

Long-term physical activity has an anti-aging effect at the cellular level

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Intensive exercise prevented shortening of telomeres, a protective effect against aging of the cardiovascular system, according to research reported in *Circulation: Journal of the American Heart Association*.

Researchers measured the length of telomeres — the DNA that bookends the chromosomes and protects the ends from damage — in blood samples from two groups of professional athletes and two groups who were healthy nonsmokers, but not regular exercisers.

The telomere shortening mechanism limits cells to a fixed number of divisions and can be regarded as a "biological clock." Gradual shortening of telomeres through cell divisions leads to aging on the [cellular level](#) and may limit lifetimes. When the telomeres become critically short the cell undergoes death. The 2009 [Nobel Prize](#) in Physiology or Medicine was awarded to researchers who discovered the nature of telomeres and how chromosomes are protected by telomeres and the enzyme telomerase.

"The most significant finding of this study is that physical exercise of the professional athletes leads to activation of the important enzyme telomerase and stabilizes the telomere," said Ulrich Laufs, M.D., the study's lead author and professor of clinical and experimental medicine in the department of internal medicine at Saarland University in Homburg, Germany.

"This is direct evidence of an anti-aging effect of physical exercise.

[Physical exercise](#) could prevent the aging of the [cardiovascular system](#), reflecting this molecular principle."

Essentially, the longer telomere of athletes is an efficient telomere. The body's cells are constantly growing and dividing and eventually dying off, a process controlled by the [chromosomes](#) within each cell. These chromosomal "end caps" — which have been likened to the tips of shoelaces, preventing them from fraying — become shorter with each [cell division](#), and when they're gone, the cell dies. Short telomeres limit the number of cell divisions, Laufs said. In addition, the animal studies of Laufs and colleagues show that the regulation of telomere stabilizing proteins by exercise exerts important cellular functions beyond the regulation of telomere length itself by protecting from cellular deterioration and programmed cell death.

In the clinical study, researchers analyzed 32 professional runners, average age 20, from the German National Team of Track and Field. Their average running distance was about 73 kilometers (km), a little over 45 miles, per week.

Researchers compared the young professional athletes with middle-aged athletes with a history of continuous endurance exercise since their youth. Their average age was 51 and their average distance was about 80 km, or almost 50 miles, per week.

The two groups were evaluated against untrained athletes who were healthy nonsmokers, but who did not exercise regularly. They were matched for age with the professional athletes.

The fitness level of the athletes was superior to the untrained individuals. The athletes had a slower resting heart rate, lower blood pressure and body mass index, and a more favorable cholesterol profile, researchers said.

Long-term exercise training activates telomerase and reduces telomere shortening in human leukocytes. The age-dependent telomere loss was lower in the master athletes who had performed endurance exercising for several decades.

"Our data improves the molecular understanding of the protective effects of [exercise](#) on the vessel wall and underlines the potency of physical training in reducing the impact of age-related disease," Laufs said.

Source: American Heart Association ([news](#) : [web](#))

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