

Review

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Posted Date: 24 September 2025

doi: 10.20944/preprints202509.1969.v1

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Review

Repurposing Monensin for Inflammation and Immunity: A Comprehensive Review

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Abstract

Monensin, a polyether ionophore antibiotic used extensively in veterinary medicine, has recently generated interest for its possible anti-inflammatory effects. Monensin has commonly been referred to as a coccidiostat and growth promoter used in livestock; however, monensin has significant effects on cellular ion homeostasis and intracellular signaling pathways, which may lead to clinically relevant immunomodulatory effects. Here, we will summarize the current knowledge regarding the anti-inflammatory mechanisms of monensin, including both in vitro and in vivo studies. Evidence indicates that monensin can inhibit nuclear factor kappa B (NF-κB) activation, inhibit the phosphorylation of mitogen-activated protein kinases (MAPK), and inhibit TLR-mediated signaling, which is important for the reduction in pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6 expression. In animal models, monensin has been reported to improve survival during endotoxemia, improve microglial activation during neuroinflammatory conditions, or attenuate gut injury in models of colitis or arthritis. Monensin has also modulated tumor-associated inflammation, suggesting broader use of the drug in the field of oncology. Although these preliminary findings are encouraging, the limitations of safety, poor pharmacokinetics, and the limited use in veterinary medicine have restricted its translation to human medicine. Advances in delivery systems, structural analogues, and repurposing options have the potential to establish its therapeutic applicability. In summary, monensin presents as a promising albeit understudied candidate for anti-inflammatory therapy. There is a clearly defined need for further mechanistic studies, toxicological assessments, and translational research studies

Keywords: ionophore antibiotics; NF-κB signaling; MAPK pathway; TLR4; sepsis and endotoxemia; cancer-associated inflammation

1. Introduction

Inflammation is a key immune response to damaging noxious stimuli, including infections, toxins, and tissue injury [1]. Acute inflammation serves as an important part of host defense and tissue repair, but its continued presence in a chronic state leads to many debilitating diseases, including rheumatoid arthritis, cardiovascular diseases, degenerative diseases of the nervous system, and cancer [2,3]. Conventional anti-inflammatory therapies comprising nonsteroidal anti-inflammatory drugs (e.g., aspirin or ibuprofen) and corticosteroids are limited by safety issues, diminished effectiveness in the long term, and a lack of specificity for molecular pathways of inflammation [4,5]. There is a pressing need for new therapeutic agents with different mechanisms of action and improved safety profiles.

Monensin, a polyether ionophore isolated from *Streptomyces cinnamonensis*, has traditionally been used in veterinary medicine as a coccidiostat and growth promoter. The primary mechanism of monensin relies on its recognition for the selective transport of monovalent cations, especially sodium

and potassium, across biological membranes [6,7]. By changing the landscape of ionic homeostasis, systemic perturbations occur. In recent years, monensin has received more research attention focusing on not only its antimicrobial activities, but also the immunomodulatory and anti-inflammatory properties of this compound, with particular attention to injury in vivo or inflammatory disease. Experimental studies have shown that monensin has anti-inflammatory effects through reduced calcium influx in cells, inhibition of important signaling pathways, including NF- κ B and MAPK, and inhibition of the release and expression of pro-inflammatory cytokines like TNF- α , IL-1 β , and IL-6 [8–11].

These findings are promising; however, systematic and complete assessments of the anti-inflammatory activities of monensin have not occurred, with most studies being preliminary or limited in scope (i.e., not comprehensive). A unified understanding of the molecular mechanisms and preclinical supporting evidence for anti-inflammatory activity may lead to repurposing for specific therapeutic applications in inflammatory disease.

The goal of this review is to provide detailed and cohesive information on the anti-inflammatory effects of monensin, including cellular and molecular mechanisms, experimental evidence from models, and future clinical applications.

2. Main Body

2.1. Chemical and Pharmacological Profile of Monensin

Monensin is a polyether ionophore antibiotic first discovered in the late 1960s, emerging from the soil bacterium *Streptomyces cinnamomensis*. Monensin is a member of a family of natural polyethers that can be characterized by their highly oxygenated backbone and their ability to make a hydrophobic complex with a monovalent cation - specifically Na⁺, and K⁺, and to a lesser degree Li⁺ [12,13]. Monensin uses this characteristic to act as a mobile carrier for ions, and this results in the collapse of an ionic gradient (ionic gradients are essential for microbial survival) across lipid bilayers. This type of action explains why monensin has such effective antimicrobial and anticoccidial activity [14].

From a pharmacokinetic standpoint, monensin has been one of the most widely used drugs in veterinary medicine. Historically, it has been used as a feed additive in livestock (cattle and poultry), primarily to prevent coccidiosis and improve feed efficiency [15]. Through its ability to alter the fermentation pattern in the rumen, monensin has been shown to improve propionate production and decrease methane production, thereby improving energy metabolism in ruminants [16]. The energy metabolism effects when monensin is used as a feed additive, although beneficial from an animal husbandry perspective, are intimately linked to its therapeutic action, which is ionophoric in nature [17].

Interestingly, and beyond its noted antimicrobial and metabolic regulatory activities, monensin has additional biological effects at the cellular level. Monensin alters the sodium and calcium balance inside cells, which then influences vesicular trafficking, lysosomal pH, and apoptotic signaling [18]. These pleiotropic effects have generated interest lately, especially in cancer and immune system research, as they may be contributing to the current potential pharmacotherapy applications for monensin as an anti-inflammatory and anticancer agent.

2.2. Molecular Basis of Inflammation: A Brief Overview

Inflammation is a process of the host that is adjustable in nature for the purpose of defense against infection and injury. Inflammatory response consists of a series of molecular signals, which begin from pattern recognition receptors (PRRs), such as Toll-like receptors (TLRs) and NOD-like receptors (NLRs). Interactions of receptors with agonists lead to the activation of intracellular signaling pathways, eventually merging onto transcription factors, mostly NF- κ B and activator protein-1 (AP-1) [19,20].

The NF- κ B pathway is the main mechanism of inflammatory signal transduction. In the absence of stimulation, NF- κ B dimer localizes to the cytoplasm as a complex with reference to inhibitory proteins such as I κ B [21]. In the case of stimulation by a potent agonist (lipopolysaccharide), for example, phosphorylation of I κ B occurs and promotes its ubiquitylation and degradation, releasing NF- κ B to the nucleus where it can induce gene transcription for pro-inflammatory markers, such as TNF- α , IL-1 β , and IL-6 [22]. Similarly, the mitogen-activated protein kinase (MAPK) family (Erk1/2, Jnk, and p38) can regulate cytokine expression, along with cell survival under inflammatory conditions [23].

The other pathways include Janus kinase/signal transducer and activator of transcription (JAK/STAT), interferon regulatory factors (IRFs), and NLRP3 inflammasome activation, which are also key players. These systems coordinate acute and chronic inflammatory responses. The dysregulation of these signaling networks may lead to a long list of diseases that include autoimmune disease, cardiovascular disease, neurodegeneration, and, conversely, cancer. To assess the therapeutic potential of monensin, it is necessary to understand the relationship with these pathways.

2.3. Anti-Inflammatory Mechanisms of Monensin (In Vitro Evidence)

2.3.1. Modulation of NF- κ B Signaling

NF- κ B is widely accepted as the master regulator of an inflammatory response. Like the more selective NSAIDs (Celecoxib, Ibuprofen), monensin shows very robust suppressive actions against the initiation of inflammatory responses mediated by monocytes and macrophages. Multiple studies show that monensin inhibits (or suppresses) NF- κ B activation caused by endotoxins [11]. Activated dendritic cells treated with LPS and monensin failed to translocate the nuclear p65 subunit of NF- κ B and suppressed transcriptional upregulation of TNF- α and IL-6. Overall, by blocking NF- κ B signaling, monensin suppresses the initiation of the inflammatory cascade at the early stage of the signaling response to a nucleated cell, whereas classical NSAIDs inhibit only at the cyclooxygenase enzymes [24].

2.3.2. Inhibition of MAPK Pathways

Monensin inhibits the MAPK signaling pathway as well. In macrophage models exposed to bacterial products, monensin inhibited phosphorylation of ERK1/2 and p38 MAPK, resulting in decreased expression of pro-inflammatory cytokines and chemokines. This shows evidence of dual effects as it inhibited not only acute inflammation, but transcriptional programs related to chronic inflammation [8].

2.3.3. Regulation of TLR Signaling and IRF3 Activity

TLR4 is an essential Bacterial LPS receptor and is a primary driver for sepsis-related inflammation [25]. Observations in cancer cell models have demonstrated that monensin reduces TLR4 and TLR7 expression, as well as IRF3 activation [26]. Since the TLR4/IRF3 axis regulates not just interferon production but the entire epidemic of inflammatory cytokine release, monensin's downregulation of this pathway can be an advantage if TLR signaling is hyperactivated (i.e., colitis and cancer-associated inflammation).

2.3.4. Effects on Ion Homeostasis and Vesicular Trafficking

Monensin, an ionophore, affects intracellular concentrations of Na⁺ and Ca²⁺ [27]. An increase in Na⁺ influx increases intracellular Ca²⁺ levels; elevated Ca²⁺ may alter calcineurin and NFAT-dependent signaling pathways [28]. It is important to note that monensin also alkalinizes endosomes and lysosomes [29]. Monensin interferes with proton gradients, thereby alkalinizing the endosomes and lysosomes. In macrophages, this activity prevents inflammasome activation and reduces

secretion of IL-1 β [30]. Collectively, these results suggest this special ionophoric activity is directly involved with its immunomodulatory effects.

2.4. Preclinical Evidence of Anti-Inflammatory Effects (In Vivo Studies)

2.4.1. LPS-Induced Endotoxemia Models

Studies done on animals using LPS-induced endotoxemia have demonstrated that the administration of monensin reduces levels of systemic cytokines and enhances survival. Treated animals had lower TNF- α and IL-6 serum concentrations with lessening of histopathological damage in the liver and spleen. Thus, monensin may counteract cytokine storm-like events [31,32].

2.4.2. Neuroinflammatory Models

In studies using rodent models of neuroinflammation, monensin decreased microglial activation and reduced IL-1 β release with increased neuronal survival. These results imply potential therapeutic uses for monensin in neurodegenerative diseases that feature pathological microglial overactivation [33,34].

2.4.3. Models of Chronic Inflammatory Diseases

In preclinical models of colitis, monensin treatment exhibited effects that ameliorated structural damage to colonic mucosa, levels of inflammatory cell infiltration, and per gene brand/cytokine expression in all tissue samples [35]. In preclinical models of arthritis, monensin demonstrated reductions in joint swelling and reduced cartilage erosion. This suggests monensin may have potential as a treatment for autoimmune and rheumatologic disorders [36].

2.4.4. Cancer-Associated Inflammation

Monensin inhibited tumor progression in xenograft models, partially by inhibiting TLR4-mediated inflammation in the tumor microenvironment. Therefore, monensin may exert both direct anticancer activities and modulate tumor-promoting inflammation, at least in part, by preventing or inhibiting inflammatory signaling [26,37].

2.5. Comparative Insights: Monensin Versus Conventional Anti-Inflammatory Drugs

Monensin is able to influence inflammation via fundamentally different mechanisms compared to its more established predecessors, NSAIDs and corticosteroids. Instead of inhibiting COX enzymes (NSAIDs) or suppressing transcription of inflammatory genes (corticosteroids), monensin acts upstream from DA and inflammation by disrupting ionic homeostasis and multiple signalling pathways, including NF- κ B, MAPK, and TLR signalling [8,11,26].

This poly-target profile could provide it with advantages as a treatment for diseases that are underlain by redundant inflammatory pathways. However, monensin's safety profile, largely related to cardiotoxicity and hepatotoxicity at high doses, is correspondingly narrower than that of traditional agents. Moreover, unlike NSAIDs and corticosteroids, it has never been approved for human consumption and is therefore not immediately clinically translatable. This contrast highlights the potential and challenges of repurposing monensin as a treatment for inflammation [24].

2.6. Clinical Perspectives and Drug Repurposing Potential

The repurposing of veterinary drugs for human diseases appears to have momentum, as illustrated by the repurposing of ivermectin and doxycycline for novel therapeutics.

Monensin, a vet drug with demonstrated broad anti-inflammatory activity, is a candidate for repurposing. However, there are several significant hurdles:

- Toxicity and safety: Monensin has dose-dependent toxicity associated with use in mammals, and after accidental overdose in livestock, has been associated with cardiac and hepatic adverse effects [38,39].

- Pharmacokinetics: Poor solubility and bioavailability limit the opportunity for systemic use.
- Regulatory issues: Monensin is currently licensed only for use in veterinary species, and there are no clinical trials in humans.

To overcome these issues, developing strategies to nanoformulate (liposomes, polymeric nanoparticles, etc.) or chemically modify to produce monensin analogues, as well as creating combination therapy with existing immunomodulators to be able to use a lower, safer dose of monensin, are being considered. Future clinical perspectives will be contingent upon overcoming these pharmacological and safety issues.

2.7. Future Directions in Research

Despite the promising preclinical results, there are several important areas we are still lacking in when it comes to our understanding of monensin as an anti-inflammatory drug. First, we need systematic toxicology and pharmacokinetics assessments in mammalian models. Second, we need comparative data against commonly utilized anti-inflammatory medications. Finally, we could continue to increase the likelihood of a successful clinical pathway by figuring out how to enhance its structure to produce safer derivatives.

In addition, we should consider the possible role of monensin in the therapeutic treatment of specific diseases, such as neurodegenerative disorders, autoimmunity, and inflammation associated with cancer. With the strides being made in drug delivery systems and medicinal chemistry, monensin could one day serve as a lead compound for the development of new anti-inflammatory therapeutics.

3. Conclusion

Historically used as a veterinary antibiotic and coccidiostat, monensin is drawing interest as a compound with anti-inflammatory effects. Although its anti-inflammatory actions are multifaceted (e.g., alterations to NF- κ B and MAPK pathways, TLR/IRF3 stimulation, and disruption of ion homeostasis), monensin is shown to inhibit production of the pro-inflammatory cytokines in vitro and lower inflammation in a variety of animal models. Clinical use of monensin remains limited due to toxicity and regulatory issues. Unfortunately, these translate to obstacles for repurposing monensin in human medicine, although continued advances in drug repurposing, formulation science and structural modifications of other derivatives may provide hope. Overall, monensin represents an exciting, yet largely unexplored option for the next generation of anti-inflammatory drugs.

Acknowledgments: The author has no acknowledgments to declare.

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