HOW NSAIDS WORK

NSAIDs have been widely used in both human and animal medicine for decades and offer excellent analgesia and anti-inflammatory properties (reduction of pain, fever and swelling). The discovery by researchers in the human medical field that the target of aspirin, the oldest NSAID in current use, is an enzyme called cyclooxygenase (COX) revolutionized the understanding of these drugs. This enzyme is the rate-limiting step in the production of prostaglandins from a fatty compound called arachidonic acid, which is cleaved from the membrane walls of cells during normal bodily function and during inflammation. Prostaglandins are local hormones that result in alteration in bodily function, depending upon their location and the type of prostaglandin.

The finding of COX seemingly simplified understanding of the mechanism of action of NSAIDs, but the recent discovery of more than one form of COX (COX-1, COX-2, and COX-3) has once again highlighted the complicated nature of these drugs. For instance, while many drugs inhibit all of the forms of COX, some drugs target specific COX enzymes, and have differing effects as a result. Nonetheless, this has also provided the opportunity for pharmaceutical companies to develop medications that have a more targeted action.

In general, it has been shown that COX-1 is responsible for producing prostaglandins that regulate normal bodily functions such as protection of the gastrointestinal tract from injury, whereas COX-2 is typically not present in most tissues, including the gut and joints, unless there is inflammation. COX-3, the most recently discovered COX enzyme, appears to be involved in control of pain and fever at the level of the brain, and is the target of drugs such as Tylenol® and dipyrrone (a drug previously used in horses in the United States).