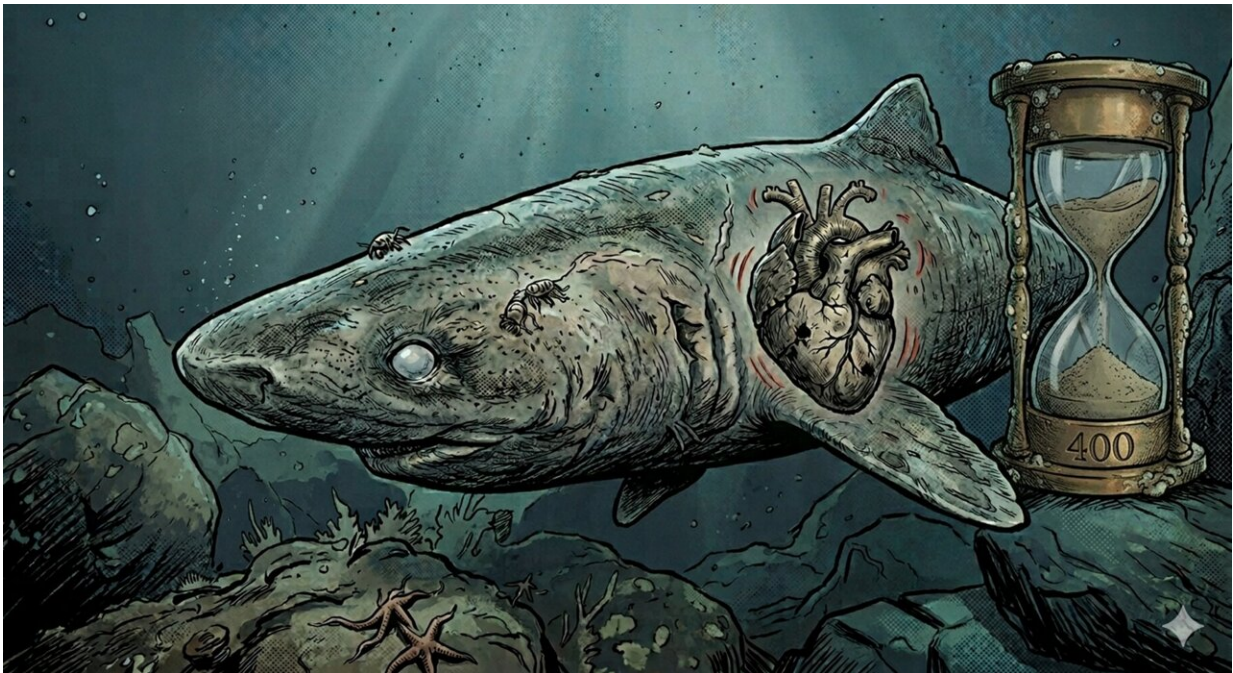


# How can a heart beat for centuries? A lesson from the Greenland shark

April 28 2026, by Kerstin Wagner

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The Greenland shark (*Somniosus microcephalus*), the longest-living vertebrate (~400 years), shows strong cardiac aging. Yet this doesn't limit its lifespan, suggesting exceptional resilience—maintaining function despite age-related damage. Credit: FLI / Kerstin Wagner; AI-generated with Google Gemini

The Greenland shark (*Somniosus microcephalus*) is one of the longest living vertebrates on Earth, with an estimated lifespan of up to 400 years or more. Its extraordinary lifespan, extremely slow growth, very low

metabolism, minimal swimming activity, and late sexual maturity—reaching about 150 years—make it a unique model organism for researching aging processes and the biological principles of longevity.

Early studies of long-lived animal species have shown that these species often possess particularly efficient systems for [DNA repair](#), tumor defense, and immune system regulation. Initial genomic analyses of the Greenland shark suggest that genes associated with anti-inflammation, cancer protection, and the stabilization of cellular processes also play a central role in this species.

An international research team led by Prof. Alessandro Cellerino of the Scuola Normale Superiore in Pisa, Italy, and Leibniz Chair at the Leibniz Institute on Aging—Fritz Lipmann Institute (FLI) in Jena, Germany, has now systematically investigated age-related changes in the heart tissue of the Greenland shark in detail for the first time.

The study, now [published](#) in *Aging Cell*, focused on one question: whether the Greenland shark is protected against typical signs of cardiac aging—such as fibrosis, oxidative stress, and functional decline—or whether it is able to compensate for the deleterious effects of aging.

For comparison, the researchers analyzed two other fish species in addition to the Greenland shark: the short-lived [turquoise killifish](#) (*Nothobranchius furzeri*), an established model organism for accelerated aging, and the deep-sea shark, also known as the lanternshark (*Etmopterus spinax*), which is phylogenetically related to the Greenland shark but has a significantly shorter lifespan ~ 10 years.

The study examined classic markers of cellular aging, including lipofuscin deposits and 3-nitrotyrosine as an indicator of oxidative stress, to draw conclusions about how efficiently their cells can prevent or

repair damages. Animals with low levels of these markers often exhibit better antioxidant defense systems, more efficient protein and cell repair mechanisms, and a lower accumulation of cellular damage over time.

## **Age-related changes in heart tissue**

Results from the study suggest that the heart of the Greenland shark exhibits significant structural and molecular changes typically associated with aging processes. These included pronounced fibrosis of the heart muscle, i.e., increased deposition of connective tissue. Such changes can reduce the elasticity of heart tissue and impair pumping function in the long term. In addition, a significant accumulation of lipofuscin (age-related pigment) was observed in the heart muscle cells. This breakdown product forms when cellular components are not completely eliminated over the lifespan and is considered a classic sign of cellular aging.

There were also indications of mitochondrial damage—the "powerhouses" of the cells—as well as enlarged lysosomes, which are responsible for the breakdown and recycling of cellular material. Both are also indications that the cells have been under heavy strain over a long period of time. Additionally, elevated levels of 3-nitrotyrosine, a marker for oxidative and nitrosative stress, were found. This suggests that increased protein-altering oxidative processes have taken place in the heart tissue.



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"All in all, the Greenland shark samples analyzed showed clear signs of classic aging at the molecular and tissue levels," explains Prof. Cellerino. "These findings demonstrate that this species also undergoes aging processes in its heart tissue."

It is striking, however, that these structural and molecular changes do not appear to be accompanied by a corresponding loss of function. Despite the clear structural and molecular signs of aging, the studied animals were alive and swimming. "The same lesion in a human heart would not be compatible with life," says Prof. Cellerino. "The findings therefore suggest that the Greenland shark is not protected against aging-associated lesions but is remarkably capable of compensating for its effects."

In the context of earlier genomic studies on the Greenland shark, it was shown that the Greenland shark possesses well-developed mechanisms to repair damage to its DNA and maintain genetic stability. Among other things, changes were found in [genes involved in DNA repair](#) and cell protection, some of which are present in the form of additional gene copies. These could help detect and correct genetic damage more efficiently, thereby protecting the cells over very long periods of time.

The study results suggest that the Greenland shark does not become extremely old because it avoids the aging process, but rather because it can effectively buffer the consequences of aging over very long periods of time. Its longevity is therefore primarily an expression of high biological resilience, which allows it to largely maintain body functions despite progressive cellular damage. Understanding the mechanisms that enable biological resilience could provide entirely novel pathways to promote healthy aging.

"Research on the Greenland shark was spearheaded by Professor John Fleng Steffensen from the University of Copenhagen, who described its extreme longevity and has been a powerful force in promoting research on this species. He passed away last year, I dedicate this work to his memory," concludes Prof. Cellerino.

**More information:** Elena Chiavacci et al, Resilience to Cardiac Aging in Greenland Shark *Somniosus microcephalus*, *Aging Cell* (2026). [DOI: 10.1111/accel.70505](https://doi.org/10.1111/accel.70505)

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