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RESEARCH REPORTS

A Research Update for the Veterinarian from Affinity Petcare

# Canine Obesity

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Obesity is best regarded as a disease. Its prevalence in dogs in developed countries means that obesity could be regarded as the most common nutritional disease that affects dogs, although the associated conditions seen as a consequence of obesity are indeed quite varied. There is much recent research about obesity in dogs, and this has helped to identify risk factors, the precise nature of associated diseases and the best ways for treatment using diet and exercise.

## INTRODUCTION

Obesity is defined as an accumulation of excessive amounts of adipose tissue in the body. In humans, epidemiologic data had demonstrated an increasing morbidity and mortality risk with increasing body fat mass. In dogs, Lawler *et al* (2008) have demonstrated that in Labrador Retrievers a fat mass above 25 % is associated with increased insulin resistance, which independently predicts lifespan and chronic diseases. Obesity is also one of the most common nutritional diseases in dogs and cats. In recent studies reported from Europe, USA. Australia and Brazil, between 17 and 44% of dogs are reported as overweight or obese (Figure 1).

## RISK FACTORS

Epidemiological studies have highlighted several different risk factors associated with canine obesity levels, some examples are shown in Figure 2.

## OBESITY AS A DISEASE

Detrimental effects of obesity and increased fat mass on health are due mainly to mechanical stress (joint, heart...) and

## INCIDENCE

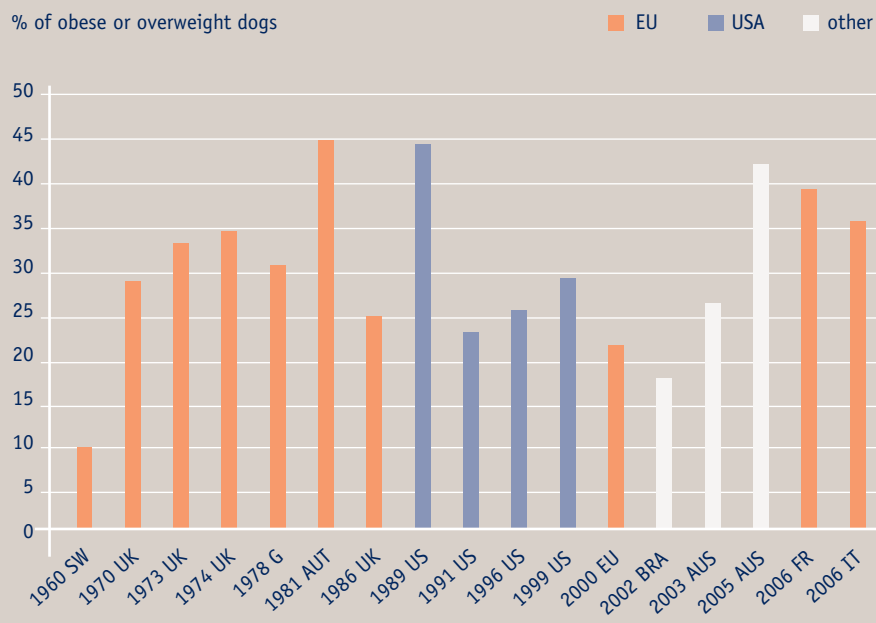



**Figure 1.** Incidence of obesity in dogs across several industrialized countries

**SW** Sweden; **UK** United Kingdom; **G** Germany; **AUT** Austria; **US** United States of America; **EU** Europe; **BRA** Brazil; **AUS** Australia; **FR** France; **IT** Italy. (Adapted from Mason, 1970; Anderson, 1973; Meyer *et al*, 1978; Steininger, 1981; Edney & Smith, 1986; Crane, 1992; Sloth, 1992; Wolfsheimer, 1994; Armstrong & Lund, 1996; Lund *et al*, 1999 and 2006; Jerico & Scheffer, 2002; Robertson, 2003; Mc Greevy *et al*, 2005; Colliard *et al*, 2006; Mussa *et al*, 2006)

metabolic changes from excess fat (with increasing secretion of inflammatory adipocytokines).

## Life expectancy

Several pathologies have a higher prevalence in overweight and obese dogs, like orthopaedic disease, diabetes mellitus, abnormalities in circulating lipid profiles, cardiorespiratory disease, renal and urinary disorders, neoplasia, dermatological diseases... (figure 3 and table 1). Therefore, we can presume that obesity may decrease longevity of the dogs, as has been reported in humans. From the data of Kealy *et al* (2002) and Larson *et al* (2003), it appears that dogs fed *ad libitum* all their life have higher body fat and serum triglycerides, insulin and glucose concentration, and their median life span was significantly lower (almost 2 years less) compared with dogs that were 25 % restricted in their energy intake.

## Orthopaedic diseases

Othopaedic diseases, including traumatic or degenerative disorders (osteoarthritis [OA], humeral condylar fractures, cranial cruciate ligament rupture, intervertebral disc disease) are clearly more frequent in obese dogs. Excessive body weight of obese dogs will increase the mechanical stress on joints and precipitates osteoarthritis. It has been shown that dogs fed *ad libitum* for a long period of time have a higher frequency and greater severity of hip, shoulder and elbow osteoarthritis than energy-restricted dogs (Kealy *et al* 2000; Kealy *et al* 2002). Clearly, body weight reduction in osteoarthritic obese dogs improves the degenerative process and reduces pain by reducing stress on joints. There is a great benefit from the application of weight reduction diets for obese dogs with OA and lameness, as has been shown by Rocksin *et al* (2007).

## Obesity and inflammatory state

In dogs as in humans, adipose tissue is now considered as an endocrine organ that secretes cytokines called adipokines, some of which are pro-inflammatory and implicated in the pathologies linked to obesity. In humans, obesity is characterised by a chronic, systemic, low grade inflammatory state. The leukocyte count, TNF-`, and CRP are increased in obesity associated with insulin resistance and promote inflammation. It is suggested that inflammation may be a potential mechanism whereby obesity leads to insulin resistance and other chronic pathologies.

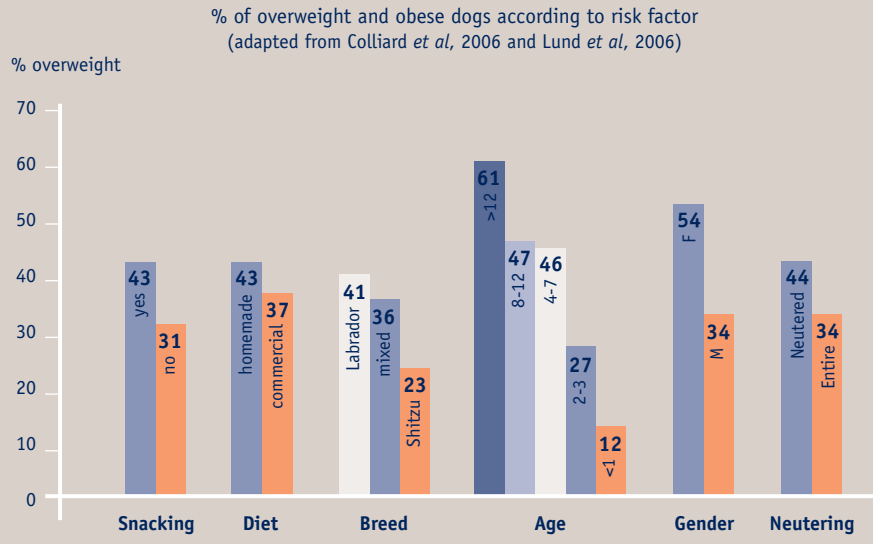



**Figure 2.** Risk factors for canine overweight or obesity: in a population with an average 34-39% overweight and obese


**Figure 3.** Percent of dogs affected by selected diseases; related to body condition [overweight and obese vs. lean dogs] (adapted from Lund *et al*, 2006)

* LUTD: Lower urinary tract disease

* Mobility problems: arthritis/osteoarthritis, lameness, musculoskeletal disease and/or ruptured cruciate ligament

## Obesity, insulin resistance, diabetes mellitus and hyperlipidaemia

Even if obesity is not clearly related to type 2 diabetes in dogs, unlike in humans, some authors have reported that 61% of obese dogs present with hyperinsulinaemia, glucose intolerance or both (Mattheeuws *et al*, 1984b). A syndrome similar to "human central obesity" has been observed experimentally in dogs by *ad libitum* feeding of a high fat diet: these dogs became obese, with increased abdominal fat mass and showed progressive development of insulin resistance and hyperlipidaemia (Rocchini *et al*, 1987; Van Citters, 2002; Bailhache *et al*, 2003; Kim *et al*, 2003;). In energy-restricted dogs, a better insulin sensitivity and lower glucose concentration are associated with a greater life expectancy (Kealy *et al*, 2002; Larson *et al*, 2003).

## Respiratory conditions, exercise and heat intolerance

Exercise intolerance and respiratory difficulties are frequently associated with canine obesity (Rocksin *et al*, 2007). There is also a correlation between obesity and tracheal collapse. In practice obese dogs are frequently inactive and lethargic.

## Cardiovascular disease and hypertension

When a substantial excess of body weight is observed in dogs, the frequency of cardiovascular diseases is also increased (Edney and Smith, 1986). It has been shown that blood pressure is higher in overweight dogs compared to those of ideal body weight, but there is no clear evidence about the origin and the mechanism of hypertension in obese insulin resistant dogs.

## Renal and urinary disease

Obesity induced by high-fat food for dogs results in glomerular hyperfiltration, sodium retention and hypertension (de Paula *et al*, 2004). Histological and functional changes that occur in the kidney in the early stage of obesity have been studied in dogs. In obese dogs, compared to lean dogs, kidney weight, plasma renin activity, insulin concentration, arterial pressure, glomerular filtration rate and renal plasma flow was higher. Histological changes are also observed in Bowman's capsules, mesangial matrix, glomerular and tubular membranes, … All these changes may be the precursors of more severe glomerular injury associated with prolonged obesity.
Overweight dogs have also been shown as having a higher risk of developing calcium oxalate calculus that can reduce kidney function (Lekcharoensuk *et al*, 2000).

## Anaesthaesia and clinical examination

Risks during anesthaesia are greater when a dog is obese, and this needs to be considered when deciding about surgery on obese dogs.

## Cancer

In dogs, obesity at 1 year of age and 1 year before diagnosis was found to be related to prevalence of mammary tumors (Sonnenschein *et al*, 1991). An increased risk of developing transitional cell carcinoma of the bladder has been reported for overweight dogs (Glickman *et al*, 1989).

## Early versus chronic obesity

Generally, obesity develops in 2 phases: a dynamic phase followed by a static phase. During the dynamic phase of

| Disease/Disease category | Obese (4.5 < BCS ≤ 5.0) n = 1099 | Overweight (3.5 < BCS ≤ 4.5) n = 6302 | Normal and Under-weight (1.0 < BCS < 3.5) n = 14,353 |
|---|---|---|---|
| Arthritis/Osteoarthritis | 4.2% | 4.0% | 2.4% |
| Dermatopathy | 18.9% | 18.6% | 17.2% |
| Diabetes mellitus | 0.7% (OR = 2.6), | 0.4% | 0.3% |
| Dystocia | 0.0% | 0.1% | 0.2% |
| Gastrointestinal disease | 6.6% | 7.3% | 7.3% |
| Heart disease | 3.8% | 3.4% | 3.9% |
| Hypertension | 0.0% | .08% | .03% |
| Hyperadrenocortism | 0.6% | 0.5% (OR = 2.4), | 0.2% |
| Hypothyroidism | 3.3% (OR = 2.8) | 1.5% (OR = 1.4), | 0.8% |
| Intervertebral disc disease | 1.7% | 2.0% | 1.8% |
| Lameness | 4.3% | 3.7% | 2.9% |
| Lower urinary tract disease (LUTD) | 3.6% | 3.6% (OR = 1.3) | 3.0% |
| Musculoskeletal disease | 8.6% | 8.3% | 6.1% |
| Neoplasia | 10.3% (OR = 1.4). | 7.6% | 5.3% |
| Oral disease | 34.1% | 30.5% (OR = 1.1) | 25.7% |
| Pancreatitis | 0.9% (OR = 2.2), | 0.5% | 0.4% |
| Paresis/Ataxia | 0.3% | 0.2% | 0.3% |
| Renal disease | 1.1% | 0.8% | 0.9% |
| Reproductive disease | 0.5% | 1.0% | 1.4% |
| Respiratory disease | 3.5% | 3.2% | 3.6% |
| Ruptured cruciate ligament | 1.6% (OR = 2.1), | 1.1% (OR = 1.7), | 0.5% |

**Table 1.** Disease and disease category* prevalence by body condition category for adult dogs (from Lund *et al*, 2006)

BCS = body condition score on 5-point scale
OR = ODDS RATIO
*Dog was reported to have at least 1 disease in categories given

obesity, in parallel with body weight and fat gain, the metabolism of the dog changes (increased plasma leptin and thyroid hormones). During this phase, blood parameters (insulin, glucose, lipids, ...) remain in the normal range and no physiological alteration are observed.
In contrast, once the static phase has begun, chronically obese dogs present with modifications to blood lipids and insulin. Therefore, treatment should be applied as soon as possible to restore optimal body composition and blood parameters. Most of the metabolic modifications are easily reversed by energy restriction and body weight loss (Jeusette *et al*, 2005a, b).
A recent dietary intervention clinical study with a low fat, high protein diet showed a decrease in blood triacylglycerol, cholesterol, insulin and fructosamine with weight reduction (Rocksin *et al*, 2007).
The study clearly showed an improvement of dogs' well-being: mobility, dyspnoea, heat and exercise intolerance (Rocksin *et al*, 2007), confirming other experimental or clinical trials with high protein diets (Diez *et al*, 2002; Jeusette *et al*, 2005b, Impellizerri, 2000, Carciofi *et al*, 2005; Saker and Remillard, 2005).

## TREATMENT - DIETARY INTERVENTION

Treatment for overweight and obese dogs is always indicated when some of the pathologies described earlier have arisen. Dietary intervention with energy intake restriction is the only obvious treatment for weight reduction. A low-energy diet in unrestricted quantity is not sufficient to control BW in overweight dogs (Jeusette *et al*, 2006b).
A strong caloric restriction (40 %) has been the only documented means to increase life span in rodents and in various invertebrate species. However, this approach is difficult to apply in humans due to an unsatisfactory permanent sensation of hunger and secondary side effects (decreased libido, serotonin, heat adaptation).
In dogs, 30% food restriction for 3 months decreased total plasma glutathione level, modified the fatty acids pathway and decreased the proinflammatory ratio of fatty acids in red blood cell membranes (Torre *et al*, 2006a).
A life long 25% food restriction resulted in lower body weight, body fat, serum triacylglycerol, triiodothyronine, insulin and glucose concentrations. These parameters are considered as ageing markers that may have value as signals for preventive medical intervention or treatment earlier in life (Kealy *et al*, 2002).
Different methods may be employed to achieve energy restriction: a reduction of food offered or a reduction of energy density (energy dilution). Restriction simply of the amount of a food offered is not recommended because it would mean a substantial reduction of the quantity of food eaten, while at the same time increasing a lack of satisfaction of the dog due to high hunger sensations. Also there is a potential deficit in nutrients such as some minerals, vitamins, oligoelements, essential fatty acids and amino acids.
Increasing satiety from a food is an additional approach, such as increasing level of proteins and medium chain fatty acids. The mechanism of satiety is not fully understood, but includes hormonal control and feedback in the gut.
For weight reduction programmes, the use of a complete diet designed specifically for obese dogs is always recommended. The traditional method of lowering food energy density is to reduce fat and increase fibre. Increased water and/or decreased physical density of a dry food (g/cm$^3$) can also be applied. In addition to the traditional low fat, high fibre energy reduced diet, increasing protein or reducing high glycaemic index carbohydrates have also been used with reasonable success in both humans and dogs.

## PREVENTION

### During growth

Hip dysplasia, osteochondrosis, radius curvus and hypertrophic osteodystrophy are recognized as being linked to an excess of body weight during growth (Hedhammer *et al*, 1974; Dammrich *et al*, 1991).
Obesity and excessive energy intake also result in modifications of hormonal secretions (IGF-1, thyroid hormones...) whose consequences remain to be evaluated in dogs. In small size dogs, the consequences of a high-energy intake on the osteo-articular system are less significant but may still lead to obesity in young adults. Energy needs during growth are of course higher than in adulthood, but excessive energy intake has to be prevented: just as in adults, the body condition score needs to be optimal during growth.

### After neutering

A strict feeding plan is recommended after neutering because neutered females have lower energy requirement, and their feeding behaviour is modified to favour weight gain (Figure 4) (Jeusette *et al* 2004b and 2006a). In neutered female dogs, body weight gain is prevented after surgery by a 20-30% energy restriction.

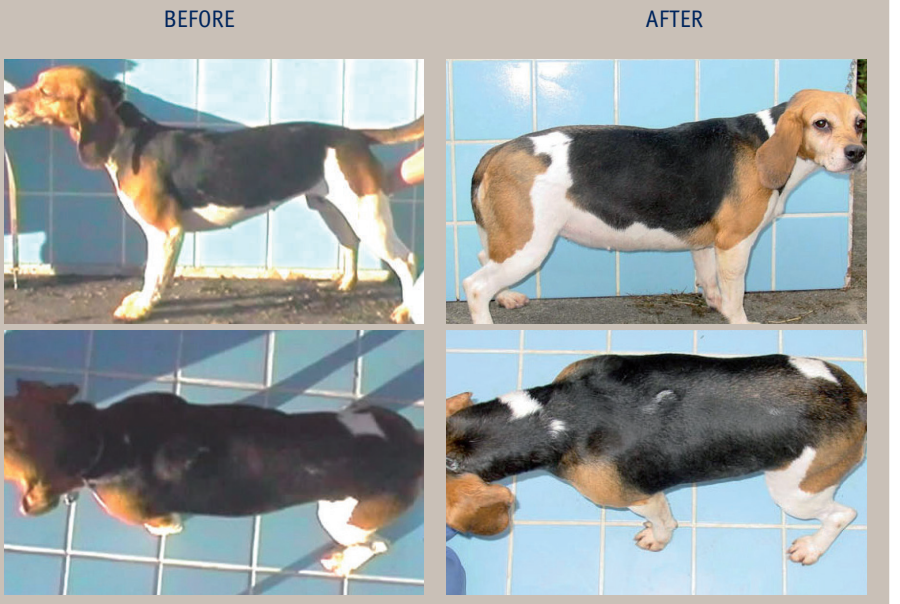



**Figure 4.** Violette before gonadectomy and after 5-months *ad libitum* feeding following gonadectomy: a substantial body weight gain is observed (+30%) that consists exclusively of body fat gain (96%)

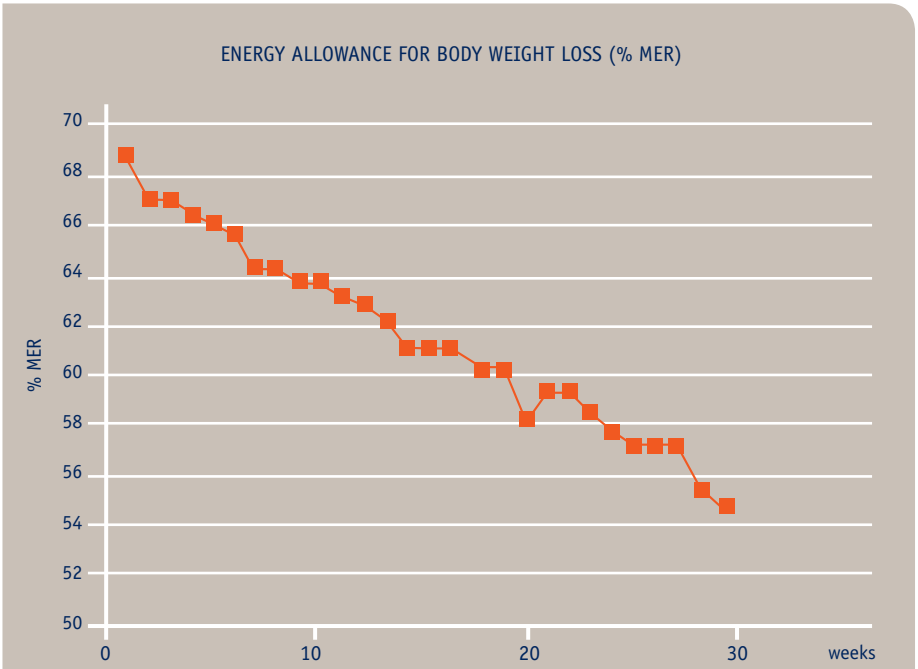



**Figure 5.** Energy allowance (in % MER with MER = 132 kcal/kg $^{0.75}$) for body weight reduction during a 6-months period: energy allowance has to be decreased with time to maintain body weight loss

## Body weight loss and energy intake

During a body weight loss programme, energy restriction should induce a weekly weight loss of 1-2% starting body weight. A body weight loss of more than 2% is not recommended because it favours a loss of muscular mass. It has been shown that dogs with a slow body weight loss (1.14%/week) more easily maintain their ideal body weight following the body weight loss programme (Laflamme and Kuhlman, 1995).

Depending on the protocol, applied energy restriction can vary greatly. Jeusette *et al* demonstrated recently that energy intake of a dog had to be reduced by 46% from its former state of obesity to obtain an optimal weight loss rate. However, initial energy intake is rarely known. Recent experimental research have obtained good weight loss results (1.14-1.46% BW loss/week) at 75 to 82% MER, MER being estimated to 132 kcal/ kg ideal BW $^{0.75}$. However, experimental data are difficult to extrapolate to clinical applications and in clinical trials reported bodyweight loss rate are generally lower (between 0.68 and 0.88% per week). In the last edition of the NRC, maintenance energy requirement of most pet dogs is estimated to be between 95 and 130 kcal/kg ideal BW $^{0.75}$. On average, a 40% restriction (60% of energy requirement) is a reasonable restriction level for weight loss management in clinical practice.

In a recent study, rate of bodyweight loss reached -1.2±0.4% with 66±6% MER during the first 3 months, but decreased for the next 3 months in the study. A significant decrease in energy allowance with time is necessary to maintain the BW loss rate (70±1% MER to induce, 66±8% MER (-9%) after 3 months, 56±7% MER (-20%) to finish) and even with this reduction of energy intake, BW loss rate decreased with time (Figure 5) (Rocksin *et al*, 2007).

A stricter recommendation at the beginning of the weight loss programme carries the risk of causing a higher body weight loss rate during the first months and with it a greater risk of weight rebound (Laflamme *et al*, 1995).

## Body weight maintenance: avoiding weight regain

Energy requirement is significantly lower after weight loss, compared to the obese phase. Jeusette *et al* (2004a, 2005b) quantified maintenance energy requirement after weight loss as 29% lower than before weight loss. This is probably linked to a modification of the body composition [inevitable loss of muscular mass] and metabolism [thyroid hormones, leptin, ghrelin…] (Laflamme and Kuhlman, 1995; Daminet *et al*, 2003; Jeusette *et al*, 2004a). Healthy dietary habits have to be put in place for the whole of a dog's lifetime to avoid weight regain.

# IMPORTANCE OF NUTRIENTS

## Protein

In humans, the conventional dietetic approach recommends a low-fat, high-carbohydrate, energy-deficient diet for weight reduction. However, interest in low-carbohydrate/ high-protein diets is increasing.

With a typical reduced energy food for overweight dogs and cats using low fat and high fibre, the additional swapping of protein for non-fibre carbohydrates should be neutral from a caloric point of view. However, increasing protein to the detriment of carbohydrate has numerous advantages.

- **Proteins improve body composition:** studies from 1988 and 1999 showed that a high protein (62% metabolizable energy) low carbohydrate (7% ME) diet for weight reduction in dogs led to better maintenance of lean body mass compared with a control diet (35% metabolisable energy from protein and 35% from non-fibre carbohydrate) taking the same time to achieve the target body weight (Laflamme and Hannah, 1988 and Hannah, 1999). This has been confirmed by more recent studies: compared to a high fibre normal protein diet (24% protein, 24% starch, 39% total dietary fibre, DM basis), a high protein low starch diet (48% protein, 5% starch, 31% total dietary fibre DM basis) induced slow body weight loss rate (<2%) and a better preservation of muscular mass (Diez *et al*, 2002). In another trial, a high protein very low-carbohydrate diet (45% ME protein, 5% ME carbohydrate) compared with a low fat high carbohydrate diet (36% ME from protein, 46% ME from carbohydrate) obtained the same weight reduction (2.18 ± 0.29% loss of BW/week) over 8 weeks with a higher daily energy intake (547 ± 6 kcal EM/day versus 495 ± 5 kcal EM/day) (Jeusette *et al*,2006b).

Muscle mass preservation is very important for long term weight management because energy expenditure depends on the muscle mass. A substantial loss of muscle mass causes a lower energy requirement and thus favours metabolic resistance to weight loss and a potential for weight rebound.

- **Proteins enhance energy expenditure:** protein has a lower net energy content compared to fat or carbohydrate because the efficiency of energy utilization of protein is lower and more heat is produced by the cells when they utilize protein.

This can explain why in dogs, a high protein diet allows higher energy intake (+10%) to achieve the same BW loss rate in experimental condition; and in clinical conditions, a trend for higher body weight loss rate has been observed for the same energy intake with a diet higher in protein (Rocksin *et al*, 2007).

- **Proteins improve palatability:** a high content of animal proteins could help to improve the palatability of low energy diets.
- **Proteins offer better satiety:** in dogs, satiety could be related to a threshold caloric load in the intestine. Proteins require the highest caloric load to induce satiety and a high protein content of the diet could be necessary to achieve sufficient satiety (Geoghean *et al*, 1997). High-protein high-fibre diets have been shown to increase satiety in dogs as evidenced by reduced voluntary food intake (Weber *et al*, 2007).
- **Proteins have no proof of being deleterious for the kidney:** there is a lack of epidemiological evidence of a link between high protein intake and chronic renal failure in both dogs and cats. It has been demonstrated that phosphorus is a key factor. A high protein diet in obese patients does not necessarily mean increased protein intake as the maintenance level of ingestion may effectively be retained due to caloric dilution.

A trial was conducted to compare, in clinical field conditions, 2 weight loss diets with different protein, starch and fibre content on weight-loss characteristics and blood parameters in obese dogs. Both diets resulted in healthy weight loss with few differences between diets. However, the high-protein moderate-starch diet induced slightly higher BW loss rate for the first 3 months, for a similar energy intake, and with lower blood triglycerides, insulin and fructosamine than the higher starch diet after 6 months. No side effects of the high-protein diet were observed (Rocksin *et al*, 2007).

## Fat

Fat level is generally reduced in obesity diets formulated for pets as the most efficient way to decrease the energy density and thus allow relatively higher food intake. Replacing fat by complex carbohydrate for example reduces gross energy content but also increases metabolic energy expenditure. Indeed, energy expenditure is 9-12% higher with carbohydrate than with fat. Fat is used more efficiently for oxidation to ATP (higher net energy) and for fat deposition in adipose tissue. A minimum of 5% fat is recommended for complete diets to cover the need for essential fatty acids and liposoluble vitamins. A source of essential fatty acids (linoleic and linolenic fatty acids) is necessary (vegetable and fish oil). A recent study has shown that when feeding a low carbohydrate rather than a low fat, the higher level of fat associated with the controlled energy intake allowed an adequate rate of body weight loss but with a more palatable food.

## v6/v3 fatty acids

As mentioned previously, obesity is characterised by a chronic, systemic, low grade inflammatory state and inflammation may be a potential mechanism whereby obesity leads to several pathologies.
In dogs, it has been shown that the v6/v3 fatty acid ratio (v6/v3) modulates inflammatory reactions. A ratio of between 5 and 10 reduces the production of inflammatory mediators without side effects, and could therefore be beneficial for obese dogs. However, besides the ratio of v6/v3, a minimum quantity of long chain v3 fatty acids (DHA-EPA) is necessary to achieve health effects from their supplementation (Hall *et al*, 2006).
Canine diets that aim to provide this recommended ratio and minimal quantity of long chain v3 fatty acids need to be supplemented with a natural source of DHA (from fatty fish or marine algae) and EPA (from fatty fish or filamentous fungi).

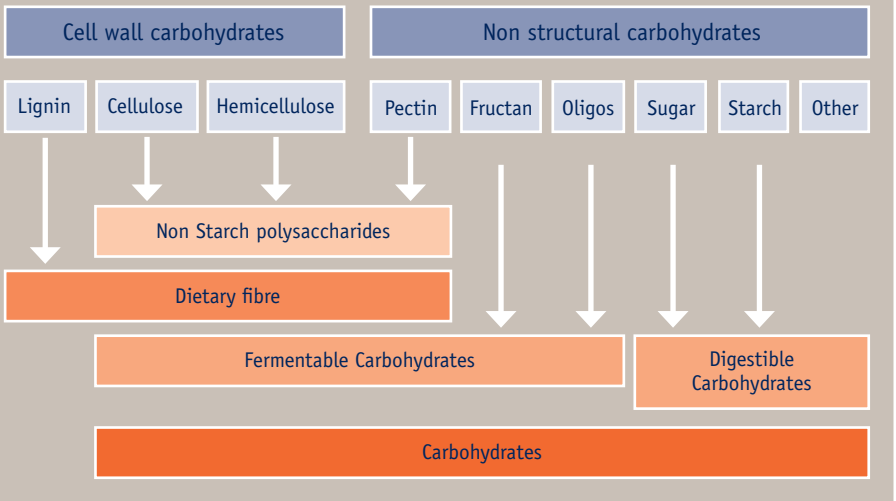
## Medium chain fatty acids (MCTs)

Coconut oil is the main natural source of medium chain fatty acids: C8 Caprylic acid (7 %); C10 Capric acid (6 %); C12 Lauric acid acid (45 %) and C14 myristic acid (16 %). From the literature reviewed, it appears that MCTs increase energy expenditure, result in faster satiety and facilitate weight control when included in the diet as a replacement for fats containing long chain triglycerides.
Animal and human studies have shown that MCTs are readily oxidized in the liver, leading to greater energy expenditure. Most animal studies have also shown less body weight gain and decreased size of fat depots after several months of consumption. Furthermore, both animal and human trials suggest a greater satiety effect of medium-chain triglycerides compared with long-chain triglycerides.

## Digestible Carbohydrates

In dry pet foods, a minimum amount of non-fibre digestible carbohydrates (figure 6) in the form of starch is necessary for their extrusion. Obese dogs could have impaired glucose control and have difficulties in blood glucose clearance, with blood glucose levels remaining higher for a longer period of time than lean dogs. For this reason starch sources that elicit a low postprandial glycaemic response would be beneficial for obese dogs with impaired glucose control. The amylose content and starch particle size varies between cereals, changing their degree and rate of intestinal breakdown. In dogs, barley results in lower blood insulin levels from 20 to 240 minutes after a meal and a lower insulin area under the curve than other more commons cereals uses in dog food.

**Figure 6. CARBOHYDRATES (from Boisen and Verstegen, 2000)**



### Fibres

The definition of fibre relates mainly to the matrix that composes vegetable cell walls, but different type of biochemical substances are included in this term, as indicated in Figure 6, which indicates the classification of carbohydrates.
There is contradictory evidence from various reported studies about the effects of moderate to high fibre diets on food intake.

**Fibre-rich foods–potential benefits:**

- Fibres dilute the diet's energy concentration
- Depending on the diet structure, some soluble fibers can delay gastric emptying and induce a slower absorption of nutrients
- Insoluble fibres are a component of faeces, inducing a more voluminous dietary bolus and accelerating the bowel transit
- Fibres can reduce postprandial glycaemia in diabetic dogs
- Fibres can help to reduce concentration of blood lipids
- Satiety

**Fibre-rich foods–potential disadvantages:**

- Decrease in palatability
- Reduction in nutrient digestibility
- Increased faecal frequency, quantity of faeces, water content of faeces and diarrhoea.
- Fibres can reduce bioavailability of some micronutrients.
- Highly fermentable fibres may induce flatulence in dogs.

### L-Carnitine

L-carnitine is a conditionally essential, vitamin-like nutrient that helps fatty acid oxidation in the mitochondria. Gross *et al* (1998) and Allen *et al* (1999) showed that a diet supplemented with 0.03 % carnitine improved lean body mass with a trend toward greater body weight loss of dogs in a weight reduction programme.
Another study with neutered female dogs has confirmed these results: dogs receiving a weight loss diet, supplemented with 0.005% or 0.01% carnitine had a greater weight and fat loss and a higher fat free mass (Sunvold *et al*, 1999).

### Antioxidants

Oxidative stress may be the unifying mechanism underlying the development of co-morbidities in obesity. In humans, evidence suggests that a clustering of oxidative stress sources exists in obesity: hyperglycaemia, hyperleptinaemia, increased tissue lipid levels, inadequate antioxidant defenses, increased rates of free radical formation, enzymatic sources within the endothelium, and chronic inflammation (Vincent and Taylor, 2006).

In dogs, it has previously been shown that oral administration of green tea polyphenols (EGCG), grape seed or citrus polyphenols (naringin) can significantly decrease dog blood oxidation biomarkers, can alter expression of genes involved in inflammation and insulin resistance and can improve lipid profile (Torre *et al*, 2006a and 2006b; Salas, 2009; Serisier *et al*, 2008).

## EXERCISE

Lifestyle modification is very important for the wellbeing of dogs during obesity treatment, as has been shown by Jeusette *et al*, 2006c, who demonstrated that dogs fed a low energy diet *ad libitum* lost weight with increased physical exercise (Figure 7).
Exercise without weight loss has also been shown to be healthy for obese dogs: heart rate recovery (average heart rate during recovery as a percentage of heart rate during exercise) of overweight dogs was faster when exercised every day (Kuruvilla and Frankel, 2003). Owners should be encouraged to increase the level of any form of exercise for their dog as part of a weight loss programme, alongside the advice on feeding regime.

## CONCLUSION

Obesity is a common disease of companion dogs. Due to the significant health consequences of obesity, and because obesity can decrease life span, prevention and treatment are important priorities, which should be approached through a modification of lifestyle and diet.
A veterinary follow-up that is individually adapted to each dog is a key to the success of the treatment due to the variation of individual energy requirements, latent metabolic resistance to weight loss and a potential for weight rebound following weight loss. In the end, owner compliance is the key factor for prevention and treatment of canine obesity. Therefore, the veterinary practitioner has a crucial role firstly to motivate and then to assist owners about the individual weight loss programme that is proposed along with their responsibility to the wellbeing of their pets followed by continuing support to the owner to help with their compliance to the programme and a resulting satisfactory long-term outcome for their dog.


**Figure 7**. Body weight evolution with and without physical activity

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