



Small Animal Nutrition

Mini Series

Session One: Obesity – Why is Controlling Weight So Hard?

Marge Chandler DVM MS MANZCVS
DACVN DACVIM MRCVS



Obesity - Why is Controlling Weight So Hard?

ML Chandler

Obesity is the number one nutritional problem affecting pets of first world countries and affects more than 50% of cats and dogs. Pet obesity has variable definitions, e.g. 15 to 20% above ideal body weight or greater than a 7/9 body condition score. The prevalence in dogs and cats, like in humans, is increasing.

Owners' recognition of their pet's overweight body condition is often limited. In one study only 30% of dog owners recognized that their dog was overweight; in a PDSA study, 71% of owners of fat pets were "shocked" when told their pet was overweight. One third of cat owners underestimated cat's body condition score (BCS), especially with overweight or long haired cats, and in a survey by the Pet Food Manufacturer's Association, 80% of owners thought their pet's body shape was "perfect". This is similar to parents' assessment of their children, where in one study 32% of children were overweight or obese; however, only 12% of their parents recognized this.

A nutritional assessment can provide detailed information about the pet's diet and assessment of weight, BCS, and muscle condition. The World Small Animal Association (WSAVA) has guidelines for these assessments (<http://www.wsava.org/nutrition-toolkit>). The BCS is determined using a combination of visual appearance and palpation, e.g. if there a waist apparent and palpation of the amount of fat felt over the ribs. Videos for doing BCS are available on the WSAVA website. The BSC evaluates body fat, but it is possible to be overweight and still have muscle loss, especially in diabetic or other ill pets or the elderly. Muscle mass scoring systems are based on palpation of muscle over the skull, scapulae, spine and pelvis.

There are well-known adverse effects on health for excessive body fat (Box 1).

Diabetes mellitus (cats)
Insulin resistance (cats and dogs)
Worsening signs of arthritis or other musculoskeletal disorders
Increased risk of pancreatitis (dogs)
Increased anaesthetic and surgical complications
Worsened heat and exercise intolerance
Complications from cardio-respiratory disorders
Some dermatological diseases
Some forms of neoplasia
Incontinence (especially female dogs)
Tracheal collapse
Chronic bronchitis
Earlier mortality than lean animals

Box 1. Some of the health implications and disorders with increased risk in obesity

We know how to achieve weight loss in pets and people: consume fewer calories than are expended; however, the pathogenesis of obesity is not as simple and direct as uncontrolled gluttony. Obesity is a complex disorder involving energy metabolism and satiety control. Multiple genetic and environmental factors control regulation of food intake, resting metabolic rate, thermic effect of food, energy expenditure and efficiency during work, and the gut microbiome.

Box .2 Some of the factors which increase the risk of obesity in dogs and cats

Breed, some are more obesity prone Genetics, affects metabolism and activity levels Gut microbiota Neutering, at any age Age (middle aged) Palatable free choice diets, especially high fat diets Feeding snacks, treats, table scraps Using a large bowl or feeding utensil Not weighing or measuring the amount fed Sedentary lifestyle, indoor cats Overweight owner (dogs only) Older owners Owners unaware of ideal body condition and weight, infrequent weighing of pet

Three common causes of obesity in pets are overeating, decreased exercise, and lower metabolic rate. While activity and eating behaviours contribute substantially to the development of obesity, there is now much knowledge about regulation of eating and energy balance. Further, while these have, at least partially, a genetic basis, there is also evidence that gene expression can be modified.

To better understand the roles of appetite and satiety and potential interventions, we need to understand the complex physiology of these drives.

APPETITE and SATIETY

Years ago food intake was thought to be solely controlled by “glucostatic” regulation, where fasting blood glucose would fall and stimulate the feeding centre in the hypothalamus. The subsequent post prandial increase in glucose was believed to activate the satiety centre, which inhibited the feeding centre. The control of food intake is now known to be much more complicated, and is often overridden in humans by social, cultural and environmental factors and highly palatable foods. These factors likely also result in increased food intake in pets.

Regulation of feeding and energy balance involves both short term regulation of meals and longer term regulation of overall energy balance and body fat. Short term control of food intake involves the central nervous system and gastrointestinal tract hormones. Long term regulation of food intake and adiposity involves input from the adipose tissue (fat), producing several endocrine and paracrine mediators, including leptin and adiponectin. Some of the gut hormones also have a role in longer term control.

Distension of the stomach activates stretch receptors which transmit satiety signals, although generally eating stops before the stomach is this distended. Central and peripheral signal communicate information about the current state of energy balance to key brain regions, including the hypothalamus and brainstem, often from the gut via the vagal nerve. Hunger and satiety represent coordinated responses to these signals.

Gastrointestinal (GI) signalling

Ghrelin – the hunger hormone

The only GI hormone which is orexigenic (increases appetite) is ghrelin, sometimes called the hunger hormone. It is secreted by gastric epithelial cells and by hypothalamic neurons and acts on the hypothalamic feeding centre. The concentration of plasma ghrelin peaks before a regular meal, and in humans, its release is accompanied by feelings of intense hunger. Blood concentrations of ghrelin are lowest shortly after a meal.

Plasma ghrelin is suppressed in proportion to the calories ingested, although dietary fat appears to suppress ghrelin less potently per calorie than carbohydrates or protein. This may reduce satiety on a high fat diet, leading to potential weight gain which already is a risk of high fat diets. The rapid postprandial drop in ghrelin is attenuated in obese humans compared to lean ones and obese people may be more sensitive to appetite stimulation by it.

Ghrelin suppresses fat utilisation in adipose tissue. Ghrelin also has beneficial cardiovascular, and anti-inflammatory effects. These positive effects may limit the use of ghrelin antagonists as potential treatments for obesity.

Satiety signals

Cholecystokinin (CCK) is secreted post prandially from the small intestine, and reduces food intake in addition to its effects on the pancreas and gallbladder. In humans, it has a short half-life of 1 to 2 minutes, limiting its use as an appetite suppressant. Chronic administration of CCK alone also does not result in weight loss. Protein and fat stimulate increased release of CCK from the gut. CCK acts both peripherally and centrally to prolong a feeling of satiety.

Peptide YY

Peptide YY (PYY) is secreted by the L cells of ileum and large intestine. It is a satiety signal and decreases food intake, likely via a pathway through the vagal afferent nerve to the hypothalamic arcuate nucleus. Peptide YY also inhibits fasting small bowel motility and gastric emptying. There are suggestions that some of the anorectic effects are due to nausea in humans. A veterinary drug (tiripatide) is marketed for helping control obesity in dogs which has its effect partially via PYY. This drug results in effective weight loss while being given, although without management changes by the owner the chance of weight re-gain is very high.

Oxyntomodulin

Oxyntomodulin is released from the L cells of the intestine and inhibits food intake. When injected in humans it results in decreased calorie intake and an increased energy expenditure from increased voluntary activity. It also increases heart rate in rodents. As it has a short duration of action, it required 3 times daily injections which limits usefulness, although analogues may be developed.

Adipokines

Lipid cells are highly active and adipose tissue has many endocrine functions. The term adipokines refers to factors released from adipose tissue that regulate energy metabolism; they also affect cardiovascular function, reproductive status, and immune function. Leptin and adiponectin are two of the better studied adipokines.

Leptin is a hormone that helps regulate body weight by signalling an increased amount of fat stored and then decreasing food intake (and may increase energy expenditure). It increases with increased amounts of white body fat. It also stimulates angiogenesis and decreases insulin sensitivity. It has the negative health effects of being pro-inflammatory, prothrombotic, and pro-oxidant. Some obese individuals develop leptin resistance and can develop very high concentrations without a marked decrease in food intake.

Adiponectin is produced by mature adipocytes. It is a key adipokine that regulates carbohydrate and lipid metabolism. It enhances insulin sensitivity and is anti-inflammatory. There is less in obese individuals, so the positive effects are only seen in lean animals. Weight loss in the obese increases adiponectin in most species including cat; however, one study in dogs did not show a decrease in high molecular weight adiponectin after weight loss, and adiponectin may not be involved in changes in insulin sensitivity in dogs. It may be that obesity decreases adiponectin concentrations in intact but not in neutered dogs.

Box 3 Some of the hormones, neuropeptides (neurotransmitters) that cause increased food intake (orexigenic peptides) and those that decrease food intake (anorexigenic) are listed below. Many more exist than are listed.

<u>Orexigenic (stimulating appetite)</u>	<u>Anorexigenic (decreasing appetite)</u>
Ghrelin	<u>Leptin</u>
Neuropeptide Y (in hypothalamus)	Peptide YY (PYY)
Agouti-related peptides (hypothalamus)	Glucagon like peptide -1 (GLP-1)
Endogenous opioids	Cholecystokinin
Melanin concentrating hormone	Oxyntomodulin
	Orexin A
	POMC melanocortin neurons (hypothalamus)
	Corticotropin releasing hormone (CRH)
	Thyrotropin releasing hormone (TRH)

Other Non-GI hormones. *Thyrotropin-releasing hormone (TRH)* can be a potent inducer of satiety in experimental animals and this may act by the direct inhibition of LH enkephalin-mediated feeding behaviour. Diurnal rhythm will also affect appetite, for example, in nocturnal hunters.

Obesity and the Gut Microbiome

In humans and rodents, and probably other animals, the gut bacteria and other micro-organisms and their environment and products differ in obese and lean individuals. The gut microbiome is influenced by the maternal microbiota, diet, environment, antibiotic use, and probiotics. Fibres which function as prebiotics, i.e. those that enhance the growth of beneficial bacteria in the gut, may shift the microbiome in obese animals to a one more similar to that of leaner animals and may improve insulin sensitivity. Studies of the gut microbiota in obese individuals (often laboratory rodents) compared to lean animals show that there are changes enabling the microbiota to extract more energy from the diet and interact with host epithelial cells to indirectly control energy expenditure and fat storage. Short chain fatty acids (SCFA) such as butyrate, acetate and propionate are produced by the gut bacteria fermentation of dietary fibre. These SCFA are absorbed into the circulation and serve as an energy source. They also may act in the gut as signalling molecules for receptors which may regulate energy balance and induce the release of the anorexic peptide YY.

MANAGING OBESITY AND WEIGHT LOSS PROGRAMMES

There are three aspects to managing obesity:

- 1) prevention of weight gain
- 2) weight loss in the overweight
- 3) decreasing the risk of weight re-gain after loss

Preventing obesity

Preventing obesity is of great importance as it is easier to avoid obesity than to lose weight. Obesity in childhood predicts adult obesity and likely the same is true for puppies and kittens, although lean puppies and kittens may still become overweight as adults.

Neutering reduces the daily energy requirement of dogs and cats by 24–33%, regardless of the age of neutering. The reduction is due to a reduction in basal metabolic rate and/or increase drive for food intake. It is imperative to discuss feeding with owners at the time their pet is neutered to decrease the risk of obesity. Owners should be taught to body condition score their pet, and provided with a suggestion for frequency of weighing.

Obesity Management Programmes

Three steps to effectively managing obese patients are 1) the recognition of obesity and establishing client commitment, 2) the development of a program which meets the needs of the pet and owner, and 3) communication and follow-up with the practitioner. Components included in weight loss programmes are diet, exercise, and behaviour modification. For successful weight loss in pets, the owner must undertake significant behaviour changes. Specific recommended changes include measuring, monitoring and recording food intake, monitoring body weight and condition, and increasing the pet's exercise.

Numerous recommendations, equations and methods have been published for caloric restriction for weight loss. A good starting point is to use resting energy requirement (RER) at ideal weight (iRER). Resting energy requirement = $\text{body weight (kg)}^{0.75} \times 70$. Using 80% of iRER is sometimes recommended but may result in a drastic reduction in food intake. A programme should sufficiently restrict calories to produce weight loss without being so severe that the pet is chronically hungry, possibly begging, and the owner feels guilty about enforcing the diet. Severe restriction is also more likely to result in regaining weight after the diet has ended.

Diets designed for weight loss are usually low in calories and fat, with other nutrients increased to avoid deficiencies. Protein is often increased to help maintain lean body mass and possibly promote satiety. Fibre content may be increased to a moderate or high level to increase satiety. While most commercial weight loss products can be used successfully, there is a large variability in caloric density. "Lite" diets may not be sufficiently low in calories for weight loss, and feeding less of a maintenance food may result in a deficiency of protein, minerals or vitamins.

Starvation will result in weight loss, but causes excess loss of lean body mass, affects body functions (e.g. cardiovascular system), risks causing potentially fatal hepatic lipidosis in cats, and is ethically questionable.

A weight loss programme should be tailored to the individual dog or cat and closely monitored. While in research animals weight loss of 1 to 2.5% per week is possible, in owned pets weight loss of 0.8 to 1% for dogs and 0.5 to 1.0% for cats is more realistic.

As owners enjoy giving treats or may use them as training rewards, incorporating treats at 10% or less of total calories may be considered. Treats may include hand-fed low calorie pet diet, low calorie foods (baby carrots, popcorn, or cooked vegetables) or limited commercial low fat treats.

Food composition and food form may influence total caloric intake in dogs and cats. Cats usually consume more calories when fed dry commercial foods compared to canned foods. Dogs tend to have an increased appetite when fed canned food and improved satiety when offered high protein/high fibre foods.

Using smaller food bowls or feeding cups may result in smaller meals. Ideally food should be weighed as even with the use of graduated measuring cup food portions are unreliable compared to weighed food portions using a kitchen scale. Minor variations in food intake can lead to weight gain over time.

Dividing the daily food into multiple meals can decrease hunger and the owner's perception of their pet's hunger, as well as possibly use a few calories from the thermic effect of food. Cat owners should know that normal cat feeding behaviour involves multiple small meals, so that if the cat only eats a small amount of the food offered it does not necessarily mean that the cat does not like the food, and it may not be necessary to offer a different more palatable food immediately.

Regular exercise expends energy, builds and maintains lean muscle mass and reduces boredom-related eating in people and in pets. Providing scheduled exercise, enrichment toys can help with prevention of weight gain in dogs and cats. Feeding food or treats in feeding balls can increase the energy output. Exercise is encouraged for most pets, although joint, heart or other disorders may limit the amount possible.

Preventing weight re-gain after weight loss

Over 90% of people who lose weight regain some or all of it within 5 years. Similarly, in one study nearly 50% of 33 dogs which had lost weight regained it, especially if they had been switched back to a maintenance diet. After weight loss, metabolic rate and energy expenditure decrease so that fewer calories are required to re-gain the weight than to put it on initially. Weight loss may result in decreased plasma concentration of leptin, increased levels of ghrelin, and suppressed fat oxidation, all leading to a risk of weight gain.

Owners may report that they are feeding very little, that the pet begs, and it is impossible to maintain the weight loss. Similarly, a plateau may be reached during weight loss. While reaching an ideal weight may have been the goal, it is important for owners to realize that any weight loss is beneficial. In humans, 5-10% weight loss decreases the risk of cardiac disease and metabolic syndrome, and in dogs a 6 to 8% loss is associated with improvement in the signs of arthritis.

In humans, factors shared by those who avoid significant weight regain are: maintaining a high level of physical activity, eating a low fat diet, and active monitoring of body weight. Encouraging owners to exercise dogs and play with cats is important. Continued use of an appropriate low fat or weight loss diet is recommended as they may regain weight on a maintenance diet. Continued veterinary or weight clinic visits aid in monitoring and provide an opportunity for assessment and counselling. Pets in less structured programmes are more likely to regain lost weight.

To date, the only intervention proven scientifically to improve quality of life and concurrently extend lifespan in pets is caloric restriction and maintenance of a lean body condition. Obesity has numerous adverse effects on health and longevity, but implementation of a weight loss programmes and maintenance of a good body condition in dogs and cats has continued to be a challenge for vets, pets and owners.