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FELINE OBESITY: CAUSES, CONSEQUENCES AND MANAGEMENT

Jacque S. Rand, DVM; Delisa J. Appleton BVSc (Hons).
Brisbane, Australia.

CAUSES OF FELINE OBESITY

Regulation of food intake and body weight involves a complex balance between long-term control of fat mass involving insulin, adrenal steroids and leptin signals to the CNS and short-term, meal-related signals. Cats will normally limit their food intake to their energy requirements. However, in some instances cats appear unable to regulate energy balance. Our research has demonstrated that despite elevated circulating leptin levels in obese cats associated with increased fat mass, they continue to overeat and gain weight. This paradox of increased leptin concentrations in obesity has been observed in other species and is hypothesized to be a consequence of 'leptin resistance'.

A number of risk factors have been identified for obesity. Physical inactivity, extended sleeping periods and indoor confinement are associated with an increased incidence of obesity. Neutering results in weight gain, predominantly as fat mass in both genders. With neutering, reductions in roaming and in general physical activity occur along with increased food intake and the efficiency of energy utilization. Neutered male cats are more likely than females to gain excessive weight. The incidence of feline obesity increases with age peaking between the ages of six and eight years. Many pure bred cats such as Siamese or Abyssinians are leaner than most mixed breed cats, suggesting a genetic influence on body weight may exist in cats, as it does in humans. Chronic administration of corticosteroids and progestins, particularly megestrol acetate, induce polyphagia resulting in weight gain. The human-animal-relationship of owners of overweight cats is characterized by a higher intensity of the bond between owner and cat, and by increased humanization compared with the relationship in owners of normal weight cats.

CONSEQUENCES OF FELINE OBESITY

Obesity in companion animals has been shown to predispose to, or cause a number of medical conditions including glucose intolerance and diabetes mellitus as well as surgical and anaesthetic complications, reproductive compromise, non-allergic skin conditions, lameness, feline lower urinary disease, hepatic lipidosis and immuno-incompetence.

We investigated the effect of weight gain on glucose tolerance, insulin sensitivity, in cats. Cats in our study developed hyperinsulinaemia and reduced insulin sensitivity with weight gain. In fact, the insulin sensitivity index (S_i) as measured by Bergman's Minimal model, was 53% lower in cats after weight gain, indicating that obese cats were significantly less sensitive to insulin compared to when they were of normal-weight. Cats with the highest basal insulin concentrations after weight gain were also the most insulin resistant. Glucose effectiveness (S_G), was also significantly decreased in obese cats indicating that glucose utilization, independent of insulin, was reduced. Other characteristic metabolic features of obesity evident in the

obese cats included basal hyperinsulinaemia and an exaggerated insulin response to glucose during the glucose tolerance test. Half the cats were identified as developing impaired glucose tolerance with weight gain. Before gaining weight, these cats tended to have higher insulin concentrations, and lower insulin sensitivity and glucose effectiveness than cats that maintained normal glucose tolerance with weight gain. Our results suggest that some cats may have an underlying predisposition to develop glucose intolerance, and if these cats become obese and inactive, they may be more at risk of developing overt type 2 diabetes mellitus. Hypertriglyceridaemia was also present in our cats after becoming obese.

In the same study, we documented a three-fold increase in plasma leptin concentrations in cats as a result of weight gain. We also demonstrated the existence of a strong relationship between leptin and insulin resistance in both lean and obese cats similar to that reported in humans, however it is still unclear as to whether leptin is causally involved in this relationship.

Unlike their ancestors, modern domestic cats no longer hunt for food, have become relatively inactive and tend to overeat resulting in an increased incidence of overweight and obese cats. These changes parallel those that have occurred in the urbanization of many of the world's indigenous populations that have high levels of diabetes. In addition, cats are predominantly fed commercial diets, many of which contain significant levels of highly digestible cereal which cats as obligate carnivores, did not evolve to eat. These factors increase the demand for insulin production from the pancreatic beta cells and lend credence to the hypothesis of 'beta-cell exhaustion' as an important element in the pathogenesis of feline type 2 diabetes mellitus.

In both cats and humans, amyloid deposition in the pancreatic islets is the most consistent histological finding. Amyloid is formed from amylin (IAPP), a hormone which is co-secreted with insulin. In diabetic cats, a large amount of amyloid is deposited in the islets, replacing the cells and leading to a reduction in overall beta cell mass.

ASSESSMENT AND MANAGEMENT OF FELINE OBESITY

A safe and effective weight reducing regime involves restricting caloric intake to 60%-75% of estimated caloric requirements for ideal body weight (approximately 60 kcal/kg of ideal body weight per day), with the aim to maintain weight loss at approximately 1.0 to 1.5% per week. Regular weight checks, initially fortnightly then monthly, with appropriate adjustments of intake should be made. There is considerable individual variation in caloric requirements between cats, and food intake needs to be individually adjusted to ensure appropriate weight loss. Additionally, methods to increase energy output such as backyard activity enclosures, laser pointers and food cubes should be instituted.

Failures typically occur when the effect of the human-animal relationship overrides the owner's commitment to achieve weight loss, or when the calories calculated are in excess of the cats needs, and the cat does not have noticeable weight loss. The client becomes discouraged, and does not return for repeat visits, and the opportunity for intervention is lost.

Making weight loss happen: increasing physical activity

The cornerstone of management of obesity is that calories need to be limited, and physical activity increased. Increasing physical activity in cats is not as easy as in dogs, although preliminary data indicate the importance of increasing physical activity in a weight management program. In a recent study, active play for 10 min daily produced the same weight loss as calorie restriction (Giles 2003). In a weight loss study over 8 weeks, cats given environmental enrichments such as additional food dishes, water bowls and litter boxes, plus climbing towers, window perches, scratching posts, cat spas, grooming supplies and toys, had significantly increased activity levels as measured by a activity monitor, and had a trend to greater weight loss compared to cats that did not have an enriched environment (Trippany, J.R., Funk).

Other strategies that may aid in increasing physical activity are to feed cats using devices which require physical activity to release food. For example, food can be hidden inside a treat ball, and physical activity is required to get the ball to roll so food is released. Placing the food at the top of stairs or on top of a climbing frame, can help increase physical activity. Because cats kept indoors are physically less active than cats with access outdoors and are at greater risk of diabetes, allowing the cat outside for limited hours in day-light may help in achieving ideal body weight. However, this needs to be balanced by the health risks of car accidents and cat fights. Modular units are available commercially which allow outside access around the garden, but protect the cat from these risks.

Making weight loss happen: Calorie Restriction

For a weight loss program to be successful and safe, calories should be restricted to 80% of current intake. However, if accurate assessment of the cats current intake is not possible, feeding 60-70% of postulated daily energy requirement (set at 60 kcal/kg ideal weight) is the best solution (ie. 36 – 42 kcal/kg of ideal weight), provided the owner is warned that this level may still be too high to achieve weight loss for their cat. This value is the best estimate of a safe and effective calorific intake to achieve initial weight loss when the energy requirement of the cat is unknown. With repeated checks, eventually the calorie intake that provides effective weight loss will be determined. However, it may take up to 2 months of visits to determine this, if there is inadequate dietary history available at the start of the trial. It is important to realise that the energy requirements often change with weight loss, and morbidly obese cats with very low energy requirements may have an increased energy demand, once they achieve substantial weight loss and become more active. In other cats, weight loss plateaus after a time, presumably because of more efficient utilisation of energy, and further energy restriction is required for continued weight loss.

Making weight loss happen: Dietary manipulations that may aid weight loss and reduce the adverse metabolic effects of weight loss.

Several dietary manipulations have been investigated including the addition of fibre and altering macronutrient sources.

Fibre

Traditionally, a high fiber diet has been promoted for weight management in dogs and cats, although there is little scientific evidence to substantiate its effectiveness. Additionally, unwanted side effects such as increased fecal bulk and frequency of defecation may contribute to the lack of compliance of such diets in clinical practice. The inclusion of dietary fermentable fiber however, may be beneficial in the management of obesity. Fermentation of fiber by intestinal bacteria produces short chain fatty acids (SCFA) which in turn, stimulate the release of proglucagon from the intestinal lining cells. Proglucagon is further broken down to glucagon-like peptide-1 (GLP-1), which increases insulin secretion from the pancreatic islets.

Protein

The provision of meals containing adequate amounts of amino acids, particularly essential amino acids, is necessary for the anabolic effect of insulin on muscle protein synthesis. In a study of nine diabetic cats, a high protein, low carbohydrate diet has been shown to induce satiety as well as reduce the dose of insulin required. In another study in normal cats, compared to a high protein and low carbohydrate diet, a low protein and high carbohydrate diet produced hyperinsulinaemia and decreased NEFA mobilization which may in the long term, lead to weight gain, obesity and beta-cell 'exhaustion' in cats fed ad libitum. Anecdotal evidence indicates that some cats changed to a high protein-low carbohydrate canned diet will self-restrict energy intake and spontaneously lose weight, although the majority need to have calories restricted for weight loss to occur.

Carbohydrate

The proportion of calories ingested as carbohydrate is one of the main determinants of post-prandial glucose and insulin concentrations. Diets with moderate to low carbohydrate content (<25% of calories) are the most appropriate for preventing diabetes in predisposed cats and for managing diabetes. Dietary carbohydrate source is also a consideration when developing diets for both healthy cats as well as for obese, glucose intolerant cats. Certain carbohydrate sources such as sorghum and barley have been shown to lower the post-prandial glucose and/or insulin responses in dogs and cats when compared with rice. The inclusion of these types of starches in feline diets may improve glucose control in cats, reduce insulin and possibly amylin secretion, thereby minimizing pancreatic amyloid deposition and beta-cell 'exhaustion'. Therefore, diets with restricted carbohydrate content and formulated using low-glycemic increase carbohydrates are likely important for the prevention or management of obesity-induced glucose intolerance and diabetes.

RECOMMENDED READING

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