WHY DO WE AGE AND DIE?

Biologists have been arguing about it for seventy years, and they still don't agree.
Three main theories have been on the table since the 1950s.
The first, coming from Peter Medawar (1), says aging is essentially evolutionary negligence. In the wild, most animals die young — from predation, starvation or disease. So, natural selection barely notices what happens to old individuals. Harmful genetic variants that only cause damage late in life simply accumulate in the genome, invisible to selection. Aging, according to this view, is what happens when no one is watching.
The second, proposed by George Williams (2), is darker. Some genes are good for the young you and bad for the old you. They get selected because the reproductive benefit early outweighs the survival cost later. You're not just passively aging; evolution actively traded your old age for your reproductive success. A Faustian bargain encoded in your DNA.
The third, by Tom Kirkwood (3), is the most pragmatic. The body is a disposable soma. Energy is finite. You can invest it in reproduction, or in maintaining the machinery of the body. Evolution, reliably, favored reproduction. The biological systems keeping you alive were never built to last forever; they were built to get you to reproductive age and a bit beyond.
Three theories. All plausible. All partially supported by observations. But for decades, what was missing was direct genetic evidence, specific variants, specific mechanisms, specific windows of time in which each theory actually operates.
That's exactly what a study recently published in Nature by Arends et al. (4) set out to do.
The researchers followed 6,400 genetically diverse mice from puberty to natural death, measuring survival day by day. Then, using a method that maps mortality across successive survival cohorts, they essentially asked the question: which genes matter at 400 days, which at 600and which at 800. They identified 59 genomic loci that modulate aging and lifespan.
And here is where it gets interesting.
Some loci show sign reversals: an allele that reduces mortality at young age increases it at old age. That's antagonistic pleiotropy. Williams was right: the Faustian bargain is real, and it's written at specific addresses in the genome.
Other 30 loci, they call Soma, mediate a direct trade-off between body mass during reproductive life and subsequent lifespan. Bigger, heavier mice die earlier. Kirkwood was right too: the energy invested in growth is energy not invested in longevity.
Some loci, on the other hand, become relevant only late in life, well past the reproductive phase, exactly where Medawar predicted selection would go blind. Mutation accumulation is confirmed at the genomic level.
What this study shows, for the first time in such a detail, is that all three mechanisms operate simultaneously, but in different genes, at different ages and differently in males and females through almost entirely separate genetic networks.
Aging is not a single thing. It is not a program. It is not a clock. It is the sum of evolutionary compromises that made sense when selection pressures were about surviving to reproduce, not about living to 90.
The question "why do we age?" turns out to have three answers, all true, all running in parallel, all written into the same genome.

(1) Medawar, P.B. (1952). An Unsolved Problem of Biology. H.K. Lewis & Co., London.
(2) https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1558-5646.1957.tb02911.x.
(3) https://www.nature.com/articles/270301a0
(4) https://www.nature.com/articles/s41586-026-10407-9