

New insights into why humans are more susceptible to cancer and other diseases

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Chimpanzees rarely get cancer, or a variety of other diseases that commonly arise in humans, but their genomic DNA sequence is nearly identical to ours. So, what's their secret? Researchers reporting in the September issue of the *American Journal of Human Genetics*, have found that differences in certain DNA modifications, called methylation, might play a role.

The researchers discovered hundreds of genes that display different patterns of methylation between the two species. These different patterns of methylation lead to different levels of expression, and many of the genes involved are linked to specific human diseases. Given that environmental factors can affect DNA methylation, these results might help researchers to better understand how differences in genetics and environmental exposure contribute to differences, including different disease vulnerabilities, between the two species.

DNA methylation doesn't change a cell's underlying <u>genetic information</u>, but it does affect <u>gene activity</u> and can have a profound impact on processes such as aging and the development of disease. By using new state-of-the-art techniques to look at methylation maps and gene expression in the brains of chimpanzees and humans, the investigators found that changes in DNA methylation at least partially explain the divergence of gene-expression patterns between these species.

In addition, differentially methylated genes showed striking links with specific neurological and <u>psychological disorders</u> and cancers to which



modern humans are particularly susceptible, suggesting that changes in <u>DNA methylation</u> might be linked to the evolution of humans' vulnerability to certain diseases.

"Our results hint, but by no means provide proof, that epigenetic divergence—or changes of chemical properties of DNA—may be particularly important for some disease-related phenotypes that are pertinent to modern humans," says senior author Dr. Soojin Yi, from the Georgia Institute of Technology. "Such findings, in the long-term, may contribute to the development of better therapeutic targets for some human diseases," she adds.

Provided by Cell Press

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