

# Sleep and Exercise May Reduce Mutation-Driven Inflammation

Accrual of genetic alterations in blood cells—a process called clonal hematopoiesis—drives inflammation, which can lead to atherosclerosis.

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## At a Glance

- Lifestyle factors may reduce inflammation caused by some mutations in blood stem cells, according to an analysis of human and mouse data.
- The results suggest improving sleep and exercise may curb the health consequences of this inflammation.

When cells divide, their DNA can change or mutate. These mutations can lead cells to multiply faster and outnumber cells without the mutation. As we age, this often occurs in the stem cells that produce our blood and immune cells.

This accrual of genetic alterations in blood cells—a process called clonal hematopoiesis—drives inflammation. Inflammation can lead to atherosclerosis, a buildup of fatty plaques in the arteries. This narrows the arteries and raises risk for heart attacks and strokes. Healthy lifestyle habits can reduce inflammation. However, it remains unclear how lifestyle factors affect clonal hematopoiesis and its consequences.

A team of NIH-funded researchers, led by Dr. Cameron McAlpine of the Icahn School of Medicine at Mount Sinai, recently set out to examine this. The results were published on June 10, 2026, in [Nature](#).

The researchers looked at data on physical activity and blood genomic alterations in more than 90,000 people participating in either the UK Biobank study or the NIH-run All of Us research program. They studied whether moderate-to-vigorous physical activity was linked with clonal hematopoiesis caused by mutations in a gene named DNMT3A or by mutations in other genes.

People who got more moderate-to-vigorous physical activity each day were less likely to have

clonal hematopoiesis caused by common mutations in one of three genes. However, exercise did not affect clonal hematopoiesis caused by mutations in DNMT3A.

The researchers also investigated how sleep and exercise affected clonal hematopoiesis in mice. The mice had mutations in one of four genes: Jak2, Tet2, Trp53 or Dnmt3. These mutations caused genetic alterations in blood cells to accumulate. Poor sleep increased the accumulation in mice with mutations in Jak2 and Tet2 but not the other two genes. Exercise decreased that accumulation. This suggests that different mutations result in different sensitivities to lifestyle factors.

Plaque buildup in arteries was worse in mice with clonal hematopoiesis. When clonal hematopoiesis was caused by mutations in three of the genes, poor sleep exacerbated, and exercise reduced, plaque buildup. Sleep and exercise did not affect plaque buildup in mice with the Dnmt3a mutation, consistent with the research team's findings in humans.

The team focused on a mutation in the Jak2 gene. The mutation led to genetic alterations in blood cells and plaque buildup that were affected by sleep and exercise. They found that in immune cells from mice with this mutation, poor sleep boosted inflammasome activity. The inflammasome is a molecular machine that drives inflammation. The resulting inflammation led to worse plaque buildup.

When mice with the same mutation exercised, a particular set of neurons in a brain region called the locus coeruleus was more active. This boosted the amount of the hormone noradrenaline in the mice's blood. Noradrenaline reduced inflammation, thereby reducing plaque buildup.

The study's results suggest that our sleep and exercise habits may influence clonal hematopoiesis and cardiovascular health. Importantly, those effects appear to depend on which specific mutation is driving clonal hematopoiesis.

"We now have the ability to use genetics to not only develop new therapies, but to tailor lifestyle management and treatment," McAlpine says.

This [research summary](#) was published by the National Institutes of Health on July 14, 2026.