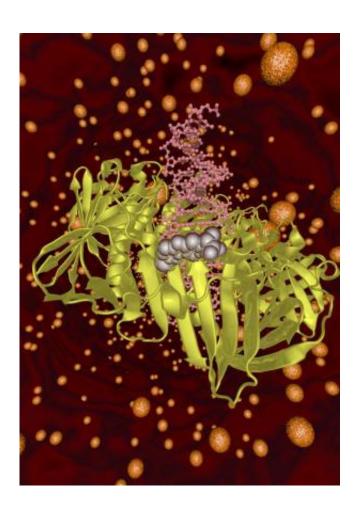


Commonly used pain relievers have added benefit of fighting bacterial infection

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This image shows nonsteroidal anti-inflammatory drugs bound to bacterial protein. Credit: Aaron Oakley

Some commonly used drugs that combat aches and pains, fever, and inflammation are also thought to have the ability to kill bacteria. New



research appearing online on March 13 in the Cell Press journal *Chemistry & Biology* reveals that these drugs, better known as NSAIDs, act on bacteria in a way that is fundamentally different from current antibiotics. The discovery could open up new strategies for fighting drugresistant infections and "superbugs."

"We discovered that some anti-inflammatory drugs used in human and veterinary medicine have weak antibiotic activity and that they exert this secondary activity by preventing <u>bacteria</u> from copying their DNA, which they need to do in order to multiply," explains senior author Dr. Aaron Oakley of the University of Wollongong, in Australia. The researchers analyzed three NSAIDs: bromofenac, carprofen, and vedaprofen. The more commonly known NSAIDs, which include aspirin, ibuprofen, and naproxen, were not tested.

Dr. Oakley and his team identified that anti-inflammatory drugs bind to and inhibit a specific protein in bacteria called the DNA clamp. The DNA clamp, which is conserved across bacterial species, is part of an enzyme that synthesizes DNA molecules from their nucleotide building blocks.

The discovery comes at a time when there is a pressing need for new classes of antibiotics. "The fact that the bacteria-killing effect of the anti-inflammatory drugs is different from conventional drugs means that the NSAIDS could be developed into new kinds of antibiotics that are effective against so-called superbugs," says Dr. Oakley. "This is important because the <u>superbugs</u> have become resistant to many—and in some cases most—of the available antibiotics."

More information: *Chemistry & Biology*, Yin et al.: "DNA Replication is the Target for the Antibacterial Effects of Non-Steroidal Anti-Inflammatory Drugs." <u>dx.doi.org/10.1016/j.chembiol.2014.02.009</u>



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