Review Article

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Osteoarthritis in cats: what we know, and mostly, what we don't know. . . yet

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Abstract

Osteoarthritis (OA) is a degenerative joint disease that is considered the primary source of chronic pain in cats, affecting well over a quarter of the feline population. Despite its prevalence, detection and diagnosis rates remain low, as many owners are unaware of the signs of feline OA. There is limited knowledge regarding the management of feline OA, with only 29 publications available, many of which lack rigorous methodology. Furthermore, most research focuses on the efficacy of non-steroidal anti-inflammatory drugs, while proposed alternatives to alleviate feline OA pain – such as food restriction, weight loss, adjunctive musculoskeletal treatments with biologics, physiotherapeutic modalities and lifestyle changes – are primarily based on human clinical studies and veterinary research on other species, which introduces a high risk of bias. New promising avenues are being explored with anti-nerve growth factor monoclonal antibodies; however, the long-term effects of repetitive administration, optimal conditions for administration and specific indications have yet to be described. Research from the *Groupe de recherche en pharmacologie animale du Québec* (GREPAQ) on pharmacological and non-pharmacological therapies for feline OA suggests that a shift in the OA management paradigm may be warranted. An omega-3 enriched diet has demonstrated therapeutic efficacy comparable to standard pharmacological treatments, without side effects and with high compliance. In addition, it was equally effective for cats with severe OA as for those with moderate OA. By establishing a theoretical framework for feline OA management based on robust scientific evidence, veterinarians will be better equipped to select treatments tailored to the diagnosed (or suspected) manifestations and mechanisms of OA pain, ultimately improving the health and well-being of their feline patients. Future research should explore the concomitant use of different therapeutic approaches, as they may offer superior outcomes compared with a single treatment through additive or synergistic effects.

Plain language summary

A narrative review about feline osteoarthritis in 2025

For decades, we have heard that osteoarthritis is the most common degenerative disease affecting cats, at least in Western countries. At the same time, reports have regularly highlighted that osteoarthritis is largely underdiagnosed and difficult to manage in cats.

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This narrative review includes the most up-to-date information about osteoarthritis, particularly as it pertains to cats, rather than extrapolating data from other species affected by the same disease. It addresses the importance of osteoarthritis in the feline population and the behavioral signs associated with osteoarthritis, as well as the mechanisms of joint degeneration and the establishment of consequences on nervous system function. Finally, all pharmacological and non-pharmacological (nutraceutical supplements, diets, physiotherapy, etc) therapeutic approaches listed for managing feline osteoarthritis were assessed based on their efficacy evidence and the quality of the referenced studies.

Osteoarthritis can be detected earlier in cats. Radiography is critical for confirming the disease. The authors listed all management approaches in decreasing order of their efficacy:risk of side effects ratio. A recently published study testing a therapeutic diet enriched in marine omega-3s, turmeric extract and hydrolyzed collagen found the diet to be equally effective as, or even better than, all previously tested drugs with the same validated outcomes. Moreover, the diet was equally effective in confirmed severe or moderate osteoarthritis and demonstrated good compliance with eating, as well as no adverse effects.

This evidence has the potential to change the current paradigm of feline osteoarthritis management.

Keywords: Degenerative joint disease; detection; diagnosis; chronic pain management; therapeutic efficacy; pharmaceutical; non-pharmacological

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Disease importance

Osteoarthritis (OA) is a degenerative joint disease that is clearly recognized as the primary source of chronic pain in cats.[1] The lumbar or lumbosacral region and the appendicular joints (hip, stifle, hock and elbow) are commonly affected.[2] Descriptive studies have reported a wide prevalence range of 16–91% of cats (primarily for radiographic OA) aged 0.2–20.0 years.[2–10] This variability arises from discrepancies in the specific feline populations studied, which differ according to age, sex, breed, weight and geographical areas, as well as from small sample sizes and the complexity of OA, which complicates diagnosis and prevalence assessment.[10–12] Nevertheless, when compiling the prevalence data available to date, 25.6% of cats (n = 454/1772, aged 0.2–20.0 years) showed radiographic evidence of OA in at least one appendicular joint.[2–10] However, these figures primarily represent clinical prevalence and are likely underestimated; post-mortem evaluations suggest an estimated clinical prevalence of 2.5% compared with a true prevalence of 20%.[10–12] This indicates that the prevalence of feline OA is likely much higher than 25.6%, especially from the onset of the animal's adult life.

As in humans, feline OA is typically associated with aging and the 'wear and tear' of joints. Consequently, OA is considered predominantly an idiopathic or primary disease in cats, with clinical signs often appearing later in life. Although there is a strong correlation with age, epidemiological studies have shown that structural damage due to OA can be observed in cats as young as 1 year old.[6] Congenital articular malformations, developmental conditions such as elbow or hip dysplasia, and traumatic events involving a joint such as ligament ruptures or fractures can also lead to secondary OA.[13] Other factors, including diet, obesity, sterilization, genetics, breed and environment, are generally considered risk factors that can influence the development and progression of OA, although their impact may vary by species.[5,14] Although these risk factors (as well as size and weight) are recognized in canine OA,[15] their influence has not been as clearly demonstrated in feline OA.

What are the signs?

In the early stages of OA, regardless of the species, the clinical signs are usually subtle, intermittent and insidious before becoming permanent in severe stages.[16,17] However, lameness and stiffness do not appear to be the primary clinical signs of OA in many cats.[18] It has been suggested that this may be linked to the bilateral nature of feline OA (as observed in original kinematic gait analyses of healthy and osteoarthritic cats[19]), their agility and ability to adapt their mobility, or their tendency to hide pain to protect themselves from predators.[9]

In the absence of lameness, it is often the behavioral changes in cats – such as alterations in jumping activity or stair use, reduced mobility, increased rest, isolation and aggression – that prompt veterinary consultations.[18] However, owners frequently perceive these behavioral changes as normal for an aging animal, which may contribute to the underdiagnosis of feline OA despite its prevalence.[9,17] Ultimately, untreated chronic pain induced by OA directly impacts the cat's quality of life.[20] A systematic review on indicators of health-related quality of life in cats with degenerative joint disease found that increased

pain and decreased mobility negatively correlated with physical appearance, energy, vitality, mood, sociability, and overall physical and mental well-being.[21] Identifying the specific moment that led owners to notice their cat's malaise may be key to enabling veterinarians to diagnose OA effectively.

The diagnosis of OA is based on the animal's medical history, the veterinarian's assessment of muscle atrophy, altered joint mobility, decreased range of motion, joint degradation and painful reactions upon palpation. In cats, signs such as joint thickening, synovial effusion, reduced range of motion and crepitus are less obvious than in dogs.[9] Reactions to palpation must be interpreted cautiously by veterinarians, as many cats dislike being touched, even when their joints are normal and pain-free.[9,14] In the validation process of a clinical metrology instrument, namely the Montreal Instrument for Cat Arthritis Testing, for use by Veterinarians (MI-CAT(V)), the response to palpation and manipulations (even when performed by an experienced board-certified surgeon) was excluded because of its lack of specificity.[22] Thus, radiography is considered the reference examination for the clinical diagnosis of OA.[17] However, radiographs do not provide a definitive diagnosis. In fact, there is little association between radiographic OA findings and limb function or pain reported by veterinarians and owners.[14,23,24] Moreover, radiography is not sensitive enough to detect the early stages of OA,[25] the positioning of the animal during the procedure can be challenging and may affect radiographic interpretation,[9,25] and contrast resolution may limit the veterinarian's ability to accurately assess an affected joint.[26] Despite these limitations, a study comparing cats diagnosed as OA-positive through both radiographic and orthopedic examinations (n = 32) vs only orthopedic examination (n = 10), with a balanced distribution of sex in both groups, found that only cats diagnosed through both methods were effectively affected by OA.[27] There was a significant difference in objective validated outcomes, such as podobarometric gait ('force plate') analysis (PGA) and peripheral sensitization (as assessed through von Frey aesthesiometer-evoked paw withdrawal threshold), with a trend observed in actimetry.[27] The radiographic scoring method used in this study[27] is simple and has been previously validated against more sensitive MRI techniques.[28] Unfortunately, despite the growing recognition of the importance of feline OA, many clinicians lack the necessary experience to detect feline joint abnormalities on radiographs.[9] Therefore, the dilemma surrounding the lack of effective pain management for feline OA is not only due to owners under detecting OA-associated behavioral changes, for which alteration in normal daily activity due to pain is the most significant manifestation, but is also exacerbated by the high rate of underdiagnosis among veterinarians.

What is osteoarthritis?

The complex interactions between structural changes in the joints and the sensory-discriminative and affective-motivational nervous pathways characterize OA as an inflammatory and nociplastic chronic pain disorder.[29,30] If OA leads to progressive joint degeneration, whether slowly or rapidly, it affects the entire organ, including the cartilaginous matrix, subchondral bone, joint capsule, synovial membrane and fluid, as well as menisci, ligaments, tendons and muscles.[31–33] As the cartilage begins to thin, the joint instability worsens, the tendons and ligaments experience increased stress, eventually stretching abnormally, which can aggravate synovial effusion and the formation of osteophytes.[31,32] The damage to the various articular structures makes movement more difficult, resulting in stiffness, reduced joint mobility and pain. In addition, decreased use of the affected limb may lead to muscle atrophy and articular sclerosis, perpetuating a vicious cycle.[31–33]

Sensory and sympathetic nerve fibers, along with their neurotransmitters, play a crucial role in the pathophysiology of OA pain.[29] The innervation of different joint components, such as the periosteum, subchondral bone, ligaments, menisci and synovium, is modulated by the local release of sensory neuropeptides from afferent neurons, leading to the phenomenon of neurogenic inflammation that exacerbates the release of inflammatory mediators from cellular damage.[29,30,34] These neuro inflammatory mediators, such as nerve growth factor (NGF), contribute to increased growth and responsiveness of pain fibers.[35] Elevated levels of NGF in the synovial fluid of dogs with chronic lameness due to OA have been documented compared with healthy joints;[36] however, this has not yet been demonstrated in cats. Unfortunately, the literature remains controversial regarding the modifications of nerve fibers during the development of OA.[29,37,38] Nevertheless, peripheral sensitization, characterized by allodynia and hyperalgesia, and central sensitization, manifested by the spinal wind-up phenomenon, have been demonstrated in cats.[39,40] There is no doubt that plasticity of the nervous system (Figure 1), along with structural changes, is critically implicated in the pathogenesis of feline OA.[29,39,40]

How is osteoarthritis managed?

Since OA itself is currently incurable, the aim of treatment is to alleviate the negative consequences of pain and mobility impairment on the animal's affective state (including anxiety, depression and sleep disturbances), cognitive function, social relationships and overall quality of life. This review proposes a classification of pharmacological and non-pharmacological therapeutic approaches, based on their efficacy and the quality of the referenced studies. The quality of each study was evaluated through

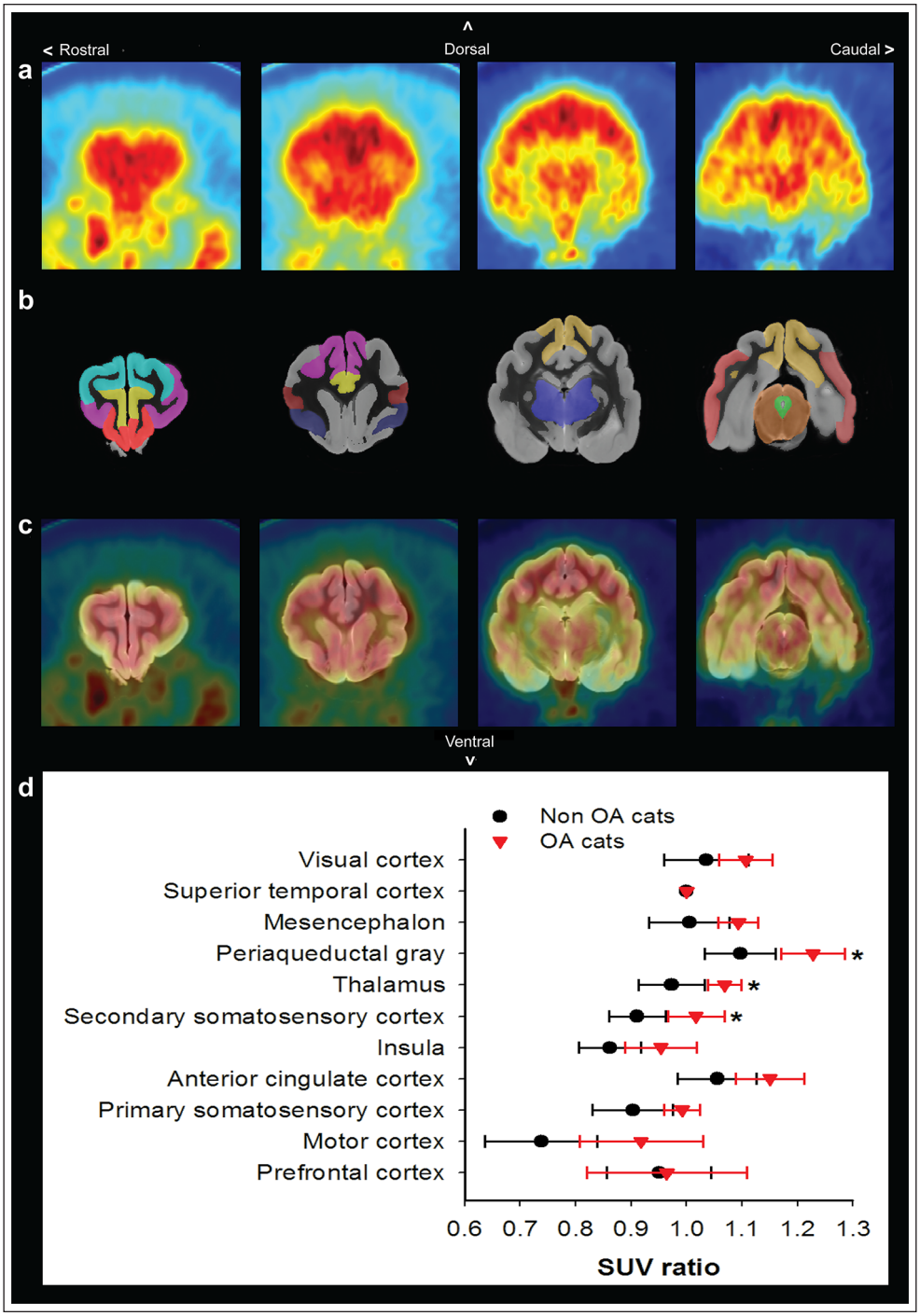

Figure 1 Cat brain activation in the presence of chronic osteoarthritic pain. Four transverse slices: (a) cat brain imaged with [^{18}F]-FDG using a small animal PET scanner; (b) brain ROIs segmented from MR images; (c) PET signal co-registered with MR images; (d) after the cartography, the mean metabolic activity in the brain ROI was expressed as a ratio of SUVs and standard deviation. The brain metabolism in the secondary somatosensory cortex, thalamus and periaqueductal gray matter was significantly increased ($P \leqslant 0.005$) in cats with osteoarthritis compared with healthy cats. Brain plasticity in cats suffering from chronic pain is associated with sustained nociceptive inputs and increased activity of the descending modulatory pathways. FDG = fluorodeoxyglucose; MR = magnetic resonance; PET = positron emission tomography; ROI = region of interest; SUV = standard uptake value; SUV ratio = $SUV_{ROI}/SUV_{Superior\ temporal\ cortex}$. Courtesy of GREPAQ (adapted from Guillot et al[39])

its experimental design, such as randomized, blinded, prospective studies that include a placebo group, and the use of objective, validated pain assessments favored over subjective non-validated scales, as well as their statistical analysis adequacy.

Regardless of the method of assessment, a placebo response can occur when testing therapeutic efficacy. Moreover, subjective outcomes are more likely to generate a placebo effect than objective measures. The caregiver placebo effect is a well-known phenomenon that refers to a perceived positive effect on companion animals based on subjective outcomes in the absence of improvement in objective measures.[41] Subjective evaluation methods can be biased because of a caregiver's anticipation of benefits, which obscures potential evidence of a substantial treatment effect. For instance, in a secondary analysis of five analgesic trials conducted in feline OA, an average placebo response of 68% (reference interval 54–74) was calculated based on scores from an owner-completed clinical metrology instrument, the Client-Specific Outcome Measure (CSOM); however, only 36% of these were associated with actimetry success, highlighting the caregiver placebo effect.[42] Another owner-completed clinical metrology instrument, the Feline Musculoskeletal Pain Index (FMPI), exhibited similar weaknesses to the CSOM. Two studies showed no responsiveness of the FMPI to first-line pharmacological treatment, with a high placebo success that masked any detection of treatment effect, and no relationship was found with objective actimetry outcomes.[43,44]

Thus, validated objective measures such as actimetry monitoring (Figure 2a), PGA (Figure 2b) and quantitative sensory testing (Figure 2c), along with the subjective MI-CAT(V) scale (see Appendix A in the supplementary material), which has high metrological properties, are highly valuable to establish an accurate therapeutic efficacy profile. These measures have been repeatedly reported to differentiate between healthy and diseased cats (specificity),[22,27,45] and to detect treatment effects (sensitivity) from medications such as meloxicam,[27,46,47] firocoxib,[48,49] gabapentin,[22] tramadol[45,46] and therapeutic diets.[50] Moreover, the MI-CAT(V) scale demonstrated a low placebo response (<15%) and validated discriminatory abilities in OA severity (absent – mild – moderate – severe) and inter-/intrareliability.[49,50] Unfortunately, many studies on feline OA lack a comparative placebo group (not controlled), are consequently non-randomized and non-blinded, and use subjective, non-validated outcomes that may not be reliable, specific or sensitive to treatment. Therefore, the quality deficiencies in the resulting findings cast doubt onto the conclusions drawn regarding feline OA pain management.

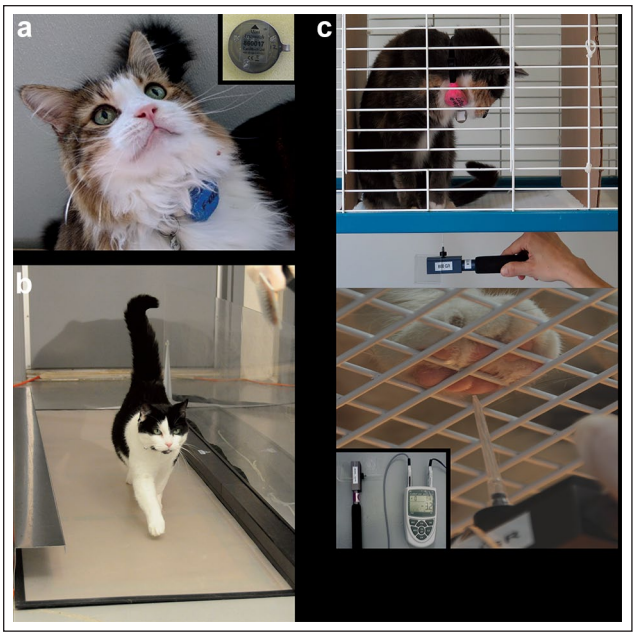

Figure 2 Illustrations of feline osteoarthritic chronic pain measurement methods. (a) An actimetric device (Mini-Actiwatch; Respironics) is a collar-mounted chip containing a tri-axial accelerometer that continuously records a cat's physical activity, typically using a 2-min epoch over a follow-up period of 4–6 months. This form of big data monitoring requires specific expertise in data extraction and has been validated as specific, sensitive and reliable (see details in the text). In cats living in groups, the placebo group shows a light, progressive and continuous decrease in nighttime actimetry, while effective analgesia is reflected in stable or even increased nighttime actimetry. (b) An osteoarthritic cat crossing the sensing walkway mattress (Walkway System WE4; Tekscan) at a regular velocity and in a straight line allows for the recording of ground reaction forces on each limb with every step. This method is widely recognized as the gold standard for detecting biomechanical alterations. Responsiveness to analgesic treatment is indicated by increased force measurements. The technique is specific, sensitive and reliable (see details in the text). (c) Peripheral somatosensory sensitization is evaluated by applying an electronic von Frey esthesiometer to the plantar/palmar paw surface. Gradually increased pressure is applied using a mechanical von Frey polypropylene probe (Rigid Tip 0.7 mm^2 of surface 28G; IITC Life Science) fitted to a handheld force transducer to determine the paw withdrawal threshold. The tip is placed perpendicularly onto the plantar/palmar surface of the four paws while the cat is standing up in a cage that is specifically designed for this evaluation, with distractions such as treats or toys. The stimulus continues until the paw is withdrawn or elevated, or any other reaction such as vocalization, agitation, jumping or avoidance, is observed, at which point the force levels are cut off. Courtesy of GREPAQ

Pharmacological treatments

Pain management in cats with OA is complex, as no clear guidelines have been established yet. Nevertheless, analgesics are usually prescribed to manage the pain. They can often be reduced or discontinued (using a therapeutic windows approach where the treatment is reintroduced when needed); however, some animals' conditions require long-term medication, which can increase the

Table 1 Literature search: pharmaceutical osteoarthritis management options

Online database search: PubMed, Google Scholar	Script (keywords)	Articles recorded (n)	References
NSAID	(cat OR feline) AND osteoarthriti* AND (NSAID OR meloxicam OR carprofen OR coxib OR piprant)	16	14, 22, 27, 43, 44, 46–49, 57–63
Anti-NGF mAb	(cat OR feline) AND osteoarthriti* AND (NGF OR monoclonal antibody)	3	67–69
Opioid	(cat OR feline) AND osteoarthriti* AND (opioid OR tramadol)	4	22, 45, 46, 64
DMOADs	(cat OR feline) AND osteoarthriti* AND (DMOAD OR pentosan OR hyaluronic OR doxycycline OR tiludronate OR steroid OR synovetin)	0	0
	(cat OR feline) AND osteoarthriti* AND (ARA 3000 BETA OR botulinum)	0	0
Amantadine	(cat OR feline) AND osteoarthriti* AND (amantadine)	1	66
Gabapentin	(cat OR feline) AND osteoarthriti* AND (gabapentin)	2	22, 65
Capsaicin	(cat OR feline) AND osteoarthriti* AND (capsaisin)	0	0

DMOAD = disease-modifying osteoarthritis drug; mAb = monoclonal antibody; NGF = nerve growth factor; NSAID = non-steroidal anti-inflammatory drug

risk of side effects. This risk must be balanced against the recurrence of OA-associated pain if the medication is not administered. In the past 24 years, a total of 23 articles on the efficacy of various pharmacological treatments such as non-steroidal anti-inflammatory drugs (NSAIDs), anti-NGF monoclonal antibodies (mAbs), tramadol, gabapentin and amantadine, have been published (Table 1). However, scientific evidence of their efficacy remains surprisingly limited because of the lack of studies with robust experimental designs.

NSAIDs are often prescribed by veterinarians as a first-line treatment for OA pain management, despite their known side effects (eg, gastrointestinal erosions/ulcerations, nephrotoxicity, hepatotoxicity, etc).[51] The risks associated with the long-term use of NSAIDs for OA management are particularly serious in cats affected by chronic kidney disease (CKD), as nephrotoxicity is one of the main side effects.[51] This is of major relevance considering that OA and CKD are often concomitant diseases in the feline geriatric population.[52] Retrospective studies initially suggested that meloxicam could slow CKD progression and increase survival.[53,54] However, even low-dose meloxicam treatment (0.02 mg/kg/day) over a 6-month period has been found to significantly worsen proteinuria in treated CKD/OA cats compared with placebo, in addition to gastrointestinal adverse effects.[55] This is problematic, as an increase in the urine protein:creatinine ratio can be associated with hypertension and the progression of azotemia,[56] requiring caution in the use of meloxicam in OA cats. The fear of side effects remains a major constraint in the use of NSAIDs for OA management in cats.

Only 16 scientific articles have been published on NSAID efficacy in feline OA (Table 1), including meloxicam (n = 13),[14,22,27,43,44,46,47,57–62] robenacoxib (n = 1)[63] and firocoxib (n = 2).[48,49] Of these, only nine studies used objective methods of assessment, six of which were published by the authors' group: Groupe de recherche en pharmacologie animale du Québec (GREPAQ).[22,27,46–49] Overall, the evidence of NSAID efficacy cannot be denied, but the intensity of the response to treatment and responder rates are lower than what might be expected.

For instance, meloxicam was reported to have a positive effect on actimetry.[22,27,44,46,47,58] However, Lascelles et al[58] reported that treated cats increased their activity levels by only 9.3% compared with baseline, with no significant difference from placebo. Similarly, Adrian et al[63] reported a 5.7% increase in activity counts over a 6-week course of robenacoxib treatment, with only 39.4% of cats receiving the NSAID increasing their activity counts by more than 10% (ie, responders to treatment) compared with baseline, which is puzzling considering that 32.8% of the control group showed a placebo effect.

In contrast, Klinck et al[47] observed a real treatment effect of meloxicam, compared with placebo, with a 23.8% increase in activity and a correlated 17.6% reduction in the Montreal Instrument for Cat Arthritis Testing, for Caretakers/owners (MI-CAT(C)) alteration score. Firocoxib induced similar intensity changes, with a 17% increase in nighttime actimetry monitoring (NAM) and a 28% reduction

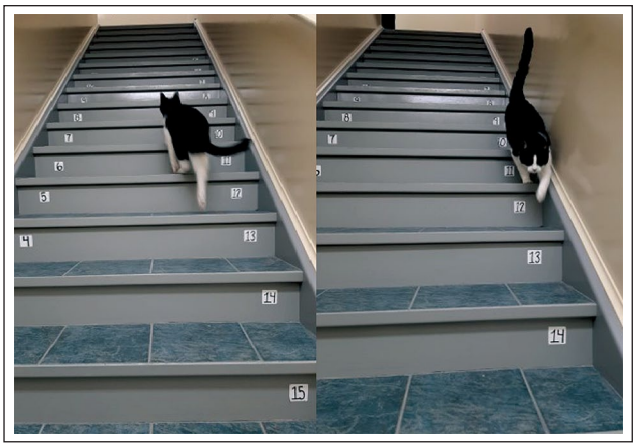

Figure 3 The stairs assay compliance test evaluates fatigue associated with chronic osteoarthritic pain. Using a standardized 16-step staircase (each step 20 cm high), the cats are encouraged to ascend to the top, where they receive a treat or another positive reward (eg, petting). They are then encouraged to descend and are rewarded again. Over a closed period of 4 mins, the cats are prompted to complete as many ascents and descents as they are able. This outcome measure has been recently validated by GREPAQ.[48] For each assay, the time (in seconds) to ascend, the number of stairs climbed before stopping, and the time and number of stairs descended before stopping are recorded. Courtesy of GREPAQ

in MI-CAT(V), associated with a real treatment effect on PGA and the stairs assay compliance test (Stairs; Figure 3).[48,49] It is interesting to note that no study published before 2016[43,44,57–61] demonstrated a real treatment effect of meloxicam, mostly because of the use of non-validated subjective scales, which were contaminated by a significant caregiver placebo effect. In addition, numerous studies reported a negative rebound effect with NSAID withdrawal, particularly observed with low doses of meloxicam.[27,44,47,62] Moreover, Delsart et al[49] reported a negative rebound effect with firocoxib, but only in cats with a mild degree of OA, while more severely affected OA cats maintained their benefits on most tested outcomes for 4 weeks after NSAID withdrawal.

Other pharmacological treatments published in the feline OA efficacy literature include tramadol, gabapentin, amantadine and frunevetmab. Four articles have been published on tramadol (see Table 1), with all studies including objective assessment methods.[22,45,46,64] Tramadol (3 mg/kg PO q12h) demonstrated a real treatment effect over 3 weeks of administration, showing improvements in actimetry (+24.4%), peak vertical force (PVF; +7.8%) and somatosensory hypersensitization.[22,45,46] Reduced dosage (2 mg/kg PO q12h), over 5 days of treatment, led to a smaller increase (+18.7%) in actimetry.[64] Gabapentin (10 mg/kg PO q8h over 30 days) has been found to slightly improve actimetry and somatosensory hypersensitization in a small sample of cats[22] or to have no positive effect on feline OA (no analgesia, sedation) at a reduced dosage (10 mg/kg PO q12h over 14 days).[65] Similar sedation effects on actimetry, but with a positive change in CSOM, were observed with 3 weeks of amantadine (5 mg/kg/day PO).[66] Lastly, three studies examined the efficacy of frunevetmab, a treatment using felinized mAb to target NGF.[67–69] Only 2/3 studies used actimetry as an objective method of assessment, but the results showed contradictory effects. Gruen et al[67] found a 10% increase in activity over a 6-week postinjection period, without corresponding results on CSOM or FMPI. However, a decrease in activity was observed during an 8-week follow-up (with two monthly injections), along with a treatment effect noted on the clinical metrology instruments after the second injection.[68] Nonetheless, the authors suggested that frunevetmab could potentially be a solution to feline OA pain. Indeed, the third study reported – with the CSOM – that 76% of treated cats responded to frunevetmab 56 days after injection, with effects carrying over until day 84.[69] However, this result is heavily outweighed by the 65% caregiver placebo effect obtained on day 56 ($P = 0.030$), which increased to 68% at day 84 ($P = 0.080$), eliminating the significant difference between the two groups.[69] Currently, guidelines on case indication (mild, moderate or severe OA; early or advanced OA) and on the potential safe and efficient use (safety of drug combinations, emergence of subpopulations more sensitive to side effects) of anti-NGF mAbs are lacking.

Disease-modifying OA drugs (DMOADs)

DMOADs are hypothesized to provide pain relief by reducing synovial inflammation, protecting cartilage through viscosupplementation, and aiding repair by targeting mediators of cartilage and bone tissue renewal. Experimental murine or canine models have previously shown promising structural effects.[70] However, to the best of the authors' knowledge, the evidence of DMOAD effectiveness in clinical canine OA remains inconclusive. Nevertheless, long-term 'structural' agents such as polysulfated glycosaminoglycan (Adequan; American Regent Animal Health), pentosan polysulfate sodium (Cartrophen Vet; Biopharm Australia), hyaluronic acid and others are still routinely used in canine OA management. Cartrophen has been used off-label to treat feline OA, despite the fact that no randomized, controlled and blinded clinical trials on DMOAD efficacy and safety for feline OA management have been conducted to date. More importantly, a case study reported acute and severe hemorrhage after Cartrophen injection in a 14-year-old castrated male Cornish Rex.[71] Based on the lack of evidence, the off-label use of such DMOADs is not recommended until further research has been completed.

Capsaicin

The properties of capsaicin have been studied in the management of chronic OA pain in both human and veterinary

medicine.[72–74] Capsaicin acts on transient receptor potential vanilloid-1 pain receptors and proinflammatory neuropeptides to desensitize the receptors, block the conduction of pain signals and provide an effective pain-killing effect.[72–74] In a reversible equine lameness model, capsaicin-treated horses showed a significant decrease in their lameness scores and other pain-indicating parameters.[75] Several research papers have been published on the effects of capsaicin injections in healthy knee joints and induced stifle arthritis in cats. He et al[76] demonstrated an inhibition of the response to mechanical and chemical stimuli in the afferent neurons of the healthy joint, while Inman et al[77] reported a decrease in histopathological inflammation scores with capsaicin injection in antigen-induced feline arthritis. However, Marshall et al[78] found that the immunocytochemical depletion of proinflammatory neuropeptides in capsaicin-injected joints did not correlate to a decreased synovium inflammation score in antigen-induced feline arthritis. No studies have been conducted on natural feline OA models, but topical capsaicin could serve as an interesting treatment alternative, if accepted.

Non-pharmacological approaches

Pharmacological approaches have not mitigated the challenges of OA management, including the lack of a cure targeting its root cause, the high cost of effective pharmaceutical options, the risk of side effects, and poor compliance with long-term treatment. Consequently, client interest in alternative therapies has been increasing and several non-pharmacological approaches have emerged in the field of feline OA management. As a result of the nature of the original compounds and favorable results from in vitro evaluations, company marketing has positioned most of these products as supportive for joint health, particularly for cartilage. However, the specific regulations for the commercialization of natural health products do not mandate the demonstration of efficacy in OA-affected animal patients, and limited safety or toxicity data are required. Because supplements are not regulated in the same ways as diets and drugs, it is essential to exercise caution in ensuring that the specific supplements suggested to your clients meet your individual standards for quality control, cost, palatability and convenience of dosing. Indeed, quality control and manufacturing standards are determined by the company, and certificates of analysis are often not available. Numerous studies have shown that many products did not meet their label claims (for more information, please refer to Consumer Laboratory, http://www.consumerlab.com/).[79–82]

It is estimated that 10–45% of dogs and cats in the USA[83] are fed a pet supplement or nutraceutical, with mobility supplements for hips and joints being the most frequently purchased. There is evidence of effective feline OA management with omega-3 based supplements, mainly marine oil and green-lipped mussels that contain high levels of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).[50,84–87] Omega-3 long-chain polyunsaturated fatty acids, such as EPA and DHA, can have a natural anti-inflammatory action, which may help relieve discomfort associated with OA. These molecules produce less potent inflammatory mediators when metabolized by the arachidonic acid cascade enzymes and stimulate specialized pro-resolving mediators, thereby promoting the animal's endogenous resolution of the inflammatory response.[88–90]

Collagen-derived dietary supplements (either undenatured collagen type II or hydrolyzed), cannabidiol, and plant-based and other composite nutraceuticals have been found to aid in OA management in dogs; however, in their systematic review and meta-analysis, Barbeau-Grégoire et al[86] concluded that a clear determination of their efficacy was impossible because of a lack of strong evidence, in contrast to EPA and DHA.

Nevertheless, existing results on the efficacy of nutraceuticals in cats are extremely limited, as only seven studies have been conducted on OA in cats (Table 2).[50,62,84,85,87,91,92] Two of these studies tested the efficacy of glucosamine-chondroitin sulfate using owner[62] and veterinary assessments,[62,91] as well as owner subjective questionnaires, CSOM, FMPI and general quality of life (QoL), and objective actimetry,[91] without showing any treatment effect. This is in accordance with results found in previous reviews in dogs, thus glucosamine-chondroitin should not be prescribed for feline OA management.[86] One composite supplement, based on eggshell membrane (hydrolyzed collagen), was tested in a non-randomized, non-blinded, non-controlled, prospective study using non-validated measurement tools, and the results were inconclusive.[92]

The other four studies evaluated various nutraceuticals centered on omega-3s.[50,84,85,87] Of the four prospective, negatively controlled studies, three used questionnaires completed by owners[85] without reaching intergroup statistical difference, either in the partially validated CSOM, QoL[84] or non-validated questionnaires completed by owners.[87] Recently, the GREPAQ tested the efficacy and safety of an experimental therapeutic diet, enriched in omega-3s EPA and DHA, turmeric extract and hydrolyzed collagen, compared with a control diet. This study showed intergroup differences with the MI-CAT(V), as well as in three objective and validated outcomes (hindlimb PVF, Stairs and NAM).[50] Indeed, the MI-CAT(V) indicated intergroup differences between week 8 and week 16, while hindlimb PVF and Stairs (total number of stairs climbed up and down) reached intergroup differences only at week 16, with no side effects.[50] The GREPAQ study[50] showed similar results to those previously described by Lascelles et al[84] in the objective actimetry outcome, with activity counts increasing in the test diet and decreasing in the placebo diet, along with intergroup differences.

Table 2 Literature search: non-pharmaceutical osteoarthritis management options

Online database search: PubMed, Google Scholar	Script (keywords)	Articles recorded (n)	References
Food restriction and weight loss	(cat OR feline) AND osteoarthriti* AND (weight OR diet OR management OR reduction OR restriction)	0	0
Therapeutic diets and nutraceuticals	(cat OR feline) AND osteoarthriti* AND (therapeutic diet OR diet therapy OR nutraceutical OR supplement)	7	50, 62, 84, 85, 87, 91, 92
Adjunctive musculoskeletal treatment with biologics			
Mesenchymal stem cells	(cat OR feline) AND osteoarthriti* AND (mesenchymal stem cells OR MSC OR stromal vascular fraction OR SVF)	0	0
Platelet-rich plasma	(cat OR feline) AND osteoarthriti* AND (PRP OR platelet-rich plasma OR plasma rich in growth factors OR PRGF OR platelet-derived growth factor OR platelet-derived OR platelet gel OR platelet concentrate OR PRF OR platelet-rich fibrin OR ACP OR autologous conditioned plasma OR APS OR autologous protein solution OR platelet lysate OR platelet supernatant)	0	0
Physiotherapeutic modalities			
Photobiomodulation therapy	(cat OR feline) AND osteoarthriti* AND (photobiomodulation OR light therapy)	0	0
Extracorporeal shockwave treatment	(cat OR feline) AND osteoarthriti* AND (extracorporeal shockwave treatment OR ECSWT)	0	0
Nuclear magnetic resonance therapy	(cat OR feline) AND osteoarthriti* AND (nuclear magnetic resonance therapy OR NMRT OR MBST)	0	0
Transcutaneous electrical nerve stimulation	(cat OR feline) AND osteoarthriti* AND (transcutaneous electrical nerve stimulation OR TENS)	0	0
Ultrasound therapy	(cat OR feline) AND osteoarthriti* AND (ultrasound therapy)	0	0
Acupuncture	(cat OR feline) AND osteoarthriti* AND (acupuncture OR gold therapy OR gold implantation OR gold wire implants)	0	0
Hydrotherapy	(cat OR feline) AND osteoarthriti* AND hydrotherapy	0	0
Aquatic exercise	(cat OR feline) AND osteoarthriti* AND (aquatic exercise OR swimming)	0	0
Manual therapy and exercise at home	(cat OR feline) AND osteoarthriti* AND (manual therapy OR manipulations OR mobilizations OR stretching OR massage OR physiotherapy OR physical therapy)	0	0
Lifestyle			
Environmental modification	(cat OR feline) AND osteoarthriti* AND (environment OR lifestyle)	0	0
Homeopathy	(cat OR feline) AND osteoarthriti* AND homeopathic	0	0
Aromatherapy	(cat OR feline) AND osteoarthriti* AND (aroma OR olfac* OR pheromone)	0	0
Sound therapy	(cat OR feline) AND osteoarthriti* AND (sound OR noise OR music)	0	0

Obesity is known to contribute to the pathology of many diseases and has been described as a major risk factor for OA in both humans and dogs.[15] Indeed, adipose tissue secretes proinflammatory cytokines, and excess weight can increase the mechanical stress imposed on joints.[93] Thus, weight control through caloric restriction and exercise has been recognized as a key component in preventing or slowing down the progression of OA. The benefits of exercise are well established in human medicine, including improving mobility, maintaining lean muscle mass, preventing sarcopenia and reducing and controlling pain. In animals, certain studies have demonstrated that moderate exercise improved OA conditions in rat experimental models,[94–96] as well as in dogs with naturally occurring OA.[97] However, it is important to bear in mind that there is no established correlation between obesity and the prevalence of feline OA,[10,98–100] and no prospective studies on the effect of exercise and weight loss on OA have yet been conducted in cats. Scarlett and Donoghue[98] published a paper in 1998 in which data from a population of 1457 cats in the northeastern USA were prospectively studied for possible associations between body condition and specific illnesses, including lameness (presumably related to OA and soft tissue injuries). They found that compared with optimally weighted cats, obese cats were 4.9 times as likely to develop lameness requiring veterinary care and suggested that weight loss could decrease these risks.[98] This finding was confirmed 20 years later with an Australian study including 2609 cats.[99] However, a recent prospective longitudinal study on 64 cats showed no influence of weight on severe feline radiographic stifle OA, with underweight, normal-weight and overweight cats having a similar frequency of OA diagnosis.[10] Nevertheless, preventing obesity remains a relevant factor in maintaining health and longevity.[100]

Physical therapy is currently performed in veterinary medicine as an alternative and complementary form of treatment and rehabilitation for diseases or injuries. Physiotherapy often involves manual joint manipulation and mobilization, massages, stretches and therapeutic exercises, such as hydrotherapy, as well as thermotherapy and cryotherapy to improve musculoskeletal health. Other physiotherapeutic modalities include the use of instruments such as acupuncture,[101] therapeutic ultrasound,[102] electrical stimulation (neuromuscular electrical stimulation, transcutaneous electrical stimulation, peripheral electrical nerve stimulation), pulsed electromagnetic field therapy,[103–105] photobiomodulation[106–109] and extracorporeal shockwave therapy.[110–112] The evidence supporting the effectiveness of physiotherapy and other modalities in alleviating feline OA is almost non-existent, making their recommendation primarily based on extrapolations from human medicine and other species.

Other regenerative interventions, such as biologic adjunctive musculoskeletal treatments (AMTs) using mesenchymal stem cells or platelet-rich plasma (PRP), have been proposed to help reduce the progression of OA in the joint. Biologic AMTs have shown positive results in canine OA, demonstrating long-term effectiveness (90–180 days) in both clinical/subjective and objective outcomes, although with a reduced effect size (range 0.2–0.5) and safety concerns primarily related to the preparation and injection process.[113,114] In cats, mesenchymal stem cells have been primarily studied for the treatment of CKD and chronic gingivostomatitis. However, they show promise for use in other chronic inflammatory diseases, with unfortunately no mention of feline OA or any other musculoskeletal disease.[115] As for PRP, no studies have been conducted on feline OA. However, a multispecies systematic review indicated clinical benefits and disease-modifying effects of PRP injections (one or multiple) in the treatment of OA in 1251 animals, including rodents, rabbits, dogs, goats and horses.[113]

Bergh et al[116] conducted a systematic review of various complementary and alternative therapies in veterinary medicine and found four studies discussing feline OA, and six discussing canine OA and its treatment with gold therapy (four studies) and homeopathy (two studies). Nevertheless, no other therapies for feline OA were mentioned.[116]

Discussion

As mentioned before, the GREPAQ was involved in testing the effectiveness of different pharmacological and non-pharmacological treatments. This section of the review aims to compare the efficacy of meloxicam, firocoxib, tramadol, gabapentin and the tested experimental therapeutic diet (enriched in omega-3s EPA and DHA, turmeric extract and hydrolyzed collagen) through their treatment and placebo responder rates (Table 3). To ensure valid and appropriate validity and comparability, all of the presented GREPAQ data used in the following analysis have been published in peer-reviewed journals and follow the same methodologies and assessment tools.[22,27,45–50]

First, the experimental therapeutic diet[50] induced a higher rate of responders on PGA than meloxicam[27] for a similar placebo rate (Table 3). Tramadol[45,46] and firocoxib[48,49] could not be compared for PGA because the sample tested was too small,[45,46] and the PGA methodology (Effort Path) used was different.[48,49] Second, the experimental therapeutic diet[50] induced a higher rate of responders on Stairs and MI-CAT(V) than firocoxib[48,49] (Table 3). Cats that received the enriched diet revealed a 26% decrease in MI-CAT(V) scores[50] after 8 weeks of administration, similar to the 28% reduction obtained with firocoxib treatment.[48,49] Third, the experimental therapeutic diet[50] induced a higher rate of responders on NAM than meloxicam,[27,46,47] tramadol[45,46] and gabapentin,[22] but not when compared with firocoxib[48,49]

(Table 3). This experimental diet[50] yielded a maximal NAM activity increase of 31% compared with baseline, 1 week after diet withdrawal (done at week 12), which is a superior improvement compared with data available on the previously mentioned pharmacological treatments. Furthermore, the supplemented cohort (experimental) showed an overall 67% responder rate compared with 23% in the placebo group and maintained the intergroup differences over the final 2 weeks assessed during a 4-week recovery period following withdrawal of the experimental diet.[50]

The placebo effect for PGA remained similar across studies, in the range of 15–18%,[27,45,46,50] except for the firocoxib studies, which revealed a placebo effect of 25–50%, depending on the outcome.[48,49] This improvement in the placebo group might be explained by the known beneficial effect of exercise.[27] As mentioned before, the PGA methodology used in the firocoxib studies (ie, Effort Path) was different and involved running and jumping.[48,49] Previous studies have demonstrated that moderate exercise improved OA conditions in rat experimental models[94–96] and in canine natural models.[97] Thus, it is possible that the repeated physical activity involved in PGA–Effort Path completion contributed to improve outcomes, which then translated to a higher responder rate in the placebo group.[48,49] The placebo effect for Stairs was slightly higher in the experimental diet study[50] compared with the firocoxib study[48,49] (Table 3). As for NAM, the placebo effect remained in the range of 9–23% across studies.[27,45–50] Finally, MI-CAT(V) maintained its placebo effect, with 14% of cats responding to the placebo.[48–50] Such a consistently low placebo effect and responsiveness to different analgesics, in validated objective (PGA, Stairs and NAM) and subjective (MI-CAT(V)) outcomes, highlight the quality of the results observed with the tested experimental therapeutic diet, both in terms of the maximal intensity of response and the responder rate comparison to concurrent analgesics (Table 3).

What can be deduced from this secondary analysis is that this experimental therapeutic diet can potentially serve as an alternative therapy to the current standard treatment, without side effects, and is associated with high compliance to administration.[50] When administered at efficient dosages with good-quality products, it takes approximately 8 weeks for the nutraceuticals to induce a noticeable difference compared with the placebo. The residual efficacy noted during the 4-week recovery period after withdrawal of the experimental diet was explained by a persistent increase in plasma concentration of specialized pro-resolving mediators (GREPAQ confidential data, 2025). This illustrates that the experimental therapeutic diet compares favorably with current analgesic treatments available on the market in terms of the complete benefits present in both moderate or severe OA.[50] Indeed, MI-CAT(V) clusterization correlated with

Table 3 Secondary analysis of GREPAQ published data (peer-reviewed) in feline osteoarthritis management

Study		Guillot et al[27], Monteiro et al[46], Klinck et al[47]		Delsart et al[48,49]		Klinck et al[22]		Monteiro et al[45]		Lefort-Holguin et al[50]	
Treatment		Meloxicam on average		Firocoxib		Gabapentin		Tramadol		Experimental diet	
Resp/Plac (%)		Resp	Plac	Resp	Plac	Resp	Plac	Resp	Plac	Resp	Plac
Outcome	PGA*	58% (21/36)	18% (2/11)	17–67% (4–16/24)†	25–50% (2–4/8)†	NE	NE	100% (7/7)	17% (1/6)	64% (9/14)	15% (2/13)
	Stairs‡	NE	NE	60% (9/15)	17% (1/6)	NE	NE	NE	NE	87% (13/15)	31% (4/13)
	NAM§	55% (50/91)	15% (10/65)	78% (18/23)	14% (1/7)	57% (4/7)	NE	64% (9/14)	21% (3/14)	67% (10/15)	23% (3/13)
	MI-CAT(V)¶	NE	NE	65% (15/23)	14% (1/7)	NE	NE	NE	NE	80% (12/15)	14% (2/14)

*PGA responder = increase in PVF vs BSL
†Due to the differing methodology used for PGA (Effort Path for firocoxib study), the response rate was not comparable
‡Stairs assay compliance (number of stairs ascended and descended) responder = increase in number vs BSL
§NAM (overall assessment) responder = positive or null slope after linear modelization
¶MI-CAT(V) responder = decrease in MI-CAT(V) by >15% vs BSL
BSL = baseline; MI-CAT(V) = Montreal Instrument for Cat Arthritis Testing, for Veterinarians; NAM = nighttime actimetry monitoring; NE = non-evaluated; PGA = podobarometric gait analysis; Plac = placebo responder rate; PVF = peak vertical force; Resp = treatment responder rate

different degrees of alteration in severity for NAM (mild > moderate, severe) and PGA or Stairs (mild, moderate > severe).[48–50] Severe and moderate OA clusters presented the same responsiveness to the experimental therapeutic diet on all outcomes, except for NAM, where the moderate cluster improved 1 week earlier than the severe cluster.[50] This indicates that a severely afflicted OA cat would benefit similarly to a moderately afflicted OA cat from such a therapeutic diet. This evidence has the potential to change the current paradigm of feline OA management. Further research is needed to validate this hypothesis and compare different therapeutic approaches in head-to-head randomized, blinded and controlled studies.

What if none of the treatments seems to be working?

In certain severe OA cases, pharmacological and non-pharmacological approaches may be ineffective in providing adequate pain management to alleviate the clinical signs associated with OA. For these patients, salvage surgery may be considered. There have been reports of joint arthrodesis[117,118] and arthroscopic removal of osteochondral fragments[119] performed in cats with degenerative joint disease. However, surgical procedures such as femoral head and neck excision (FHNE) and total hip replacement (THR) are more routinely performed as they are also indicated for other conditions such as fractures, chronic luxation, metaphyseal osteopathy and slipped capital femoral epiphysis.[120,121] Unfortunately, reported outcomes after surgery have been inconsistent and often subjective in nature. Indeed, to the authors' knowledge, only one study assessed PVF and vertical impulse in 17 cats 1 year after a FHNE and found statistically significant lameness in FHNE cats compared with healthy cats.[122] Nevertheless, Rodino Tilve et al[121] conducted a long-term follow-up of 44 cats that underwent THR according to the feline hip registry (2010–2020) and described a significant postoperative improvement on the short-form FMPI (2022 refined version)[123] with an overall complication rate of 19.6%, similar to previous reports on canine THR (7–22%).[124,125] These surgical procedures, when indicated, can help feline patients regain use of a limb and increase their QoL.

Finally, when a case is reluctant to improve with the above-reported therapeutic approaches (weight control if necessary, omega-3s, frunevetmab, tramadol, NSAIDs, gabapentin and amantadine, listed in decreasing order of their efficacy:risks of side effects ratio), it is important to consider other sources of pain, such as neurological alterations (eg, disc herniation). Referring the case to a specialist (board-certified surgeon or neurologist) could be proposed to motivated clients.

Conclusions

To date, very little research is available regarding feline OA and many published papers lack rigorous methodology. Most of the proposed alternatives to alleviate feline OA pain still draw upon human clinical studies and veterinary research on other species, which poses a high risk of bias. Therefore, there is a great need for standardized feline OA assessment tools and methodologies to accurately evaluate treatment efficacy. Increased owner and veterinary awareness of OA, particularly early OA, and exploration of multimodal or combined therapies (to provide assurance about their synergism and benefits to the patient) are urgently needed.

Nevertheless, research on pharmacological and non-pharmacological therapies for feline OA suggests that a change in the OA management paradigm may be warranted. A diet enriched in omega-3s EPA and DHA, turmeric extract and hydrolyzed collagen has demonstrated therapeutic efficacy comparable to standard pharmacological treatments, offering a side-effect-free alternative with high compliance, no impact on body weight or condition score, and equally effective in cats with moderate to severe OA.[50] Bioavailability and mechanistic studies should be performed to better understand the influence of each nutraceutical on clinical efficacy for feline OA management. Future perspectives should explore the concomitant use of different therapeutic approaches, as they could potentially be superior to a single treatment through an additive or synergistic effect.

The first steps are to educate owners about the signs of feline OA to improve detection and diagnosis rates, and to inform veterinarians about the OA pain processes and sensitization diagnosis. By establishing a theoretical framework of feline OA management based on strong scientific evidence, veterinarians will be better equipped to choose treatments based on the diagnosed (or suspected) OA pain manifestations and mechanisms, allowing for a tailored approach in improving our feline patients' health and well-being.

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Appendix A: MI-CAT(V) scoring sheet.

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