Nanoparticles cut off ‘addicted’ tumors from source of their survival

Yale UniversityYale University

Yale biologists and engineers have designed drug-loaded nanoparticles that target the soft underbelly of many types of cancer — a tiny gene product that tumors depend upon to replicate and survive.

The novel therapy successfully stopped lymphoma in mice when injected directly into tumors, the researchers report in the Proceedings of the National Academy of Sciences, published the week of May 28.

The interdisciplinary collaboration takes advantage of a new concept in cancer research — that tumors become “addicted” to a few genetic abnormalities they need to survive, grow, and spread throughout the body.

“Thousands of genes are mis-expressed in cancer, but so far cancer researchers have only found 10 or so that cancer cells absolutely need to survive,” said Frank Slack [1], professor of molecular, cellular, and developmental biology, director of the Cancer Genetics and Genomics Program for the Yale Cancer Center, and senior author of the study.

Slack’s lab studies microRNAs, or small pieces of genetic material that determine when and where much larger genes that code for proteins are used. One of these miRNAs, miR-155, helps regulate cell survival and is overactive in many forms of cancer. For instance, mice with excessive amounts of miR-155 develop lymphoma tumors.

One of Slack’s graduate students collaborated with a student working in the lab of Mark Saltzman, the Goizueta Foundation Professor of Chemical and Biomedical Engineering and Yale Cancer Center researcher, about ways to use nanoparticles to help block actions of miR-155 in mice with lymphoma. The team discovered that injecting nanoparticles that deliver a compound that specifically targets miR-155 into tumors stopped them from growing in mice.

Slack pointed out that miR-155 is also overactive in lung cancer and many other treatment-resistant forms of the disease.

“At this point, we want to improve the technique so we can load even more of this compound into the nanoparticles and make it easier for them to enter tumor cells,” Slack said. “Ultimately, we would like to take this to human clinical trials for difficult-to-treat cancers.”

Imran A. Babar and Christopher J. Cheng were co-lead authors of the paper. Other Yale authors are Carmen J. Booth, Xianping Liang, and Joanne B. Weidhaas.

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Links:
[1] http://www.mcdb.yale.edu/facultystaff/slack.html