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Celecoxib may be effective in preventing non-melanoma skin cancers

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Celecoxib may help prevent nonmelanoma skin cancers in patients with extensive actinic keratosis, which is often a precursor to these cancers, according to a randomized clinical trial published online November 29 in *The Journal of the National Cancer Institute.*

Nonmelanoma skin cancers—including cutaneous squamous cell carcinomas (SCCs) and basal cell carcinomas (BCCs)—are among the most common cancers in the United States. The incidence of these malignancies is rising and is beginning to occur more frequently in young adults. Exposure to UV radiation is believed to be one of the main causes. Existing methods of protecting against UV light, namely sunscreens, have only been modestly effective at reducing these cancers' incidence, so researchers have begun looking for other ways to prevent them. Celecoxib is a nonsteroidal anti-inflammatory drug (NSAID) that inhibits the enzyme cyclooxygenase 2, believed to be involved in the development of UV-induced nonmelanoma skin cancers.

To determine whether celecoxib reduces the incidence of new actinic keratosis, Craig A. Elmets, M.D., of the University of Alabama at Birmingham, and colleagues, conducted a double-blind, placebo-controlled randomized trial with 240 people with actinic keratoses, or precancerous skin lesions. They looked at the number of new lesions after 3, 6, and 9 months of therapy and at 2 months following the completion of therapy.

The researchers found that the number of new precancerous lesions in

the group of people taking celecoxib and those taking the placebo was the same. However, by the end of the trial, those taking celecoxib had a statistically significant fewer number of nonmelanoma skin cancers compared with those in the placebo group.

The researchers write, "The findings of this study, which showed that the celecoxib-treated individuals developed fewer nonmelanoma skin cancers than placebo-treated individuals, suggest that cyclooxygenase inhibitors may provide an additional benefit to sunscreens in the prevention of nonmelanoma skin cancers."

The finding that celecoxib did not reduce the number of precancerous lesions was inconsistent with animal studies showing that celecoxib reduced both precancerous lesions and nonmelanoma skin cancers. However, the drug's efficacy against later stages of tumor development was consistent with findings of trials of celecoxib for colorectal adenoma.

The FDA terminated the study early, after preliminary findings from a concurrent trial of another cyclooxygenase 2 inhibitor showing an increased risk of cardiovascular adverse events. The fact that no adverse cardiovascular events were found with this trial could be because the study participants were only taking it for 9 months, the researchers point out. Another trial showed that adverse events occurring only after 1 year of rofecoxib, a cyclooxygenase-2 inhibitor.

In an accompanying editorial, Frank L. Meyskens Jr. M.D., and Christine E. McLaren, M.D., of the University of California, Irvine write that the fact that celecoxib was effective in reducing the number of nonmelanoma skin cancers but not the number of precancerous lesions may suggest that pathways of carcinogenesis differ between early- and late-stage tumor development.

The editorialists say future trials of celecoxib might try lowering the frequency of celecoxib administration, given the adverse cardiovascular events shown by other trials, or use a lower dosage in combination with other compounds with proven efficacy in colorectal adenoma.

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