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The key to fighting inflammation: Herbal COX-2 inhibition

By Thomas M. Newmark

Both the Wall Street Journal and the New York Times have recently proclaimed the revolutionary promise of COX-2 enzymatic inhibition. The Wall Street Journal described the inhibition of this enzyme as a future milestone in preventative medicine. The Times reported that COX-2 inhibition reduced arthritic inflammation and menstrual pain, and appears to "prevent" cancers of the colon, esophagus, skin and bladder.

What is this important COX-2 enzyme, and how can it be safely inhibited or kept in check?

The COX-2 enzyme is naturally created in every one of us. It controls the creation of a hormone-like substance that causes "inflammation." And not just the common inflammation many experience in a knee or shoulder, but also inflammation throughout the body, even in the brain of an Alzheimer's sufferer. Scientists now have convincing evidence that the unchecked activity of this enzyme is responsible for many forms of cancer, arthritis and Alzheimer's disease.

Amazingly, this fundamental cause of many of our most threatening diseases was only recently discovered, and drug companies immediately began the race to come up with pharmaceutical substances to inhibit the enzyme's devastating effects. Research scientists around the world supported this effort to create synthetic COX-2 inhibitors, and there was a wonderful, and surely unexpected, side benefit of that research. The scientists tried to find plant compounds and herbs to synthetically copy or mimic, and they identified many traditional herbs that contain natural and safe COX-2 inhibitors. The very process to

create synthetic COX-2 pre-prescription drugs has also led to the discovery of herbal alternatives that are naturally available, without prescription.

How does this enzyme cause harm?

It is important to understand how the enzyme works to create threatening disease conditions. The full name of the COX-2 enzyme is "cyclooxygenase-2," and its name contains the first clue as to how the enzyme works. Cyclooxygenase has the job of "oxidizing," or burning, a fat in the body called "arachidonic acid." This fat is an omega-6 fatty acid, one which occurs naturally in all of our cell membranes or walls. Picture the COX-2 enzyme as a "spark plug" that ignites this fat and cooks up inflammatory substances. Simply put, the enzyme is the spark, the fat is the fuel, and the flame of inflammation is the end result.

While we need that fire to help our bodies defeat enemies like bad bacteria and respond to trauma and injury, sometimes the fire burns too hot or gets out of control. When that happens we can be casualties of "friendly fire." We can develop some forms of cancer and arthritis, and our very brain neurons can get overheated and die.

This, fortunately, is not a hopeless situation, and one of the most popular drugs in the world helps to inhibit the fires of COX-2 inflammation. That drug is none other than aspirin, which not only inhibits the COX-2 enzyme, but also reduces the stickiness of our blood platelets, thus helping to prevent strokes

and heart attacks. Unfortunately, aspirin and related "non-steroidal anti-inflammatories" can have very serious side effects, and last year alone more people died from such side effects than from AIDS.

While "safer aspirins," have been introduced in recent years, they lack traditional aspirin's protective effect against platelet stickiness.

As we explained above, and in much more detail in our book *Beyond Aspirin*, scientists discovered many traditional herbs that can safely inhibit the COX-2 enzyme. Those herbs include green tea, ginger, turmeric, holy basil, chamomile, Chinese goldthread, barberry, Baikal skullcap, hu zhang (Japanese knotweed), rosemary, hops, feverfew and oregano. A blend of the

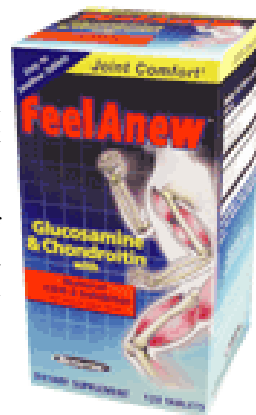
appropriate extracts of these herbs, if sufficiently concentrated, may offer substantial relief from the "fires" of COX-2 inflammation and the related diseases, without the side effects of synthetic drug inhibition. And the even better news is that when these botanicals inhibit the COX-2 enzyme, many of them also powerfully balance platelet aggregation (thus helping to prevent strokes and heart attacks) and heal ulcers. This is the genius of nature, and why cultures for thousands of years have obtained healing benefits from extracts of these botanicals.

RESEARCH & STUDIES

Nexrutine™

— Natural COX-2 Inhibitor

- ◆ Nexrutine™ was developed from a plant (*Phellodendron amurense*) used safely for more than 1,500 years in Asia.



- ◆ Nexrutine™ has a unique mechanism of action: It is a selective COX-2 inhibitor. Most herbal COX-2 inhibitors have not been evaluated for their potential to inhibit COX-1. Nexrutine™ has cell culture, animal and human data to support its efficacy and safety as well as . data that indicate that it has a characteristic that no other natural COX-2 inhibitor has, which is COX-2 selectivity.
- ◆ Inhibiting COX-1 could result in greater gastrointestinal irritation, however, Nexrutine™ does not inhibit COX-1 and is gentle on the stomach. To be most useful, the botanical must not have a pronounced inhibitory action on COX-1 activity. Nexrutine™ selectively inhibits the COX-2 enzyme without affecting the important COX-1 enzyme.
- ◆ Not only is the key constituent of Nexrutine™ absorbed, but also it has been reported to exhibit a strong moderating effect. Human data indicates that a small dosage of Nexrutine™ is needed for efficacy compared to a rather large dose for other ingredients. This equates to less pills an individual must take daily and should improve individual compliance with

Cell Culture Data:

COX-2 inhibitory activity was confirmed in human umbilical endothelial cells. Cells were stimulated with one of two agents to provoke a COX-2 response and then Nexrutine™ was added to the cell culture. COX-2 enzyme inhibition was measured and the ability of Nexrutine™ to inhibit COX-2 was compared to other selective COX-2 inhibitors. The results were favorable and indicated Nexrutine's™ COX-2 inhibitory potential. Nexrutine™ also showed little inhibition of COX-2.