Why the Concern Over Obesity: An Introduction

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Human obesity has reached epidemic proportions in the United States, Europe and much of the developed world. It has been predicted that by 2030, 86.3% of adults in the U.S. will be overweight or obese and 51.1% will be obese.[1] Concurrent with obesity are serious comorbidities, such as diabetes mellitus, cardiovascular disease, fatty liver disease, and numerous others. In the United States, the incremental costs for health care directly associated with overweight and obesity in people was estimated at well over $100 billion in 2008, or about 5 to 10% of total health care costs.[2] In places where pets share our lifestyle, they also are "enjoying" the same epidemic. Depending on the study, the proportion of dogs and cats that are overweight or obese ranges from 33 to 58%.[3-5] Further, according to a 2015 news release from the Veterinary Pet Insurance Company (*http://press.petinsurance.com/press room/438.aspx*), which insures approximately 525,000 pets, obesity-related health claims have increased and totaled nearly $52 million in 2013 alone. Whether or not all the claims are truly causally related to obesity may be debated, but the point is clear: Obesity is a significant health issue for pets and cost issue for owners.

Recent Advances in Understanding About Obesity

Obesity has been the focus of considerable research, as attempts have been made to understand the causes and to improve solutions. It is hoped that such research will provide more effective means of treating and preventing obesity and its associated comorbidities. Much of the research has focused on the metabolic changes in obesity, specifically inflammation, oxidative stress and insulin resistance.

Ongoing research has identified over 100 hormones, cytokines and other cell-signaling substances (collectively called adipokines) secreted by adipose tissue, which may contribute to obesity-related diseases.[6-8] Many have functions involved in energy balance or metabolism, pro- or anti-inflammatory regulation, or promoters of insulin function or resistance. Among the numerous inflammatory adipokines are tumor necrosis factor-α (TNFα), interleukins (IL)-1β and IL-6, C-reactive protein (CRP), and others.[7-12] TNFα influences energy metabolism, promotes inflammation and causes insulin resistance by blocking activation of insulin receptors.[6,13] IL-6, IL-1β and CRP also contribute to insulin resistance, via different pathways, as well as promote inflammation. The persistent, low-grade inflammation secondary to obesity is thought to play a causal role in chronic diseases such as osteoarthritis, cardiovascular disease and diabetes mellitus.[10,14]

Obesity is associated with increased oxidative stress, which contributes to insulin resistance and obesity-related diseases, as well as further stimulating release of inflammatory cytokines.[10,12,15] Insulin resistance, and the resulting hyperinsulinemia, may play a central role in many of the adverse effects of obesity.[16] With the exception of diabetes, most of these links have not yet been confirmed in veterinary species, but insulin resistance is a common feature of obesity in both dogs and cats.[9,11] In cats, insulin sensitivity decreases by about 30% for each kilogram of weight gain,[11] and feline obesity is associated with up to an 4-fold increased risk for development of diabetes mellitus.[3,17] Insulin resistance not only contributes to secondary diseases, it can contribute to ongoing obesity via disruptions in energy homeostasis, appetite regulation and reduction in diet-induced thermogenesis.[18,19] These effects can make reversing obesity more difficult.

The intestinal microflora has recently received attention for its role in promoting or reducing obesity.[20] Crosstalk between organs is crucial for controlling energy homeostasis. It is now well known that microflora can interact with the host through various chemical messengers. Evidence reviewed by Guerts, et al., suggests this crosstalk can influence the development of metabolic alterations associated with obesity.[20] Germ-free mice or those treated with antibiotics have less fat mass and less glucose intolerance than normal mice, whereas colonization of germ-free mice with the gut microbiota from obese donors resulted in an increase in total body-fat mass. Further, the microbiota can stimulate energy metabolism via stimulation of brown or beige adipose tissue. Emerging evidence is showing differences in gut microflora in lean versus healthy individuals, suggesting that prebiotic and/or probiotic treatment may have a role in obesity management.[20-22]

Current Practice for Managing Pet Obesity

Although there are different approaches to management of obesity in pet dogs and cats, the general approach is to estimate a subject's ideal or target body weight and the energy needed to allow for weight loss, then to recommend

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a suitable diet in amounts to deliver the estimated energy intake. Plans may include follow-up with the patient at various intervals and giving advice regarding treats for the pet and for increasing exercise and activity.

Controversy exists over the best nutrient profile for diets for obese pets, specifically surrounding the roles of carbohydrates versus fat. Some veterinarians recommend low-carbohydrate diets for weight management, especially for cats. However, scientific evidence suggests that higher carbohydrate, lower fat diets are more effective for preventing obesity, whereas little data has been published differentiating these nutrients for weight loss in cats or dogs.[23,24] Although higher protein diets increase energy expenditure, and promote greater loss of fat while minimizing loss of lean-body mass when fed in restricted amounts for weight loss, studies do not show a benefit of high-protein diets for the prevention of weight gain.[25-28]

Success with weight-loss protocols is highly dependent on the pet owner, and owners may be more willing to comply with programs that are easy to follow. Various studies have indicated that pet owners may resist or fail weight-loss programs due to the lack of recognition of their pet as overweight; real or perceived inability to exercise their pet; unwillingness to purchase therapeutic diets; or lack of mindfulness regarding how they feed their pet.[29-31] Even when successful, weight loss in pets tends to occur at a slower rate of loss in a home setting compared to rates obtained in research settings.[25,29,32-34]

Relatively little information has been published regarding maintenance of loss following weight-loss protocols. Two studies (one in dogs, one in cats) showed a reduction in energy requirements induced by weight loss, potentially increasing the risk for weight rebound.[35,36] A separate study showed lower energy requirements in cats shortly after weight loss but documented an increase in energy expenditure over time during the weight-maintenance period.[27] Despite this potential challenge, pet dogs maintained their reduced weight when they and their owners were part of a program of continued follow-up evaluations.[37]

These few studies show that it is possible to correct obesity in pets. However, given that the prevalence of overweight and obese conditions remains above 50%, the current methods for preventing and managing obesity have room for improvement.

Why Aren't We Succeeding?

Preventing pet obesity is theoretically easier than correcting it. However, this requires the pet owner to have an understanding about what a healthy body condition looks like in their pet and to actively feed the pet to achieve such a body condition. Current evidence suggests that pet owners do not recognize ideal body condition or excess body weight in their own pets.[29-31]

Thomas Webb, of the University of Sheffield, has referred to "The Ostrich Problem," in which individuals avoid or reject information or self-monitoring.[38] Instead, they "stick their head into the sand" and wait for the situation to "go away." Webb's use of this analogy refers primarily to behaviors related to individual achievement of goals and is not specific to obesity. However, this concept applies readily to both human and pet obesity and to pet owners who opt not to monitor their pet's body weight or calorie intake. I propose applying this term even more broadly. Rather than individuals, I propose that the veterinary community and others with the ability to influence pet owners have tended to apply the "Ostrich Principle" to weight management by ignoring excess body weight and risk factors that will lead to obesity and obesity-related comorbidities in their patients. As suggested by the data from Lund, et al.,[3,4] in which veterinarians neglected to diagnose overweight or obesity in 69 to 96% of overweight and obese cats and dogs, our profession may be choosing to stick our "collective heads" in the sand. Various reasons have been suggested to explain these missed diagnoses though without supportive data. Perhaps one step toward successful management of pet obesity may involve developing a better understanding about why both veterinarians and pet owners avoid diagnosing or recognizing obesity and why they avoid addressing it.

Future Directions

If one reviews the literature from the past several decades, it is clear that considerable research has been underway to better understand the causes and treatments for obesity. However, despite this, obesity remains a growing problem. It is becoming widely recognized that human obesity is not simply a dietary problem and that the solution does not rest exclusively within the medical community. Instead, a comprehensive approach will be needed to encourage changes in lifestyle, physical activity, diet, and expectations. Similar approaches may be needed in veterinary medicine and among pet owners. Some of the risk factors for pet obesity, e.g., inactivity, indoor lifestyle, and abundant food and treats, are potentially controllable factors that can be modified to reduce pet obesity. Key questions to be addressed are "How?" and "Who?"

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