Non-steroidal anti-inflammatory drugs (NSAIDs) typically reduce aches, pains, swelling, and fever by inhibiting activity of COX-1 and COX-2. In addition to the analgesic, anti-inflammatory, and anti-pyretic effects, compounds such as Acetylsalicylic Acid (Aspirin, A0819) and Piroxicam (P3269) also exhibit chemopreventive activity, decreasing the risk of colon cancer, breast cancer, intestinal cancer, and prostate cancer. This inhibition of carcinogenesis is likely due to both COX-dependent and COX-independent mechanisms.

Inhibition of COX enzymes results in an increase in prostaglandin precursor arachidonic acid. In colon tumor cells, arachidonic acid stimulates the conversion of sphingomyelin to ceramide and induces apoptosis. Animal models of colorectal cancer support this finding, showing apoptosis and inhibition of aberrant crypt foci development in animals administered Sulindac (S8145).

A separate chemopreventive mechanism includes inhibition of PDE5. Suppression of this enzyme increases levels of cGMP and activates PKG. This results in downstream activation of JNK1 and downregulation of β-catenin-mediated transcription; as a result, cell cycle arrest and apoptosis are induced and proliferation and angiogenesis are inhibited. Both sulindac and Flurbiprofen (F4481) act through this pathway, preventing development of cancers in cellular models.

LKT Laboratories carries a wide variety of NSAIDs, including both non-selective COX inhibitors and COX-2 selective inhibitors such as Etoricoxib (E7858), Meloxicam (M1644), and Celecoxib (C1644) and many more!

References: