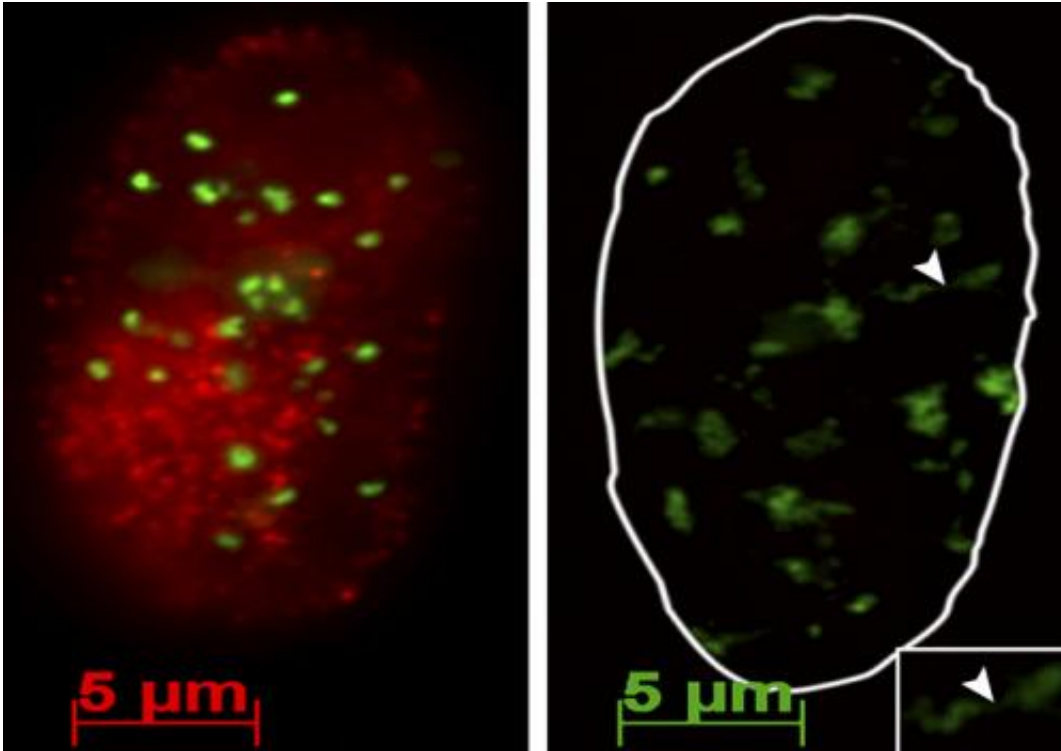


Aging cells unravel their DNA

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Satellite DNA (green) is compact in a normal proliferative cell (left) but distended in a nonproliferative senescent cell (right). A study in *The Journal of Cell Biology* identifies a common marker of senescence that could have important implications for aging and cancer. Credit: Swanson et al., 2013

Senescent cells, which are metabolically active but no longer capable of dividing, contribute to aging, and senescence is a key mechanism for preventing the spread of cancer cells. A study in *The Journal of Cell Biology* identifies a common, early marker of senescent cells that could

have important implications for tumor suppression and aging-related diseases like Progeria.

Senescent cells permanently exit the cell cycle, a process that can be triggered by the cellular changes associated with aging or by other stresses such as the expression of cancer-promoting oncogenes. Despite the importance of senescence for both aging and [tumor suppression](#), however, researchers have failed to identify any distinguishing features that are common to all types of [senescent cells](#).

Researchers from UMass Medical School discovered that the satellite DNA found at human and mouse centromeres—the points where chromosomes connect to microtubules during cell division—unraveled from its normal compact state as cells entered senescence. This unraveling—which the researchers termed senescence-associated distension of satellites, or SADS—occurred regardless of how senescence was induced and appeared to occur early in the process of cell cycle exit. Strikingly, cells from Progeria patients formed SADS as they exited the [cell cycle](#), suggesting that these prematurely arrested cells follow the same senescence pathway as normally aging cells.

The extensive unfolding of structures critical for cell division could thus prove key to inhibiting cell proliferation, in the context of both aging and limiting the proliferation of tumor cells.

More information: Swanson, E.C., et al. 2013. J. Cell Biol. [DOI: 10.1083/jcb.201306073](https://doi.org/10.1083/jcb.201306073)

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