Chronic Hepatitis C Linked to Faster Biological Aging

Successful antiviral treatment partially turns back the clock on age acceleration.

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People with chronic hepatitis C experience faster biological aging, but sustained response to antiviral therapy partial reverses this acceleration, according to study results published in the Journal of Hepatology. However, people who go on to develop liver cancer even after being cured do not experience this reversal in epigenetic aging.

“Our results suggest that epigenetic changes, or acceleration of biological age, are reversible in principle, but this requires time, while a lack of reversibility appears to be associated with the development of hepatocellular carcinoma,” wrote the researchers.

Over years or decades, chronic hepatitis C virus (HCV) infection can lead to liver complications, including fibrosis, cirrhosis and hepatocellular carcinoma, the most common type of liver cancer. While direct-acting antiviral (DAA) treatment usually leads to a cure, some effects of the disease can linger.

Recent studies have shown a link between chronic viral diseases and accelerated biological aging. Markus Cornberg, MD, of the Centre for Individualised Infection Medicine in Germany, and colleagues explored whether chronic hepatitis C is linked to accelerated biological aging and whether this could be reversed once a functional cure is achieved.

For this study, the researchers recruited 54 people with chronic hepatitis C who achieved a sustained virological response, or an undetectable HCV viral load 12 weeks after completing antiviral therapy, which is considered a cure. The participants were evaluated at three different time points: treatment initiation, the end of treatment and follow-up at 96 weeks.

The team assessed the participants’ biological age using circulating immune cells called peripheral blood mononuclear cells. They used Horvath’s clock, a well-established biochemical test that measures DNA methylation, to quantify epigenetic aging of cells.

In general, the researchers found that biological age acceleration decreased from treatment initiation through long-term follow-up and that the decrease was especially notable after completing therapy. Before treatment, people with hepatitis C had epigenetic age acceleration of

+3.12 years compared with -2.61 years in an age- and sex-matched reference group. At long-term follow-up, biological age acceleration fell to +1.37 years after being cured.

People without cirrhosis at baseline had the least epigenetic age acceleration at the start of therapy, while those who developed hepatocellular carcinoma after SVR had the greatest acceleration. What's more, participants with liver cancer showed little evidence of reversal of age acceleration even after achieving SVR.

"The results of our study suggest that chronic HCV infection leads to a general acceleration of epigenetic aging predicted by Horvath's clock and that HCV elimination by DAA therapy can partially slow, halt or reverse biological aging," the researchers concluded. "The exact mechanism leading to this age acceleration and its reversal remains elusive."

"While most clinical risk scores now take chronological age into account, it may be worthwhile to explore how biological age might improve these scores in the future," they wrote. "Biological age may be a cornerstone for the individualized clinical assessment of patients in the future, as it better reflects patients' lifestyle and environmental exposures over decades."

Click here to read the study abstract in the Journal of Hepatology.

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