MedicalXpress

The presence of roseola virus in chromosomes triples the risk of angina

June 16 2015

People whose chromosomes contain the DNA of the roseola virus are three times more likely to suffer from angina, according to a new study by researchers from the Université Laval Faculty of Medicine, the CHU de Québec Research Center-Université Laval, and the University of Washington. Details of this finding are published in the latest issue of the *Proceedings of the National Academy of Sciences*.

Roseola, also known as "sixth disease," is a very common childhood infection caused by the HHV-6 virus. Symptoms include a fever and a rash that dissipates after a few days. Once the infection has cleared, however, the virus does not disappear but instead takes up residence in the cells. In a small percentage of cases, the virus's DNA gets integrated in the chromosomes, including eggs or sperm.

"When that happens, the genetic material of the virus can be transmitted just like human genes," says the study's lead author, Louis Flamand, professor at the Université Laval Faculty of Medicine and researcher at CHU de Québec. Children are born with a copy of the HHV-6 DNA in every cell of their body. We wanted to determine whether this situation, which affects 40 to 70 million people in the world, increases the risk of developing a range of diseases."

To find out, researchers analyzed biological samples and health information of nearly 20,000 people recruited through CARTaGENE, a project to sequence the genome of a cross-section of the province of Québec's population. The analysis showed that the HHV-6 DNA was

integrated into the chromosomes of 0.6% of people tested. A cross-check with CARTaGENE medical data revealed that the prevalence of angina in this group of subjects is three times greater than in the general public.

Professor Flamand acknowledges that the nature of the link between angina and HHV-6 remains unclear. One hypothesis put forward by the researchers is that the virus affects the length of telomeres, the regions at the ends of chromosomes. By measuring telomere length in patients in their sample, researchers found that they were 24% shorter in people who were both angina sufferers and HHV-6 carriers than in non-HHV-6-carriers. "This telomere shortening may be associated with premature aging of cells lining the walls of blood vessels, and may initiate an inflammatory reaction that could lead to angina pectoris," Dr. Flamand suggests.

Detecting integrated HHV-6 in human chromosomes is easy, notes Dr. Flamand. "We could easily track this condition at birth, as we already do with hereditary diseases, and more closely monitor people whose chromosomes contain the virus."

More information: Inherited chromosomally integrated human herpesvirus 6 as a predisposing risk factor for the development of angina pectoris, *Proceedings of the National Academy of Sciences*, www.pnas.org/cgi/doi/10.1073/pnas.1502741112

Provided by Laval University