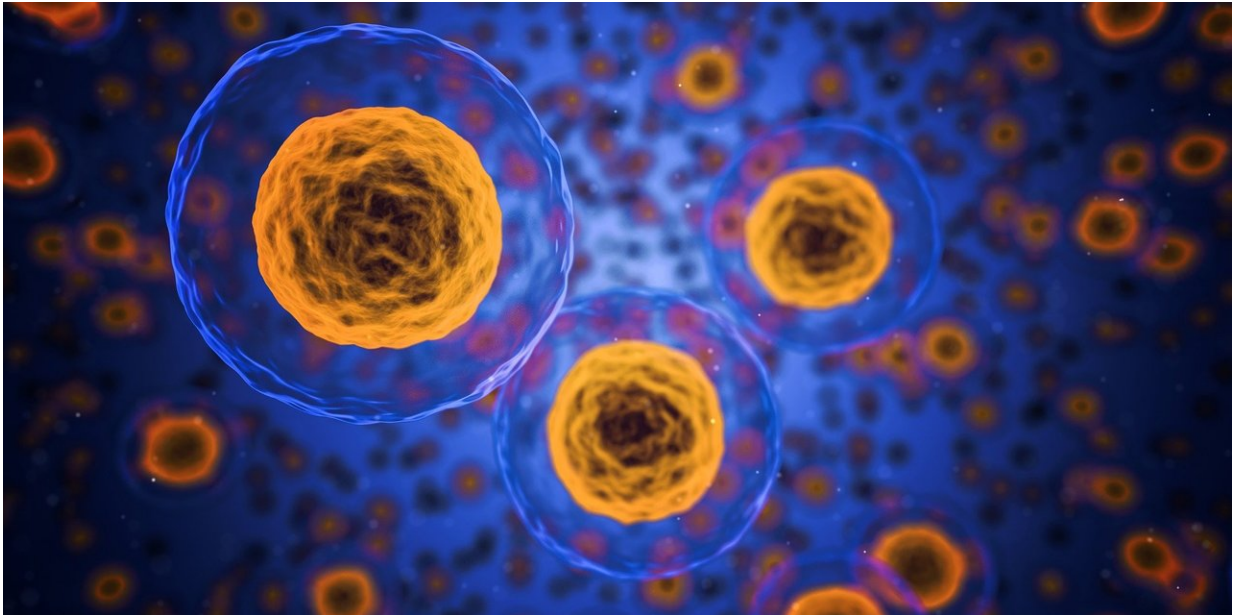


When cells cycle fast, cancer gets a jumpstart

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The progression of cancer has been studied extensively, and the key steps in this journey have been well mapped, at least in some solid tumors: Lesions to genes that confer risk of cancer accumulate and alter normal cell behaviors, giving rise, scientists believe, to early stage cancer cells that eventually swamp normal cells and become deadly.

But Yale researchers have now identified another bit of cellular chicanery that jumpstarts cancer. In at least one form of blood cancer, they report Dec. 18 in the journal *Nature Communications*, cells with

cancer-causing gene lesions can remain normal and healthy—until [cell division](#), or cycling, speeds up.

"Many people with cancer-causing genes remain healthy for many, many years," said senior author Shangqin Guo, assistant professor of cell biology and a researcher at the Yale Stem Cell Center. "So, in these cases, you have to wonder whether the dogma 'mutations cause cancer' is the complete truth."

Guo and first author Xinyue Chen, a graduate student in Guo's lab, wanted to focus on the role of cell cycling speed, which varies in normal tissue, on the formation of cancer. They studied [acute myeloid leukemia](#) (AML), an aggressive type of blood cancer that carries the lowest number of mutations among human cancers. They introduced a known leukemia-causing mutation, MLL-AF9, into the genomes of mice and tracked individual blood cells for signs of cancer. While the great majority of cells remained normal, the few cells that divided most quickly almost always became malignant.

"When a normal cell that divides quickly meets MLL-AF9, this combination creates a monster cell that is stuck in a state of perpetual fast division," Guo said. "Their more slowly cycling counterparts remain normal and do not display the malignant cancer traits, even in the presence of cancer-causing mutations."

Understanding how a normal cell crosses over to the dark side has important implications.

For example, the researchers said, certain infections can trigger a proliferation of rapidly-dividing cells to fight the pathogens, perhaps opening the door to cancer.

Also, stem cells generally experience functional decline during aging,

which requires healthier stem [cells](#) to divide more quickly to repair damaged tissue. The increased proliferation may provide an explanation of why people become more susceptible to [cancer](#) as they age, the scientists said.

Provided by Yale University

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