Why big dogs age faster

Large-scale Dog Aging Project reveals how 'jumping genes' underlie differences in lifespan and disease risks across breeds

By Joe Caspermeyer, ASU News
October 8, 2026

Across mammals, increased species size typically means a longer lifespan, ranging from only a couple of years for mice to almost 200 years for some whales.

However, when we look within species, we often see the opposite — smaller individuals outliving larger individuals. And we see this in dogs.

Dog lovers and owners have long experienced this exception, as larger dogs breeds tend to grow faster and die younger than smaller dogs. Therefore, scientists have been hard at work to explore and understand the underlying causes of these so-called “dog years” that can shorten their companion time with people.

“Dogs provide an extraordinary model for understanding aging because they show dramatic variation in lifespan within a single species,” said senior author of the study Noah Snyder-Mackler, a professor at Arizona State University’s School of Life Sciences and Center for Evolution and Medicine.

Not only do big dogs die younger, but they are also more susceptible to many age-related diseases.

Now we may know why.

A new study led by researchers at ASU offers the first compelling molecular explanation of why large dogs live shorter lives: The answer may lie in how hallmarks of aging reshape dog DNA. The study was published in Science.

And what scientists can learn about dog aging can provide an important window into human aging, too.

“Because they are companion animals, they can also be a powerful model with immediate relevance to human health," Snyder-Mackler said. "Living with us means that we share experiences and environments, while our love for our pets means we provide them food, exercise and lifelong health care – meaning we can translate many findings in our shorter-lived pets to humans.”

Jumping genes

Drawing on data from 894 dogs enrolled in the Dog Aging Project, a team of scientists mapped genome-wide patterns of DNA methylation.

DNA methylation is part of the epigenome, which influences how much genes are turned on or off without changing the sequence of DNA in each gene. The epigenome is also responsive to environmental cues, such as diet or stress, and, crucial to this work, DNA methylation is a well-established chemical signpost of aging and DNA regulation.

Their findings reveal that aging is associated with the widespread loss of these regulatory marks over time, particularly in regions of the genome known as “jumping genes” or transposable elements. A class of these jumping genes called LINE1s were found to be a key component of biological aging differences.

The LINE1s can copy and insert themselves throughout the genome, jumping from chromosome to chromosome and damaging DNA in the process. While usually kept in check by DNA methylation, their activity can increase when these regulatory marks are lost — a process that has been linked to genomic instability, cancer and other age-related diseases.

“What we found is that the epigenetic regulation of transposable elements — especially LINE1s—appears to be a major factor shaping how quickly different dogs age,” Snyder-Mackler said.

The study found that more than 40% of LINE1-associated regions in the genome lose methylation with aging, making them the most affected class of transposable elements. This loss is not evenly distributed across dogs: Larger breeds experience significantly faster declines.

On average, giant dog breeds lost approximately 35% more LINE1 methylation per year than small breeds.

“This is one of the clearest molecular signatures we’ve seen that aligns with the well-known size-lifespan tradeoff in dogs,” said co-author Blaise Mariner, also a researcher at ASU’s School of Life Sciences and Center for Evolution and Medicine. “It suggests that faster aging in larger dogs may be driven, at least in part, by reduced control over these ‘jumping genes.’”

Sex and aging

The study also uncovered surprising differences between females (which contain two X chromosomes, or XX) and males (which are XY).

LINE1s on the X chromosome were found to be more methylated in males than in females. This suggests that females may experience higher activity of these elements, potentially influencing sex-specific aging patterns.

“This was an unexpected result,” said Brianah McCoy, who co-led this work as part of her PhD research at ASU. “It challenges some of our assumptions about how the X chromosome is regulated and highlights the complexity of epigenetic aging.”

Beyond specific findings, the research underscores the importance of epigenetics in shaping health and longevity. While genetic sequences remain largely fixed, epigenetic marks change over

time and in response to environmental factors, offering a powerful lens into the biology of aging.

The study's scale was also notable as one of the largest of its kind. By analyzing 864 dog DNA genomes, more than 3 million methylation sites across a large and diverse cohort of dogs had to be mapped and sequenced. Only through this huge undertaking was the team able to detect patterns that would be invisible in smaller datasets.

"Large, collaborative efforts like the Dog Aging Project are essential for uncovering these kinds of insights," Snyder-Mackler said. "They allow us to connect molecular changes to real-world variation in health and lifespan."

Dog companions

Ultimately, the findings could have implications beyond dogs to translate back to their human companions.

"Our work suggests that transposable elements may be a fundamental part of the aging process across mammals," Snyder-Mackler said. "If that's the case, targeting these elements or the mechanisms that regulate them could be a promising avenue for future therapies to extend the health span in humans."

While more research is needed to determine whether LINE1 activity is a cause or consequence of aging, the study provides strong evidence that epigenetic changes in these genomic regions are a hallmark of biological aging — and a potential driver of its variability.

As scientists continue to unravel the molecular mechanisms of aging, one thing is becoming clear: The secrets to longer, healthier lives may be hidden not just in our genes, but in how they are regulated over time.

This story originally appeared on ASU News.

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Main image



Not only do big dogs die younger, but they are also more susceptible to many age-related diseases. Now we may know why. Photo by Charlie Leight/ASU News

Text image(s)



ASU researcher Noah Snyder-Mackler has long been interested in better understanding why animals age — and applying that knowledge to human aging. His new study relied on data from 864 dogs enrolled in the Dog Aging Project. Here, he poses with his two dogs, 7-year-old Homer (bottom) and 4-year-old Juno (top). Photo by Deanna Dent/Arizona State University



After listening to a lecture by ASU Professor Noah Snyder-Mackler (left), Mirabella resident and retired attorney D. P. Stoller (right) hands out treats to Snyder-Mackler's dogs, Juno and Homer. Snyder-Mackler's latest research explores why larger dogs live shorter lives, and the findings could provide insight into human aging, too. Photo by Charlie Leight/ASU News