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Aspirin slows growth of colon, pancreatic tumor cells

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Researchers from Oregon Health and Science University and Oregon State University have found that aspirin may slow the spread of some types of colon and pancreatic cancer cells. The paper is published in the *American Journal of Physiology—Cell Physiology*.

Platelets are blood cells involved with clotting. They promote the growth of cancerous cells by releasing growth factors and increasing the response of certain proteins that regulate tumor cell development (oncoproteins). Low doses of aspirin, an anti-platelet drug, have been shown to reduce the risk of some types of gastrointestinal cancers, but the process by which aspirin hampers tumor growth has been unclear. "The current study was designed to determine the effect of inhibition of platelet activation and function by aspirin therapy on colon and pancreatic cancer cell proliferation," the researchers wrote.

The research team combined activated platelets primed for the clotting process with three groups of cancer cells:

- metastatic colon cancer (cells that have spread outside the colon),
- nonmetastatic colon cancer (cells that grow only within the colon) and
- nonmetastatic pancreatic cancer cells.

When they added aspirin to the mixture, they found that the platelets were no longer able to stimulate growth and replication in the pancreatic and nonmetastatic colon cancer cells. The metastatic colon cancer cells

continued to multiply when treated with aspirin.

In pancreatic cancer cells, low doses of aspirin stopped the platelets from releasing growth factor and hampered the signaling of the oncoproteins that cause cancer to survive and spread. Only very high doses—larger than are possible to take orally—were effective in stopping growth in the metastatic colon cells, explained the researchers.

The findings detail the interaction among platelets, aspirin and tumor cells and are promising for the future treatment of nonmetastatic cancer, according to the researchers. "Our study reveals important differences and specificities in the mechanism of action of high- and low-dose aspirin in metastatic and nonmetastatic cancer cells with different tumor origins and suggests that the ability of aspirin to prevent platelet-induced c-MYC [an oncoprotein] expression might be selective for a nonmetastatic phenotype."

More information: Annachiara Mitrugno et al. Aspirin therapy reduces the ability of platelets to promote colon and pancreatic cancer cell proliferation: implications for the oncoprotein c-MYC., *American Journal of Physiology - Cell Physiology* (2016). DOI: 10.1152/ajpcell.00196.2016

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