David Remnick liked this story so much he called me at home on a Saturday morning to congratulate me on it. But then another editor at the magazine decided it was too much like a story Malcolm Gladwell had written eight years earlier. Said I: I told you about Gladwell's story when I pitched the piece, and you didn't think it was a problem then. And besides, in the field of genetics, eight years is a lifetime. Everything has changed. (And in fact, the two stories were about entirely different technologies.) No matter: the story died.

Dan Baum
1650 Lombardy Drive
Boulder, CO 80304
(303) 546-9800
(303) 917-5024 mobile
danbaum@me.com

Aging

Late one night in 2002, Paul and Ringo died in a petri dish in a San Francisco laboratory. They were nematodes, parasitic roundworms about the size of the comma in this sentence, and their deaths would not ordinarily have been newsworthy. Millions of Caenorhabditis elegans are bred and slaughtered each year in laboratory experiments around the world. But scientists had manipulated the genes of Paul and Ringo to extend the worms' lives. C. elegans normally die after twenty days; Paul and Ringo lived six times that long. What's more, they wriggled like young healthy worms right up until the end, suggesting that they not only got more time, they got good time. They didn't reach senescence at the normal age and then linger in

decrepitude. Their aging seemed to have been slowed to one day for every six that they lived.[1] It was as though a human being had lived to four hundred, skiing and playing tennis well past three hundred and fifty.

The leap from worms to people may not be as farfetched as it sounds. All animals, ranging from single yeast cells on up to Lance Armstrong, share remarkably similar "genetic pathways." To give but one example, you can put the gene for Huntington's disease, a human ailment, into a roundworm and the roundworm will get Huntington's disease.[2] The 2002 Nobel Prize in Physiology or Medicine was awarded for the discovery that the same genetic mechanism that controls organ development and the orderly death of cells in C. elegans also works in more complicated species, including humans.[3] Figure out how to manipulate the genes in C. elegans, a nine-hundred-and-sixty-nine-cell animal a millimetre long, and you're on the road to figuring out how to do the same in people. Scientists are beginning to speculate -- publicly, and sometimes in front of rooms full of venture capitalists -- that we are on the brink of genetically engineering people to live radically longer and healthier lives. In the view of these geneticists,

---

[1] Cynthia Kenyon, UCSF., work: 415-476-9250, home: 415-641-9174, cell: 415-271-0644 or ckenyon@biochem.ucsf.edu. Her assistant is Myra Melville, 415-514-4078
[2] Cynthia Kenyon, UCSF., work: 415-476-9250, home: 415-641-9174, cell: 415-271-0644 or ckenyon@biochem.ucsf.edu. Her assistant is Myra Melville, 415-514-4078
[3] file "2002 Nobel prize"

aging is a disease – and potentially a curable one. The biggest preventable cause of death, in other words, may not be tobacco after all, but time.

The very idea flies in the face of two principles that form the bedrock of modern science. The Second Law of Thermodynamics states that things fall apart over time and that aging – whether in an animal, a rock, or a distant star -- is a process of inexorable and ungovernable molecular decay.[4] Classical evolutionary biology holds that no beneficial genetic traits appear after the age of reproduction, because such traits could not be chosen by natural selection and passed along.[5] The C. elegans experiments challenge both doctrines. They seem to demonstrate that aging is controlled by genes rather than by feckless time and that relevant genes kick in only after the worms have passed reproductive age.[6] Thus the experiments cock a snoot at both Lord Kelvin[7] and Charles Darwin. As might be expected, the scientists engaged in this work have big egos.

(line break)

---

[4] S. Jay Olshansky, University of Chicago, work: 312-996-7000, home: 847-537-7278, cell: 847-347-8585 or sjayo@uic.edu and Leonard Hayflick, home: 707-785-3181 or lenh38@netscape.net and hayflick.leonard@gene.com

[5] S. Jay Olshansky, University of Chicago, work: 312-996-7000, home: 847-537-7278, cell: 847-347-8585 or sjayo@uic.edu

[6] Cynthia Kenyon, UCSF., work: 415-476-9250, home: 415-641-9174, cell: 415-271-0644 or ckenyon@biochem.ucsf.edu. Her assistant is Myra Melville, 415-514-4078

[7] Lord Kelvin, a.k.a. William Thomson, was one of the developers of the second law of thermodynamics, also known as the law of entropy. See file “Lord Kelvin”

When Tom Johnson arrived at the University of Colorado at Boulder in 1977 to do post-doctoral work in genetics, he was warned to stay away from the post-doc across the hall. Michael Klass was working on aging, people told him with a roll of the eyes. Klass carried a double stigma. His efforts to change the way organisms age seemed to most people like trying to cancel gravity or slow the speed of light. Worse, the effort smacked of snake-oil and Ponce de Leon. Klass was among the first to try mutating single genes in C. elegans to make the worm live longer. He postulated the existence of a single lifespan-controlling gene he called "Age One." Johnson thought Klass's work was inexact – he thought it more likely that several genes were involved -- but he couldn't help being intrigued. When Klass took a job in private industry a few years later, he left his strains of roundworms to Johnson, who began his own search for the gene or genes that control lifespan.[8] "Well, that's the end of your career," Johnson's department chair told him, only half joking.[9]

Johnson is short and muscular, with thinning hair and freckles of the same ginger hue. "I'm absolutely convinced my twelve-year-old son will live substantially longer than eighty," Johnson told me as we

---

[8] Michael Klass can confirm his role in this story. 858-410-8839, cell: 619-726-1676 or michaelk@gen-probe.com

[9] Tom Johnson, work 303-492-0279, home: 303-665-5652 or tom.johnson@colorado.edu

settled into chairs in his unadorned cinderblock office on the University of Colorado campus. "He'll make a hundred. The important thing is, he'll be doing better at seventy-five than I'm doing at fifty-five. It looks like the only way to make something live longer is to make it healthier."[10] Johnson's lab is a windowless brick warren crammed with microscopes, freezers, and stacked boxes of surgical gloves. We threaded our way among half a dozen graduate students – who call themselves "worm-pickers" -- hunched over microscopes. Nate Kahn, a post-doc, opened a freezer and pulled out a small, flat plastic box perforated with three hundred and eighty-six tiny wells. "Each one contains a worm gene," he said. When the students want to see what a gene does, they defrost it, insert it into a worm, and wait to see the change it effects. "We have about seventeen thousand here, or eighty-five percent of the genome . . . we know what about five thousand do. Another five to eight thousand, we know something about them. The rest, we know nothing."[11] I peered through a microscope at what first looked like a disc covered with cracked white paint. Upon closer inspection, each crack was moving.

When Johnson started in 1982, genetic engineering was a young field and he used crude techniques -- radiation and chemicals -- to

---

[10] Tom Johnson, work 303-492-0279, home: 303-665-5652 or tom.johnson@colorado.edu
[11] Nate Kahn, 303-492-5159

mutate genes and move them around in big bunches. He was able, willy-nilly, to increase both mean and maximum lifespan in worms by about seventy percent. He couldn't prove exactly what was extending the worms' lives and his findings, published in the prestigious Proceedings of the National Academy of Sciences, were hardly noticed.[12] But he took the descendants of Klass's roundworms with him when he moved to the University of California at Irvine. Mutating and rearranging genes is laborious, the kind of thing professors get their graduate students to do, but none at Irvine would work with Johnson. "They'd been warned," Johnson told me. So he roped in an undergraduate named David Friedman to do the grunt work. ("I was just a little undergrad who didn't know any better," remembers Friedman, who is now a researcher at the Vanderbilt-Ingram Cancer Center in Nashville. "I assumed Tom knew what he was doing.")[13] Under Johnson's direction, Friedman found in 1987 that Klass had been right along: somewhere on the roundworm's genome is a single gene that controls lifespan.[14] Even more extraordinary, in Johnson's view, was the discovery that the gene's function is to shorten life; only by "turning off" the gene were Johnson and Friedman able to lengthen

---

[12] Tom Johnson, work 303-492-0279, home: 303-665-5652 or tom.johnson@colorado.edu
[13] David Friedman, 615-343-7333 or david.friedman@vanderbilt.edu
[14] Tom Johnson, work 303-492-0279, home: 303-665-5652 or tom.johnson@colorado.edu

a worm's life. "It was totally counter-intuitive," Johnson told me. They might have assumed, in good Darwinian fashion, that the gene was there for the good of the species – to kill off the parents so that they wouldn't compete with the offspring. But by the time a C. elegans dies, it has produced a hundred billion progeny. "It's hard to imagine that the competition of one parent has any relevance," Johnson said. His theory is that the species wants to keep each individual as fit as possible through reproductive age, but after that, "nature doesn't give a damn." Genetics often works in trade-offs, he said: for every benefit, a harm. Shortening life to maximize fitness in youth "may be bad for the individual, but good for the species." The reverse might also be true. A gene-altering drug that would extend human life might interfere with reproduction. In the real world, though, such a trade-off may not matter, Johnson said. "It may be you'd take this drug after you've had whatever children you want to have." [15]

With the 1988 publication of Johnson and Friedman's results in the journal Genetics, the stigma hanging over aging research lifted. Their experiments demonstrated that Age One existed and gave an idea of how it worked. Johnson and Friedman had not, however, laid their

[15] Tom Johnson, work 303-492-0279, home: 303-665-5652 or tom.johnson@colorado.edu

hands on Age One itself.[16] The race was on to find it. At this moment, Johnson more or less bowed out. The high-powered research universities were on the coasts, but at the request of his wife, an anesthesiologist, Johnson returned to the University of Colorado's department of behavioral genetics. "I don't want to sound too Buddhist," he said, "but there's a middle path in life." He added with a shrug, "Somebody will get a Nobel Prize for this, but I'm probably the better dinner companion."[17]

Five years after Johnson and Friedman published their Age One paper, a young woman at the University of California at San Francisco stepped to center stage, discovering the gene that seems to control Age One. What Cynthia Kenyon found, more precisely, was the commander of a genetic "management team" that appears to regulate the lifespan of C. elegans**.** The "team" tinkers with the endocrine system – particularly insulin and a hormone known as IGF-1 -- and Kenyon believes a similar "team" works the same way in humans. I met her at Bacco, a small Italian restaurant near her house in the Noe Valley section of San Francisco, where the headwaiter greeted her as he would a daughter coming home to dinner. Kenyon is a descendant

---

[16] Tom Johnson, work 303-492-0279, home: 303-665-5652 or tom.johnson@colorado.edu and David Friedman, 615-343-7333 or david.friedman@vanderbilt.edu

[17] Tom Johnson, work 303-492-0279, home: 303-665-5652 or tom.johnson@colorado.edu

of Mayflower passengers, thin and pale, with faint freckles and sandy hair cut in a fabulous bob. She is fifty and, appropriately for her field, looks about thirty-five. She has never been married and has no children. "I'm told that's really good bread," she said as the waiter set down a basket, but she didn't touch any. On the day she noticed that adding sugar to a worm's petri dish shortened its life, she bought a low-carbohydrate diet book and in the ensuing months lost thirty pounds that by ordinary fitness measures she didn't need to lose. Kenyon never touches sweets or starches, and drinks one glass of red wine a day. Genetics is well and good, but she is hedging her bets.

Kenyon entered the field of aging research at about the same time as Johnson and Klass, and seethed under the reigning conventions. "People were saying there are evolutionary reasons why there can't be a genetic code for aging. I thought, 'How can they know? What hubris!' Think about women. You're a girl, then you hit puberty. Then four decades later you go through menopause. Who's keeping track? How does my body know how long its been alive? Nothing in biology ever just happens. Everything is controlled."

When Kenyon turned off C. elegans's commander gene, she discovered that her worms lived twice as long, and stayed youthful. They moved like young worms, and didn't show the thickening and darkening of old worms until late in their extended lives. "It was like

every day of the worm's biological life took two days," she said. "The minute I saw the long-lived worms, I knew I wanted to start a company." Kenyon believes that a pill that would allow us to live longer and healthier would be good for the planet. "We don't have thirty-year-old national leaders and C.E.O.s," she said. "Older people are repositories of wisdom." She rummaged in her bag, briefly confusing the restaurant's ambient classical music for her cellphone's Mozart ring. "Maybe it can't be done; I want to say that," she suddenly said. "But the big surprise of the past twenty years for biologists was how uncannily similar the basic processes of life are in all organisms. We know that if you change a gene in at least one species of animal, it lives twice as long."

A pill that changes all the DNA in a human is theoretically possible. Scientists change the DNA in a worm's cells by isolating and altering a gene and implanting it in the DNA of a bacterium. They then feed the worm the genetically altered bacterium and within hours, the DNA in every cell in the worm's body has adopted the change. "What's standing in the way of doing this in humans?" Kenyon asked. "Really just luck, time, and money."[18]

For the money, Kenyon and a biologist colleague at M.I.T., Lenny

---

[18] Cynthia Kenyon work: 415-476-9250, cell: 415-21-0644, home: 415-641-9174 or ckenyon@biochem.ucsf.edu

Guarente, went to the community of biotech venture capitalists. Their firm, Elixir Pharmaceuticals, started up in late 2000 with eight million dollars in speculative cash, which has since increased tenfold without yet putting a product on the market.[19] I reached one of Kenyon's initial backers, Bob Nelson of Arch Venture Partners, on the phone at his office in Seattle. Nelson has an undergraduate degree in biology as well as an M.B.A. and has been starting biotech companies for eighteen years. "Everybody knows the little old lady who's a hundred years old, with the sister who's ninety-six; they're thin, they're vibrant, they don't feel as old as they are and they don't have the diseases other old people get," Nelson said. "You want to find why grandma is healthy and impart that to others. People who don't think there's a genetic component of aging – their emotions are getting in front of their neurons." Nelson and his partner, Cindy Bayley, speak only of developing drugs to make old people stay healthy longer; they don't promise longer lives. "Extending human life would be a side effect," Nelson said, [20] to which Bayley, when I reached her at her office in Boston, added, "You don't talk about this without talking about longevity. When you're doing one, you're doing the other."[21] Nelson and Bayley avoid hyping longer lives to avoid charges of

---

[19] Karen Roberts, Elixir investor relations, kroberts@elixirpharm.com
[20] Bob Nelson, 206-674-3266 or rtn@archventure.com
[21] Cindy Bayley, 617-877-2779 or Cbayley@archventure.com

quackery, and also because they recognize that under current Food and Drug Administration regulations, there would be no way to conduct clinical trials of a life-extending drug. The study period would far exceed the twenty-year life of a pharmaceutical patent, so by the time the drug was approved, the company that developed it would be unable to profit from it.[22] If Kenyon or anybody else gets a life-extending drug onto the market, it will almost certainly begin as a treatment for ailments other than age. Life-extension will be an "off-label" benefit, the way revived sexual function for men was an off-label benefit of the hypertension drug, Viagra.[23] It's possible that a drug capable of extending human life is already on the market but hasn't had time to show its most remarkable effect. Kenyon and Johnson both told me they thought statins – the modern anti-cholesterol drugs that include Lipitor, Prevachol, and Zocor – might prove to be life-extenders.

Statins, though, don't alter one's DNA. The suggestion that aging can be affected by tinkering with genes still infuriates some leaders in the field. Leonard Hayflick is the geneticist most offended by Cynthia Kenyon. Among other achievements, he created the cell strain that is the most widely used globally for making vaccines. All the rubella

---

[22] Karen Roberts, Elixir Pharmaceuticals, kroberts@elixirpharm.com
[23] file "history of Viagra"

vaccine in the western hemisphere, for example, is made from cells Hayflick created in his lab at the Wistar Institutive of Anatomy and Biology in Philadelphia in 1962. Hayflick discovered that the cause of "walking pneumonia" wasn't a virus, as everybody thought, but a mycoplasma, among the smallest of single-cell organisms. He developed a revolutionary oral polio vaccine.[24] He helped create the N.I.H.'s National Institute on Aging. He has received just about every important prize in genetics and gerontology short of the Nobel, and he achieved popular literary success in 1994 with How and Why We Age.[25] By 2002, Hayflick and two colleagues were so alarmed by Kenyon's and Johnson's claims that they denounced them in Scientific American, in a "Position Statement on Human Aging." "Our language on this matter must be unambiguous," they wrote. "There are no lifestyle changes, surgical procedures, vitamins, antioxidants, hormones, or techniques of genetic engineering available today that have been demonstrated to influence the process of aging."[26]

Hayflick is now mostly retired. He supervises research, via phone and fax, for Genentech, a biotechnology company in San Francisco.[27]

---

[24] Hayflick's bio, which is at the start of the file "Leonard Hayflick," containing notes of my interview with him.
[25] Hayflick's bio, which is at the start of the file "Leonard Hayflick," containing notes of my interview with him.
[26] file "position statement on aging."
[27] This story is well told in Hall, Stephen S., Merchants of Immortality, Houghton Mifflin, 2003, pages 14-41

But he lives five hours north, at the remote Sea Ranch, a thirty-five-hundred acre development pioneered in the nineteen sixties with restrictive covenants to protect the wild landscape. All the houses, which are far apart, must be clad in the same weathered redwood, and no traditional landscaping is allowed. The resulting tableau, on a foggy day, is of lonely-looking gray boxes hulking in the coastal fog, like bunkers of the Maginot Line.

Scientists nowadays commonly profit from their discoveries, in the way that Cynthia Kenyon hopes her work at U.C.S.F. will someday make money for Elixir. The development of personal computers and the Internet arguably would not have been so rapid without the partnership of industry and academe. But in the nineteen sixties, when Hayflick was young, the fruits of scientists' labor generally belonged to their employers. In 1962, Hayflick learned that Wistar was offering to sell one of his miraculous strains of cells to a private pharmaceutical company without passing any of the profits along to him. Furious that he had been relegated to, as he put it, "a concessionaire at a ballpark," Hayflick left Wistar for Stanford University. When he drove west, his precious cells, packed in liquid nitrogen, were squirreled away in the trunk of the family Buick. Hayflick's proprietary flight scandalized Philadelphia and the scientific world. The Evening Bulletin headlined its story, "Philadelphia Scientist

Drove West With More Than His Luggage." In Palo Alto, Hayflick formed his own company and began selling the strain of cells to other researchers. But he had used funds from the National Institute of Health to develop the strain, and the government treated him like a thief. One October night in 1975, investigators crept into his office and confiscated both his cells and his files. The Santa Clara County district attorney opened a criminal investigation, and when Stanford refused to back him up, Hayflick resigned. For a while, the wonder boy of genetics wandered as a pariah, roosting briefly at the University of Florida but finally securing a position at one of the rising stars of the field, U.C.S.F. It wasn't until 1981 that the government conceded Hayflick ownership of the cells, and agreed that he was entitled to the money he'd earned from them.[28] Though Hayflick may have done a favor for succeeding generations of scientists in fighting for the right to profit from his intellectual property, the battles left him bitter. He is short, sturdy, and younger-looking than his seventy-six years. But he is unsmiling and tough in the manner of Edward G. Robinson.

The interior of the Hayflick house is a spacious and lovely expanse of tile floors, exposed-beam ceilings, and Danish furniture. Ruth Hayflick served us coffee in the living room on a tray. "People

---

[28] This story is well told in Hall, Stephen S., Merchants of Immortality, Houghton Mifflin, 2003, pages 14-41

have been promising what Cynthia Kenyon is promising for thirty-five hundred years," Hayflick said as we sat down. "It's Martian biology. These people have to be stopped." I was a little taken aback by his vehemence, but geneticists are known for trashing each other publicly in a way that, say, physicists do not.[29] Most people credit James Watson for the field's lack of collegiality; he opened his 1968 account of the discovery of DNA, "The Double Helix," with this assessment of his equally famous partner: "I have never seen Francis Crick in a modest mood," and went on to say something nasty about almost everybody else involved in the discovery.[30]

Hayflick, who helped found the National Institute on Aging and has built his career on the study of the aging process, believes Kenyon is muddying the very definition of the term. "We know what causes aging; the loss of molecular fidelity over time. That's aging. It's a universal process." He compared humans to Mercedes-Benz and Yugo automobiles. "They have different life expectancies, but age at the same rate, as does everything else in the universe. I'll show you the blueprints for your car; you show me the device that tells it how to age." Hayflick is, without doubt, a Kelvinist: thermodynamics, for him,

---

[29] this according to my father in law, William Knox, who worked on the Manhattan Project during the Second World War and is professor emeritus of physics at University of California at Davis. 530-753-2788

[30] Watson, James D., The Double Helix, page 9

rules out the possibility that a gene might control aging. Scientists have not even found a way to determine an organism's biological age -- that is, what percentage of its life has passed. "Tree rings only count chronological time, and we don't have rings," he said. Hayflick praised Kenyon's "Nobel-quality" experiments but argued that if her worms are living longer, she's learning not about aging but "the phenomenon I define to be longevity determination. Non-scientists may not care about the difference because the outcome may look the same, but they are two different processes." Living longer and wriggling youthfully does not make a worm young, he insisted. "You just got finished telling me I don't look seventy-six. If I can beat my eight-year-old grandson in a one-mile foot race, does that mean I'm younger than he is? If you assume that motion equals youth, then children who are paralyzed are ninety years old."

We talked for four hours. When I asked Hayflick whether his objection to Kenyon's work was semantic – aging versus longevity determination – he said Kenyon's life-extended worms probably aren't as healthy as she thinks they are. If there's a benefit to the mutation – longer life – there is doubtless a trade-off, he said, perhaps in cognitive function. "You can't show me anything they've done that gives both longer and healthier," he said. But even if Kenyon's worms are somehow living longer and staying healthier, he said, "To make

the leap from worms to people is unconscionable, and bad science, and a form of self-aggrandizement." Young C. elegans can go into a kind of suspended animation called a dauer phase, to endure times of low food and harsh conditions, and then emerge from dauer and reproduce when conditions improve. "Humans aren't big worms," Hayflick said. "We don't have a dauer phase." But for the sake of argument, he was willing to allow that a pill to make people live longer and healthier might be possible. Then the question for Hayflick becomes, who would want to take such a pill? He can imagine interpersonal nightmares, in which, "your wife doesn't want to do this; then she's getting old and you're not." He believes that if you ask comfortably retired people in their seventies and eighties whether they want to slow the aging process and stretch out life, "they'll say you're crazy." And as for the majority of people on the planet, he said, living with hunger and violence and dictatorships – forget it. "This question can't be answered by everybody on the planet the same way," he said.

He walked me out to my car as the light was fading. "They have to stop fooling themselves and the public" he said of Kenyon and Johnson. "The more you promise the public that they can live a greater number of years, the more popular you're going to be, the more conferences you'll be invited to, the more articles you'll have written about you, the more grants you'll get." He shook my hand, and

returned to his fog-shrouded house.[31]

[line break]

Gary Ruvkun is the molecular biologist at Massachusetts General Hospital who, in 1995, found the Age One gene itself, two years after Kenyon identified the "team" it is a part of. "I'm agnostic on whether it will really happen," he said when I asked him on the phone about the prospect of hundred-and-fifty-year-old people. "I have a problem with people trying to start companies based on this. I wouldn't invest in one." On the other hand, "the force of history is against the Luddites," he said. What mostly convinces Ruvkun that there is something to Kenyon's work is the eerie and largely unexamined immortality of DNA. "A thirty year old sperm – a sperm from a thirty-year-old man – and a thirty-year-old egg meet, and they make a thirty-year-old fertilized cell, right? But now the clock is reset, and we say this life is just beginning. Why is that? The material is thirty years old. Or older, if you consider that it came from the generation before, and the generations before that." Unlike Hayflick, Ruvkun considers the discovery of the similarity in basic genetic function between C. elegans and people – the research that won the 2002 Nobel Prize -- to have been a wake-up call. Geneticists, in his view, need to re-think the

[31] Leonard Hayflick, home: 707-785-3181 or hayflick.leonard@gene.com and lenh38@netscape.net

possibility of vastly longer human life spans. "There's no fundamental reason it can't be done."[32]

S. Jay Olshansky was one of Hayflick's co-authors of the scornful "Position Statement on Human Aging." A sociologist at the University of Chicago with a specialty in the demographics of aging, he also did the coursework, but no dissertation, toward a PhD in molecular and evolutionary biology. When I asked him about broader implications of the nematode research, he gave me a demographer's answer, "I'm not a nay-sayer. I love the work and think it should continue. If we don't find a way to slow aging, we're going to see catastrophically expensive increases in frailty and disability among the elderly, which will hit in 2011 and increase exponentially."

Contrary to what seems intuitive, he said, doubling people's life spans would not wildly overpopulate the planet. The population growth rate is defined as the difference between birth- and death-rates – the number of births or deaths per thousand people in a year. The national population growth rate was historically high in 1950, when there were forty-five births and fifteen deaths for every thousand people. The difference – thirty per thousand – came out to a growth rate of three percent. By 2000, when fifteen babies were born for every ten people

---

[32] Gary Ruvkun, 617-726-5959 or ruvkun@molbio.mgh.harvard.edu or ruvkun@frodo.mgh.harvard.edu

who died, the rate had fallen to half a percent. Were people to become immortal – a death rate of zero – while the birth rate remained at fifteen, the growth rate would be only one and a half percent, or triple what it is now, but only half the 1950 rate.[33] Olshansky likes to think about such questions but is under no illusions that a long-life pill is imminent. Kenyon's worms might wriggle attractively, he told me, but "you can't ask it how its cognitive function is." And his study of genetics tells him Hayflick is right: that there is no such thing as a gene that controls aging. All Kenyon is doing, he said, is tinkering with "longevity determination," not the aging process.[34]

"Who cares?" asked Tom Johnson when I saw him again. "If I'm living longer and healthier, do I care why?" Johnson, who set off the race for the hundred-and-fifty-year human lifespan, said that for years he was a "typical scientist," ignoring the broader implications of his work. It wasn't until he reached his fifties that he started thinking about what he calls "the big philosophical questions." "I think there's nothing we could do that could cause a more basic shift than have everybody double their life spans. Everything would change. It would be immoral not to recognize that. Insurance, annuities, tenure, social

---

[33] S. Jay Olshansky, home 847-537-7278, cell: 847-347-8585, or sjayo@uic.edu and file "Olshansky's immortality chart."
[34] S. Jay Olshansky, home 847-537-7278, cell: 847-347-8585, or sjayo@uic.edu

security -- all of that would be completely transformed."[35] The very thought of hundred-and-fifty-year-old people makes some of his undergraduates blanch, he said. "They worry that if their parents never die they'll never have a life."[36]

The idea of super-seniors makes some bioethicists throw up their hands in horror. Daniel Sulmasy, chair of the Ethics Department at St. Vincent's Hospital on Eleventh Street, draws a line between longevity research and the use of gene therapy to treat illnesses, or such commonplace practices as encouraging people to quit smoking to extend their lives. "With genetic treatment of illness we're repairing what we are," he said. "But here we're proposing changing the kind of thing we are." Then there's the question of medical scarcity. "If we're spending resources trying to live to a hundred and fifty instead of curing malaria or bringing affordable drugs to the Third World to combat H.I.V. – if you don't see a problem with that, I'd have to question your ethics."[37] Daniel Callahan, director of International Programs at the Hastings Center, a bioethics research institute in Garrison, New York, called the idea of genetically extending human life, "a bad idea, absolutely. What present problem in our society

---

[35] Tom Johnson, work 303-492-0279, home: 303-665-5652 or tom.johnson@colorado.edu

[36] Tom Johnson, work 303-492-0279, home: 303-665-5652 or tom.johnson@colorado.edu

[37] Daniel Sulmasy, 212-604-8280 or daniel_sulmasy@nymc.edu

would be solved by people living radically longer lives?" Callahan, who is seventy-four-years-old, sees no correlation between a longer life – even if it's healthy -- and a happier one. "There's such a thing as getting tired of life. You hear it all the time, 'whatever happened to so-and-so? He's not sick, he's not dead, he's just not writing anymore.' The scientists are making a solipsistic argument: 'I want to live longer, therefore society should allow everybody to live longer.'" Cynthia Kenyon's idea that the world would benefit by being filled with older and wiser people is "one of the great myths," he said. "Maybe that's true in an agricultural society, where things remain the same. In our society, where things change so rapidly, it's very hard for older people to have anything relevant to say to younger people. You rarely see older people consulted, except for Alan Greenspan."[38]

[line break]

The morning after our dinner, I visited Kenyon at her lab in San Francisco. It's in the Genentech Building, a stunning, modern giant marooned in the vast, bleak industrial area beyond Pacific Bell Stadium. One wall of Kenyon's big office is made almost entirely of glass, but it faces a particularly grim tableau of construction sites and warehouses. She opened her Powerbook laptop to show me through-the-microscope videos of young worms sliding transparently through

---

[38] Daniel Callahan, (845) 424-4040 ext 222 or callahand@hastingscenter.org

fields of bacteria, leaving trails. Then she showed me old worms, thicker, dark with fat deposits, sluggish. Finally, she called up a film of one of her genetically engineered worms, which at thirteen days, or twice the normal lifespan, was slightly dark but moved with a young worm's vigor. "Isn't it pretty?" Kenyon asked, beaming at the screen as though she were watching a six-year-old daughter score a soccer goal. Between 1993 and 2002 Kenyon was able to increase the extension of her worms' lives from two-fold to six-fold; she showed me one at a hundred-and-forty-four days old, and once again, it looked about the same as a young worm. "They don't look miserable, but we can't ask them," she said without taking her eyes from the screen. Kenyon is trying to develop reliable tests of their cognitive function. "They move toward things they like, and away from things they don't like."

I mentioned that some of her critics find her conclusions inconsistent with the theory of evolution, and she looked up sharply. "This isn't a theory," she said, gesturing at the screen. "This is what we see. It doesn't matter what you think evolution is." Then, apparently speaking of Charles Darwin, she said, "He's the one who has to reconcile his theory with the facts we're finding in the laboratory." Her face grew tense, her eyes wet. "Hayflick!" she spat. "He's the old guard. They have these . . . what I call preconceptions of

how it's going to be." She put her hand on my computer. "Maybe you should listen to this and stop typing," she said. "We never thought there was anything you could do about aging, because of people like Olshansky. The past is represented by Olshansky. He can call this whatever he likes, but it's happening."

I mentioned the ethicists' objections and she stopped me. "It's a natural human desire not to want to deteriorate. Read Shakespeare's sonnets if you don't believe me. Society adapts," she said, bouncing in her chair. "Society will change. If there's a problem, society will change to accommodate it. We've already changed the demographics just by curing infections and disease. We have a lot more old people relative to young people than we used to. Is that a problem? No. It's something we have to deal with, and we're dealing with it."

Her face was flushed with anger. "Who's to say what's ethical and not ethical? It's so easy to tell a horror story, but we're talking about keeping people who are young, productive and active, young, productive and active longer."

End