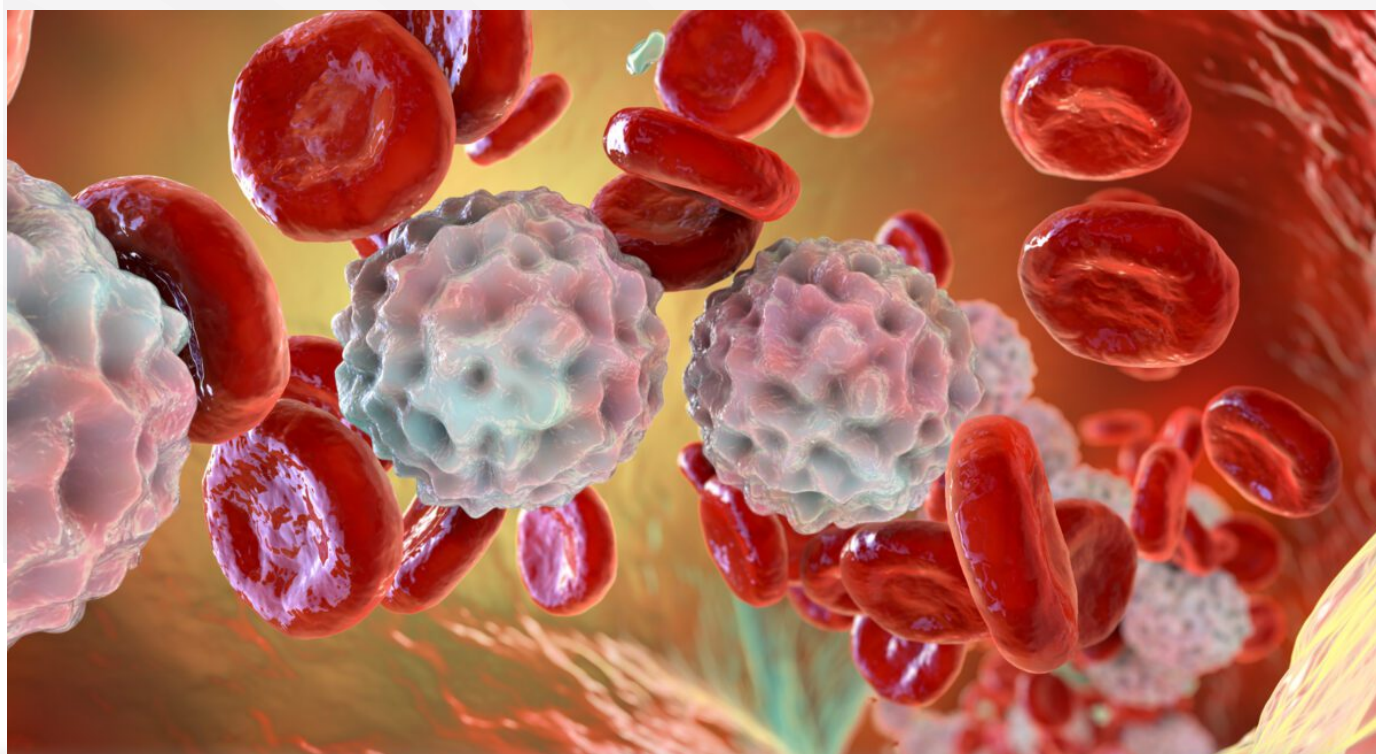


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Calico Stories

Uncovering New Signs of Aging in Blood



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As we age, our blood cells can develop genetic mutations that increase our risk of cancer and other aging-related diseases. This is known as clonal hematopoiesis. To provide a foundation for future efforts to intervene in the aging process, scientists have been studying the biological mechanisms behind clonal hematopoiesis.

Research Expands Prior Understanding

In a new study published in *Nature Genetics*, scientists at Calico, along with our collaborators at the Wellcome Sanger Institute and the University of Cambridge, have expanded on prior knowledge by identifying 17 additional genes that trigger clonal hematopoiesis. While these genes have long been known, researchers were not previously aware of their link to this aging phenomenon.

In genomics, it is often hard to distinguish between genetic mutations that are actually driving a biological phenomenon and genetic mutations that are present but have no actual effect in that particular situation. The progress made in this study was not just finding the 17 genes, but generating data to prove that mutations in these genes are actually causing clonal hematopoiesis.

That discovery was made possible through a number of contributors: the UK Biobank, a repository of genetic and clinical data from more than 200,000 volunteers; Sanger scientists, who sequenced blood samples from the biobank; and Calico scientists, who performed deep data analysis to spot driver genes. As added validation of the findings, the genetic mutations were associated with higher rates of infection and cancers of the blood among study participants.

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With the addition of these 17 genes, scientists can now learn even more about clonal hematopoiesis. Eventually, a comprehensive list of driver mutations could be used for diagnostic purposes, potentially offering early detection of aging-related diseases such as blood cancers and related myeloproliferative diseases.

Based on what is known today about clonal hematopoiesis, "this suggests that blood aging is akin to things like cancer or neurodegeneration, so it's a clear and present danger in the aging cycle," Cohen adds. "The idea that we acquire mutations as we age is a problem for those of us interested in slowing down aging."

Read the full article, "Analysis of somatic mutations in whole blood from 200,618 individuals identifies pervasive positive selection and novel drivers of clonal hematopoiesis," in [Nature Genetics](#).

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