Latent CMV infection induces telomere shortening

Published on July 2, 2015 at 8:50 AM

Molecular, cellular and clinical changes that arise from an infection with a latent virus and result in a decrease in longevity.

The telomeres are repetitive DNA sequences at each end of our chromosomes. Studies show that in every cell division, the telomere is shortened. As a result, the telomere limits the cell to a fixed number of divisions and a limited life span. An essential part of human cells they affect how our cells age - as people with longer telomeres live longer lives. Surprisingly, people who are infected with a latent virus, that is, an asymptomatic virus, have shorter telomeres. This is an important observation and a great mystery. Is the virus causing the telomere shortening, and how? And if this is the case, what does it mean in terms of the relationship between the latent viruses and longevity?

Now, an article "The Latent Cytomegalovirus Decreases Telomere Length By Microcompetition" by *Hanan Polansky* and *Adrian Javaherian*, published in Open Medicine by De Gruyter Open, provides some answers to these questions. As it turns out, a certain gene, called telomere repeat binding factor 2 (Terf2), belongs to a complex of six telomere-associated proteins, termed shelterin. The protein produced by this gene protects the chromosome ends of the DNA from damage, and controls telomere maintenance by the telomerase enzyme. When does a cell produce the Terf2 protein? After receiving a signal that tells a transcription factor called GABP to bind the Terf2 gene. One can think of GABP as a finger that pushes the "ON" button on the Terf2 gene. Now consider a case where a latent virus called CMV infects the cell. As it turns out, the CMV virus also uses the GABP transcription factor to press the "ON" button on its own genes. When the CMV virus steals the GABP "fingers" from the Terf2 gene, there are no fingers left to press the "ON" button on the Terf2 gene, and the Terf2 gene fails to produce the Terf2 protein. What is the result of a shortage of Terf2 proteins? Short telomeres. In his book "Microcompetition with Foreign DNA and the Origin of Chronic Disease", Dr. Polansky used the term Microcompetition to describe this event.

Most people are infected with a latent virus. For instance, CMV is present in more than 70-80% of individuals by the age of fifty. A study by Gkrania-Klotsas et al. showed that individuals with signs of CMV have higher all-cause mortality compared to uninfected individuals. Polansky and Javaherian (both from The Center for the Biology of Chronic Diseasein Valley Cottage, NY) explain the molecular, cellular and clinical changes that result from a latent infection with the CMV and the consequent decrease in longevity.

There are many implications of the Microcompetition theory. Not only does it suggest new targets for drug development, but it also puts forward new behavioral changes that may be taken on the part of the infected individual in order to decrease the chances of developing a major disease and increase longevity. A common misconception that only the active phase of a viral infection can cause harm proves inadequate, as the Microcompetition theory shows that the latent phase is the most dangerous one. These findings can be crucial to further research into viral infections.

Source:
http://www.degruyter.com/
