Medical Xpress

Smoking and heavy alcohol use are associated with epigenetic signs of aging

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Cigarette smoking and heavy alcohol use cause epigenetic changes to DNA that reflect accelerated biological aging in distinct, measurable ways, according to research presented at the American Society of Human Genetics (ASHG) 2015 Annual Meeting in Baltimore.

Using data from the publicly available Gene Expression Omnibus, Robert A. Philibert, MD, PhD and colleagues at the University of Iowa and other institutions analyzed patterns of DNA methylation, a molecular modification to DNA that affects when and how strongly a gene is expressed. Prior research had shown that methylation patterns change in predictable ways as people age, as well as in response to environmental exposures, such as cigarette smoke and alcohol. In these earlier studies, Dr. Philibert's laboratory identified two specific locations in the genome, base pairs cg05575921 on the AHRR gene and cg23193759 on chromosome 10, at which methylation levels were highly associated with smoking and alcohol consumption, respectively.

In fact, they showed, DNA methylation levels at these two locations was a better measure of substance use than people's self-reported estimates. Thus, in this follow-up study, Meeshanthini Dogan, MS, and Dr. Philibert used methylation levels as a proxy for tobacco and alcohol consumption. They estimated each person's biological age using a previously validated epigenetic "clock" based on methylation levels at 71 locations in the genome, as measured by the widely used Infinium HumanMethylation450 BeadChip. Then, they calculated the difference between biological age and chronological age, and assessed the

relationship between tobacco and alcohol use and premature aging.

They found that all levels of exposure to smoke were associated with significantly premature aging. Interestingly, moderate alcohol use - about one to two drinks per day - was correlated with the healthiest aging, while very low and high consumption were linked to accelerated aging.

"These new tools allow us to monitor smoking and alcohol use in an objective way, and to understand their effects quantitatively," Ms. Dogan said. "Furthermore, our methods could be used to analyze any set of 450 BeadChip data, which means that existing data can be used to identify new patterns and that all such results can be easily compared."

"Being able to objectively identify future smokers and heavy alcohol users when they are young, before major health issues arise, can help providers and public health practitioners prevent future problems, improve quality of life, and reduce later medical costs," Dr. Philibert added.

The researchers' next step is to unravel the details of how methylation patterns change in response to lifestyle changes during the life course, so that their assessments can be more informative.

"For example, we want to study how the intensity of current tobacco and alcohol use and cumulative levels of use throughout a lifetime affect methylation, including what happens when a person quits smoking or drinking," Ms. Dogan said. "By clarifying at what point the epigenetic changes become tougher to stop or reverse, we can inform decisions about how best to use the limited public health resources we have."

Provided by American Society of Human Genetics