

Evolution Didn't Design You for Long Life — Can Science Change That?

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STORY AT-A-GLANCE

- › Jacob Kimmel, president and co-founder of NewLimit, a company that develops reprogramming medicines for aging, discusses how evolution only optimized humans to survive through reproduction, not a long lifespan
- › Aging reflects weak evolutionary pressure for longevity, which leaves your cells vulnerable to decline after their most productive years
- › High mortality in early human history favored intelligence for survival, with fluid intelligence peaking in early adulthood to match evolutionary priorities rather than preserving cognitive strength into later life
- › Unlike microbes, humans didn't evolve antibiotics due to genetic stability constraints. Instead, your adaptive immune system emerged as a flexible defense, leaving traces of ancient viral battles in your DNA
- › Epigenetic reprogramming challenges evolution's limits by reversing cellular decline, offering a way to extend healthspan

Human life today is the result of millions of years of evolution, shaped by forces that favored survival and adaptation. You might think that the same process would have extended health and resilience into later life. Yet longevity was not a priority in the evolutionary blueprint, and the result is a body that wears down with age rather than one designed for lasting vitality.

This paradox is the focus of an interview on the Dwarakesh Podcast, featuring Jacob Kimmel, president and co-founder of NewLimit, a biotechnology company developing reprogramming medicines for aging.¹ In their discussion, Kimmel shares his insights into how evolution shaped the limits of human lifespan and what modern science can do to change that trajectory.²

The Evolutionary Trade-Offs That Left Humans Aging Fast

Your body's aging process reflects choices made by evolution, balancing survival against a complex web of constraints. Kimmel identifies three key factors that explain why natural selection didn't equip you with a longer, healthier lifespan. By viewing evolution as an optimization process with limited resources, he unpacks why your cells and systems decline over time, revealing trade-offs that favored immediate needs over long-term vitality.³

- **Evolution only needed you to reach reproduction** — Natural selection favored traits that carried humans into their childbearing years and allowed them to raise children, but it applied little pressure beyond that point. As Kimmel explains, in human and primate history, the daily chance of dying (what he called the “baseline hazard rate”) from infection, predators, or accidents was extremely high.

If most lives ended around 40, there was no evolutionary incentive to shape traits that would keep you vigorous at 60. “The number of individuals in the population that are going to make it later in that lifespan, where using some of your evolutionary updates to try and push your lifespan upward, is relatively limited,” Kimmel said.⁴

- **This high hazard rate also influenced traits like intelligence** — Longer childhoods made it possible for humans to develop larger, more capable brains, but stretching adolescence too far carried the risk of dying before reproduction. This is reflected in your fluid intelligence, the ability to reason, solve new problems, and think flexibly without relying on prior knowledge or experience,⁵ which peaks around your 20s or 30s.

Evolution optimized for cognitive prowess when you were most likely to contribute to the group, not later in life. Mathematical discoveries often occur before age 30, suggesting your brain's peak aligns with the age of maximum population contribution during evolutionary history.

- **Evolution may have even favored shorter lifespans** — Kimmel explains that, from the perspective of the “selfish gene,” older individuals who are less fit and still consuming resources could reduce a group's overall survival.

If you live longer but contribute fewer calories or gather fewer resources than younger members, your extended presence actually lowers the group's fitness. In this sense, evolution tends to favor turnover, giving younger and more productive individuals the chance to propagate genes more effectively. According to Kimmel:

“There is a notion by which a population being laden demographically with many aged individuals, even if they did have fecundity persisting out some period later in life, is actually net negative for the genome's proliferation and that really a genome should optimize for turnover and population size at max fitness.”⁶

- **Longevity sits within the constraints on evolution's optimization process** — Kimmel describes the genome as a set of parameters and natural selection as an optimizer with limits. Mutation rates need to stay low to prevent catastrophic errors such as cancer, and small population sizes restrict how many genetic variants can be tested.

At the same time, your ancestors were locked in a constant battle with infectious disease, which absorbed much of evolution's attention. These constraints left little room to fine-tune traits related to longevity, even if longer life might have offered some benefit.

Kimmel stresses that aging is not a single flaw that evolution could have easily corrected, but a multi-causal process shaped by many layers of molecular regulation. The decline in your cells' function comes from accumulated changes in gene expression

and resilience, not from one defect. This complexity explains why evolution didn't simply "fix" aging and why interventions need to target multiple pathways to extend your healthy years.

Why Humans Didn't Evolve Their Own Antibiotics

When Kimmel discussed the evolutionary limits on human biology, he pointed to antibiotics as an instructive example. Your body's ability to fight infections relies on intricate defenses, but you might wonder why evolution never equipped you with built-in antibiotics like those produced by microbes. Instead, your immune system evolved as a flexible alternative to antibiotics, shaped by pathogens.⁷

- **Microbes produce antibiotics through a unique evolutionary advantage** — With vast population sizes and extremely high mutation rates, bacteria and fungi engage in chemical arms races, churning out molecules like antibiotics to outcompete rivals. This process allows microbes to rapidly adapt, producing diverse compounds that target specific competitors in their environment.
- **Humans, by contrast, could never evolve along this path** — Our mutation rates need to stay relatively low in order to protect the stability of our complex genomes. Rapid mutation at microbial levels would lead to catastrophic consequences, most notably uncontrolled cancer. This constraint means that while microbes thrive on variation, mammals depend on genetic stability to survive from one generation to the next.
- **Because of these biological limits, humans developed a different defense system** — Instead of producing chemical antibiotics internally, you evolved an adaptive immune system capable of learning and remembering threats. This approach provides flexibility without relying on high mutation rates. It also allows your body to respond to a wide variety of pathogens across your lifetime, even as they change and adapt.

- **Your DNA still carries the marks of past battles with pathogens** — Over millions of years, infectious diseases shaped survival, and the genetic record shows the defenses your ancestors developed against those threats. These remnants serve as evidence of how strongly microbes directed human evolution, even when the pathogens themselves disappeared long ago. Kimmel points to one striking example:

*"We have a gene called TRIM5alpha. It actually binds an endogenous retrovirus that is no longer present, but was at one point actually resurrected by a bunch of researchers. It was demonstrated that this is the case. We have this endogenous gene which basically fits around the capsid of the virus like a baseball in a glove and prevents it from infecting."*⁸

Targeting the Epigenome as a Path to Youthful Function

Evolution has set boundaries around what your body can develop. Kimmel notes that one of the most promising ways to move beyond those boundaries is by targeting the epigenome, the layer of chemical and structural markers that regulates which of your genes are turned on or off.⁹

- **The epigenome explains how identical DNA produces different cell types** — For instance, a kidney cell and an eye cell carry the same genetic code, yet they perform distinct tasks because the epigenome programs them differently.
- **The main levers of this system are transcription factors** — These are proteins that bind to DNA and direct gene activity, turning certain genes on and others off. Kimmel describes them as conductors of an orchestra — they don't perform the functions themselves but determine which instruments play, when they enter, and how they interact. In the same way, transcription factors set the rhythm of cellular behavior.

- **Epigenetic reprogramming restores youthful patterns of gene activity** — With age, the epigenome drifts, leading to weaker cell performance. By steering transcription factors in specific ways, the goal is to return aged cells to a state where they function as effectively as they did when they were young, without altering the DNA sequence itself. For example, a liver cell would remain a liver cell but regain the ability to clear toxins efficiently, and an aged T cell would recover its capacity to fight infections.
- **Kimmel contrasts this with the Yamanaka factors** — Discovered by scientist Shinya Yamanaka, these factors strip away a cell's specialized identity, turning it into a blank slate that could become any cell type. Kimmel notes that this process, while powerful, carries risks because it disrupts the cell's role in your tissues.
- **The size of that space is one of the biggest scientific challenges** — There are thousands of transcription factors, and when you consider the possible combinations, the number of potential interventions rises into the trillions. Testing every possibility in the lab is impossible, which is why computational tools have become essential.
- **This is where computational tools come into play** — Machine learning models can analyze massive amounts of experimental data and help pinpoint which transcription factor combinations are most promising to test. Instead of working blindly through endless options, researchers can use this technology to chart a focused path.

In this sense, the effort is not just about understanding aging, but about building a new kind of toolkit for medicine — one that can push discovery forward and expand what treatments are possible.

Approaches to Cellular Delivery

Delivering transcription factors into your cells is another central challenge to epigenetic reprogramming. Today, there are two main modalities for doing this, but they rely on technologies originally developed for other fields of medicine, such as gene therapy and vaccines, and both have trade-offs.¹⁰

- **Lipid nanoparticles (LNPs)** — These “fat bubbles” that resemble cell membranes are taken up by tissues like the liver, which naturally absorb fat. They are the same technology used in mRNA vaccines, where they carry RNA into cells. In reprogramming, they can deliver RNA instructions for transcription factors.

However, Kimmel points out that LNPs have physical limits in how they travel through the body, making them unlikely to serve as a lasting solution. I’ve also covered their risks before, including in the context of mRNA shots, in “[HIV mRNA Vaccines Continue to Fail in Clinical Trials](#).”

- **Viral vectors** — Another common method borrows from viruses, which have evolved specifically to enter cells. One example is AAV (adeno-associated virus), which can carry DNA payloads into certain cell types. Kimmel likens AAV to a small delivery truck — it can bring in whole genes but has limited cargo space.

Researchers engineer these viral sequences further to restrict where the genetic payload is active. However, viral vectors always carry some degree of immunogenicity, raising risks of immune reactions and toxicity.

- **Future solutions may resemble the systems your own body already uses** — The immune system already has cells that patrol tissues, sense problems, and release targeted responses. These engineered immune cells could eventually take on the role of couriers for reprogramming therapies, delivering them with precision and safety that current methods cannot achieve. According to Kimmel:

“Ultimately, we’re probably going to have to solve delivery the way that our own genome solved delivery. We have the same problem that arose during evolution ... We have cell types in our body, T cells and B cells, which are

effectively engineered by evolution to run around, invaginate whatever tissues they need to.”¹¹

While delivery remains one of the practical hurdles for reprogramming therapies, Kimmel also points to a broader challenge in medicine — the pace of discovery itself. Even if you solve how to move therapies into cells, developing those therapies in the first place is slowed by the cost and limitations of traditional lab work. This is where he introduces the idea of “virtual cells.”

How Virtual Cells Could Transform Drug Discovery

“Eroom’s Law” is a term coined by inverting Moore’s Law, which Kimmel explains is the “doubling of compute density on silicon chips every few years.” That steady progress has fueled decades of rapid advances in technology. In biopharma, however, the opposite trend has held true. Since the 1950s, the number of new medicines discovered per billion dollars invested has steadily declined, and this decline has persisted across multiple technological eras.¹²

- **Computational models help reduce the trial-and-error bottleneck** — A major challenge in drug discovery is the dependence on trial and error in living systems. Each experiment is costly, slow, and narrow in scope, leaving progress constrained by the physical bottlenecks of the lab.

Kimmel explains that accurate computational models could shift much of this process into silico, allowing researchers to simulate biology with far greater speed and scale than traditional experiments.

- **What virtual cells are** — Virtual cells are computer-based simulations of how real cells behave. By capturing how genes are expressed, how proteins interact, and how pathways respond, they create a digital environment where interventions can be tested.

In practice, this means scientists could simulate how transcription factors or other therapies change gene activity and cell function, then filter out unpromising approaches before moving to the lab.

- **Virtual cells expand what can be tested** — The benefit is not only speed but also the ability to explore ideas that would be impractical in physical labs. Entire classes of hypotheses could be tested computationally, widening the scope of discovery beyond what current resources allow. This doesn't eliminate the need for lab work, but it means that only the most promising interventions reach that stage, saving time and cost.
- **Kimmel frames this shift as essential to breaking free of Eroom's Law** — Without it, drug development will remain constrained by slow, expensive cycles that hinder innovation. With it, medicine could move toward a future where discovery scales more like computing, driven by the ability to model biology in silico.

By modeling entire cells in silico, the trial-and-error cycle of drug discovery could be transformed. For more on how emerging technologies are reshaping health, see [“Smart Medicine — Harnessing Augmented Reality and AI to Transform Health.”](#)

Economic Approaches to Future Treatments

Finally, the interview shifts from the science of reprogramming to the economics of how future medicines might be brought to patients. As medical science advances, the way you access and pay for transformative therapies is poised to evolve. Kimmel outlines the challenges and opportunities in funding and delivering therapies that could extend your healthy years.¹³

- **One model already under discussion is pay-for-performance** — This is where the cost of a therapy depends on its real-world effectiveness for you. For long-lasting treatments like those targeting aging, insurers face challenges because patients

may switch providers before they experience benefits. Linking payment to measurable health improvements ensures you receive therapies that work while addressing payers' concerns about covering high upfront costs.

- **Another shift could involve direct-to-consumer access** — In this model, drugs might be made available to patients in ways that resemble consumer products, bypassing some of the traditional channels that rely heavily on insurers and intermediaries. This approach could simplify your access to innovative drugs, particularly for chronic conditions or aging-related therapies.
- **The development of these therapies often begins with small biotech firms** — “The industry has sort of bifurcated where smaller biotechs like ours take on most of the early discovery,” Kimmel said. Meanwhile, larger firms step in later to manage clinical trials, regulatory approval, and global distribution. This division of labor reflects how risk and expertise are distributed in the sector.

The future of medicine depends not only on scientific breakthroughs but also on how therapies reach you. From the limits set by evolution to the use of epigenetic reprogramming, delivery systems, and virtual models of biology, each part of the discussion points to the same conclusion — your cells already hold the capacity for repair. With the right inputs and carefully designed tools, that potential can be unlocked to restore youthful function and extend not just lifespan but healthspan.

Frequently Asked Questions (FAQs) About Evolution, Aging, and Cellular Reprogramming

Q: Why didn't evolution make humans live longer?

A: Evolution shaped your body to survive long enough to reproduce and raise children, not to remain healthy for decades afterward. High risks from infections, predators, and accidents meant most people never lived past 40, so there was little

pressure to optimize traits for old age.

Q: What is fluid intelligence, and why does it decline with age?

A: Fluid intelligence is your ability to solve new problems and think flexibly without relying on past experience. It peaks in your 20s or 30s, when evolution most strongly favored cognitive abilities that supported survival and group contribution. As you age, this capacity naturally declines because evolution placed less value on maintaining peak cognition later in life.

Q: Why didn't humans evolve their own antibiotics?

A: Microbes like bacteria and fungi produce antibiotics because they mutate quickly and exist in huge populations. You can't adopt that strategy because high mutation rates would destabilize your genome and increase your risk of cancer. Instead, you evolved an adaptive immune system that learns and remembers threats across your lifetime.

Q: What is epigenetic reprogramming?

A: Epigenetic reprogramming targets the epigenome, the chemical and structural markers that control which of your genes are switched on or off. By adjusting these markers, aged cells can be nudged back toward youthful patterns of activity without changing their DNA sequence.

Q: What are virtual cells, and why do they matter?

A: Virtual cells are computer-based simulations of how real cells behave. They let researchers model gene activity, protein interactions, and cellular pathways in silico. This allows millions of interventions to be tested virtually before the best ones move into lab experiments, boosting efficiency.

Sources and References

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