

The inhibition of COX-2 is mostly responsible for the therapeutic effects of NSAIDs, while the adverse gastrointestinal (GI) effects of NSAIDs are attributable to the inhibition of both COX-1 and COX-2, which decreases prostaglandin synthesis, impairs the maintenance of GI mucosal integrity, and results in erosion, ulceration, and bleeding [3,4]. Coxibs selectively inhibit COX-2 and thus inhibit inflammation while preserving most of the homeostatic functions of COX-derived prostaglan