

Scientists show commonly prescribed painkiller slows cancer growth

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Authors of the new study included The Scripps Research Institute's (left to right) Research Assistant Smitha Kota, Research Associate William Guerrant, Associate Professor Joseph Kissil, Research Assistant Scott Troutman and Research Associate Vinay Mandati. Credit: The Scripps Research Institute.

Scientists from the Florida campus of The Scripps Research Institute (TSRI) have found that one of the most widely prescribed pain and anti-inflammation drugs slows the growth rate of a specific kind of cancer in animal models and suggests the medication could have the same effect on other types of tumors.

The new study, published online ahead of print by the journal *Cancer Research*, focused on the effects of celecoxib (Pfizer's Celebrex).

Celebrex targets an enzyme called "cyclooxygenase-2" (COX-2), which is linked to pain and inflammation. This enzyme is also critical in the creation of prostaglandins, compounds that act like hormones and play a role in promoting tumor growth. COX-2 expression is typically low in normal tissue, but high in multiple types of cancers.

"We were actually interested in determining what a particular signaling pathway does in [cancer](#)," said TSRI Associate Professor Joseph Kissil, who led the study. "In the process, we found that it activates genes that promote survival of [tumor cells](#) and that they do so by turning on enzymes involved in inflammation, including COX2, which anti-inflammatory drugs like Celebrex inhibit."

The researchers went on to conduct animal studies tracking the effects of celecoxib on the growth of cancer cells from a tumor type known as neurofibromatosis type II (NF2). In humans, NF2 is a relatively rare inherited form of cancer caused by mutations in the anti-tumor gene *NF2*, which leads to benign tumors of the auditory nerve.

Animals received a daily dose of the drug, and tumor growth was followed by imaging. Analysis of the results showed a significantly slower tumor [growth rate](#) in celecoxib-treated models than in controls.

Using various approaches, the new study also showed that a signaling

cascade known as the Hippo-YAP pathway is involved in these results and that the protein YAP is required for the proliferation and survival of NF2 cells and [tumor formation](#).

"Our study shows that COX2 inhibitors do have an effect on the tumor cells," said TSRI Research Associate William Guerrant, the study's first author. "They also have an impact on inflammatory responses that play a role in [tumor growth](#). It's possible that in other cancers these effects might actually be stronger because of the drug's impact on inflammation."

More information: W. Guerrant et al, YAP mediates tumorigenesis in neurofibromatosis type 2 by promoting cell survival and proliferation through a COX2-EGFR signaling axis, *Cancer Research* (2016). [DOI: 10.1158/0008-5472.CAN-15-1144](#)

Provided by The Scripps Research Institute

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