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Scientists decode how anthrax toxin proteins might help treat cancerous tumors

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Scientists from the National Institute of Allergy and Infectious Diseases (NIAID), the National Institute of Dental and Craniofacial Research (NIDCR), and the National Heart, Lung and Blood Institute (NHLBI), all parts of the National Institutes of Health (NIH), describe how combining engineered anthrax toxin proteins and existing chemotherapy drugs could potentially yield a therapy to reduce or eliminate cancerous tumors. The findings, based on testing in mice, will appear this week in the Early Edition of the *Proceedings of the National Academy of Sciences*.

The bacterium *Bacillus anthracis*, which causes the deadly anthrax disease, produces a toxin made of three proteins that individually are non-toxic. Because the proteins can be engineered to suppress tumor growth they have emerged as a potential cancer therapy. Until now, however, scientists have been unsure how the anthrax toxin proteins control tumor growth.

In this study, NIH scientists used mouse models to show that anthrax toxin proteins work by specifically targeting the cells that line the inner walls of the blood vessels feeding the tumor. The proteins, which reach these cells through a surface receptor called CMG2, prevent the cells from reproducing. Because the toxin does not target the tumor cells themselves but rather the host-derived blood vessel cells, the strategy could be efficacious for a wide range of tumor types, the NIH team notes.

Unfortunately, the immune system produces antibodies in response to

the anthrax toxin proteins, making additional courses of treatment ineffective. To circumvent this problem, the investigators examined in mice whether a regimen of the chemotherapy drugs pentostatin and cyclophosphamide (PC) could block production of the antibodies that neutralize the anthrax toxin proteins. Mice were inoculated with tumors and treated with one of the following regimens: saline (for use as a placebo), anthrax toxin protein therapy, PC, or a combined regimen of anthrax toxin protein therapy and PC. After four cycles of therapy (42 days), all mice receiving the combined regimen were alive, whereas mice in the other groups had to be euthanized due to tumor growth. In addition, the investigators could not detect any neutralizing antibodies in the combined regimen group, even after the fourth round of therapy. Together, the results showed that the combined anthrax toxin protein and PC therapy has durable, anti-tumor effects worthy of further exploration, according to the authors.

More information: Solid tumor therapy by selectively targeting stromal endothelial cells, *PNAS*, www.pnas.org/cgi/doi/10.1073/pnas.1600982113

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