Record Nr. Autore Titolo	UNINA9910817130503321 Radi Zaher A Comparative pathophysiology and toxicology of cyclooxygenases / / Zaher A. Radi
Pubbl/distr/stampa	Hoboken, N.J., : Wiley, c2012
ISBN	1-283-57607-4 9786613888525 1-118-35191-6
	1-118-35190-8 1-118-35188-6
Edizione	[1st ed.]
Descrizione fisica	1 online resource (338 p.)
Disciplina	615.9/5137
Soggetti	Cyclooxygenases - Pathophysiology Cyclooxygenases - Toxicology
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Note generali	Description based upon print version of record.
Nota di bibliografia	Includes bibliographical references and index.
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 Acetic Acid 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 Anthranilic Acid Derivative ns-NSAIDs on the GI Tract; Effects of Anthranilic Acid Derivative ns-NSAIDs on the GI Tract; Effects of Anthranilic Acid Derivative ns-NSAIDs on the GI Tract; Effects of Anthranilic Acid Derivative ns-NSAIDs on the GI Tract; Effects of Anthranilic Acid Derivative ns-NSAIDs on the GI Tract; Effects of Anthranilic Acid Derivative ns-NSAIDs on the GI Tract; Effects of Anthranilic 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

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	Role of Xenobiotic GlucuronidationAging 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 PhosphorylationRole of Peroxisome Proliferator-Activated Receptor g; 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 ConditionsEffects 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