Canine Hip Dysplasia: Reviewing the Evidence for Nonsurgical Management

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Objective: To systematically review the evidence available for nonsurgical management of hip dysplasia (HD).

Study design: Literature review.

Methods: Databases (Pubmed, Veterinary Information Network) were searched for clinical studies on nonsurgical management of HD in dogs. The evidence in each study was reviewed and assigned a score (I–IV) based on previously reported levels of evidence.

Results: Fourteen articles were identified that met the inclusion criteria, including 3 Level IV, 4 Level III, and 7 Level II studies. Methods of nonsurgical management reviewed included: activity restrictions, weight management, acupuncture, modulation of joint disease by polysulfated glycosaminoglycans, mesenchymal stem cell therapy, and extra corporeal shock wave therapy.

Conclusion: Weight management is an effective and important component of managing dogs with HD and associated osteoarthritis. Techniques that modulate the progression of joint disease may also be beneficial for treating dogs with HD. Further studies are needed to investigate other methods of managing HD such as hydrotherapy and physical rehabilitation.

Hip dysplasia (HD), a heritable condition that results in laxity of the coxofemoral joint, is one of the most common orthopedic diseases affecting dogs.[1] In skeletally immature dogs, coxofemoral subluxation, joint capsule stretching, cartilage erosion, and subchondral bone fracture often produce pain and lameness.[1] Disease progression can lead to degenerative and inflammatory changes within the joint and development of osteoarthritis (OA) in mature dogs.[1]

Treatment of HD can be broadly categorized into surgical and nonsurgical management. Surgical options include juvenile pubic symphysiodesis (JPS), triple pelvic osteotomy (TPO), femoral head and neck ostectomy (FHO), and total hip replacement (THR). Nonsurgical management typically involves a multimodal approach including activity modification, weight reduction through dietary control, pain management, and pharmacologic modulation of joint disease. The decision to pursue surgical treatment in an individual dog with HD is typically based on owner expectations of the dog's intended function, the dog's physical condition, the owner's financial resources, the surgeon's experience, and the response to nonsurgical treatment. Clinicians have a responsibility to present available options and assist the client in determining which method of treatment is best suited for the well-being of their dog.

An invaluable tool in this decision-making process is the evaluation of scientific data that supports or refutes the treatment options being considered. This is the foundation of evidence-based medicine that has been defined as "the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients."[2] However, clinicians and pet owners must recognize that the validity and importance of individual studies are highly variable; therefore, strength of evidence should be reviewed critically.

Ranking systems have been developed by several organizations (eg, United States Preventative Services Task Force, the American Academy of Neurology, and the Centre for Evidence-Based Medicine) to aid in the evaluation of data and determination of the quality of evidence.[3] Aragon and Budsberg proposed Levels of Evidence to be used for veterinary orthopedic surgery (Table 1).[3,4] Development of this scale, along with the creation of the Veterinary Evidence Based Medicine Association, is a starting point for veterinarians to incorporate evidence-based medicine into their practice.

Our purpose is to provide a systematic review of the available evidence for nonsurgical methods of treating HD and associated OA in dogs using Evidence Classes.[4] Our intent is to aid veterinarians in determining appropriate therapeutic options when managing dogs with HD and OA.

Table 1 Level of Evidence Proposed by Aragon and Budsberg[4] for Evaluation of Orthopedic Surgery

Evidence Class	Study Design	Examples/Comments
I	Evidence derived from multiple, randomized, blinded, placebo-controlled trials in the target species	Systematic reviews (ie, metaanalyses)
II	Evidence derived from high quality clinical trials using historical controls	Randomized-controlled clinical studies on animals that developed the disease naturally and is performed in a laboratory setting
III	Evidence derived from uncontrolled case series	Nonrandomized, prospective case comparison studies; prospective comparison studies that are not truly randomized with limited subjective influence, prospective case series that include subjective clinical impressions to objective gait analysis
IV	Evidence derived from expert opinion, and/or extrapolated from research or physiologic studies	Retrospective case comparison; research (nonclient owned) studies

MATERIALS AND METHODS

Pubmed and Veterinary Information Network (VIN) databases were searched (date of search: October, 2008) for the following terms: "hip dysplasia," "hip (osteo)arthritis," "physical therapy," "conservative management arthritis," and "exercise therapy."

Exclusion criteria included: scientific abstracts, review articles, primary evaluation and/or comparison of anti-inflammatory pharmaceuticals, treatment of OA in joints not including the hip, articles not printed in English, species other than dogs, and nontreatment-based articles (eg, radiographic signs of early HD).

Fourteen articles were found that met the inclusion criteria, including 3 Level IV, 4 Level III, and 7 Level II studies (Table 2). Manuscripts were grouped by the primary means of treating HD, and the level of evidence provided by each study addressed individually.

RESULTS

Activity Restriction

The first retrospective study examining the effects of conservative management of HD was published in 1987 by Barr et al.[5] Outcome of restricted activity, defined as "short leash walks only," was investigated in 50 dogs with clinical and radiographic HD at <1 year of age; dogs were 1–11 years of age when evaluated. Outcome measures evaluated were radiographic appearance of OA, owner-assessed functional abilities, and subjective gait evaluation. Thirty-eight dogs (76%) had no or intermittent hind limb lameness, and 72% were considered to have normal exercise tolerance by their owners. No correlation between radiographic severity of OA and clinical outcome was identified. The authors concluded that despite initial radiographic evidence of HD in all dogs, and progression of radiographic signs in 89% of dogs, spontaneous improvement of clinical signs often occurred, implying that surgical treatment of immature dogs may be premature or unnecessary.

This study provides **Level IV** evidence. In addition to the limitations inherent to retrospective studies, the outcome measures used were subjective, and details of how activity or gait scoring was conducted were not provided. Statistical evaluation was not performed, or at least not reported, for outcomes other than radiographic score. This study was not controlled for the age of the dogs at follow-up evaluation. HD has subsequently been shown to have a bimodal age distribution and although this pattern was not documented at the time of Barr's publication, it is a limitation of the study. Dogs that were initially evaluated at a younger age may have eventually developed overt clinical signs referable to HD. Finally, factors such as the dogs' weight and body condition were not reported or considered. The relationship of body weight and condition on the development of clinical and radiographic signs of OA would have been interesting variables to evaluate.

Ten years later, another group compared long-term results of conservative management of skeletally immature dogs with HD to dogs that have 1 of 2 surgical procedures: TPO or FHO.[6] This retrospective study consisted of 20 dogs (7 treated conservatively, 8 that had TPO, and 5 that had FHO) reevaluated ≥20 months after initial diagnosis or treatment. Conservative management was defined as "controlled regular exercise, weight control, and analgesics." Outcome measures included radiographic evaluations and both client and investigator assessment of lameness, pain, range of motion, and daily activity. Dogs that had TPO had less pain and lameness and were able to perform daily functions better than dogs in the other groups. It was concluded that dogs managed conservatively had worse outcomes compared with either surgical technique, based on significantly worse lameness, pain, and musculature scores in the conservative group compared with TPO and FHO groups. The authors suggested that results of conservative treatment may be worse than previously reported by Barr.

The evidence in this study is **Level IV**. By assigning numerical scores for statistical comparison, the outcome measures used were more robust than those of Barr; however, significance was not reported for any outcome measure. Scores were assigned subjectively and investigators

Table 2 Summary of Studies Evaluating Nonsurgical Treatment of Hip Dysplasia and Osteoarthritis in Dogs, Grouped by Level of Evidence

Level	Title of Study (reference no.)	Date	N	Study Design	Primary Outcome Measures	Conclusion
II	Lifelong diet restriction and radiographic evidence of OA of the hip in dogs (10)	2006	48	Longitudinal cohort study	Radiographic evidence of OA	Restricted feeding resulted in lower body weight, delayed and decreased severity of radiographic OA, and a longer life span
II	Evaluation of PSGAG for the treatment of hip dysplasia in dogs (12)	1994	84	Double blind, randomized, placebo-controlled, multicenter clinical trial	Orthopedic examination score (lameness + pain + range of motion)	No statistical improvement with PSGAG; trend toward improvement with 4.4 mg/kg
II	Effect of adipose-derived mesenchymal stem cells in dogs with chronic OA of the hip (14)	2007	18	Double blind, randomized, placebo-controlled, multicenter clinical trial	Orthopedic examination score (lameness, pain, range of motion, functional disability)	Statistically significant improvement in lameness, pain, and range of motion
II	Evaluation of an oral antioxidant as a treatment for OA secondary to HD (17)	1998	18	Double blind, randomized trial	Lameness evaluation, range of motion, owner assessed function	Improvement with treatment
II	Evaluation of implants of gold wire at acupuncture points in the dog as treatment for OA induced by HD (21)	2001	38	Double blind, randomized, placebo-controlled clinical trial	Lameness, pain, range of motion, owner assessed function	No improvement with treatment; placebo effect noted
II	Gait analysis of dogs with HD treated with gold bead implantation acupuncture (22)	2002	19	Double blind, randomized, placebo-controlled clinical trial	Force plate, kinematic analysis, subjective lameness examination, owner-assessed function, and pain	No improvement with treatment; treated dogs had increased lameness
II	The pain relieving effects of the implantation of gold beads into dogs with hip dysplasia (23)	2006	78	Double blind, placebo-controlled clinical trial	Lameness score, pain, owner-assessed function, and behavior	Significant improvement in mobility and decreased pain in treated dogs; placebo effect noted
III	Effect of weight reduction on clinical signs of lameness in dogs with hip OA (9)	2000	9	Nonblinded prospective clinical trial	Lameness score, body condition and weight, pain during manipulation of hip	Weight loss is significantly associated with decreased lameness
III	Effect of PSGAG on COMP, C-reactive protein, MMP-2 and -9, and lameness in dogs with OA (13)	2007	16	Prospective nonrandomized clinical trial	Lameness score, blood MMP-2, -9, COMP, and C-reactive protein levels	Significantly decreased lameness and COMP in treated dogs
III	Effects of radial shockwave therapy on the limb function of dogs with hip OA (20)	2007	24	Nonblinded prospective study	Force plate analysis	Treated dogs developed symmetric gait and increased weight bearing on effected limbs
III	Two-year follow-up study of the pain relieving effect of gold bead implantation in dogs with hip OA (24)	2007	73	Nonblinded prospective study	Lameness score, pain, owner assessed function and behavior	Improved function and decreased pain 24 months after treatment
IV	Long-term results of conservative management of hip dysplasia in growing dogs (5)	1987	50	Retrospective study	Gait evaluation (subjective)	Approximately 50% of dogs did not have gait abnormalities at follow-up; many dogs had normal gait despite radiographic OA
IV	Long-term results of conservative treatment, FHO, TPO for treatment of HD in immature dogs (6)	1997	20	Retrospective study	Owner assessed activity and pain questionnaire; Orthopedic examination score (lameness, musculature, pain, range of motion)	Dogs treated without surgery had poorer outcomes; dogs treated with TPO had less pain and lameness and improved function
IV	Long-term outcome of nonsurgical management of dogs with clinical hip dysplasia (7)	2007	74	Retrospective study	Owner assessment of pain and function and use of analgesics; Orthopedic examination (lameness, musculature, pain, range of motion, body condition)	The majority of dogs had clinical lameness, pain and/or impaired activity; many dogs had other concurrent orthopedic abnormalities

were not blinded to the treatment the dog received. Because conservative treatment was used as a control group (investigators were primarily concerned with the results of surgical intervention), bias may have been present when the dogs were evaluated. Dogs were not randomly assigned to treatment groups, and the overall number of dogs in each group was small (power was not reported). Whereas this study may justifiably provide evidence supporting the benefit of TPO in dogs with HD, the results do not invalidate the option of conservative management.

Farrell et al evaluated long-term outcome of dogs with HD managed without surgery.[7] This **Level IV** retrospective study evaluated 74 dogs using owner-completed questionnaires designed to assess pain, lameness, exercise tolerance, and frequency of administration of medication. Thirty percent of dogs were reevaluated by the investigators and assigned scores for lameness, muscle mass, body condition, range of motion, and pain on manipulation of the coxofemoral joint. Nonsurgical management was defined as weight control, activity restrictions, and administration of pain-relieving medication as needed. Most dogs were clinically affected in 9 of 11 outcomes measured. The authors concluded that a significantly higher proportion of dogs managed conservatively was clinically affected compared to dogs in the Barr[5] and Plante[6] studies. In contrast to previous studies, which excluded dogs affected with concurrent orthopedic disease, 40% of dogs in Farrell's study were clinically affected by other orthopedic conditions. Dogs were also older than those included in previous studies.

Despite the low levels of evidence provided by these 3 retrospective studies, the results suggest that additional intervention would be beneficial and potentially able to improve outcomes in dogs with HD managed without surgery.

Weight Control Through Dietary Restriction

Excessive body weight has been shown to contribute to the progression and symptoms associated with OA in people.[8] Two separate groups of investigators hypothesized that weight reduction achieved through dietary management would also directly affect the development of OA in dogs with HD.[9,10]

Impellizeri et al,[9] prospectively evaluated 9 dogs that were 11–12% overweight and had lameness attributable to HD. Dogs were fed 60% of the calories needed to maintain their initial weight and were evaluated until there was at least a 10% decrease of their initial body weight. Outcome measures included body weight and hind limb lameness scores. Dogs were not placed on a specific exercise regimen and maintained their typical level of activity (sedentary for all dogs). All dogs lost between 11–18% of their initial body weight and showed decreased lameness at the midpoint and end of the study period. Pain medication was not administered to any dog during the study and none of the dogs were censored during the study because of pain. It was concluded that weight reduction alone resulted in considerable

improvement in clinical lameness in overweight dogs with hip OA.

Although this was a prospective study, dogs were not randomized to a treatment group. Rather, this was a **Level III** case comparison study. Whereas the prospective nature of the study adds strength to the level of evidence, the outcomes evaluated remained subjective. The authors acknowledge that future studies would benefit from objective measurements of body condition (such as dual energy x-ray absorptiometry) and force plate analysis of lameness. Nonetheless, this study does provide evidence for the importance of weight restriction in the management of HD.

In a longitudinal cohort study, 48 Labrador Retrievers in a research colony were followed from birth until death.[10] As puppies, these dogs were paired by litter, sex, and weight and then randomized to either control or limit-fed groups. One pair mate was fed ad libitum (control group) and the other pair mate was fed 75% of the volume the control dog consumed daily (treatment group). All dogs were fed a diet with the same composition and were housed under the same conditions with equal opportunities for exercise. Body weight and condition were recorded, and coxofemoral radiographs evaluated. The limit-fed group weighed 26% less than the control group of dogs over 12 years. Interestingly, while control dogs developed radiographic signs of OA at a mean of 6 years, the limit-fed dogs did not develop similar signs until 12 years of age. By study end at 14 years, 83% of control dogs had radiographic signs of OA compared with only 50% of the limit-fed dogs. Dogs in the treatment group lived, on average, 2 years longer than ad libitum-fed dogs.

These results provide noteworthy, **Level II** evidence for the impact of body weight on the development of hip OA in dogs. However, as demonstrated by Barr et al,[5] radiographic appearance of OA does not always correlate with clinical disease. Dogs can have severe arthritic changes on radiographs but may not have signs of pain and/or lameness. Yet, because a smaller percentage of dogs developed radiographic signs of OA when body weight was limited, it can be assumed that an even smaller number would have shown clinical signs associated with hip OA, if functional outcomes such as gait analysis and pain assessment had been performed.

Modulation of Degenerative Joint Disease

The hallmark of HD is coxofemoral laxity and progressive development of OA.[1] Laxity results in abnormal joint loading, causing osteochondral damage, and initiation of inflammatory and degenerative processes.[11] Inflammatory mediators such as interleukin 1 and tumor necrosis factor α, along with free radicals are released into the joint.[11] Inflammatory cytokines cause the release of matrix metalloproteinases (MMP) from chondrocytes that cause breakdown of the cartilage matrix and loss of proteoglycan. This leads to distorted cartilage mechanics and further joint injury. These degenerative processes become a vicious cycle, resulting in cartilage erosion, subchondral bone sclerosis,

joint capsule thickening, periarticular new bone formation, and associated pain and loss of function.[11] Several investigators have focused on altering these molecular processes, by inhibiting various steps in the inflammatory cascade and administering substances that may aid restoration of cartilage matrix to treat HD and OA.[12–14]

Polysulfated glycosaminoglycans (PSGAG) are a semisynthetic product structurally similar to the glycosaminoglycans in articular hyaline cartilage. PSGAGs have been shown to stimulate collagen synthesis and inhibit collagen breakdown in vitro.[15] In 1994, DeHaan et al evaluated the efficacy of 3 different doses of PSGAG to ameliorate clinical signs ascribed to HD in adult dogs. This was a controlled, double blind, multicenter study providing **Level II** evidence.

Dogs aged 2–10 years with overt clinical signs and radiographically confirmed with bilateral HD were recruited from 9 veterinary centers. Dogs were randomly assigned to 1 of 4 treatment groups (3 different doses of PSGAG and 1 placebo control). Investigators and owners were blinded to each dog's treatment. Dogs were administered 8 intramuscular injections over 1 month. Outcomes measured included lameness, pain, and range of motion scores, in addition to general physical examination and routine blood work to monitor for adverse reactions. Dogs were examined before each treatment and 1 week after the last treatment. Eighty-four dogs met the study inclusion criteria. Dogs administered the intermediate treatment dose (4.4 mg/kg) had the most improvement in orthopedic scores, and dogs in the placebo group showed least improvement; however, there was no statistically significant difference between any treatment groups and placebo.

This was a well-conducted, placebo-controlled, prospective study. Although a trend toward improved orthopedic scores was observed for treated dogs, the authors were unable to conclude that PSGAGs were effective in treating dogs with severe hip OA. It is possible that the outcome measures used were not sensitive enough to detect a difference in treatment groups. Statistical power was not reported, so it is also possible that a type II statistical error (accepting the null hypothesis when it is incorrect) occurred. Additional study limitations include short follow-up period and subjective outcome measures of lameness and pain. Also, it would have been useful to evaluate joint fluid and markers of collagen degradation before and after PSGAG administration.

The effects of PSGAGs on biomarkers of degenerative joint disease were investigated in a prospective, nonrandomized clinical trial of 16 dogs with OA and 5 normal dogs administered 5 mg/kg PSGAG intramuscularly.[13] Dogs were evaluated and scored for lameness before and after 8 treatments with PSGAG. Blood samples were analyzed for MMP-2 and -9, cartilage oligomeric matrix protein (COMP), and C-reactive protein. MMP-2 and -9 degrade collagen and other components of articular cartilage can be increased in synovial fluid from arthritic joints.[16] COMP is a substrate for MMP and increased levels have been identified in diseased cartilage, joint fluid, and serum of dogs

and people with arthritis.[13] C-reactive protein is a marker of acute inflammation.[13]

Twelve OA (75%) dogs had significantly improved lameness scores ($P = .001$) after treatment. Serum COMP levels decreased significantly after PSGAG administration ($P < .001$). Interestingly, dogs that did not have improved lameness scores had significantly higher ($P = .013$) COMP levels than dogs that did improve. No significant differences were found in MMP-2 or -9 levels after PSGAG treatment. C-reactive protein in OA dogs was not significantly different from control dogs before or after PSGAG.

This study provides **Level III** evidence. Dogs were not randomized to treatment groups. Outcomes measured represented both subjective and objective data and were well correlated, in that dogs that improved on subjective lameness examination also had decreased COMP. Whereas this study included dogs with forelimb OA as well as dogs with hip OA, dogs with hind limb lameness improved significantly more after treatment than dogs affected in the forelimbs. This study did not provide long-term follow-up; however, it did provide reasonable evidence to support the use of PSGAGs in the treatment of OA. Further studies would be necessary to determine duration of treatment effect.

Antioxidant Impellizeri et al conducted a prospective study to examine the effects of an oral antioxidant as a treatment for OA in dogs with HD.[17] Eighteen dogs (1–13 years) were randomized to either the treatment (Proanthozone multivitamin antioxidant) or placebo group; both owners and investigators were blinded. Outcome measures included investigator evaluation of lameness and range of motion and owner-reported functional assessment. Data were collected at biweekly intervals for 14 weeks. This study reported an improvement in the treatment group at a significance level of 0.1.

According to the proposed Evidence Classes, this would represent a **Level II** study; however, no details other than those listed above were provided about methods of evaluation (scoring system) or outcomes. The individual scores for each of the groups were not reported and results did not include any data other than the significance level. Whereas this study may have been a well-conducted evaluation, providing evidence to support the use of antioxidants in the treatment of HD, the dissemination of the information was weak, leaving the critical reader questioning the true level of evidence.

Mesenchymal Stem Cells (MSC) Attempts to modulate the biological effects of degenerative joint using adipose-derived MSC have been investigated in dogs with hip OA.[14] MSC are multipotent cells that can differentiate into fat, bone, and cartilage cell lineages and unlike embryonic stem cells, can be collected from adult bone marrow and adipose tissue.[14] Autologous MSC have demonstrated therapeutic effects in experimental studies, including improved tendon healing in horses and increased meniscal tissue regeneration and delayed OA progression in a goat model.[18,19]

Based on the apparent efficacy of MSC in other species, Black et al examined the effects of autologous MSC in dogs with clinical and radiographic signs of hip OA using a multicenter, randomized, double blind, placebo-controlled trial that included 18 dogs (1–11 years old). Treatment consisted of surgical collection of fat followed by intraarticular (coxofemoral joint) injection of isolated, resuspended MSC. Control dogs underwent fat collection and were injected with a placebo. Owners were instructed not to alter the amount of pain medication the dog had been receiving and to leash walk the dog twice daily. Dogs were evaluated and assigned a numerical score for lameness, pain, range of motion, and functional ability on days 0, 30, 60, and 90. Owners also completed a functional ability questionnaire at each time point.

Dogs treated with MSC improved significantly in veterinarian assessed lameness, range of motion, and pain scores at all time points; owner evaluated scores improved, but not significantly, compared with control dogs. This was a well-conducted study that provides **Level II** evidence in support of the use of MSC in dogs with hip OA. Whereas the results were promising, the outcome measures were subjective. Nonetheless, clinical use of MSC in dogs with OA, along with additional studies using objective measures of efficacy are warranted.

Extracorporeal shockwave therapy (ESWT) is a modality in which a high-energy sound wave is applied transcutaneously, to increase tissue cytokine and growth factor expression and reduce pain associated with chronic inflammation.[20] Meuller et al investigated physical alteration of the joint environment in dogs with hip OA by means of radial EWST. This was a nonrandomized, nonblinded, prospective study in which 18 dogs with unilateral or bilateral hip OA were treated with EWST 3 times, at weekly intervals. Six dogs with hip OA were not treated and served as controls. Outcome measures were obtained by force plate gait analysis, and dogs were evaluated before and 6, 12, and 24 weeks after treatment.

Dogs treated with ESWT developed a more symmetrical gait, with peak vertical force and vertical impulse equalizing between limbs; however, untreated dogs also had similar changes in peak vertical force at 6 weeks. The authors concluded that radial ESWT resulted in increased weight bearing on limbs with OA, and effects of 3 treatments may last at least 12 weeks. The importance of evaluating both peak vertical force and vertical impulse was also acknowledged.

This is a **Level III** study that provides support for the use of ESWT based on objective data. There were a number of study limitations apart from the lack of randomization and blinding of investigators. It was not stated whether there was significant difference between dogs in each group for age, body weight, or initial disease severity. The authors did not state if dogs with additional orthopedic or neurologic abnormalities were excluded. Furthermore, it was not stated whether additional methods of disease management, such as administration of analgesics were permitted. Finally, while objective outcome measures provide a strong

level of support, inclusion of subjective data including pain evaluation would have added to the clinical relevance of this study.

Acupuncture

Several prospective clinical studies have evaluated outcome of dogs treated for OA with acupuncture. Hielm–Bjorkman et al conducted a double blind study in 38 dogs with HD and OA randomly assigned to receive either gold wire inserted at acupuncture points or sham treatment (skin pierced at nonacupuncture points).[21] Dogs were evaluated prospectively before and 4, 12, and 24 weeks after treatment. Veterinarians assessed dogs for pain, lameness, and joint range of motion. Owners completed questionnaires 7 times throughout the study, evaluating their dog's pain, behavior, and functional abilities. Results failed to show a statistically significant improvement in dogs treated with gold wire implantation, and a placebo effect was noticed with both owners and veterinarians reporting improvement in 50–60% of the sham-treated dogs.

This study provides **Level II** evidence that does not support this method of acupuncture in the management of dogs with hip OA. This was a well-conducted and well-reported trial. The authors make a point of reporting statistical power and significance for all of the outcomes evaluated, thoroughly discuss confounding factors, such as some dogs receiving medication whereas others did not, and present theories for lack of significant improvement in treated dogs. An obvious limitation of the study was lack of objective outcome measures; nonetheless, had force plate analysis detected decreased lameness in treated dogs, lack of corresponding clinical improvement would leave the clinician weighing the importance of such objective data.

Another group of investigators used objective outcome measures (kinetic and kinematic analysis) to evaluate gold bead implantation at acupuncture points in dogs with HD and OA.[22] This was a prospective, double blind clinical trial in which 19 dogs were randomly assigned to treatment or control groups. Objective and subjective data (lameness score and owner questionnaire) were collected at 0, 1, and 3 months after treatment. There was no significant improvement in both objective and subjective variables and, in fact, dogs in the treatment group were more likely to have increased lameness according to their owners. This study contributes additional **Level II** evidence against the use of gold beads inserted at acupuncture points in dogs with hip OA.

Conversely, Jaeger et al published evidence supporting the efficacy of gold bead implantation as a treatment for dogs with coxofemoral OA.[23,24] The first of these studies provides **Level II** evidence and was a double blind, placebo-controlled trial that included 78 dogs.[23] Outcome measures were subjective, including veterinarian and owner assessment of lameness, pain, function, and behavior measured at 0, 2, 12, and 24 weeks. There was a significantly greater improvement in treated dogs, yet a placebo effect was also seen.

With **Level III** evidence, Jaeger et al reported long-term follow-up for 73 dogs from the initial study[24]; 66 dogs that had previously been treated with placebo were subsequently treated with gold bead implantation, and 7 dogs remained in the control group. The trial was no longer blinded. Investigators and owners assessed the dogs using the same subjective variables from the first study 24 months after initiation of the blinded portion of the study. Improved function and reduced pain continued to be reported by owners and veterinarians at study end. This study may have had additional bias, because the observers were no longer blinded. Nonetheless, the authors concluded that the study provided additional evidence in support of acupuncture for treatment of HD and OA, and that improvement after treatment may be sustained for at least 24 months.

The contrasting results between the Jaeger studies and those of Hielm–Bjorkman and Bolliger may be explained by the difference in case numbers: a greater number of dogs were included in Jaeger's study, resulting in adequate power to achieve significance. The trial by Hielm–Bjorkman also admitted dogs that received surgical intervention for HD, whereas dogs that had only been managed with nonsteroidal anti-inflammatory drugs were accepted into Jaeger's trial. In addition to the contrasting study designs, acupuncture requires experience and proficiency, and therefore, differing results may also be based on clinical technique.

Conclusions

The nonsurgical management of HD and OA encompasses a wide range of therapies. The traditional concept of conservative management is an ambiguous definition of activity limitation and pain management. Three studies report **Level IV** evidence regarding the use of this method of treatment—1 study in support and the other 2 contesting activity restriction as an adequate method of managing HD and OA. On the other hand, 2 well-conducted prospective trials report positive, **Level II** evidence in support of weight restriction through dietary limitation as an effective means of managing hip OA. A substantial component of the information reviewed in this manuscript involves studies aimed at modulating the molecular events associated with OA. Two studies provided **Level III** and 3 studies presented **Level II** evidence in support of various methods of attenuating degenerative and inflammatory processes within the joint. Finally, conflicting evidence was available for the use of gold beads implanted at acupuncture sites: 2 **Level II** studies refute whereas 1 **Level III** and 1 **Level II** support this method of treatment.

This manuscript is a systematic review rather than a metaanalysis, and therefore, new results are not described. However, the purpose of this review is to summarize previous studies that evaluate nonsurgical methods of managing HD and associated OA so that veterinarians can make evidence-guided treatment recommendations (Table 1). Levels of evidence can be used to categorize studies based on their design; however, other factors such as sample size and study limitations should be taken into consideration when drawing conclusions from the literature.

Summarily, conclusions that may be drawn from this review are that weight management is an important aspect of preventing and managing OA, and modulation of joint disease through the use of PSGAGs, MSC, or possibly ESWT may be beneficial. What can also be gleaned from this review is the lack of **Level I** evidence in the veterinary literature and the need for studies investigating physical rehabilitation therapies, such as exercise and hydrotherapy, in dogs with HD and OA.

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