A review of osteoarthritis and obesity: current understanding of the relationship and benefit of obesity treatment and prevention in the dog

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Keywords

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Summary

Obesity is an increasingly important health problem for both man and dog. Osteoarthritis (OA) is a significant cause of pain and disability in both species. A link between obesity and OA has been established in man, though the exact mechanism of the relationship remains to be fully elucidated – current research supports both biomechanical and biochemical theories. There is good evidence (class I*) to support weight loss as an effective treatment for human knee OA. In the dog, the relationship is just beginning to be investigated. The results of one study in dogs (class IV evidence*) suggest that preventing the development of overweightness and obesity reduces the prevalence of hip dysplasia and OA of the hip and other joints. Three other studies (class III and IV evidence*) support weight loss as an effective treatment for OA in affected overweight and obese dogs. Further research could yield greater understanding of the pathophysiology of this relationship, perhaps identifying novel therapeutic targets. Confirmation and better understanding of the positive effect of treating and preventing obesity on symptoms and prevalence of OA is likely to be valuable in the campaign against canine obesity.

* Classes of evidence detailed in Table 1.

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Introduction

The World Health Organisation has declared obesity to be the most important health problem currently facing the Western world (1). In humans, body mass index (BMI) is calculated using the formula; body mass (kg) / height $(m)^2$, and obesity and overweightness are defined as a BMI = 30 and = 25 respectively (2) . In the United States in 2004, 33% of adults were considered obese and 17% of teenagers overweight (2). A study published in 1986 estimated the prevalence of canine obesity to be 24% and a similar study published in 2005 gave a figure of 41% (3, 4). It is widely suspected among veterinarians that the prevalence of obesity among populations of domestic dogs is increasing. Currently obesity and overweightness in the dog are arbitrarily defined as relative bodyweight = 120% and = 110% respectively (5). Relative bodyweight is calculated by dividing actual weight by an estimated ideal body weight and multiplying by 100%. Osteoarthritis (OA) is the most common cause of pain and physical disability in man, and is likely to be of similar significance in the dog, with an estimated 20% of adult dogs affected (6, 7). Obesity has consistently been identified as a risk factor for the development of OA in man, with the strongest evidence supporting a relationship with knee OA. It is now recognised that reducing the prevalence of OA and its associated burden on health services requires a commitment to tackling the obesity pandemic (8).

Aim of review

The purpose of this review is to summarise current theories surrounding the relationship between obesity and OA in man, and to systematically review the literature pertaining to a similar relationship in the dog. Two questions are raised in the systematic review: first – does prevention of canine overweightness and obesity reduce OA prevalence, and second – will weight loss alleviate clinical signs of pain and disability in overweight and obese dogs with OA?

Obesity and osteoarthritis in man

Obesity has consistently been identified as a risk factor for development of knee and hand OA, and for the progression of knee OA (9–11). The relationship between obesity and hip OA is not so convincing: a meta-analysis performed in 2002 found only moderate evidence to support it, and two recent studies did

not find any association (9, 10, 12). The mechanisms by which obesity can affect the development and progression of OA (in those joints where a relationship has been established) is the subject of a significant volume of current research in human rheumatology.

Obesity, joint biomechanics and osteoarthritis

Joint loading is essential for the maintenance of healthy cartilage, but a reduction or an increase in loading outside normal physiologic levels, or a change in the direction of joint forces (e.g. with joint instability) can be detrimental (13). Current understanding of knee OA in man suggests that initiation of the disease requires not only an increase in load, but also that joint kinematics are altered so that weight-bearing is shifted to areas of cartilage incapable of sustaining such loads (13). Joint kinematics may be altered by some primary abnormality of congruity or stability, or by a load-bearing shift secondary to excess bodyweight itself. By altering joint kinematics and increasing ambulatory load, obesity may have roles both in the initiation and progression of OA (14).

Illustrating the importance of joint kinematics are two studies that examined the influence of knee alignment. The first study demonstrated that there was a significant relationship between BMI and radiographic severity of OA in human knees with a varus, but not valgus alignment (15). The authors suggested that the increased axial load on articular cartilage is concentrated across the medial joint compartment by varus malalignment, and that such malalignment, combined with a long moment arm with respect to the centre of gravity at the knee, may explain the strong link between obesity and knee OA (15). The second study found that the progression of knee OA was positively associated with increasing BMI, but only where moderate malalignment (valgus *or* varus) was present (16). Again the theory is that malalignment acts to concentrate the increased joint forces caused by obesity thus precipitating cartilage damage.

The biomechanical aspect of any relationship between obesity and OA is far from being completely understood. However, there is considerable evidence to suggest that the link between knee OA and obesity is, at least in part, a biomechanical one and that malalignment may be an important mediating factor (15, 16).

Obesity, adipokines and osteoarthritis

Obese people are at increased risk for developing OA of certain joints of the hand. As these joints are non weight-bearing, this suggests a metabolic rather than mechanical association between obesity and OA (11). Adipose tissue can no longer be considered a simple energy reserve – it has many functions beyond the storage of triglyceride and release of fatty acids. The adipocyte is capable of synthesising and releasing a variety of molecules with immunological or endocrine function including the 'adipokines' leptin and adiponectin. Indeed, it has been proposed that adipose tissue should be thought of as an organ in its own right (17).

Leptin is a peptide hormone, produced by the adipocyte, which has received much attention in attempts to understand the relationship between obesity and OA. It plays a major role in regulating appetite through activation of hypothalamic receptors, but also participates in various other biological processes – inflammation and immune function in particular (18). There is a growing body of evidence to suggest that leptin has a detrimental effect on articular cartilage, and a role in the pathogenesis of OA (19). A key paper on leptin was published recently by Simopoulou and colleagues who hypothesised that OA is a metabolic disease caused by systemic and local factors including altered lipid metabolism (20). They demonstrated that in people with OA of the knee or hip, leptin levels were significantly elevated in synovial fluid compared to serum and that chondrocytes cultured from healthy or arthritic cartilage could express leptin mRNA and protein. Expression of leptin and Ob-Rb (leptin receptor) mRNA was significantly increased in chondrocytes cultured from cartilage affected by OA compared to normal tissue, and in advanced OA compared to minimally affected cartilage. In severely affected cartilage, leptin mRNA expression was significantly increased in obese compared to normal weight patients, suggesting a local as well as systemic hyperleptinaemia in these individuals. Leptin has an inhibitory effect on the long-term growth of cultured chondrocytes, and induces the production of interleukin-1 (IL-1), matrix metalloproteinase-13 (MMP-13) and MMP-9 in a dose-dependent manner, which signifies a catabolic effect on chondrocyte metabolism (20).

The biochemical relationship between adipose tissue and arthritis is unlikely to be mediated by leptin alone. Adiponectin is another cytokine synthesised and released by fat, and by other tissues. It is present within the systemic circulation and is also produced by local joint tissues such as the adipocytes of the infrapatellar fat pad and synovial fibroblasts (17). Treatment of synovial fibroblasts from joints affected by OA with adiponectin *in vitro* induces production of pro-MMP-1 and IL-6 which play key roles in cartilage destruction (17).

It was recently demonstrated that human and murine chondrocytes express functional adiponectin receptors, and that treatment of these cells with adiponectin induces expression of nitric oxide synthase type II (NOS2), IL-6, MMP-3, MMP-9 and monocyte chemoattractant protein-1 (MCP-1). Nitric oxide (NO) (produced by NOS2) controls cartilage functions including loss of chondrocyte phenotype, chondrocyte apoptosis and extracellular matrix degradation. Interleukin-6, MMP-3, MMP-9 and MCP-1 are all mediators of cartilage degeneration (21). An understanding of the role of adipokines in OA pathophysiology is important as they may represent target molecules for novel therapeutic compounds.

Other obesity related diseases and osteoarthritis

Cardiovascular disease appears to have an association with both obesity and OA. It has been proposed that atherosclerosis of subchondral bone microvasculature and resulting bone ischaemia may contribute to the progression of OA (22). This raises the question of whether treatment of hypercholesterolaemia could help to slow the progression of the disease (22).

An association between diabetes and hand OA has been demonstrated and one theory to explain this association focuses on advanced

glycation end products (AGE), which form within many tissues as part of the ageing process (23). The deposition of these products is accelerated by diabetes mellitus, including obesity-induced type 2 diabetes (23). The AGE accumulate in articular cartilage and may have several detrimental effects on that tissue; cross-linking of AGE causes increased stiffness of collagen and compromises the mechanical properties of cartilage (23). Chondrocytes express a receptor for AGE, stimulation of which results in activation of inflammatory pathways and MMP-13 production (24). The AGE increase matrix degradation and decrease proteoglycan synthesis (25). If AGE play a role in OA pathogenesis, then prevention and treatment of type 2 diabetes could indirectly reduce OA prevalence. In addition, administration of compounds such as pyridoxamine that inhibit AGE formation could represent a novel therapy for OA (23).

An understanding of the complex relationship between obesity and OA is slowly evolving. It is conceivable that from any one of the four potential contributing factors discussed here (biomechanics, adipokines, vascular disease, and diabetes), novel therapies for treating OA might be developed that would actually modify the disease process. Some of these treatments could be of benefit to canine as well as human patients, but only if we further develop our understanding of this relationship in the dog.

Benefit of weight loss in human osteoarthritis

Although the pathogenesis and relationship of obesity and OA may require further elucidation, and may yield valuable pharmacologic therapies in the future, the evidence to support weight loss itself as a therapeutic intervention for obese people with OA is difficult to dispute – at least in the case of knee OA. A recent meta-analysis showed that a 5.1% reduction in bodyweight within a 20 week period could significantly reduce self-reported disability in obese patients with OA of the knee (26). Similar studies examining the effect of weight loss on symptomatic hand OA do not yet exist within the literature, to the authors' knowledge.

Table 1 Levels of evidence. Modified from Aragon and Budsberg (2005) and reproduced from Innes (2007) (27, 28).

Evidence class	Study design	Examples, comments
I	Evidence derived from multiple, randomised, blinded, and placebo-controlled trials in the target species.	**Systematic reviews** (e.g. meta-analyses). Advantages of meta-analyses include: • objective appraisal, • large number of subjects, • improved estimates of association, • assimilation of large quantities of information, • findings developed on a common scale, and • improved quality of primary research.
II	Evidence derived from high quality clinical trials using historical controls.	**Randomised-controlled clinical studies.** Studies that are done on animals that developed the disease naturally and are performed in the laboratory setting. Historical controls are thought to be less reliable than randomised controls.
III	Evidence derived from uncontrolled case series.	**Non-randomised, prospective case comparison studies**. Examples include prospective case series that include subjective clinical impressions to objective gait analysis.
IV	Evidence derived from expert opinion, or are extrapolated from research or physiological studies.	**Retrospective case comparison studies**. Studies on research subjects (non-client owned) are also included in this class.

Obesity and osteoarthritis in dogs

Investigative method

By searching the Medline database from 1950 to 2008 via PubMed using the following search terms; 'dog and obesity and osteoarthritis', 'dog and weight loss and osteoarthritis' and 'dog and dietary restriction', papers or abstracts were identified that described either the effect of becoming overweight or obese on the development and progression of OA, or the effect of weight loss on clinical signs of OA in overweight or obese dogs. Any suitable publications known to the authors but not revealed by the database search were also included. The identified studies were classified based on their quality according to a scheme proposed for examination of evidence in veterinary orthopaedic surgery (27, 28) (▶Table 1). Eleven prospective studies were identified that described treatment or prevention of overweightness and obesity in dogs and the associated effect on OA (▶Table 2).

Effect of preventing overweightness and obesity on the development of osteoarthritis in the dog

The papers reviewed in this section (29–36) are the result of an experimental study that examined the effect of food restriction in Labrador Retrievers that were genotypically predisposed to hip dysplasia. Forty-eight dogs from seven litters of experimental animals were paired by sex and bodyweight and randomly divided into 'control-fed' and 'limit-fed' groups of twenty-four. The control group was initially fed *ad libitum*, and then at around three-years-old their intake was reduced to 62.1 kcal of metabolisable energy per kilogram of ideal bodyweight per day. The limit-fed group was given 75% of the food consumed by the control group. The magnitude and rate of bodyweight gain were significantly less in the limit group over the first two years of life. Mean body-condition scores at 12-years-old were 4.6 and 6.7 out of nine for limit-fed and control dogs respectively. The

Table 2 Studies that have examined the effect of treatment or prevention of overweightness and obesity on osteoarthritis in dogs.

Reference number	Method: treatment or prevention?	Co-morbidity examined	Evidence class
29	Prevention	Hip dysplasia	IV
30	Prevention	Radiographical signs of hip osteoarthritis	IV
31	Prevention	Radiographical signs of osteoarthritis in multiple joints	IV
32	Prevention	Radiographical signs of hip osteoarthritis	IV
33*	Prevention	Radiographical and pathological evidence of elbow osteoarthritis	IV
34	Prevention	Radiographical and pathological evidence of shoulder osteoarthritis	IV
35	Prevention	Various diseases including osteoarthritis; lifespan	IV
36	Prevention	Lifespan and causes of death	IV
37*	Treatment	Clinical signs of hip osteoarthritis (objective outcome measure)	IV
38	Treatment	Clinical signs of hip osteoarthritis (subjective outcome measure)	III
39	Treatment	Clinical signs of osteoarthritis (various joints, subjective and objective outcome measures)	III

* Abstract in proceedings.

dogs were examined at intervals throughout their lifetime for evidence of disease. Because experimental animals were used, this study can be considered class IV evidence, but the use of client-owned animals would have been extremely difficult and may have precluded the use of a randomised, controlled design. On average, the control dogs became overweight and the limit-fed dogs maintained a body condition score very close to normal; this study compared the overfed (control-fed) and optimally-fed (limit-fed) dogs.

Radiographic examination of the hip joints was performed when the dogs were 30, 42, 54, 78 and 104-weeks-old. Measurement of the Norberg angle on a standard (hips extended) radiograph of the pelvis at 30-weeks-old revealed significantly less dysplasia among the limit-fed dogs. Norberg angles were measured again when the dogs were two-years-old; hip dysplasia scoring was also performed at this time using both the Orthopedic Foundation for Animals and Swedish Kennel Club systems. At two-years–old, 16 of the 24 control dogs and seven of the 24 limit-fed dogs showed radiographic evidence of hip dysplasia. The reason why limiting food intake reduced the incidence of hip dysplasia is unknown, and may not be the result of decreased bodyweight alone, but could also be a consequence of decreased growth rate. Without a complete understanding of the mechanism, it can still be said that dietary restriction appears to be an environmental change that can improve the phenotype of animals genotypically predisposed to hip dysplasia. The authors of this study recognised potential concerns, in that this approach may perpetuate hip dysplasia in the canine population; however they felt that efforts to reduce the incidence of dysplasia were justifiable and required. This study highlights the need for a canine hip dysplasia test that is not influenced by environmental factors (29).

Pelvic radiographs were repeated when the dogs were three- and five-years-old. These and all previous images were examined for evidence of OA. At the age of one year, there was a significant difference in the frequency and severity of coxofemoral OA between the two groups of dogs: sclerosis of the craniodorsal portion of the acetabulum was present in seven of the control-fed and none of the limit-fed dogs. At two-years-old, 10 of the 24 control dogs showed radiographic evidence of hip OA compared with one of the 24 limit-fed dogs. By the age of three-years, 12 of the 23 control dogs showed OA versus three of the 23 limit-fed dogs (1 dog from each group had died by this time). The number of dogs affected by hip OA in each group did not change between the ages of three and five, though the radiographic severity did increase in both groups. Bodyweight among the limit-fed group was 25% less than the control-fed group, and it was significantly correlated with the severity of OA. On the basis of their results, the authors recommend that dogs be maintained in 'slender' body condition throughout their period of growth and adult lives to reduce the incidence of hip OA (30). At eight-years-old, 15 of the 22 control-fed and three of the 21 limit-fed dogs had radiographic hip OA, with greater severity in the control group (both of these findings were statistically significant). Bilateral hip OA was more common than unilateral in a ratio of 2:1 (31).

In 2006, another paper was published on the development of hip OA in the same dogs (23). Confusingly, prevalence of hip OA seemed to have decreased among the control dogs from when these results were first reported. This discrepancy is probably because only one investigator interpreted the radiographs, whereas the median OA score of three investigators' interpretation had previously been reported . When the dogs died (end of life – EOL) 20 of the 24 control-fed and 12 of the 24 limit-fed dogs had radiographic hip OA. The hip joints were not examined at postmortem. ▶Table 3 illustrates the development of hip OA in the two groups of dogs during their lifetime.

Radiographic examination of the elbow, stifle and shoulder joints was performed when the dogs were eight-years-old; the prevalence of OA affecting multiple joints was significantly greater in the control group. Ten of the 22 control dogs had OA in two different joints versus one of the 21 limit-fed dogs. Eight of the 22 control dogs and four of the 21 limit-fed dogs had elbow OA; this difference was not significant, however the radiographic OA severity was greater in the control group. At EOL, elbow OA was more prevalent when assessed radiographically,

however there was no difference in prevalence when the joints were examined postmortem. An explanation for the difference in prevalence for radiographic and postmortem OA of the elbow is not given, however it could be due to over interpretation of the radiographs. Histopathologic severity of elbow OA was greater in the limit-fed dogs at EOL (33). Two of the control dogs had radiographic stifle OA at eight-years-old; none of the limit-fed dogs did (31).

The most recently published paper by this group focuses on shoulder OA (34). Radiographic examination of the shoulder was performed at six- and eight-years-old . Gross and histopathological evaluations of the various components of the shoulder joint were performed at EOL. The radiographic and pathological prevalence of shoulder OA in both groups is illustrated in ▶Table 4.

Similar to elbow OA, severity but not prevalence of shoulder OA was lower at the ages of six- and eight-years in the limit-fed dogs. There was not any difference in prevalence or severity at EOL; though it should be noted that median lifespan among the limit-fed dogs was 1.8 years longer. Ninety-one percent of all dogs had histopathological evidence of shoulder OA at EOL; this high overall prevalence of OA is striking. Radiographic and pathological evidence of shoulder OA were poorly correlated.

Overall, diet restriction reduced the prevalence and severity of OA, and had by far its most significant effect on OA of the hip joint. This was almost certainly a consequence of a reduced prevalence of phenotypic hip dysplasia in the limit-fed dogs. By comparison, prevalence of shoulder OA was high in both groups of dogs, but it was not significantly different between them. Lesions consistent with osteochondrosis were not found at postmortem, and for this reason the authors propose that the shoulder OA in this population of dogs was primary in nature (34). If this is the case then it can be suggested that the effect of diet restriction on development of primary OA (OA with no apparent predisposing factor) is not as significant as its effect on hip joint laxity and subsequent (secondary) OA.

Osteoarthritis was the most common chronic disease to develop in both groups. The control dogs required institution of long-term treatment for OA on average three years earlier then the limit-fed dogs. This is the first indication of the clinical significance of OA diagnosed radiographically – it seems that the control dogs displayed clinical signs earlier as well, however lameness was not evaluated either subjectively or objectively. It is interesting to note that debilitating OA was a leading cause of death (euthanasia) in both groups. Seven limit-fed versus 11 control dogs were euthanatized because of OA at mean ages of 11.5 and 13.1 years respectively. In general, causes of death between the two groups of dogs were similar; it was the time of death that differed; with limit-fed dogs living (on average) 1.8 years longer (35, 36).

In summary, from the series of publications resulting from this long-term study (29–36), it seems that maintaining Labradors at a body condition score of around five out of nine for life may:

- reduce the incidence of hip dysplasia,
- reduce the incidence or severity of OA depending on the joint in question,
- delay the need for treatment of OA (and of other chronic diseases),
- delay the need for euthanasia due to chronic disease (OA was a leading cause of euthanasia),
- delay natural death due to disease other than OA.

Table 3
Prevalence of radiographic hip osteoarthritis at selected ages and at end-of-life in limit-fed and control dogs.

Dog group	Limit-fed (%)		Control-Fed (%)	
Reference number:	30, 31*	32**	30, 31*	32**
1-year-old	0	--	29	--
2-years-old	4	4	42	25
5-years-old	13	13	52	39
8-years-old	14*	14	68*	64
End-of-life	--	50	--	83

* Prevalence of hip osteoarthritis at eight-years-old in this column is derived from reference 31.
** Reference 32 was published after reference 30 and 31 and gives different values for prevalence of hip OA among the control population, possible due to differences in the method of radiographic interpretation.

Table 4
Radiographic and pathological prevalence of shoulder osteoarthritis in limit-fed and control groups of Labradors.

Age (years)	Examination method	Limit-fed (%)	Control-fed (%)
6	Radiographical	43	68
8	Radiographical	62	81
End-of-life	Radiographical	83	74
	Gross pathology	83	91

Benefit of weight loss in canine osteoarthritis

Sixteen overweight and obese dogs with osteoarthritis (OA) of the hips showed improved hindlimb function with a reduction of body condition score: original scores of *six to eight* improved to *four to five* based on a scale of *nine* (37). The improvement was demonstrated by comparison of kinetic gait analyses at the beginning and end of weight loss: peak ground reaction force increased and time of the stride propulsive phase decreased. Increased peak vertical force (PFz) was observed in both fore and hindlimbs and ranged from 0.24 to 0.98 N/kg, with the greatest increase being observed in the weakest hindlimb. The decrease in time of the propulsive phase of the stride ranged from 10.9 to 14.2 ms. The increase in PFz seen in the forelimbs was interesting, but the greatest increase of 0.52N/kg seen in the strongest forelimb was very modest (equivalent to 5% bodyweight).

Even though it was statistically significant, it may not be biologically significant. The decrease in time of the propulsive phase of the stride suggests that the dog's gait had changed with an increased limb velocity, which may suggest improved joint comfort.

This clinical trial demonstrated an objective improvement in lameness with weight loss. It is difficult however to visualise the significance of the reported changes in gait parameters without an accompanying description of clinical signs, subjective gait assessment, or the owners' perception of their dogs' level of disability. Also, the data remains unpublished and can therefore only be considered, at best, class IV evidence.

Subjective outcome measures were utilised by a different clinical trial that also examined the effect of weight reduction on lameness caused by OA, and represents class III evidence (38). Nine dogs that were 11–12% greater than their estimated ideal bodyweight and had clinical and radiographic signs of hip OA completed the study. A 40% reduction in caloric intake resulted in weight loss of between 11 and 18% over a period of 10 to 19 weeks. By the midpoint of the weight loss period, mean bodyweight had decreased significantly from 39.0 to 36.6 kg (a 6.2% decrease). This was accompanied by a significant decrease in body condition score and in subjective lameness score using numerical rating and visual analogue scales. The major limitation of this study was the opposite to that of the previously mentioned work; there was no objective confirmation of the observed improvement in lameness.

The final study identified in this area was a prospective clinical trial that did combine subjective and objective outcome measures, and also represents class III evidence (39). Twenty-nine dogs with a body condition score of four to five out of five were enrolled. Selection criteria dictated that lameness was observed in one limb only and that OA was present in that limb. Dogs with OA of the hip, elbow, stifle and shoulder were included. The weight loss program was designed to produce a one percent reduction in bodyweight per week. Bodyweight, subjective lameness and pain scores were evaluated monthly. Kinetic gait analysis was performed bimonthly using four force-plates mounted in a treadmill. Asymmetrical weight distribution between limbs affected and unaffected by OA was demonstrated using symmetry indices, calculated for PFz and vertical impulse (IFz) by dividing greatest by least values for contralateral limbs (e.g. sound limb divided by lame limb). Weight loss was combined with physiotherapy: dogs were randomly allocated to an intense (group 1) or moderate (group 2) physiotherapy program. Owners of dogs in both groups were instructed to perform massage, passive range of motion and to gradually increase levels of controlled exercise. Group 1 dogs were additionally treated in a physiotherapy clinic twice weekly; this treatment included application of transcutaneous electrical nerve stimulation.

A difference in weight loss was first detected at day 90, and after six months group 1 had lost more weight than group 2 (13.6% of initial body weight versus 9.3%). Lameness scores decreased significantly, starting at day 30 in group 1 and at day 60 for group 2. Pain scores also decreased, again the difference reached significance sooner in group 1 (at 60 versus 90 days). The difference in lameness scores between groups 1 and 2 was significant only at days 30 and 180. There was not any difference in pain scores between the two groups. Symmetry indices for PFz and IFz in group 1 were significantly improved (i.e. closer to 1) at each re-evaluation. In group 2, only the symmetry index for PFz improved, and only at day 120. There was no difference in weight loss between the two groups at day 60, however significant improvement in PFz and IFz symmetry indices was observed for group 1. This suggests that at day 60, the objective improvement in lameness shown in group 1 may have been the result of intensive physiotherapy rather than weight loss. A comparison of indices between groups indicated a greater degree of symmetry in group 1, but only for PFz. Overall, the dogs in group 1 appeared to show a more substantial improvement in lameness than those in group 2. It is difficult to interpret this result because two treatment variables were examined simultaneously – degree of weight loss and intensity of physiotherapy – and it was not possible to separate their effects. It can be said that a combination of the two treatments would effectively reduce disability in dogs with OA.

Conclusion

Current research suggests that in man, obesity is a risk factor for the development of hand OA, and both the development and progression of knee OA (9–11). There is good evidence to support weight loss as an effective treatment for knee OA (26). The exact mechanism of the relationship between obesity and OA is not completely understood, but likely involves both biomechanical and biochemical factors (15–25).

At present, based on four studies that represent class III and IV evidence, we have a limited comprehension of the links between growth, developmental orthopaedic disease, OA and obesity in the dog. The work of Kealy and colleagues suggests that restricting energy consumption during growth may reduce the incidence of hip dysplasia; however a recent experimental study did not confirm this relationship (29, 40). It is accepted that hip OA in the dog is initiated by abnormal joint kinematics, secondary to underlying laxity, and that OA in other joints usually occurs secondary to some initiating process (e.g. osteochondritis dissecans, cranial cruciate ligament rupture) (41). Obesity may be an important factor in driving the progression of OA by increasing the load factor of such abnormal joints; we do not know to what extent obesity itself may alter canine joint biomechanics. It has been shown that obesity causes a systemic hyperleptinaemia in the dog, but leptin's potential importance in the pathogenesis of canine OA is at present unknown (42). In order to devise improved treatment and prevention strategies, a more complete understanding should be sought, but at the same time we must consider the existing evidence, and make suggestions based on it.

It is generally acknowledged that the prevalence of overweightness and obesity in domestic dogs is increasing. It is the authors impression that the problem of canine obesity is not effectively dealt with by the majority of veterinarians. The reasons for this probably include ignorance about the importance of obesity, disinterest in its treatment, and a reluctance to confront clients on the issue. In addition, even those veterinarians that are pro-active in treating and preventing obesity will meet with resistance and non-compliance from many pet owners. On a positive note, there has been a recent move within the

veterinary profession to highlight the scale and significance of obesity in companion animals (43). Evidence that demonstrates, to both veterinarians and their clients, the health benefits of obesity treatment and prevention must surely be fundamental to any such campaign. This review shows, when OA as a disease is associated with obesity, that we have a limited but growing body of supportive evidence for two recommendations; maintaining dogs at a normal body condition score throughout their lives can be recommended to optimise synovial joint health, and bodyweight reduction will alleviate clinical signs of OA in obese and overweight dogs.

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