

Epigenomic abnormalities predict patient survival in non-Hodgkins lymphoma

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Think of the epigenome like a giant musical mixing board, turning up or down the expression of various genes. A University of Colorado Cancer Center study published today in the journal *PLOS Genetics* shows that in cancer, not only can genes themselves go bad, but abnormal changes in the epigenetic mixing board can unfortunately change the expression of these genes. Researchers hope to play the role of sound engineers, controlling these harmful epigenomic changes to turn down cancer itself or perhaps sensitize cancers to existing drugs.

The epigenome's primary tool – and by far the easiest to study – is methylation: it attaches little [methyl groups](#) to [DNA sequences](#) near the genes to silence or promote their expression.

"Not only do we see more abnormal methylation in non-Hodgkin [lymphoma patients](#) than in healthy B-[cell populations](#), but there are three distinct subtypes of the disease in the clinic, each more aggressive than the next. These three clinical [trajectories](#) of non-Hodgkins lymphoma were distinctly marked by their levels of abnormal methylation," says Subhajyoti De, PhD, CU Cancer Center investigator and assistant professor at the CU School of Medicine.

In other words, methylation patterns predict patient survival. Here's how it works:

DNA should be methylated in a consistent way – you get a certain, standardized amount of methyl "residue" attached to your genes. Sure

enough, that's the case in healthy B-cells. Subhajyoti and colleagues show that in cancerous B-cells, the level of [DNA methylation](#) from cell to cell varies wildly. And the more wildly the level of DNA methylation varies, the more aggressive is the cancer. It's as if, in the body, you want a consistent epigenome that maintains the methylation of the healthy status quo –when a willy-nilly epigenome drops methylation randomly here and there, it promotes non-normal cells, like cancer.

So abnormal methylation is certainly correlated with not only cancer, but with the aggressive behaviors of cancer subtypes. But what exactly is the functional role of this methylation?

"We think that in addition to genetic mutations that cause cancer, epigenetic changes probably play a subtle role that allows the cancer to thrive within our body," Subhajyoti says.

There are drugs that affect the epigenome's ability to methylate and so control genes – some of which crescendo or decrescendo the amount of methylation across the board, and some of which affect the amount of methylation on certain genetic products. Does one of these drugs hold the key to muting cancer?

Subhajyoti hopes to find out.

"For the last 50 years, the scientific community pushed to identify the genetic drivers of cancer, but now in the past five or six years we've expanded the search into the epigenome as well," Subhajyoti says. "We now expect to find that both genetic and epigenetic abnormalities are important for initiation and maintenance of cancer."

Provided by University of Colorado Denver

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