Advances in Dietary Management of Obesity in Dogs and Cats

Richard F. Butterwick and Amanda J. Hawthorne

Waltham Centre for Pet Nutrition, Waltham-on-the-Wolds, Melton Mowbray, Leicestershire, LE14 4RT, UK

ABSTRACT Recent evidence in humans has reemphasized the importance of specific lifestyle behaviors such as activity level on energy requirements. A recent survey of adult pet dogs has shown a clear association between the level of activity and energy requirement, and suggests that current feeding recommendations may overestimate the energy requirements of adult dogs. Although a reduction in feeding guides may help to reduce the risk of overfeeding and subsequent development of obesity in adult dogs, there is considerable individual variation in energy requirements, which emphasizes the importance of tailoring feeding practices to the individual. Diet clearly has a critical role to play in both the prevention and treatment of obesity. We have evaluated the effect of different dietary regimens on the treatment of obesity in companion animals. In cats, increased energy restriction results in more rapid weight loss. However, this is associated with less favorable changes in body composition. In dogs, we have evaluated the potential benefit of insoluble and soluble dietary fiber on satiety in dogs that have been restricted to an energy intake appropriate for weight reduction. Results of a series of studies have failed to show any benefit of either fiber type on satiety in energy-restricted dogs. J. Nutr. 128: 2771S–2775S, 1998.

KEY WORDS: obesity dogs cats fiber intake body composition

The ability to gain weight can be viewed as a natural defense against periods of food shortage. Hibernating animals represent an extreme example of this trait, whereas certain Third World populations exhibit more moderate, albeit profound, seasonal changes in body weight that are associated with fluctuations in food supply (Prentice et al. 1981). In these circumstances, weight gain can be viewed as a positive trait, enabling the individual to accommodate subsequent weight loss. In the developed world, the situation is very different; food shortages are generally rare, and it is perhaps no coincidence that obesity is reaching epidemic proportions in humans (Gregory et al. 1990, Kuczmarski et al. 1994) and is of increasing concern in companion animals (Edney and Smith 1986, Scarlett et al. 1994). The negative health implications (Bray 1996) and socioeconomic costs (Seidell 1995) associated with obesity in humans emphasize the need to develop strategies for both the prevention and treatment of this condition. Although the health implications of obesity are less well defined in dogs (Markwell and Butterwick 1994), there is little doubt that obesity has an effect on quality if not quantity of life in this species.

Despite recent proposals that dogs represent a potentially useful model for research on the pathogenesis of obesity in humans (Stock 1996), there have been relatively few studies in this area. This review will focus on aspects of treatment rather than prevention of obesity because this reflects the majority of work conducted in companion animals. However, comparative data from other species will be referred to in the context of obesity prevention because this may have future application to the control of obesity in companion animals.

CAUSES OF OBESITY

Recent advances have made significant contributions to our understanding of the causes of obesity. Identification of a genetic basis for obesity in rodent models resulting from single-gene mutations such as leptin and the leptin receptor have provided an insight into the metabolic control of energy metabolism and the physiologic basis of defects underlying the development of obesity (see reviews by Roberts and Greenberg 1996, Rosenbaum et al. 1997, York 1996). However, the relevance of rodent models with single-gene mutations to obesity in humans (and other species) is at present uncertain. Although obesity in humans can result from single-gene defects, these are relatively rare (Bray 1996). Nevertheless there is evidence of a significant genetic and familial association of obesity in humans (Stunkard et al. 1986 and 1990) and a breed predisposition in dogs (Markwell and Butterwick 1994). In humans (Roberts and Greenberg 1996) and presumably most other species, it has been concluded that obesity is a multifactorial, multigene condition resulting from a complex interaction of environmental and genetic components.

Notwithstanding recent developments in our understanding of the genetic basis of obesity, the identity of an underlying metabolic factor(s) has proved elusive. For the lay person and indeed certain health professionals, the underlying cause(s) and most effective treatment(s) for obesity remain shrouded in mythology. It is not uncommon for physicians in both medical and veterinary professions to have obesity explained by pa-
Patients or clients as an abnormal medical condition unrelated to diet—"it's due to my metabolism" syndrome.

Early dietary surveys of food intake in obese individuals suggested that obese subjects may have a lower metabolic rate compared with nonobese subjects, thereby predisposing them to obesity. However, recent studies have shown that obese subjects habitually underreport food intake (Lichtman et al. 1992), undermining the concept that a defect in energy metabolism is responsible for the development of obesity. Studies investigating the relationship between energy expenditure and obesity have provided conflicting results, which may be due in part to methodological issues. It has been suggested that the relatively small difference in energy intake and energy requirement required to cause a positive balance sufficient to cause obesity, given adequate time, may well be beyond the sensitivity of current techniques (Roberts and Leibel 1998).

**TREATMENT OF OBESITY**

Irrespective of the underlying mechanism, obesity is a result of energy intake exceeding energy expenditure with the resultant storage of the surfeit as adipose tissue. If excess energy intake is the cause of this condition, then the remedy, decreased energy intake, would appear to be a simple solution. However, a relationship between food supply and obesity should not be viewed as the exclusive cause, or even causal, because other factors such as activity level may be contributory.

**Energy expenditure.** A recent review of historical epidemiologic data has revealed some interesting secular trends associated with obesity (Prentice and Jebb 1995). In the UK over the past 40 years, in contrast to a trend of increased prevalence of obesity, there has been a decline in the average daily consumption of energy and fat. It has been concluded that the apparent decline in energy (and fat) consumption intake and paradoxical increase in obesity prevalence can be explained only if there has been a much larger decline in energy expenditure. Although historical measures of energy expenditure are not available, secular trends of physical activity (or inactivity), based on proxy indicators such as number of cars owned per household and hours spent viewing television, show a close association with obesity. Although these can be regarded only as circumstantial, they indicate that total daily energy expenditure and the level of physical activity, in particular, may be critical factors underlying the development of obesity.

We have recently reviewed (unpublished data) energy intake in a single breed of dog and examined the relationship between the level and type of activity with energy requirement. Information on food intake and activity level was obtained using either a questionnaire sent out to dog owners or from a feeding study. Questionnaires were sent to owners of pet, show and working Border Collies. Owners were asked about the diets they fed their dog (type, brand and daily allowance), the incidence and type of any treats fed and level of activity and type (pet or working) of activity. Of the 309 questionnaires completed, energy intake was assessed in 39 dogs that were fed a single brand of dry diet and had a quantifiable intake of treats. Metabolizable energy intake was determined from proximate analysis of the diets [Association of American Feed Control Officials (AAFCO) 1995] and the reported amount the dogs consumed. Dogs that were still growing (<12 mo old) or were overweight were excluded from the analysis because these were not considered representative of the nonobese adult population. In a separate study, food intake was recorded using a food diary in both working and pet Border Collie dogs. Dogs were fed two complete dry diets of known energy content in amounts sufficient to maintain weight for 8 wk each in a randomized block design. Body weight was monitored over the duration of the study, and food intake was recorded using a food diary during the final week of each feeding period. Energy intake was determined as a mean of the intake of the two diets in dogs who had a quantifiable intake of snacks and treats. Data were obtained from 12 pet and 17 working dogs. Energy intake and activity levels of pet and working dogs were considered separately. Data from both studies were pooled because there was no significant difference in energy intake between the two sources of information (questionnaire or food diary) in either pet or working dogs.

The majority (70%) of pet dogs received between 1 and 3 h of daily exercise (Table 1). Of the remainder, a similar proportion had <1 h (17.5%) or >3 h (12.5%) exercise. A higher percentage (25%) of working dogs were active for >3 h/d. Of the remainder, a similar proportion had <1 h (39.3%), or between 1 and 3 h (35.7%) of daily exercise. Although it was not possible to assess the intensity of exercise, food intake averaged across all activity levels supports the concept that intensity of activity was higher in working [691 kJ/(kg body weight)0.75 · d] compared with pet dogs [496 kJ/(kg 0.75 · d)]. Pet dogs exercised for >3 h/d had a significantly (P < 0.05) higher energy intake than dogs receiving <3 h of daily exercise (Table 2). The distinction between activity categories for working dogs was less clear, which in part may be due to the wide variation in energy intake (and presumably intensity of activity) of dogs that exercised between 3 and 6 h/d. However, energy intake of dogs that exercised between 1 and 3 h/d was significantly (P < 0.05) higher than those that exercised <1 h/d.

These data indicate that there is considerable variation in the energy requirement of adult dogs, which may be due in part to differences in both the level and type of activity. There was considerable variation, however, in the energy intake of dogs within distinct exercise categories (Table 2). Although this may reflect differences in the intensity of activity, other factors such as the efficiency of energy utilization and environmental differences may also contribute to individual variation, which emphasizes the need to tailor feeding practices to individual requirements.

In this survey, the vast majority of pet dogs (87.5%) received either <1 h or between 1 and 3 h of daily exercise. Taken together, the mean energy intake of these dogs was ~462 kJ/(kg0.75 · d). This value is considerably lower than current recommendations [532 kJ/(kg0.75 · d)] of energy requirements for adult dogs (NRC 1985). Although food intake based on food diaries or recall is subject to variation, these data suggest that current recommendations, based on studies in kennel dogs (NRC 1985), may overestimate the energy requirement of the vast majority of adult dogs in the pet population and potentially contribute to the development of obesity. It is likely that the discrepancy between the NRC

### Table 1

<table>
<thead>
<tr>
<th>Lifestyle</th>
<th>n</th>
<th>&lt;1 h</th>
<th>1–3 h</th>
<th>3–6 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pet</td>
<td>40</td>
<td>7</td>
<td>28</td>
<td>5</td>
</tr>
<tr>
<td>Working</td>
<td>28</td>
<td>11</td>
<td>11</td>
<td>6</td>
</tr>
</tbody>
</table>

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**TABLE 1**

Reported activity levels in pet and working Border Collie dogs.
(1985) recommendation for energy requirements of adult dogs and data from our food intake survey is due to differences in activity levels. These findings are in agreement with recent data in humans, indicating that low activity levels may be a significant factor underlying the development of obesity (Prentice and Jebb 1995). In light of these findings, further work should be conducted to evaluate the energy requirement of free-living adult dogs under different activity conditions.

Dietary treatment of obesity. A reduction in energy intake is the obvious dietary route to achieve weight loss. The most appropriate method for reducing energy intake, and the degree of energy restriction required to achieve weight loss, in particular promoting fat loss while minimizing loss of lean body mass in dogs and cats, is unclear.

Effect of energy restriction on weight loss and body composition in cats. Loss of lean body tissue appears to be an inevitable or an obligatory physiologic response to weight reduction in humans (Forbes 1987). However, excessive loss of lean body tissue is not desirable because functional tissue losses will have to be replaced. In humans, initial body weight (or body fat content) and the degree of energy restriction are key factors influencing the composition of weight loss (Forbes 1987, Prentice et al. 1991). Although severe energy restriction results in rapid weight loss, this is associated with relatively high losses of lean body mass (Prentice et al. 1991). In companion animals, weight reduction programs have been developed principally on the basis of changes in body weight and maintenance of good health (Butterwick et al. 1994b, Markwell et al. 1990). Although this has provided information on efficacy and safety of weight reduction regimens for both dogs and cats, until recently there has been limited data on the effect of weight loss on body composition.

In cats, moderate energy restriction to ∼60% of adult maintenance requirements at target body weight over an 18-wk period resulted in a weight loss of ∼1%wk with the majority of weight loss from body fat (90%) and minimal loss of lean body tissue (8%) (Butterwick and Markwell 1996). Increasing energy restriction to 45% of adult maintenance requirements at target body weight resulted in a greater rate of weight loss averaging ∼1.3% over an 18-wk period (Butterwick et al. 1995). Although this level of energy restriction proved safe in cats, it resulted in an increase in the proportion of weight loss from lean body mass (19%) and a relative decrease in the proportion of weight loss from body fat (80%) compared with the cats restricted to 60% of adult maintenance energy requirements at target body weight. These data suggest that higher rates of weight loss in cats may have an undesirable effect on body composition.

The only comparable data in dogs appear to be from a study in which obese dogs were fed diets differing primarily in fiber content (Borne et al. 1996). Dogs fed a high fiber diet lost proportionately more body fat and less lean body tissue than dogs fed an equivalent amount of a low fiber diet. Because both diets were reported to have been fed at an equivalent energy intake, these findings are difficult to reconcile with those in humans (Prentice et al. 1991), unless the conditions of the study (i.e., initial body weight, degree of energy restriction, exercise and/or protein intake) were not standardized. It is possible that dietary fiber may represent a significant source of energy, particularly in high fiber diets. A digestible energy value for dietary fiber in mixed diets for humans of ∼8.4 kJ/g has been proposed (British Nutrition Foundation 1990). It is not clear in the study of Borne et al. (1996) whether dietary fiber was considered as a source of energy because neither the method used for measuring energy content of test diets nor the source of dietary fiber was specified. It is possible that the net energy content of the high fiber diet in the study of Borne et al. (1996) may have been under- or overestimated and resulted in an energy intake different than that specified; this could account for some of the differences in body weight and composition observed. There are limited data on the digestibility of dietary fiber in dogs or cats, and it is clear that further research is required to define the contribution of dietary fiber to energy intake in companion animals. This has particular relevance to weight reduction diets, particularly those that are high in fiber because a precise value of the energy content of the diet is critical in ensuring a controlled reduction in energy intake.

Effect of dietary fiber on food intake in dogs. In human and small animal medicine, dietary fiber has been included in foods in an attempt to overcome hunger and increase compliance during weight reduction programs. However, evidence demonstrating a clear effect of fiber on food intake in humans is equivocal (Burley et al. 1987, Burley and Blundell 1990, Krotkiewski 1984, Levine et al. 1989). Furthermore, long-term studies indicating a clinical benefit of dietary fiber in weight reduction programs are lacking.

Notwithstanding these observations, and despite the fact that relatively few studies have specifically evaluated the satiating properties of dietary fiber during energy restriction in dogs or cats, both insoluble and soluble fibers have been used in some weight control products for companion animals as a means to restrict energy intake. This philosophy to a large extent has been directly translated from the approach in humans in which the idea of dietary “fiber” as an aid to slimming diets has been enthusiastically embraced by the lay public and some health professionals (British Nutrition Foundation 1990).

We have conducted a series of studies to evaluate the role of dietary fiber on food intake and satiety in dogs. In these studies, diets with different levels of fiber were fed at the same restricted energy intake, thereby mimicking conditions during weight reduction. An objective measure of satiety was provided through intake of a challenge meal. This consisted of

### Table 2

<table>
<thead>
<tr>
<th>Activity level</th>
<th>n</th>
<th>Pet energy intake</th>
<th>Working energy intake</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>kJ/kg0.75</td>
<td>kJ/kg0.75</td>
</tr>
<tr>
<td>h/d</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1</td>
<td>7</td>
<td>502 ± 123.4</td>
<td>598 ± 134.3</td>
</tr>
<tr>
<td>1–3</td>
<td>28</td>
<td>452 ± 106.7</td>
<td>770 ± 83.3</td>
</tr>
<tr>
<td>3–6</td>
<td>5</td>
<td>741 ± 275.7</td>
<td>728 ± 247.3</td>
</tr>
<tr>
<td>All</td>
<td>40</td>
<td>496 ± 164.8</td>
<td>691 ± 169.9</td>
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1 Values in the same column with different subscripts differ P < 0.05; values are means ± so.
offering dogs on two occasions during each study a second meal exactly 3 h after consumption of the test diet. Dogs were allowed 15 min access to the challenge meal, after which intake was measured. This technique therefore provided an objective measure of the degree of inhibition of food intake (satiety) resulting from consumption of the preceding test diet. Because test diets were fed (and consumed) at the same restricted energy intake, this provided a realistic assessment of satiety in dogs during weight reduction and prevented any confounding effect of different energy intakes on satiety.

In the first study (Butterwick et al. 1994a), moderate levels of different dietary fibers, composed principally of insoluble dietary fiber, were added to a commercial low calorie diet, resulting in diets that differed only in fiber content. Results of this study demonstrated that the dietary fibers, at the inclusion level used, had no effect on satiety as measured by the challenge meal intake. Although the diet with the highest fiber content resulted in a fiber intake of 9.2 g/d, which is comparable to fiber doses used in studies in humans (Burley et al. 1987, Burley and Blundell 1990, Krotkiewski 1984), it was lower than that found in some commercial high fiber weight reduction diets.

In a subsequent study, using identical methods, we evaluated diets to which much higher levels of either an insoluble or soluble fiber had been added (Butterwick and Markwell 1997) (Table 3). The soluble fiber was added at somewhat lower amounts than the insoluble fiber because high intake of soluble fiber can cause bloating and abdominal cramps. The amount of insoluble fiber (a-cellulose) added was designed to reflect the amount and type of insoluble fiber commonly found in commercial high fiber diets recommended for weight reduction.

Results of this study confirmed our initial findings, demonstrating that, when fed in allowances appropriate for weight reduction, diets that result in very high fiber intakes have no beneficial effect on satiety. The fiber intake of dogs receiving the highest fiber diet in this study was 20.4 g/d, which when expressed as a metabolic body weight basis, greatly exceeded the doses previously used in humans studies. In fact, in a nonobese human male, this would be equivalent to a daily dietary fiber intake of ~120g/d. Even at this level of fiber intake, we were not able to demonstrate any benefit on satiety. The contribution of dietary fiber to energy intake, particularly in high fiber diets, is a potential confounding factors in studies of this nature. In our studies, the energy content of the test diets was calculated either by an equation based on Atwater factors (AAFCO 1995) or by assigning an energy coefficient to dietary fiber of 8.36 kJ/g. Although these methods may result in either an under- or overestimate of the metabolizable energy content of the diet, the energy values produced by both methods were in close agreement and resulted in differences of <3% in the diet with the highest fiber content.

The role of fiber as a component of dietary management in weight reduction programs has recently been reviewed (British Nutrition Foundation 1990). It was concluded that, despite the popular assumption that dietary fiber is an aid to slimming programs, current evidence supporting this view is inconclusive. Control of food intake is a complex and multifactorial process, and the concept that food intake can be controlled by the dry matter or fiber content of the diet in nonruminant species is overly simplistic. Results of our studies show that in dogs that are restricted in energy intake to a level currently recommended for weight reduction, neither soluble nor insoluble fiber influences satiety. In the light of findings from these studies, perhaps the preoccupation with high fiber diets for the management of obesity in companion animals should be reappraised, and more attention should be focused on the nutritional adequacy of these diets when fed at a restricted energy intake. There is a clear need for well-designed long-term studies to demonstrate whether dietary fiber treatments are more effective than other regimens in the companion animal.

In conclusion, recent advances in the study of genetics have provided new insights concerning the genetic basis of obesity. However, considerable effort is required to elucidate the mechanisms underlying the development of obesity. It is clear that obesity is not simply a case of excess energy intake or "gluttony" because nondietary factors such as lifestyle behaviors may contribute to the development of this condition. Research in companion animals has focused on various aspects of the treatment of obesity, such as the effect of energy restriction on body composition and the role of dietary fiber. However, the current epidemic of obesity in humans and companion animals, and the almost inevitable recidivism that occurs after weight loss in obese subjects emphasizes the need for further research on the prevention of this condition.

**LITERATURE CITED**

Association of American Feed Control Officials (1995) AAFCO Official publication. Atlanta, GA.


| Fiber content of test diets and fiber intake of dogs fed |
|---|---|---|---|---|---|
| Diet | CL1 | CL2 | SF1 | SF2 | IF1 | IF2 |
| TDF g/MJ | 3.1 | 3.6 | 4.7 | 9.5 | 16.3 | 23.7 |
| IDF g/MJ | 0.9 | 0.9 | 1.3 | 2.4 | 13.9 | 21.7 |
| SDF g/MJ | 2.2 | 2.7 | 3.4 | 7.1 | 2.4 | 2 |

1 TDF, total dietary fiber; IDF, insoluble dietary fiber; SDF, soluble dietary fiber; CL1, control low-energy diet; CLC2, control low-energy diet; SF1, medium soluble dietary fiber diet; SF2, high soluble dietary fiber diet; IF1, medium insoluble dietary fiber diet; IF2, high insoluble dietary fiber diet.


