The inhibition of COX-2 is mostly responsible for thetherapeutic effects of NSAIDs, while the adverse gastrointestinal(GI) effects of NSAIDs are attributable to theinhibition of both COX-1 and COX-2, which decreasesprostaglandin synthesis, impairs the maintenance of GImucosal integrity, and results in erosion, ulceration, andbleeding [3,4].

Coxibs selectively inhibit COX-2 and thusinhibit inflammation while preserving most of the homeostaticfunctions of COX-derived prostaglan