How One Molecule Involved in Most Leukemias May Lead to Targeted Therapies

Researchers have identified another molecule that may lead to the development of more effective targeted medications.

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Nearly all human cancers, and particularly blood cancers, involve dysregulated gene expression – the wrong genes are switched on or the right ones are switched off. The molecule responsible for switching genes on and off is called a transcription factor. Identifying which transcription factor is misbehaving and how is often the key to developing effective cancer treatments.

In the case of leukemias, recent studies have helped to clarify which molecule is to blame: a transcription factor called MYB (pronounced “mib”), when suppressed, almost entirely eliminated tumor growth in mice. Now, data emerging from researchers at Sloan Kettering Institute has shed light on the how: both how normal MYB function goes wrong within cancerous cells, and how its cancerous activity may be blocked by future therapies.

The team, led by former Damon Runyon Clinical Investigator Alex Kentsis, PhD, has identified another molecule, known as a co-factor, named CBP/P300 that binds to MYB and disrupts its normal function. Inhibiting CBP/P300’s ability to bind to MYB – but still allowing MYB to bind with other co-factors – prevented cancer-causing genes from being expressed in the observed cells. This finding suggests a mechanism by which MYB could be selectively targeted without knocking out its function in healthy cells.

The inhibiting agent used for this study is not safe for use in humans, but demonstrating that it works is an essential step toward developing a drug that is safe and ready to be tested in clinical trials.

While the rise of personalized medicine allows for highly customizable cancer care, the sheer number of cancer subtypes has presented a challenge to drug developers. Research like this, which seeks to elucidate the underlying mechanism of tumor growth in nearly all blood cancers, could lead to the development of a drug that is more widely applicable for a greater number of cancer patients.

As Dr. Kentsis told Memorial Sloan Kettering Cancer Center, “We want to understand the universal

rules that control cancers, so that we can develop therapies that could work for many different types of cancers for most people.” His lab’s recent findings are certainly a step in that direction.

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