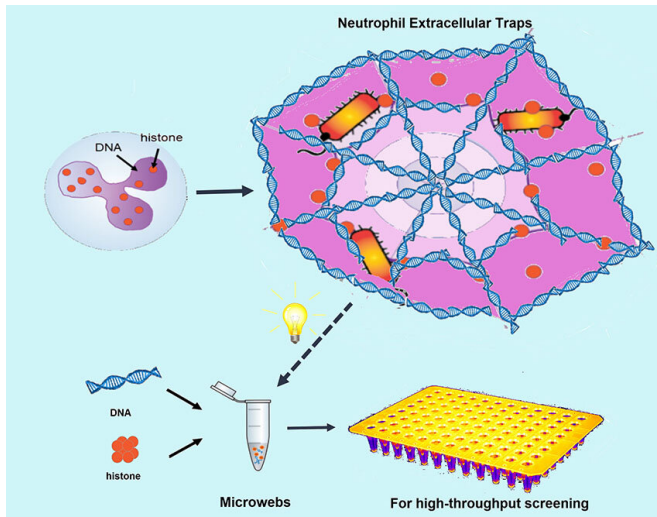


Understanding white blood cells' defense mechanisms could lead to better treatments

31 January 2019, by Kylie Urban



Credit: University of Michigan

Experiencing a bacterial infection? You're generally prescribed antibiotics by your doctor. But how exactly do those antibiotics and your white blood cells work in tandem to improve your infection?

"The human body's first line of defense against [bacteria](#) are certain white blood cells called neutrophils," says J. Scott VanEpps, M.D., Ph.D., assistant professor of emergency medicine at Michigan Medicine. "One of their weapons are neutrophil extracellular traps, also called NETs."

The traps are microscopic networks of fibers made primarily of DNA that are produced by the neutrophils to capture bacteria. But how exactly they work, VanEpps notes, is still unclear.

"The exact function and mechanism of NETs remains a bit of a mystery likely because they have different functions for different situations," he says. "And it's very hard to isolate these NETs in the

laboratory and study them in detail."

VanEpps is a co-senior author of a new study, published in *Advanced Materials*, that investigated if there was a way to successfully create NETs in the laboratory to better understand how NETs can capture bacteria.

"We were hoping to provide a bottom-up engineering approach to better understand how the immune system fights bacteria, specifically the immune system's weapon: NET," says VanEpps, associate director at the Michigan Center for Integrative Research in Critical Care.

Recreating and studying networks

VanEpps explains that he and the team found their first surprising finding during the recreation of the NETs.

"Although there are literally hundreds of different ingredients in natural NETs, we were able to recreate a lot of their structure and function with just two ingredients and determined the optimal ratio of those ingredients," he says. "They look and function very similar to NETs produced by those neutrophil [white blood cells](#) and the synthesis method is much simpler than isolating them from neutrophils."

After producing the microwebs, the research team studied them more closely to provide a more detailed understanding of how NETs can capture, and the second surprising finding of the study, even kill bacteria.

"We studied how our microwebs capture *E. coli* and found they could also kill it," VanEpps says. "This led to us examining how microwebs might play a role with antibiotics.

"Importantly, we found that the dose of antibiotic required to kill *E. coli* (including a colistin resistant

strain of *E. coli*) was less when our microwebs were present."

VanEpps' co-senior author, Shuichi Takayama, Ph.D., a professor at Georgia Tech, says the findings are an example of teamwork.

"This research was an outstanding team effort," Takayama says. "Microbiology from the VanEpps lab, NET-like materials contributions from the Moon lab, NET expertise from the Knight lab, colistin resistance expertise from the Weiss lab and Yang Song, Ph.D., leading the effort, made this all come together."

Future research

VanEpps notes this study can be a jumping off point for future research.

"Now that we have a platform, we can synthesize microwebs with different NET components to more precisely describe how the immune system uses NETs in different situations," he says.

This understanding of the body's natural killing of bacteria could also help in developing new treatments for infections.

"The knowledge gained in this study could be helpful in the future in designing new and better antibiotics that mimic the body's natural defense mechanisms, as well as potentially change how we dose antibiotics given the potential synergy between the immune system and certain antibiotics," VanEpps says.

"That's critical as we have not developed a new class of [antibiotics](#) in over 30 years and resistance continues to increase."

More information: Yang Song et al, Antimicrobial Microwebs of DNA-Histone Inspired from Neutrophil Extracellular Traps, *Advanced Materials* (2019). [DOI: 10.1002/adma.201807436](https://doi.org/10.1002/adma.201807436)

Provided by University of Michigan

APA citation: Understanding white blood cells' defense mechanisms could lead to better treatments

(2019, January 31) retrieved 5 December 2020 from <https://phys.org/news/2019-01-white-blood-cells-defense-mechanisms.html>

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