

# Genome wide association study of epigenetic aging rates in blood reveals a critical role for TERT

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Researchers from several institutions, including, UCLA, Boston University, Stanford University and the Institute for Aging Research at Hebrew SeniorLife, analyzed blood samples from nearly 10,000 people to find that genetic markers in the gene responsible for keeping telomeres (tips of chromosomes) youthfully longer, did not translate into a younger biologic age as measured by changes in proteins coating the DNA. This study was recently published in the journal *Nature Communications*.

DNA methylation age is a biomarker of chronological age and predicts lifespan, but its underlying molecular mechanisms are unknown. In this genome-wide association study, researchers found gene variants mapping to five loci associated with intrinsic epigenetic age acceleration (IEAA) and gene variants in three loci associated with extrinsic epigenetic age acceleration. Variants in the gene called Telomerase Reverse Transcriptase (TERT) on chromosome 5 that were associated with older IEAA were also associated with longer telomeres indicating a critical role for TERT in regulating the epigenetic clock, in addition to its established role of compensating for cell replication-dependent telomere shortening.

Co-author Douglas P. Kiel, M.D., M.P.H, Director, Musculoskeletal Research Center and Senior Scientist at Hebrew SeniorLife's Institute for Aging Research said, "We calculated the epigenetic aging rate for

each person using a previously described epigenetic clock method. Next, we related the epigenetic aging rate to millions of genetic locations (SNPs) across all of the chromosomes. Then we studied the SNPs that had very significant associations with epigenetic aging rates. To our surprise, one of these locations was the TERT locus. The finding is surprising because this was not a study of [telomere](#) length. TERT is a subunit of the enzyme telomerase, which is a widely known enzyme because it has been touted as an anti-aging enzyme. It has been called a modern fountain of youth. However, some scientists have pointed out that it is unlikely to become a source of anti-aging therapies. Our study highlights the error in the notion that activation of telomerase (as advocated by some) will cure aging. Instead, our study shows that an anti-aging therapy based on [telomerase](#) expression would be accompanied by continued aging."

Provided by Harvard Medical School

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