

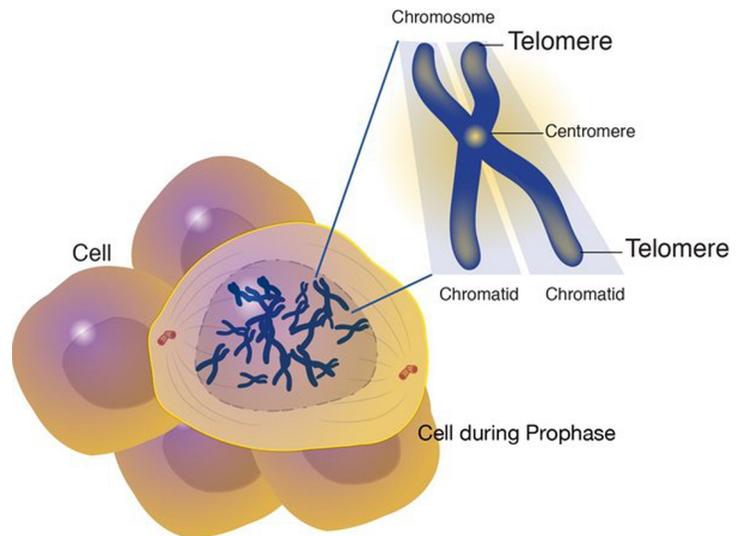
# Telomere length linked to catching a cold in preliminary study

By Eryn Brown

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Test subjects with shorter immune cell telomeres faced an increased risk of catching a cold, researchers wrote Tuesday in [JAMA](#) ([abstract here](#).)

It was the first time the DNA structures had been implicated in acute illnesses in healthy, relatively young people, wrote study leader Sheldon Cohen, a psychologist at Carnegie Mellon University, and colleagues at other institutions.



Telomeres are DNA “caps” that sit at the end of chromosomes and protect them from damage. They shorten each time a cell divides, ultimately getting whittled away to such an extent that the cell can no longer divide, loses its ability to function properly and dies.

In studies over the years, shorter telomeres in people have been connected to [aging](#) and aging-related disorders like [cancer](#), heart disease and [dementia](#). But according to the authors of the JAMA paper, researchers hadn’t determined how telomere length might relate to more acute bouts of illness in otherwise healthy, younger adults.

To gauge whether there was any relationship, the team conducted an experiment on 152 healthy adults, ages 18 to 55, in the Pittsburgh area. Researchers first collected blood to measure telomere length in leukocytes, or white blood cells. Then they placed the study participants in quarantine for six days, administering nasal drops containing a virus that causes the common cold (rhinovirus type 39) after the first 24 hours.

Over the course of the experiment, 69% of the participants, or 105 people, were infected with the virus; 22%, or 33 people, developed colds.

Overall, shorter telomeres in four types of blood cells were associated with greater risk of infection, with the strongest link occurring in a type of T-cell called CD8CD28- cells. What’s more, shorter

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telomere length in CD8CD28- cells was also associated with clinical illness (that is, getting a cold). The connection between shorter telomeres and infection got stronger with increasing age in study subjects.

The researchers speculated that, when challenged with viral antigens, T-cells with shorter telomeres might not proliferate as well as T-cells with longer telomeres, making it harder for the body to clear away virus-infected cells.

“A provocative possibility is that telomere length is a very stable marker of disease susceptibility, with associations between telomere length and clinical outcomes beginning to emerge in early adulthood,” they wrote. But they also cautioned that their data were preliminary, and that any clinical implications remained unknown.

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