# Non-steroidal anti-inflammatory drug's potential for ecotoxicity

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**ABSTRACT**

Numerous Cu (II) complexes of NSAIDs with improved anti-inflammatory activity and decreased Gastrointestinal (GI) toxicity have been developed as a result of the suggested curative qualities of Cu-based Non-Steroidal Anti-Inflammatory Medicines (NSAIDs). Since many of today's anti-inflammatory medication regimens, especially those based on NSAIDs, remain either woefully ineffective or linked to significant renal, GI, and cardiovascular side effects, it is important that these low toxicity Cu medicines eventually find their way to a wider human market. However, it is still unclear where Cu-NSAIDs' anti-inflammatory and gastric-sparing effects come from. Apart from their commonly reported Superoxide Dismutase (SOD)-like activity in vitro, their potential to affect copper metabolism has been a subject of contention. Relatively little is known, however, regarding how they eventually control the inflammatory process and/or immune system.

**Key Words:** *Anti-Inflammatory Drugs, Superoxide Dismutase*

## INTRODUCTION

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) are some of the most often prescribed medications in the world, mainly for the treatment of osteoarthritis and other chronic musculoskeletal disorders' symptoms. Additionally, NSAIDs are the standard colon cancer chemo preventive drugs and cut colon cancer risk and mortality by roughly half. The substantial toxicity of NSAIDs, however, restricts their use. A wide range of adverse events related to NSAID use have been documented, including renal side effects, gastrointestinal side effects (including dyspepsia, gastrointestinal bleeding, and even perforation), and a few other side effects (including hypersensitivity reactions and distinct salicylate intoxication).

It has traditionally been necessary to provide gastro protective medications with NSAIDs, such as H2 receptor antagonists, proton pump inhibitors, or prostaglandin analogues (most commonly misoprostol), although COX-2 NSAIDs alone are now an alternative. Some older NSAIDs have significant COX-2 activity (COX-2 selective, which include etodolac, meloxicam, nabumetone, and nimesulide). For their COX-2 activity, more recent NSAIDs (COX-2 specifically include celecoxib and rofecoxib) have been developed. NICE guidelines state that COX-2 NSAIDs and non-selective (COX-1 or conventional) NSAIDs without gastro protection are equal in lowering pain and increasing physical functioning in persons with arthritis, and that COX-2 NSAIDs are linked with fewer gastrointestinal problems. Lister-3 and Lister-4 discovered that a number of NSAIDs had equivalent efficacy at equivalent dosages. Crohn's Disease (CD) and ulcerative colitis are the two most common immune-mediated chronic or recurrent GI tract disorders known as Inflammatory Bowel Diseases (IBD). IBD is defined by a chronic intestinal inflammatory process, with environmental variables (such smoking or NSAIDS), genetics, host intestinal flora, and the host immune system all playing a role in the disease's development. Steroidal Anti-Inflammatory Drugs Among the most popular treatments for a variety of inflammatory disorders are drugs. Approximately 60 million Americans frequently take NSAIDS. Concern over the development of gastrointestinal toxicity, such as mucosal injury in the form of erosions and ulcers, upper GI, small bowel or colonic haemorrhage, and infrequently perforation and obstruction due to stricture formation, is the principal barrier to the use of NSAIDS. Additionally, non-specific colitis and small intestine inflammation caused by NSAIDS may result in recurring bleeding or protein loss. Sharply defined or circumferential ulcers, which are typically curable following drug termination, are endoscopic characteristics of NSAIDS-induced colonic injury. Over 100,000 hospital admissions for GI complications are attributed to NSAIDS use by arthritis patients in the US each year, with lower GI symptoms accounting for around one-fifth of those admissions. According to a review of the literature on the negative effects of NSAIDS on the lower gastrointestinal tract, using NSAIDS in patients with lower GI bleeding, perforation, and severe diverticular illness was statistically significantly more likely to result in negative outcomes. It has been argued frequently that there may be a link between the use of NSAIDS and the development or recurrence of IBD. But it's challenging to come to firm conclusions given the absence of controlled prospective trials. There have been several proposed pathways for NSAIDS-induced GI damage in general and in IBD patients in particular. Enhanced Enterohepatic Circulation, Drug-Enterocyte Adduct Formation, NSAIDS-induced intracellular ATP shortage, and increased mucosal permeability are a few of these.

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However, their impact on prostaglandin production is the most talked-about mechanism. Prostaglandins are essential for mucosal defence, microcirculation upkeep, and immune system modulation in the colon. Intestinal ulcers and an aggravation of Dextran Sulphate Sodium (DSS)-induced colitis were produced in experimental animals utilising active vaccination against prostaglandins E2, F2A, and D2, as well as suppression of COX-1 and COX-2. The early and frequent clinical relapse of has been linked to decreased prostaglandin synthesis caused by COX-1 and COX-2 inhibition.