



PAIN MANAGEMENT AND SELF-MEDICATION BEHAVIOUR IN NON-HUMAN ANIMALS: A COMPARATIVE REVIEW

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ABSTRACT

Pain is a vertebrate universal, yet mechanisms of pain management in field conditions remain poorly understood. While laboratory research emphasizes opioid analgesia, wild animals routinely encounter pharmacological compounds with analgesic properties. This review examines pain management behaviours across animal taxa, explores self-medication patterns with naturally available analgesics, considers evolutionary trade-offs between pain suppression and injury awareness, and discusses how anthropogenic analgesics alter pain mediated or environmentally imposed ecological costs: masking injury related behavioural cues essential for healing, social cohesion, and predator avoidance. Understanding these mechanisms is critical for predicting wildlife population responses to pharmaceutical contamination of food chains and water systems.

Keywords: Self-medication, Pain behaviour, Wildlife pharmacology, NSAIDs, Behavioural ecology.

INTRODUCTION

Pain is a constraint, not a failure. A Zebra with a torn ligament that does not feel pain will maintain full running speed, converting a treatable injury into a fatal one. A songbird with wing damage will attempt flight it cannot complete. Evolution maintains pain despite its obvious costs because pain stops animals from compounding injuries. Yet animals often face competing demands: an injury exists, but so does the need to feed, reproduce, or escape threats. This creates pressure for pain management. Wild animals respond by reducing activity, favouring injured limbs, spending more time grooming affected areas. These pain-avoidance behaviours are costly they increase vulnerability to predators and starvation. When these costs mount sufficiently, some animals appear to seek pain relief. The question is not whether animals manage pain, but how and at what cost.

Self-Medication: Documented Cases & Mechanisms

Self-medication in wild animals is not anecdotal. Chimpanzees and gorillas selectively consume plants with antimicrobial and anti-inflammatory properties (bitter leaf

plants, figs with parasiticides). Injured individuals show targeted preference for these plants over normal dietary items (Huffman, 2001). Bears consume willow bark, which contains salicylates (the active precursor to aspirin), particularly during injury or when showing signs of arthritis. Primates (macaques, langurs) consume clay and tree resins after parasitic infection or gastrointestinal upset compounds that bind toxins and reduce inflammation. Elephants preferentially consume anti-inflammatory plants when exhibit lameness. Several bird species apply crushed ants and beetle secretions to plumage; recent research indicates this serves not only parasite control but also pain relief (Lozano, 1998). What remains unknown is the mechanism of action. Do these animals understand pharmacological effects, or do they respond to immediate sensory changes (taste, temperature, topical sensation)? The distinction matters for understanding cognition. Both explanations, however, indicate that behavioural pain management is a real strategy refined across evolutionary time.

Ecological Mismatch: Pain Relief Without Natural Constraints

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Wild animals that self-medicine face a natural dosing constraints availability is limited. A bear must locate and consume willow bark. A chimpanzee spends hours foraging for medicinal plants. The dose of active compounds is modest, intermittent, and titrated by effort expended. This constraint vanishes when analgesics become freely available through pharmaceutical contamination or human provisioned food sources. Injured animals no longer rest or limit activity they continue normal behaviour despite injury. Healing is delayed. Damage accumulates. Animals become progressively more vulnerable. Paradoxically, pain relief enables pain-inducing behaviour. Evidence from captive animals receiving chronic NSAIDs shows increased fracture risk, delayed wound healing, and altered gait patterns that overload already damaged joints. In wild populations, injured animals are typically culled by predation before long term costs become visible. In semi-captive or provisioned populations (habituated primates, urban bears, zoo animals), chronic pain suppression correlates with progressive mobility loss and premature death.

Comparative Pharmacology: Species Difference in Drug Response

Analgesic safety profiles in humans are calibrated to human physiology. Animals are not scaled-down humans. Interspecies differences in drug metabolism, clearance, and toxicity are substantial.

Paracetamol: Humans metabolize it via glucuronidation and sulfation. Many carnivores and some primates lack sufficient glucuronidase, leading to toxic accumulation. Documented cases of toxicity occur in wild canines, felids, and primates exposed to human waste or contaminated prey. The safety margin is dramatically narrower in these taxa.

NSAIDs: NSAIDs inhibit prostaglandin synthesis across all mammals, but downstream effects differ dramatically. Renal impairment appears earlier in species with naturally lower baseline glomerular filtration rates. Diclofenac is

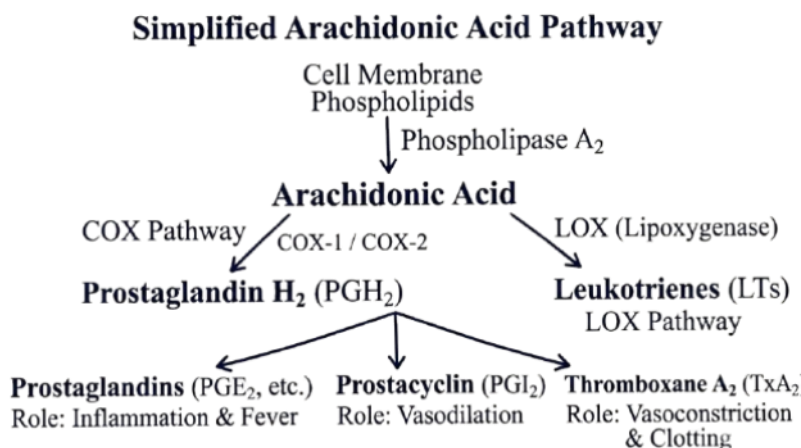
notorious for causing fatal kidney failure in vultures feeding on livestock carcasses treated with the drug; this single factor has collapsed entire vulture populations across South Asia (Green *et al.*, 2004). Many herbivores tolerate NSAIDs poorly due to differences in gastric pH and mucosal protection. NSAIDs work by inhibiting cyclooxygenase (COX) enzymes responsible for producing prostaglandins.

COX-1 inhibitors: It helps in following, supports normal platelet functioning, maintains kidney blood flow, Protects the stomach lining

COX-2 inhibitors: Developed to reduce gastrointestinal bleeding in humans, these agents still cause significant renal effects in animals, particularly species with naturally variable water availability (ungulates, arid-zone specialists). Safety data in wildlife are limited.

Metabolism of Natural NSAIDs in animals

Absorption: Natural NSAIDs such as omega-3 fatty acid, boswellic acid from *Boswellia*, curamin from turmeric, salicin from willow bark are absorbed from the gastrointestinal tract. Bioavailability varies depending on compound and animal species. Liver metabolism (Hepatic metabolism) Phase-I. In liver oxidation, reduction and hydrolysis of NSAIDs are catalysed by cytochrome P450 enzyme, and converted it into more polar metabolites. Phase-II. In phase-II glucuronidation, sulfation, methylation, glutathione conjugation reaction takes place which increases water solubility, making excretion easier. Excretion. Water soluble metabolites are excreted through urine. Some metabolites are secreted into bile and given out through sweating. Examples: Cats have limited glucuronidation capacity, making them more susceptible to toxicity from many NSAIDs and some plant derived phenolics. Dogs metabolise most of natural NSAIDs more effectively compare to cats but inappropriate dose. Cattles, sheep and goats metabolise plant NSAIDs in different ways because rumen microbes alter them before absorption (Figure 1).



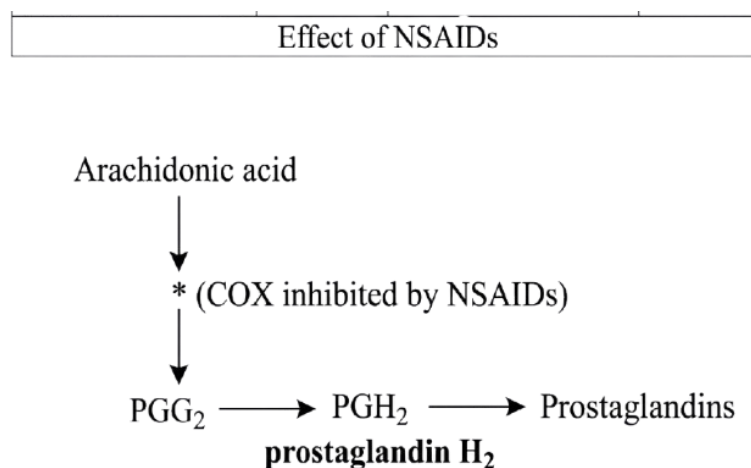


Figure 1. Simplify arachidonic acid pathway

PGH₂ is rapidly converted by tissue specific enzymes into different proteinoids.

PGE₂ provides pain, fever, inflammation, gastric protection.

PGI₂ (Prostacyclin) – Vasodilation and inhibition of platelets aggression.

Chronic Pain in Wild Populations: Prevalence and Ecological Consequences

We have almost no systematic data on pain-related morbidity in wild animals. This gap is not accidental pain is invisible unless animals display obvious behavioural signs, and such signs are easily misinterpreted. What is known: Arthritic and degenerative joint disease are common in long-lived species (elephants, whales, great apes, large predators). Age-related pain is likely endemic but undocumented. Fracture and wound pain from predation, intraspecific conflict, falls, and vehicle strikes is episodic but significant in many populations. Parasitic and infectious from intestinal parasites, skin infections, and dental disease is endemic. Chronic strain pain from repetitive movement may be underappreciated in migratory species birds undertaking intercontinental flights, cetaceans diving to extreme depths, ungulates in seasonal mass migrations. Chronic pain has ecological consequences. Affected animals show reduced vigilance, slower predator detection, diminished competitive ability, and reduced reproductive investment. In populations where pain is prevalent, these cumulative costs shape population dynamics.

Analgesic Overuse in Captive and Provisioned Animals

Animals cannot self-administer pills. Overuse occurs when caregivers provide chronic analgesics for injuries that should be managed by rest and natural healing. Several patterns emerge: Zoo and captive animals on chronic NSAIDs show higher fracture rates during aging, delayed healing of surgical sites, and progressive lameness. Prolonged pain suppression interferes with the behavioural cues (reduced weight-bearing, postural changes) that guide healing. Captive primates receiving long-term analgesia for healed injuries often develop movement constraints incompatible with wild reintroduction. Rehabilitated

raptors medicated during recovery may lack pain-avoidance behaviours necessary to prevent re-injury post-release.

Rethinking Pain management in wildlife: A framework

Current approaches to animal pain management often mirror human medicine: suppress pain to improve welfare. But animals' welfare is not identical to human comfort. A framework for responsible pain management might include: Context-specific dosing. Short-term analgesia for acute, treatable injury (fracture, laceration) differs fundamentally from chronic suppression of degenerative conditions. Behavioural monitoring. Pain suppression should not eliminate injury-appropriate behaviour (reduced activity, guarding the affected area). If an analgesic prevents an animal from self-protecting an injury, it is contraindicated. Environmental modification. Rather than medicate an animal in an unnatural environment, modify the environment. Soft substrates for arthritic animals, water-depth management for diving mammals. Population-level perspective. In wild populations, some pain-mediated mortality may be ecologically acceptable and genetically important, removing animals unable to recover from significant injury.

Pharmaceutical Contamination of Aquatic Systems

Analgesics are now quantifiable contaminants in aquatic systems globally. NSAIDs and paracetamol are consistently detected in rivers, streams, and groundwater in regions with high pharmaceutical use. Fish exposed to NSAIDs exhibit behavioural changes reduced antipredator behaviour, altered migration patterns, impaired reproduction at concentrations below acute toxicity thresholds. Aquatic invertebrates show altered development and reduced immune function. Amphibians display disrupted metamorphosis and compromised predator-avoidance behaviour. The mechanism is not straightforward. A fish does not experience subjective pain relief. Rather, its

physiological changes in ways that reduce defensive vigilance and increase vulnerability. This is not a benefit for the fish.

CONCLUSION

Pain is not a medicinal problem to solve universally. It is an ecological signal that shapes behaviour, guides healing, and maintains population fitness. Wild animals manage pain through behavioural, physiological, and biochemical strategies refined over evolutionary time. Our recent capacity to chemically suppress pain, combined with increasing pharmaceutical contamination of animal environments, far outpaces understanding of consequences. The responsible approach is not to assume pain relief is universally beneficial for wild animals. Instead, the critical questions are: What does this pain do? What happens when we remove the signal? Will the animal recover, or deteriorate faster? These questions demand collaboration across evolutionary biology, ecology, and veterinary medicine.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest

ETHICS APPROVAL

Not applicable

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AI TOOL DECLARATION

The authors declare that no AI and related tools are used to write the scientific content of this manuscript.

DATA AVAILABILITY

Data will be available on request

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