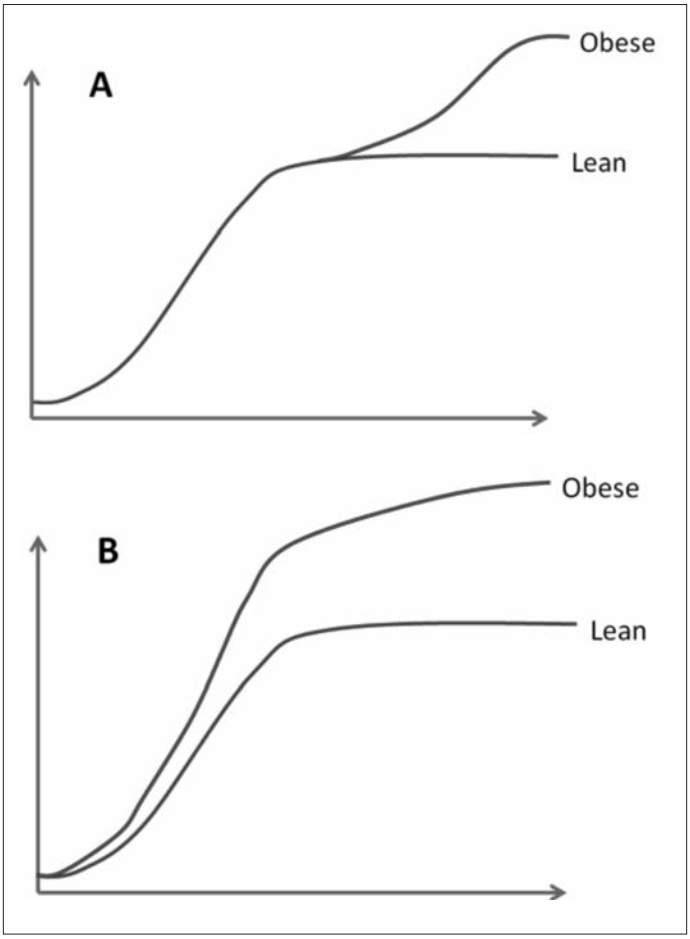
should be directed toward understanding the trajectory to obesity in client-owned animals.

The development of the obese state could follow two theoretical body mass pathways:



In pathway **A**, the individual grows normally, and then accrues excessive body fat mass in adulthood. In this pathway, it is suggested that the obese phenotype is more determined by environmental factors, or factors that were operative during adulthood, than a predetermined type. In pathway **B**, the obese phenotype is largely predetermined, and the individual's growth rate and mature body weight are greater. Genetics, the perinatal environment or early life events would appear to be most influential. In order to maximize the effectiveness of obesity prevention schemes, it is essential that we understand which broad scenario exists in each species being considered.

In humans, the risk of obesity is influenced heavily by several perinatal variables. Early risk factors for obesity include being born post-term (males), maternal overweight and obesity during pregnancy, higher birth weight or "catch up" during the early postnatal, and formula feeding rather than breast feeding.[4] Thus, the obese phenotype can be identified early in life, and the emphasis for obesity prevention must then be directed toward childhood. In veterinary science, we have made little to no progress understanding the typical trajectory of either dogs or cats largely because we have limited our research to cross-sectional surveys in adult animals in which there is no data on juvenile or neonatal factors let alone maternal or prenatal factors. Recent findings in colony animals suggest that, similar to humans, there may be significant early influences on the trajectory.[5] Previously unconsidered variables such as the influence of the enteric microflora and season of birth now need to be investigated.[6] Future studies are thus needed to elucidate the relative contribution of early or adult-onset risk factors.

Several cross-sectional studies have identified risk factors present in adult obese animals that are different than studies reporting opposite results. For instance, the provision of *ad libitum* feeding has been shown to increase the risk of feline obesity in some studies, while in others it was either not significant or protective.[7,8] Similarly, the feeding of scraps to dogs was shown to cause a decreased risk if fed frequently in one study, while in another study it was shown to be associated with an increased risk if fed infrequently.[9] However, it appears true that no particular feeding practice is a strong risk factor for cats, while dogs are less accurate at regulating intake and *ad libitum* feeding cannot be considered a sensible approach. It is tempting to suggest that this species difference relates to their ancestral dietary approaches, whereby wild canids will often consume large meals infrequently and small felids often eat small meals throughout the day. For both cats and dogs, the most consistent risk factors for obesity other than age are being neutered, low activity, and the owners' underestimation of their pet's body condition. As mentioned, there are conflicting results between studies caused mostly by differences in sampling strategies, survey questions, geography, and sample populations.

Several studies have documented the association between low activity and obesity, especially in dogs, though as most authors note, cross-sectional studies cannot differentiate causality from effect.[10] Low activity as a risk seems intuitive: Reduced activity decreases energy expenditure and, therefore, if food intake remains the same, weight gain will result. However, this statement requires that food intake remains the same for it to be true. Food intake is surprisingly accurately matched to fluctuations in energy expenditure in normal animals, thus for inactivity to lead to obesity there still needs to be dysregulation in the control of food intake. Alternatively, for activity to prevent obesity there has to be an effect of activity on the regulation of food intake. Although not studied mechanistically in either dogs or cats, it is likely that inactivity is associated with inappropriately high intake and that a certain amount of activity leads to a reduction in intake relative to the increased requirements, thus it appears that sustained "regular activity" decreases the risk of obesity. The amount and type of activity necessary has not been defined.

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The Control of Food Intake

Appropriate food intake requires control of both meal initiation (signalled by hunger) and meal cessation (signalled by satiety). In most species, oestrogen is a key negative regulator of food intake that functions by increasing the sensitivity to cholecystokinin (CCK) released during a meal and leading to satiety.[11] Gonadectomy of both male and female cats results in a significant and rapid increase in food intake, which is reversible with exogenous estrogen, and the effect is very likely to occur in bitches as well.[12] With a sustained increase in food intake as BFM increases, more of the hormone leptin is released from adipocytes, which is supposed to function as a controller of food intake promoting early meal cessation. However, this supposes that mammals have evolved control mechanisms to avoid obesity. Rather, it is likely that leptin functions less as an inhibitor of food intake, so when BFM decreases, the decreased leptin acts as a facilitator of increased hunger to restore a healthy BFM.

Most animals can regulate food intake appropriately within a wide range of energy densities, and a high-energy density per se is not predicted to lead to obesity. However, when changing from one to another energy density, there is much better compensation when changing to a low-density food than to a high-density food. Thus, when switched from a high- to low-energy density food, a cat or dog may lose weight briefly, then adapt and put weight back on to restore its original mass. However, when switched from a low- to a high-energy density diet, there is a period of over-intake and weight gain followed by a normalization in energy intake, but not a compensatory decrease to lose the weight gained.[13] This emphasizes that most mammals have evolved in an environment where access to nutrients is limiting, and the evolutionary drive is to be efficient at securing food. There has not been evolutionary pressure to protect against obesity, thus there is much more accurate defense against energy deficiency than energy excess. This probably explains the effect of feeding scraps to dogs. Thus, it may be that variety feeding, not a variable that has been specifically studied in cross-sectional studies, is more important than specific dietary characteristics.

MER Calculations and Feeding Guidelines

Feeding guides provided by pet food companies are the bane of their existence. A good manufacturer is expected to provide a feeding guide with its food, but guides are a double-edged sword. For both dogs and cats, the variation in individual maintenance energy requirement (MER) is massive.[14,15] Although the majority of animals lie within a few percentages of the mean, it is sobering to remember the consequences of an animal consuming 5% more than its MER. For a 20 kg dog with an MER of 3052 kJ, that equates to 152 kJ per day. This dog will accrue body fat at a rate of approximately 5g/day, or 1.8kg/year, which is a nearly 10% increase in body weight per year.

Even if an owner knew the exact requirements of their pet and if they use a measuring cup to measure the food, they may inadvertently measure as much as 80% more than intended.[16] To make matters worse, the amount owners feed is influenced by the size of the food bowl and scoop used to dispense dry food.[17] So, the exact amount of food an animal needs is not known. Animals of the same weight can vary enormously in their requirements, i.e., a neutered animal is unable to regulate its food intake normally, and there is great inaccuracy in the normal methods of food allocation. Thus, the emphasis from veterinarians must shift from the dietary factors to feeding to "ideal" body condition, or to maintain an established ideal weight.

Ideal Body Weight (BW)

The ideal body condition score (BCS) has traditionally been defined as 4-5/9 for dogs, and 5/9 for cats. However, though widely used and likely to be reasonable, these categorizations are not based on objective measurements of health. Just as the definition of "obese" requires consideration of the BFM at which the risk of disease or shortened life span increases, so should the definition of ideal be based on the BFM associated with the lowest disease and greatest longevity. It is likely that large-scale epidemiological studies will produce a "U-shaped" association between BCS and disease similar to the association between body mass index (BMI) and all-cause mortality in humans.[18] It may well be that ideal BCS is less than 4/9 — currently defined as "underweight." If the ideal BCS range is found to be nonoverlapping with "quality of life" or freedom from hunger, then the two will need to be traded against each other to produce an optimal compromise.

Success of Weight Loss

The definition of "success" is open to interpretation. Definitions in the literature range from "some" weight loss to attainment of the target (ideal) weight. No study has yet considered maintenance of lean body weight as the definition. In addition to the definition, studies have reported varying denominators as measures of success. They have ranged from the percent of animals that completed the program to the percent of animals enrolled that met the criteria for success. Studies that report the dropout rate in home settings indicate almost 50% failure to complete the program.[19] No study has yet considered the percent of obese animals in the practice that become lean and remain lean for a significant proportion of their life.

Gene Transcription Studies

There are now several studies that have documented changes in gene transcription associated with obesity and weight loss. Typical findings include changes of the expression of genes involved in fat and carbohydrate metabolism,

oxidative defence, and proteome and mitochondrial function.[20] Although it is unquestionable that such studies expand our understanding of the metabolic derangements associated with obesity, they have not yet fundamentally altered the approach to management nor provided an argument for the superiority of assaying gene transcription over simply measuring BFM. The value of this approach to understanding obesity remains uncertain.

Dietary Composition and Risk of Malnutrition During Weight Loss

Several studies have suggested superiority of one dietary composition over another in experimental weight-loss settings. Perhaps most notable is the effect of feeding a diet that is higher in protein than that required for maintenance on reducing lean body mass (LBM) loss during weight reduction.[21,22] When feeding to produce an energy deficit to stimulate BFM catabolism and weight loss, there may be accompanying nutritional deficiency of essential nutrients. The theoretical concerns of inducing weight loss using diets formulated for maintenance have recently been raised.[23] Consistent with these concerns are reports in humans undergoing very rapid weight loss in which vitamin and mineral deficiencies are reported.[24] There is little information on malnutrition in veterinary patients undergoing controlled weight loss, and a recent study in dogs did not demonstrate any significantly deleterious changes in a selection of nutrients following weight loss of an average 0.8%/wk when fed a nutrient-dense weight-loss diet.[25] This question is important since it is unusual in practice to achieve rates of weight loss greater than 2%/wk. At that rate, there is little reason to believe that when a maintenance diet formulated to meet the Association of American Feed Control Officials (AAFCO) or European Pet Food Industry Federation (FEDIAF) requirements is fed that a clinically significant deficiency will result. Thus, if success is prioritized over theoretical concerns and if client compliance is greater with reduction of current diet rather than prescription of another diet, then preference should be given to the protocol most likely to succeed. Studies that objectively compare in a randomized manner the efficacy of prescription diets over adjustment of current diets are sorely needed.

Weight-Loss Rates

It is commonly recommended that the rate of weight loss not exceed 2% per week, and as stated, in client-owned animals, it is difficult to create a rate of weight loss that exceeds 2%/wk. The justification for avoiding faster rates is that the proportion of LBM lost is increased as the rate of weight loss increases. Certainly in humans, the proportion of LBM lost is influenced by the rate of weight loss and hence degree of caloric restriction.[26] Some loss of LBM is inevitable and unpreventable, but excessive weight loss has been argued to be an adverse effect. Unfortunately, studies in client-owned animals have not quantified the likelihood of supposed negative effects. In addition, there currently is no standard to define "excessive LBM loss." In humans, it is assumed that the LBM of an age, gender and ethnically matched lean individual has the ideal LBM, thus the optimal fat:FFM loss ratio is based on that.[27] In veterinary practice, this figure will vary with species, breed, age, sex, and neuter status. Thus, a suitable universal figure may not exist.

In human weight-loss studies, there is a general but nonuniform trend for exercise to be effective in reducing but not preventing LBM loss during hypocaloric feeding.[26] Similar studies have not been conducted in dogs or cats undergoing weight loss, but it is likely to be similar. The type of exercise, amount, intensity, and nature of meaningful benefits remain to be demonstrated. In one study, there was no spontaneous increase in activity in dogs undergoing successful managed weight reduction when no specific recommendations were made despite owner knowledge of the measurement of activity.[28] This emphasizes the need for education, monitoring and feedback to owners. However, the benefits to the animal beyond successful reduction in BFM are unknown and may even be unimportant.

In one study of 34 obese dogs, the dogs were fed a low-calorie canned food at the rate of 40% of the calculated maintenance requirement of their target weight. Despite that draconian reduction, 20/34 dogs reached the target weight with an average weight loss of 14% in 12 weeks — i.e., less than 2%/wk.[29] In another study in dogs, obese dogs were fed to 50% of the calculated MER of the dog's ideal BW. The duration of the weight-loss period was 42-161 d, with a mean of 85 ± 17 d and a rate of –2.6 ± 0.4% of wt/wk. There was no significant decrease in FFM.[30]

Following weight loss, it is not surprising that previously obese animals regain BFM if allowed *ad libitum* access to food. In the seminal study in dogs, it was shown that the more severe the energy restriction during weight loss, the greater the rebound of body weight.[31] However, it is not clear that the findings should influence our prescriptions. In that study, dogs were shifted immediately to *ad libitum* feeding once the target weight was reached, which though experimentally justified does not emulate normal practice where continued controlled feeding is recommended.

Lastly, there are uncommon but important cases were rapid weight loss is imperative. Cases of dynamic airway collapse in obese small-breed dogs pose an extreme anaesthetic risk. Exercise restriction combined with weight reduction can be effective as sole management strategies, but in extreme cases that require airway stenting, accelerated weight reduction to improve ventilation and allow safer anaesthesia is warranted. Other scenarios include surgical management of cruciate rupture using osteotomy techniques in obese patients. Surgical

outcome is impaired in the obese animal, but it is not yet known what defines the ideal preoperative management. The surgeon is tasked with weighing the values of several unknowns: the effect of a delay in surgery, the benefit of BFM reduction, and the risk of LBM reduction. It may be that the value of very rapid weight reduction (>2%/wk) outweighs the negative impact of LBM loss. Evidence rather than assumption should guide us.

Importance of Satiety and the Magic Diet

A reasonable hypothesis for an increase in the prevalence of obesity in pets is that highly palatable, energy-dense dry food diets have led to an inappropriate energy intake by pets. The energy density of the edible portion of a whole mouse, whole rabbit or whole bird is 5-10 kJ/g as fed, while most premium dry diets are around 16-18 kJ/g. This is certainly consistent with the hypothesis. However, in several studies home-prepared diets or canned diets have not been associated with a decreased prevalence of obesity.[32] Rapid obesity, at least in rodents, results with the provision of a variety of foods (cafeteria diets). A similar effect is predicted to occur in cats and dogs, especially when associated with variations in energy density.

Numerous previous studies have described the efficacy of tightly regulated caloric restriction in producing weight loss in obese dogs and cats. Indeed, it is almost axiomatic that feeding an obese animal less than its MER will produce weight loss. However, the determining factor in success or failure of weight-loss programs in clinical practice is owner compliance and accuracy. Thus, a diet that when fed in this manner can produce reliable weight loss would be a portentous development in the management of a truly globally important disease in dogs and cats and might have implications for obesity in humans. However, it is the author's opinion that the success of any weight-reduction program should be defined by long-term weight maintenance at a lean body weight and not simply by short-term weight loss.

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