Practical Weight Management in Dogs and Cats

1 Clinical Importance of Canine and Feline Obesity

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Introduction

The last decade has seen a fundamental shift in our understanding of obesity. The discovery of hormones and cytokines generated by adipose tissue (termed adipokines) has expanded fat's traditional roles as an energy storage depot, insulator, and support for abdominal organs. Fat is now recognized as the most abundant source of hormones in the body, making it the largest endocrine organ. Additionally, macrophages in adipose tissue contribute to the release of numerous inflammatory cytokines and other adipokines into the blood. As a result, overweight and obese individuals reside in a state of chronic inflammation (Figure 1.1).

This knowledge has come on the heels of an epidemic of obesity in companion animals that parallels the global obesity epidemic in human patients. The combination of serious metabolic and health consequences of obesity and sheer number of obese pets should make canine and feline obesity a priority for veterinarians. Just as veterinarians have long provided routine infectious disease and dental prophylaxis, preventive health care also must focus on nutrition counseling. Informing pet owners about disease risk factors associated with obesity and recommending appropriate dietary intake for obesity prevention and weight loss should be integrated into most preventive care examinations.

Practical Weight Management in Dogs and Cats, First Edition. Edited by Todd L. Towell.

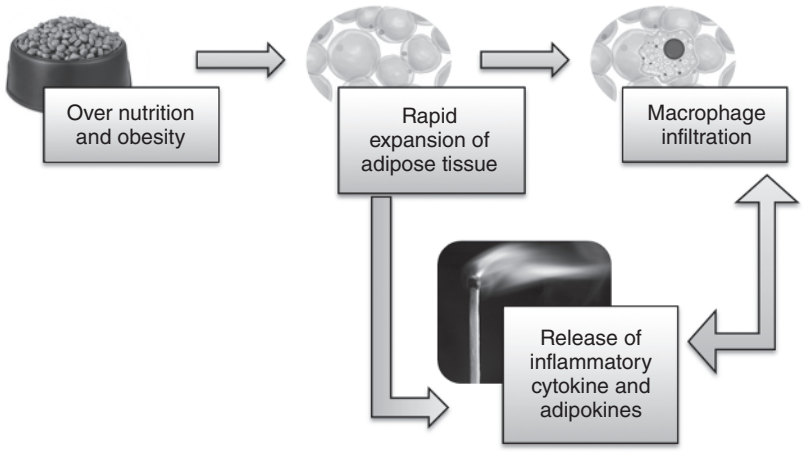

Figure 1.1. Relationship of obesity to chronic inflammation. In obese states, adipose tissue expands rapidly and adipocytes enlarge. This induces a state of local hypoxia and stress responses that recruit macrophages to adipose tissue. In the obese state, adipocytes also release cytokines, adipokines, and free fatty acids. These work locally and systemically to increase the inflammatory state within adipose tissue, liver, and muscle, and cause insulin resistance. Images from Fotolia.com.

Defining Obesity

Obesity is a disease in which excess body fat has accumulated such that health may be adversely affected. In human medicine, application of this definition is based on epidemiologic data that demonstrate increased morbidity and mortality risks with increasing body fat mass. Based on such data, criteria have been established for what constitutes "overweight" and "obese." To date, such objective criteria are not available for dogs and cats. Fat mass comprises about 15% to 20% of the body weight in dogs and cats in ideal body condition.[1–5] Pets are typically considered overweight at 10% to 20% above their ideal body weight and obese if their weight exceeds 20% to 30% more than ideal.[6,7]

One of the most difficult challenges in diagnosing obesity is determining ideal body weight and present fat mass. A patient's fat mass can be measured using a variety of methods. However, most involve some procedure or parameter that makes them unsuitable at present for routine clinical use. Because of its precision and relative ease of use, dual energy X-ray absorptiometry (DEXA) has become the standard tool for measuring body composition when performing clinical research in pet obesity. Unfortunately, access to DEXA equipment is generally limited to academic and corporate research facilities.

Similarity in shape among human patients permits the use of the body mass index (BMI), a number calculated as weight (kg) divided by height^2 (m). Similar semi-quantitative indices are not available for pets.

Because of the wide breed differences in body types, the equivalent of a BMI is unlikely to be developed for dogs. A BMI has been described for cats based on ribcage circumference and leg length measurements, but has not yet gained wide acceptance in clinical practice or research.[8] This may be partly because accurate measurements are difficult to make in an awake, active cat.

The standard method of semi-quantitatively assessing degree of adiposity in dogs and cats in a clinical setting is the use of a body condition scoring system. The advantage of assigning a body condition score (BCS) is that it provides more information than body weight, which often varies markedly, even within individuals of the same breed and gender. Veterinary clinicians and researchers most often use one of two semi-quantitative BCS systems (nine-point and five-point scales) that are based on visual and palpatory findings (Table 1.1).[4,9–12] It is important to keep in mind that these BCS systems were developed for use in healthy animals. For example, sick animals with conditions that lead to weight gain and marked muscle wasting (hyperadrenocorticism) are very difficult to score; therefore, simultaneous use of a muscle scoring system is recommended (Table 1.2).[13,14]

Prevalence of Obesity

The number of pets that are overweight or obese has reached epidemic proportions in the United States and other industrialized countries. Approximately 25% to 35% and 35% to 40% adult cats and dogs, respectively, are either overweight or obese.[15–17] Middle-aged neutered male cats and middle-aged spayed female dogs are at highest risk of becoming obese. Some purebred dogs also have higher obesity risks; these include Shetland Sheepdogs, Golden Retrievers, Dachshunds, Cocker Spaniels, Labrador Retrievers, Dalmatians, and Rottweilers.[17] Manx cats are more likely to become obese than other purebred cats. Not surprisingly, low activity level increases risk for weight gain in both species; in cats, apartment dwelling is associated with a higher risk. 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 prepare or eat their own meal.[16,17]

Veterinarians must proactively focus on obesity prevention. Wellness visits are the ideal time to regularly reassess body weight history and body condition score. The benefits of maintaining a pet in lean body condition, and the health risks that can accompany obesity, are important owner education topics. The veterinary visit for spaying/neutering is an important, but often neglected, opportunity to reassess feeding practices and discuss obesity issues with clients. Studies in cats have shown that neutering decreases metabolic rate by 25% to 33%.[18] Neutered animals, however, usually have increased fat mass. When

Table 1.1. Comparison of body condition scoring systems with body fat percentages.

5-point scale	9-point scale	% Body fat	Description
1	1	≤5	Emaciated: Ribs and bony prominences are visible from a distance. No palpable body fat. Obvious abdominal tuck and loss of muscle mass.
2	2	6–9	Very thin: Ribs and bony prominences visible. Minimal loss of muscle mass, but no palpable fat.
	3	10–14	Thin: Ribs easily palpable, tops of lumbar are visible. Obvious waist and abdominal tuck.
3	4	15–19	Lean: Ribs easily palpable, waist visible from above. Abdominal tuck present in dogs. Abdominal fat pad absent in cats.
	5	20–24	Ideal: Ribs palpable without excess fat covering. Waist and abdominal tuck present in dogs. Cats have a waist and a minimal abdominal fat pad.
4	6	25–29	Slightly overweight: Ribs have slight excess fat covering. Waist discernible from above, but not obvious. Abdominal tuck still present in dogs. Abdominal fat pad is apparent, but not obvious in cats.
	7	30–34	Overweight: Difficult to palpate ribs. Dogs: Fat deposits over lumbar area and tail base. Abdominal tuck may be present, but waist is absent. Cats: Moderate abdominal fat pad and rounding of the abdomen.
5	8	35–39	Obese: Ribs not palpable and abdomen may be rounded. Dogs: Heavy fat deposits over lumbar and base of tail. No abdominal tuck or waist. Cats: Prominent abdominal fat pad and lumbar fat deposits.
	9	40–45+	Morbidly obese: Dogs: Large fat deposits over thorax, tail base, and spine, with abdominal distension. Cats: Heavy fat deposits over lumbar area, face, and limbs. Large abdominal fat pad and rounded abdomen.

Adapted from Lusby A, Kirk C. 2008. Obesity. *Kirk's Current Veterinary Therapy XIV*. St. Louis: Saunders Elsevier.

energy expenditure is expressed on a lean mass basis, no difference in metabolic rate is noted between neutered and entire individuals (REF). An alternative explanation for the effect of neutering on obesity is an alteration in feeding behavior leading to increased food intake and decreased activity, without a corresponding decrease in energy intake.

Table 1.2. Muscle scoring system.

Score	Fat mass	Muscle mass
0	Absence of palpable subcutaneous fat over the ribs or the abdominal region	Severe muscle wasting as evidenced by pronounced decreased muscle mass palpable over the scapulae, skull, or wings of the ilia
1	Decreased amounts of palpable subcutaneous fat of the ribs or the abdominal region	Moderate muscle wasting as evidenced by clearly discernible decreased muscle mass palpable over the scapulae, skull, or wings of the ilia
2	Normal amounts of palpable subcutaneous fat over the ribs or the abdominal region	Mild muscle wasting as evidenced by slight but discernible decreased muscle mass palpable over the scapulae, skull, or wings of the ilia
3	Increased amounts of palpable subcutaneous fat over the ribs or the abdominal region	Normal muscle mass palpable over the scapulae, skull, or wings of the ilia

Adapted from Michel KE, Sorenmo K, Shofer FS. 2004. Evaluation of body condition and weight loss in dogs presented to a veterinary oncology service. *Journal of Veterinary Internal Medicine* 18:692–695.

Health Risks of Obesity

Diseases associated with obesity in human patients include hypertension, coronary heart disease, dyslipidemias, and type-2 diabetes mellitus (these conditions form the components of so-called metabolic syndrome), as well as certain cancers (breast, ovarian, and prostate), osteoarthritis, respiratory disease such as asthma, and reproductive disorders.[19–22] Studies investigating overweight or obese dogs and cats have identified many of the same chronic health problems observed in obese humans.

Obesity in companion animals is a serious medical concern, resulting in a shorter life span and greater disease morbidity. Dietary restriction can increase longevity in other species, and a landmark prospective study in Labrador Retrievers yielded the same result.[23] In this study, 24 pairs of dogs were enrolled as puppies with one dog in each pair randomly assigned to consume food *ad libitum*. The paired dog was fed 75% of the amount consumed by its counterpart. In the energy-restricted group, the mean body condition score was 4.5/9 compared to 6.8/9 in the *ad libitum* feeding group. Causes of death did not differ between the two groups, but life span was increased by 1.8 years in the energy-restricted group (median 13 years vs. 11.2 years). Dogs in the lean body condition group also had reduced risk of hip dysplasia and osteoarthritis and improved glucose tolerance.

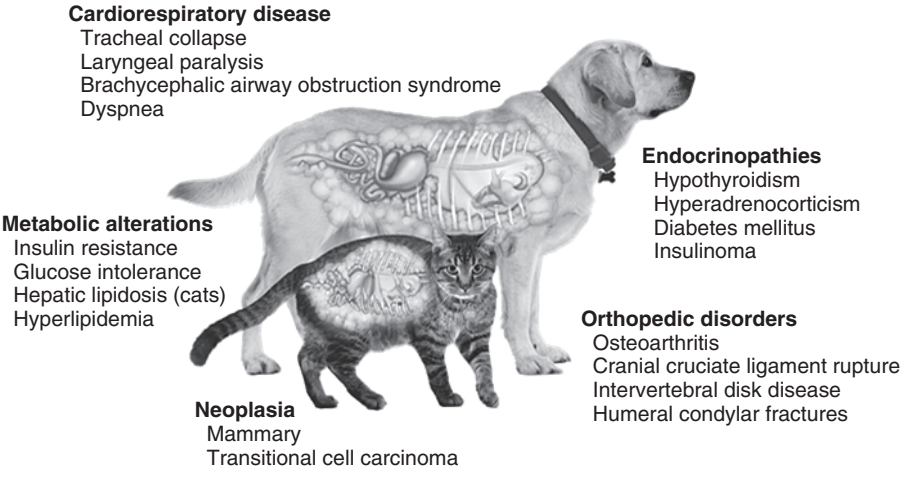

Figure 1.2. Diseases associated with obesity in dogs and cats. Courtesy Hill's Pet Nutrition, Inc.

In cats, diabetes mellitus, neoplasia, dental disease, dermatologic diseases, and lower urinary tract problems have been associated with obesity.[24] In dogs, obesity has been linked with insulin resistance, pancreatitis, cruciate ligament rupture, lower urinary tract disease, oral disease, neoplasia (mammary tumors, transitional cell carcinoma), abnormalities in circulating lipid profiles, osteoarthritis, hypertension, and altered kidney function.[17,25] In addition, although harder to measure, obesity exacerbates existing musculoskeletal problems, brachycephalic syndrome, and pregnancy, and is associated with increased anesthetic risk (Figure 1.2).

Fat as an Endocrine Organ

In addition to functioning as an energy storage site and thermal insulation, adipose tissue operates as an active endocrine organ. A variety of endocrine, paracrine, and autocrine signals are released from cells within adipose tissues. These signals are referred to as adipokines. The term adipokine is generally used to mean any protein released by adipose tissue regardless of whether it is released by adipocytes or nonfat cells.[26] To date, approximately 100 adipokines have been identified as being released from fat tissue, of which at least 24 have increased circulating levels in obese humans.[27,28] Some of these putative adipokines, such as C-reactive protein (CRP), haptoglobin, and amyloid A, are actually acute phase proteins primarily released by the liver in response to the mild inflammatory response seen in human obesity. Most of the remaining 21 also are inflammatory proteins, but the source of their elevated circulating levels in obesity is unclear and could result from release by tissues other than fat.[28]

Upregulation of the systemic inflammatory response appears to provide a critical pathophysiologic link to the wide variety of chronic diseases associated with obesity. There is increasing evidence that obesity in humans is associated with low-level inflammation that is often accompanied by hypertension and type-2 diabetes. Interestingly, some adipokines may actually have anti-inflammatory effects and circulate at higher levels in obesity as part of a homeostatic mechanism to counteract the effects of the inflammatory mediators. Interleukin 10 (IL-10) probably is such a molecule, and there is some evidence that interleukin 6 (IL-6) has dual effects.[28] Currently it is thought that the increase in visceral omental rather than abdominal subcutaneous adipose tissue best correlates with measures of insulin resistance and cardiovascular disease.[28] The state of low-grade systemic inflammation that accompanies obesity, as measured by assaying various adipokines, has now been shown in dogs to undergo reversal when weight loss occurs.[29]

Although many adipokines have been discovered, the function and physiologic relevance of most have not been identified. A handful of adipokines have been intensively studied and appear to positively or negatively impact insulin sensitivity. The metabolic role of most adipokines is complex and incompletely understood. The following are examples of adipokines that have been widely studied and have significant metabolic effects.

Leptin

The presence of an obesity mutation (*ob*) was first discovered in mice nearly 60 years ago.[30] Mice with the *ob/ob* mutation in the leptin gene are morbidly obese, insulin resistant, hypothyroid, infertile, and have defective T-cell immunity.[31] In 1994, this mutation was cloned, sequenced, and later found to code for the hormone leptin.[32] Leptin was the first adipokine identified, and its main function is to regulate body fat mass through appetite control and increased energy metabolism. As body fat mass increases, more leptin is secreted from adipocytes.[33] Leptin is able to cross the blood-brain barrier; it inhibits neurotransmitters that increase appetite and lower energy expenditure and stimulates neurons that decrease appetite and increase in energy expenditure.[34,35] Therefore, as individuals gain body fat, leptin promotes weight loss. Leptin has been called a "lipostat" because it works like a thermostat for body fat mass.[36,37] Although leptin's primary physiologic role is to regulate body fat storage, it also affects the immune, cardiovascular, and reproductive systems. In addition, leptin is capable of regulating cardiac and vascular contractility through a local nitric-oxide-dependent mechanism.[38,39]

Leptin also may enhance insulin signaling to improve intracellular glucose uptake and decrease the accumulation of lipid in peripheral tissues.[40] Lipid accumulation with cells can lead to the phenomenon of

lipotoxicity, which has been implicated in the development of peripheral insulin resistance.[41,42] Administration of leptin decreases cellular lipid stores in the pancreas, adipose, liver, and cardiac tissues of rodents.[43] Leptin deficiencies found in *ob/ob* rodents result in obesity, insulin resistance, and diabetes.[44]

As evidenced by the current obesity epidemic, leptin does not always succeed in maintaining appropriate body fat mass. In fact, obese individuals often have the highest concentrations of this hormone.[45] When leptin cannot effectively regulate appetite and energy expenditure, this is termed "leptin resistance."[46] Several mechanisms may lead to leptin resistance: genetic mutation, receptor down-regulation, decreased permeation of the blood brain barrier, and molecular interference.[47] Although genetic mutations of leptin receptors can occur in humans, they are rare and account for only a tiny fraction of people with leptin resistance.

Leptin is capable of self-regulating its physiologic action by down-regulating its receptors. A reduction in receptor numbers has been demonstrated in the hypothalamus of rodents that overexpress leptin.[47] In addition, prolonged increases in central leptin concentrations eventually diminish its physiologic actions.[48] Evidence suggests that central leptin resistance causes obesity and that obesity-induced leptin resistance injures numerous peripheral tissues, including liver, pancreas, platelets, vasculature, and myocardium. This metabolic- and inflammatory-mediated injury may result from either resistance to leptin's action in selective tissues, or excess leptin action from adiposity-associated hyperleptinemia. In this sense, the term "leptin resistance" encompasses a complex pathophysiological phenomenon. The leptin axis has functional interactions with elements of metabolism, such as insulin, and inflammation, including mediators of innate immunity, such as interleukin-6. Plasma levels of leptin and inflammatory markers are correlated and also predict cardiovascular risk in human patients.[47]

Adiponectin

Adiponectin is the most abundantly secreted adipokine in circulation with concentrations in the μg/ml range (three orders of magnitude higher than leptin).[49] Although adipocytes are responsible for secreting adiponectin, hormone levels become paradoxically lower with increased fat mass.[50] The reason behind this unusual relationship is not clear. It is speculated that increased levels of other adipokines, such as tumor necrosis factor-alpha (TNF-α), may suppress adiponectin expression. Adiponectin exerts a myriad of metabolic affects. Perhaps the most influential role of adiponectin is as an insulin sensitizer. Adiponectin is closely associated with insulin sensitivity, independent of body fat mass.[51–53]

In a study of obese rhesus monkeys, low adiponectin levels correlated with insulin resistance and preceded the onset of diabetes mellitus.[54] Prospective and longitudinal studies in human beings also demonstrate that lower adiponectin levels are closely associated with insulin resistance and future development of diabetes.[55–57] Higher levels of adiponectin are also strongly associated with reduced risk of type-2 diabetes in healthy adult human beings.[58]

Adiponectin proteins bind to each other to form complexes of varying sizes. The high molecular weight (HMW) form of adiponectin is made up of 12 or more adiponectin molecules bound together. This HMW complex is thought to be the most active form of adiponectin and is more closely associated with insulin resistance and diabetes than total adiponectin or the lower weight forms.[59,60]

Numerous clinical and epidemiological studies associate low levels of adiponectin with chronic inflammatory states such as obesity, insulin-resistance, type-2 diabetes, hypertension, cardiovascular disease and liver disease.[61,62] Adiponectin is an attractive therapeutic target. In support of its therapeutic potential, administration of recombinant adiponectin ameliorates metabolic complications in mice, and the beneficial effects of the insulin-sensitizing thiazolodinedione drugs in human patients are at least partly due to the improvement in adiponectin profiles.[63]

Tumor Necrosis Factor-alpha

TNF-α is an inflammatory cytokine expressed by a variety of cells including macrophages, mast cells, neuronal cells, fibroblasts, and adipocytes. The connection between TNF-α, obesity, and insulin resistance is unclear. Because TNF-α can be secreted by both differentiated and undifferentiated adipocytes, it was thought that the increased levels of TNF-α found in obesity were primarily due to adipocyte secretion; however, cells within the stromovascular fraction of adipose tissue, including macrophages, produce significantly more TNF-α than adipocytes.[64–66] Obesity increases macrophage migration into adipose tissue, and this is likely the cause of increased TNF-α expression.[67,68] One theory behind recruitment of monocytes and macrophages to expanding adipose tissue is that increased levels of adipocyte apoptosis and necrosis produce chemoattractant agents.[65,69]

TNF-α secretion from adipose tissue has key species differences. In mice, TNF-α is released into systemic circulation.[69] In humans, most adipose TNF-α exerts local paracrine and autocrine actions.[70,71] Obese dogs tend to have higher systemic concentrations of TNF-α.[72] The circulation patterns of TNF-α derived from adipose tissue are not well understood in cats, but mRNA expression within fat is increased with obesity.[73]

One of the primary actions of adipose TNF-α is induction of localized insulin resistance. TNF-α down-regulates genes responsible for

insulin-mediated glucose uptake into cells.[74–76] In addition to inhibiting glucose entry into cells, TNF-α decreases uptake of free fatty acids (FFA) into adipocytes and promotes lipolysis and release of FFA into circulation.[65,70,77] As a result, FFA levels increase in circulation and negatively affect insulin sensitivity in peripheral tissues.

In addition to directly influencing insulin sensitivity of adipose tissue, TNF-α can alter secretion of other adipokines involved in glucose metabolism. In particular, TNF-α inversely correlates with adiponectin and may alter its gene expression.[65,70,78,79] In contrast to adiponectin, expression of leptin and several other adipokines is increased by TNF-α.[80] In summary, TNF-α secreted from adipose tissue plays an important role in glucose and lipid metabolism at both the local and systemic levels and is a key component to inflammation associated with obesity.

Interleukin-6

Interleukin-6 (IL-6) is a pleiotropic cytokine affecting a wide variety of physiologic processes. It plays a major role in regulating inflammation, immune responses, and hematopoiesis.[81] IL-6 appears to mirror TNF-α in its interactions with other adipokines. It has an inhibitory effect on adiponectin and promotes expression of leptin, resistin, and visfatin.[80] Adipose tissue secretes up to 35% of basal IL-6 plasma levels.[71] Although adipocytes produce IL-6, other cells in the stromovascular fraction of adipose tissue also secrete the cytokine and probably contribute more to overall secretion.[81,82] Visceral adipose tissue secretes more IL-6 than subcutaneous adipose, and the concentration of IL-6 in adipose tissue is approximately 100-fold greater than that of plasma.[82,83] This implies that IL-6 plays an autocrine and/or paracrine role in adipose. One important function may be to induce lipolysis within adipocytes. Adipocytes and adipose tissue grown in culture with IL-6 demonstrate increased levels of lipolysis.[81,84] In addition, infusion of IL-6 in humans increases overall fatty acid concentration and oxidation.[81,85,86]

Several studies[87–89] demonstrate a positive relationship between IL-6 adipose expression and insulin resistance; however, a cause-and-effect relationship has not been established, and higher concentrations of IL-6 may only reflect increased adipocyte numbers. Studies showing that IL-6 closely correlates with body mass index (BMI) but not insulin sensitivity in healthy and diabetic patients support this idea.[90,91] Some of the confusion regarding IL-6's contribution to insulin sensitivity may be due to its conflicting action on the skeletal muscle, liver, and fat. In general, IL-6 appears to improve insulin's action in skeletal muscle while impairing insulin-mediated glycogen synthesis and glucose uptake in hepatic and adipose tissue, respectively.[81]

Appetite Regulation

Obesity occurs when more calories are consumed than the body needs. Understanding factors that influence the amount of food eaten is key to preventing and managing obesity. The three main components that determine food intake are environment, emotional or cognitive decisions, and metabolic regulation. Pet owners have the most control over the environment by limiting the amount of food available to their pets. However, owner compliance improves when we consider the emotional and metabolic aspects of food intake. For example, part of the reason dogs enjoy receiving treats from their caregivers is the extra attention that is given. The joy of getting a treat may be replaced with owner praise and affection in many instances. The emotional role of food intake has been studied extensively in humans and is influenced by many factors including depression, boredom, palatability, serving dish size, and social situations.[92,93]

Although it is difficult to assess many of the thoughts and emotions dogs and cats undergo while eating, some of the same factors probably influence their food intake. Cats living in apartments are more likely to be obese, and although activity level certainly impacts their weight, boredom or lack of environmental enrichment also may lead to increased food intake.[16] Increased exercise and activity may fight obesity in pets by burning extra calories and improving the emotional health of cats and dogs. The metabolic controls of appetite and food intake are numerous and complex. This discussion focuses only on a few key aspects.

The arcuate nucleus of the hypothalamus is the main central regulator of appetite. Within the arcuate nucleus there are two sets of neurons that have opposing actions. Anorexigenic neurons inhibit appetite and include the proopiomelanocortin (POMC) and cocaine-amphetamine regulated transcript (CART) neurons. The anorexigenic neurons release α-MSH, which decreases food intake and increases energy expenditure by acting on melanocortin receptors (MC3 and MC4). Orexigenic neurons increase food intake and include neuropeptide Y (NPY) and agouti-related gene transcript (AgRP). NPY inhibits POMC cells. AgRP antagonizes MC3 and MC4 receptors and reduces the anorectic effects of α-melanocyte stimulating hormone (α-MSH) (Figure 1.3). The appetite-regulating neurons in the hypothalamus are influenced by peripheral signals such as leptin and by other centers in the brain.[94]

Peripheral controls of appetite involve the entire body. The mouth is the first contact food has with the body, and when food touches the tongue or palate it stimulates the brain to continue or stop eating. Oral stimulation of acceptable food encourages food intake through cranial nerve and olfactory sensors. Dopamine and opiods are the main neurotransmitters mediating positive feedback of oral stimuli. As food moves into the stomach, a hormone called ghrelin is secreted from the gastric mucosa. Levels of ghrelin peak just before a meal is eaten and

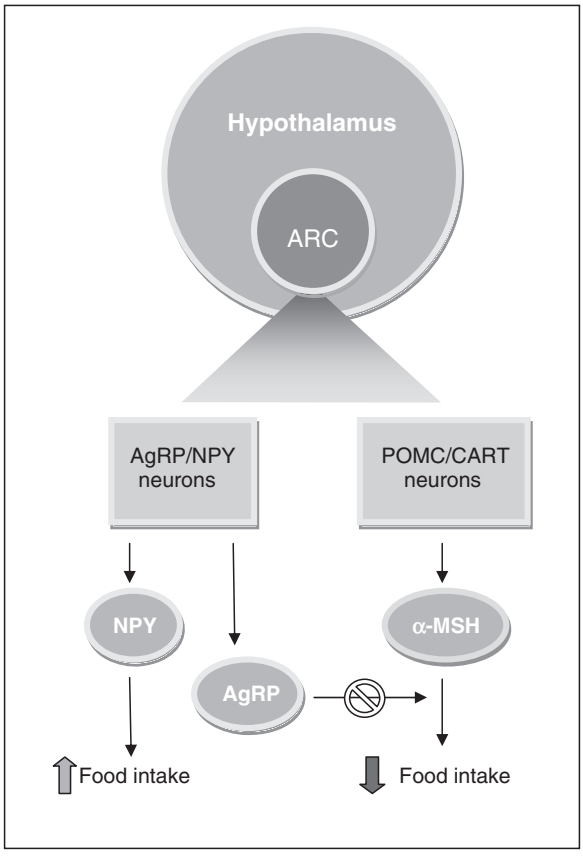

Figure 1.3. Central controls of appetite. Neuronal controls of appetite in the arcuate nucleus (ARC) within the hypothalamus of the brain. Neuropeptide Y (NPY) and agouti-related protein (AgRP) are released from neurons that stimulate appetite. Proopiomelanocortin (POMC) and cocaine-amphetamine regulated transcript (CART) neurons release α-melanocyte stimulating hormone (α-MSH), which suppresses appetite. AgRP inhibits the action of α-MSH.

rapidly decrease as nutrients enter the duodenum. It directly affects the reward system in the brain and is responsible for initiating hunger and food-seeking behaviors. While ghrelin stimulates appetite, most other signals from the gastrointestinal tract suppress feeding behavior. As the stomach expands with food, mechanoreceptors of the vagus nerve and spinal visceral afferent fibers are activated and cause release of anorexigenic peptides.[95] This is the mechanism behind the satiating effects of foods high in fiber and moisture that tend to fill the stomach without providing nutrient energy.

As nutrients move into the small intestine, proteins, monosaccharides, and fatty acids act on mucosal receptors that stimulate vagal afferent nerves and endocrine cells. Some of the key hormones released

from the small intestine are cholecystokinin (CCK), glucagon-like peptide 1 (GLP-1), and protein YY (PYY). CCK release is stimulated mostly by fats and proteins and it works by slowing gastrointestinal motility and gastric emptying. It also stimulates the vagus nerve to suppress appetite. PYY is found throughout the human intestinal tract and increases distally. The colon and rectum have the highest concentrations. PYY is released after meals from L cells in the intestine and its concentrations peak two hours after a meal. Therefore, PYY may be important in regulating the timing of meals. GLP-1 is released into circulation after a meal and is co-secreted from L cells with PYY. GLP-1 is an incretin (promotes insulin release) and also acts on the hypothalamus and the brain stem-vagus system. The pancreas also impacts appetite with its release of insulin and amylin during and after meals.[95] Insulin and amylin decrease eating through central actions that result from their cumulative release over time. As mentioned earlier, adipose tissue also regulates appetite through the release of the hormone leptin. As adipose tissue increases, leptin is released and suppresses appetite.[96]

In Practice

Explaining the health risks associated with obesity will help clients understand the importance of helping their pet's return to or maintenance of an ideal body weight.

- Obesity is a pro-inflammatory state due to cytokines released from fat. Hormones produced by fat contribute to the detrimental health effects of obesity.
- Excess body weight shortens life span. In dogs, this has been shown to be about a 15% reduction.
- Obesity has known health risks for some serious diseases that can influence both quality of life and life span in pets. Significant illnesses associated with obesity/overweight in dogs are cranial cruciate injury, osteoarthritis, and pancreatitis; in cats, these are diabetes mellitus, lower urinary tract disorders, and hepatic lipidosis.
- Diets with low caloric density that are high in fiber and moisture may help suppress appetite by stimulating stomach mechanoreceptors. Diets containing fats and proteins can decrease appetite through the release of CCK.

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