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| Nota di contenuto | COMPARATIVE PATHOPHYSIOLOGY AND TOXICOLOGY OF CYCLOOXYGENASES; CONTENTS; PREFACE; INTRODUCTION: DISCOVERY OF CYCLOOXYGENASES AND HISTORICAL PERSPECTIVE; Aspirin; Prostaglandins; Cyclooxygenases; COX-2 Selective NSAIDs; References; CHAPTER 1 GASTROINTESTINAL TRACT; Introduction; Comparative COX-1 and COX-2 Expression in the GI Tract; Effects of ns-NSAIDs on the GI Tract; Effects of Arylpropionic Acid ns-NSAIDs on the GI Tract; Effects of Enolic Acid (Oxicam) ns-NSAIDs on the GI Tract; Effects of Acetic Acid Derivative ns-NSAIDs on the GI Tract Effects of Aminonicotinic Acid Derivative ns-NSAIDs on the GI TractEffects of Pyrazolone Derivative ns-NSAIDs on the GI Tract; Effects of Salicylic Acid Derivative ns-NSAIDs on the GI Tract; Effects of Anthranilic Acid Derivative ns-NSAIDs on the GI Tract; Effects of COX-1 Inhibitors on the GI Tract; Effects of COX-2 s-NSAIDs on the GI Tract; Pathophysiology and Mechanisms of NSAID-Associated GI Toxicity; Role of Cyclooxygenase Potency; Species Differences in NSAID- Associated Susceptibility to GI Injury; GI Anatomical Differences; Enterohepatic Recirculation and NSAID Toxicity |

Role of Xenobiotic Glucuronidation Aging and Stress and NSAID GI Effects; Disruption of GI Physiological Mucosal Defense Mechanisms; GI Disequilibrium; Effects on Physiological GI Mucosal Cell Renewal Mechanisms After Mucosal Injury; Effects on Leukocyte Adhesion Molecules and Trafficking; Effects of GI Physiological Local pH, Gut Absorption, and Fasting; NSAID Topical Effect-Mediated Injury; Changes in GI Motility, Microcirculation, and Enterobacteria; Decreased Phosphatidylcholine Levels; Impaired Drug Metabolism; Role of Toll-like Receptor (TLR)-4/MyD88 and Enteric Bacteria
Role of Uncoupling of Mitochondrial Oxidative Phosphorylation Role of Peroxisome Proliferator-Activated Receptor α ; Role of Mitogen-Activated Protein Kinases; NSAID GI Injury-Associated Risk Factors; Conclusions; References; CHAPTER 2 BONE-TENDON-LIGAMENT SYSTEM; Introduction; Comparative Physiological and Anatomical Aspects of the Skeleton; Role of Prostaglandins in Skeleton Metabolism; The Process of Bone Healing and Potential Role of Prostaglandins; Inflammatory Response; Bone Resorption; Bone Formation
COX-1 and COX-2 Expression in Bone, Tendon, and Ligament During Repair and in Pathological Conditions Effects of ns-NSAIDs on Bone Healing; Effects of COX-2 s-NSAIDs on Bone Healing; Effects of ns-NSAIDs on Ligament and Tendon Healing; Effects of COX-2 s-NSAIDs on Ligament and Tendon Healing; Conclusions; References; CHAPTER 3 RENAL SYSTEM; Introduction; Comparative Physiological, Developmental, and Anatomical Aspects of the Renal System; Role of Prostaglandins in the Renal System; COX-1 and COX-2 Expression in the Kidney; Effects of ns-NSAIDs on the Kidney
Effects of COX-2 s-NSAIDs on the Kidney

Sommario/riassunto

The first thorough review of cyclooxygenase inhibitors, including their toxicity mechanisms and toxicopathological risks Cyclooxygenases (COXs) are enzymes responsible for the formation of an important class of biological mediators called prostanoids. Prostanoids such as prostaglandins mediate inflammatory and anaphylactic reactions. For those suffering from inflammation and pain, the pharmacological inhibition of COXs, with non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen, can provide relief. Yet the use of NSAIDs can trigger toxicological effects as well, lea
