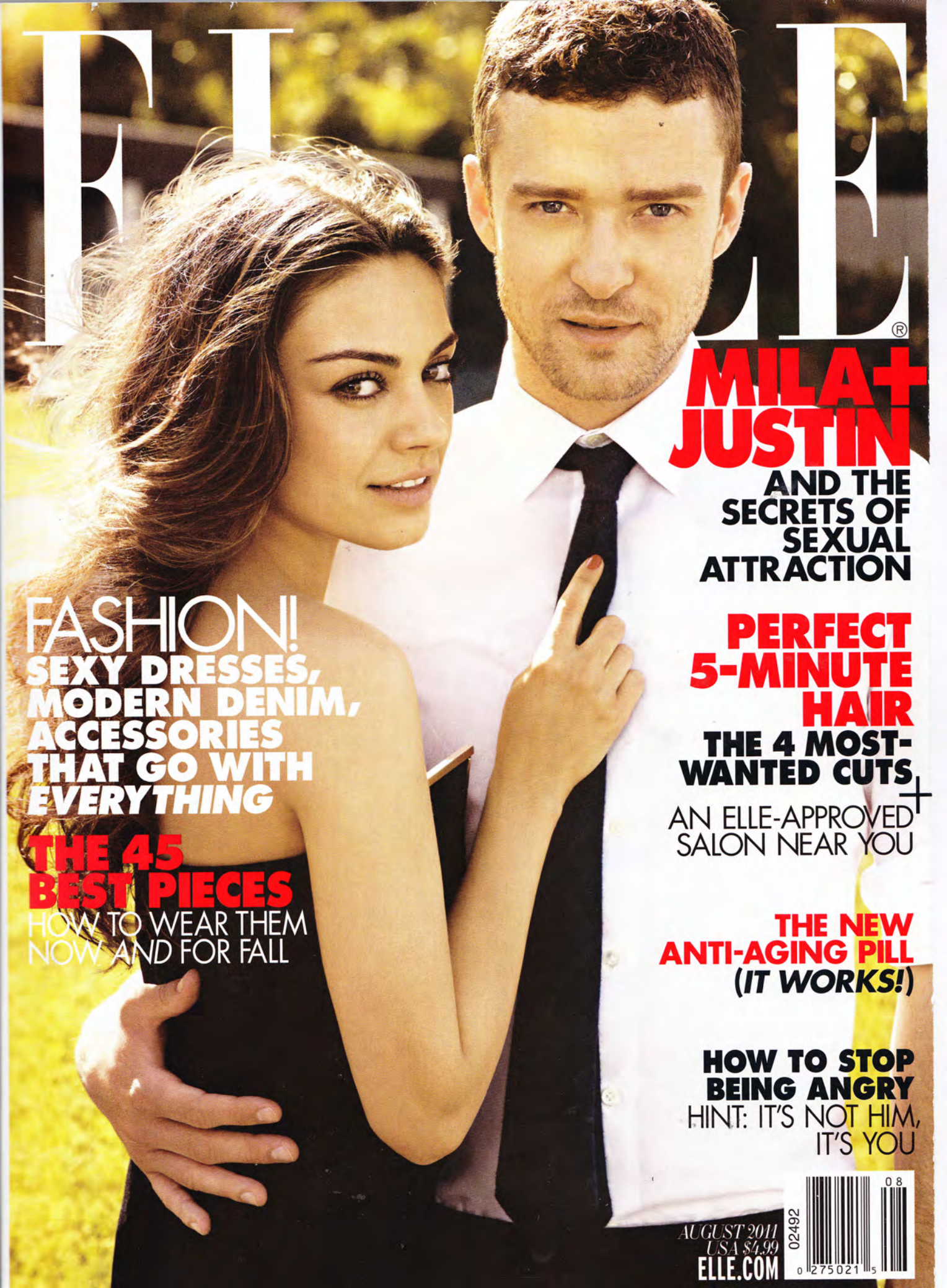
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HEY—WANNA LIVE FOREVER?

More quickly than anyone imagined, Nobel Prize–winning research is yielding promising compounds for fighting the inevitability of aging at the most fundamental cellular level. By Joseph Hooper

In case you missed the memo, research biologists can now make you immortal. Not *you*, exactly, but human cells in a petri dish. They can be made to stay firm and youthful-looking and able to divide indefinitely—the supermodels, so to speak, of cultured cells—if the enzyme telomerase is inserted into their machinery.

The actual you already has a piece of that immortality. Your reproductive cells produce lots of telomerase and in a real sense do live forever. Through sex and procreation, these cells keep dividing and creating the next generation, and the next after that, such that the egg cells in your ovaries house more or less the same genome as those in the first woman who walked the earth. The rest of you is of course *not* immortal—not even close. Our ordinary nonreproductive cells work nicely through the first decades of life, and with luck or effort hold it together as best they can in the middle decades, and then—well, you know how it ends. For all the advantages of modern life, we don't live any longer than our longest-lived ancestors, because evolutionarily, it doesn't matter what happens to us after our reproductive years; over the course of hundreds of thousands of years of human existence, we have evolved no protection from the cellular exhaustion that, providing we survive the perils of middle age, will take us in the end, whether the cause of death reads heart failure, cancer, or "she left us quietly in her sleep." And that's the last depressing thing you'll read in this article.

In 1984, University of California, Berkeley biology professor Elizabeth Blackburn and then grad student Carol Greider made the discovery that a quarter century later would win them the Nobel Prize: They identified telomerase as the enzyme that protects the DNA in our chromosomes, in effect keeping our cells—and, to some degree, us—young. But telomerase is naturally produced only minimally and intermittently in some of our cells—just enough to give disposable us a maximum life span of around 120 years. Unless, that is, someone figures out how to increase the telomerase inside our bodies. Such tinkering with the basic machinery of life has been a theoretical possibility since the 1990s, when scientists at the Bay Area biotech firm Geron and elsewhere identified the human telomerase gene. Of course, it's one thing to have the gene in hand, and another thing entirely to devise a telomerase-stimulating drug that can set back our cellular clocks (and not somehow harm us in the process).

If that future has not completely arrived, let's just say it's been a very good year for telomerase. Last November, Harvard biologist Ronald DePinho published an elegant study in *Nature* (earning him a spot on *The Colbert Report*) in which he described how he was able to turn telomerase on and off in genetically modified mice via a synthetic estrogen drug. In the "off" state, aging was accelerated—their fur turned gray, their internal organs became decrepit, their brains shrank. (The mice became miniature versions of those with Werner syndrome, who, because of a rare genetic defect that affects DNA repair, turn frail and elderly by the time they hit middle age.) When DePinho chemically turned the mice's telomerase switch back on, these effects were reversed within a month, returning the rodents to a condition he describes as "the physiological equivalent of young adults." This is what scientists call "proof of concept"—the concept being nothing less than the reversal of aging, or as DePinho puts it, "the remarkable capacity of aged tissues to rejuvenate when you remove the underlying cause."

The capstone of nearly two decades of research teasing out the effects of telomerase in mouse aging, DePinho's study raises the question of when might we try something similarly audacious in human beings. DePinho admits it's reasonable to think that a drug or a gene therapy that "upregulates" telomerase in humans might seriously reduce major illnesses of aging such as heart disease, diabetes, and Alzheimer's. (He's agnostic on the question of whether slowing down normal aging is possible.)

Photograph by Marc Philbert

Like most good researchers, he favors a go-slow, more-work-is-needed approach, given the possibility that too much telomerase could fuel the development of cancer. (The other type of "immortal" cells that, like reproductive cells, produce telomerase nonstop are cancer cells.) "We're not talking years but perhaps a decade or more before we begin to think about ways to exploit this newfound information," he says.

But events may well overtake DePinho's cautious view. Humans have been chasing immortality since the beginning of civilization. Sometimes the moral of this imperative has been that it's a bad idea (you may remember that Zeus gave Tithonus immortality but not youthfulness, condemning him to eternal decrepitude), or more often just bogus—think of Ponce de Leon searching Florida (of all places) for the Fountain of Youth. More recently, we've lived through the "melatonin miracle" of the '90s and the recent craze for the grape-skin compound resveratrol, which has lately run into a spate of negative research results.

If we can manipulate something as fundamental to our makeup as telomerase activity without inducing cancer, this genie won't easily be put back in the bottle. In the '90s, Geron tried to deliver the telomerase gene into cells by piggybacking them onto viruses, which, of course, invade cells. That gene-therapy approach failed (and the entire field is at a standstill due to as-yet-unresolved safety issues). But around 2000, Geron got lucky with a lower-tech approach—a telomerase-activating (TA) compound made from a single molecule of the Chinese medicinal herb astragalus. After many fits and starts, the company hopes to petition the FDA next year to begin human trials with its current astragalus-based drug candidate for the treatment of pulmonary fibrosis, an often fatal lung disease. Even if all goes well, the journey from clinical trials to the pharmacy shelf might take five or six years, according to Geron toxicologist Hooman Kashani.

However, in 2002, Geron—down on its luck and forced to lay off a large fraction of its workforce—licensed rights to its TA formula to New York City entrepreneur Noel Patton, enabling him to make and market a dietary supplement (for which no medical claims are allowed to be made). "Geron has never tested any of its telomerase activators in humans because it can't," Patton says. "But I can. I'm not selling a drug."

More precisely, Patton is selling hope to people willing to take a risk—himself included. ("I want to save my own ass," the 66-year-old says.) Three years ago, under close medical supervision, he and a pilot group of nearly 100 clients began taking his TA-65 supplement—the clients each paying $25,000 annually for the privilege. No new cases of cancer have so far turned up among this group, so Patton has stepped on the throttle this past year, dropping TA-65's price into the stiff-but-affordable range ($2,400 to $8,000 a year, depending on the dosage) and expanding his client numbers into the thousands.

Chasing Patton's heels is Isagenix, a company whose TA supplement-in-waiting is scheduled to launch before Labor Day. (It will be sold in doctors' offices, online, and door-to-door.) The TA properties of its one or more natural compounds (the identity of which Isagenix has so far chosen to keep to itself) have been vetted in the lab by the molecular biologist who, back in the '90s, helped identify human telomerase for Geron. John Anderson, the businessman and self-taught chemist behind the new venture, calls it his "product B," as in future billion-seller.

With their TA supplements, these companies are proposing nothing less than to fix the wrinkles *underneath* our skin—at the molecular level. How deeply from the telomerase cup of immortality can we hope to drink? Pick your fantasy scenario, in descending order of fabulousness (and increasing likelihood): Lengthen our maximum life span—to 150 years, perhaps?—to a degree that will transform our existential horizons (some scientists working on longevity believe this could happen during our lifetime); increase our "health span" by a significant margin by delaying diseases of aging—in other words, look and feel relatively great until you hit the same old longevity wall at 90 or 100 (probably a majority of researchers see this as a goal within our grasp); or at 50, your skin could pass for a 20-year-old's (telomerase is most active in cells that divide most frequently, such as those in our skin; Patton's next order of business is to partner with a cosmetics company to market a skin cream that will activate telomerase in your skin cells).

Or—none of these wonderful things may come to pass. So far, science has established that telomerase—unlike every other anti-aging elixir before it—has a profound impact on the aging process, and that it can be manipulated. Whether it will turn out to be worth any potential cancer risk to do so is still an open question. "Since we already live very many years, I think we have to tread very carefully," muses telomerase research pioneer Elizabeth Blackburn, now at the University of California, San Francisco. "Magic pills have a way of turning around and biting you." If Blackburn has her way, we will reserve the telomerase drugs for the truly sick, exploiting the enzyme in ways that can't lethally sneak up on healthy people 20 years from now.

Telomerase—unlike every anti-aging elixir before it—has a profound effect on the aging process.

"Hallo," comes a cheery voice on the phone. "It's gotten very dramatic out here. It's been hailing on us." I'm sitting in Blackburn's office at UCSF's modernistic Genentech Hall (the House That Biotech Built), and she's late, trapped in freeway gridlock as San Francisco falls apart at the seams during an unusually severe February storm. "The campus was agog," she announces as she bustles into the office a few minutes later, her umbrella trailing behind, and without missing a beat plunges into a customized lecture on the state of her telomeric research. The effect is engaging, like being tutored in molecular biology by a genteel but rather fun English aunt. (Blackburn is Australian, but she did her graduate work at Cambridge alongside luminaries such as DNA codiscoverer Francis Crick.)

Her telomerase story takes us back to the telomeres themselves. In every cell in our body, telomeres sit at the ends of 23 pairs of chromosomes, protecting them from damage so they can get on with their basic job of mitosis—dividing into two new cells. (The standard telomere analogy is to the plastic cap that prevents the end of a shoelace from fraying.) There's a fundamental problem with mitosis that researchers like Blackburn had identified by the late '70s and were struggling to solve: Every time a cell divides, the end segment of DNA fails to get copied in the two new cells.

Evolution's mechanisms for dealing with this "end-replication problem" are telomeres—repeating sequences of DNA

that with every cell division shrink a notch without any violence being done to the genetic instructions encoded in the rest of the chromosome. Telomeres thus also serve as a kind of clock for the aging cell—or as the bouncer clicking a number-counter every time someone passes the velvet rope. Since our skin, gut, and immune-system cells divide so frequently, their telomere strings would become depleted if there weren't something to replenish them. Blackburn figured there must be some sort of enzyme that synthesizes new segments of DNA and adds them to the telomeres before such cells lose the ability to divide, or die outright. She directed her second-year grad student Carol Greider to find it.

The haystack they chose to look through was tetrahymena, a pond-dwelling protozoan that comes in seven distinct sexes and is open to having sex with any of the other six. When I compliment Blackburn on having the vision to suspect that what worked in primitive single-cell organisms would work in humans, she stops me short. "That's the credo of molecular biology," she says. "Life is universal. The fundamental rules are the same." And so her Berkeley lab staff got a colony of tetrahymena reproducing like crazy, then whipped them up in a blender to see if they could detect and isolate telomerase activity. After beating her head against the wall for nine months, Greider arrived at the lab on Christmas Day 1984 to see a signature telomeric sequence show up in her lab results. Knowing, or at least strongly suspecting, that she and Blackburn had their enzyme, Greider went home that evening and rocked out to Bruce Springsteen's *Born in the U.S.A.*

Now that telomerase has given rise to big dreams, Blackburn would like to shift the emphasis away from the notion of anti-aging "magic pills" (no matter how irresistible they are to the media) and toward more practical and less chancy ways of using telomere biology to improve our everyday lives. While she continues to plumb the hows and whys of molecular structure, since 2004 she has immersed herself in an extraordinary collaboration with a young UCSF psychologist, Elissa Epel, who studies the health effects of stress, to refine and broaden the use of a single lab measurement—the average length of telomeres in white blood cells—both as a marker for our current overall health and to predict our future well-being (or not-so-well-being).

A fast-growing body of studies tells us that shorter-than-average telomeres are associated with a far greater risk of dying from heart disease or cancer. In studies that have tracked twins over time, the twin with the longer telomeres has lived longer 60 percent of the time. (And a twin who exercises more is likely to have longer telomeres.) More telling than a single reading are multiple readings taken over time. Holding steady or slowing the rate of telomere decline indicates that you've made beneficial changes resulting in a healthier metabolism overall. (Some studies suggest that natural telomerase production can add length to telomeres over short periods of time.) On the other hand, based on a preliminary analysis of data from one large study not yet published, a sharp drop in telomere length over the previous five years indicates that a subject stands an excellent chance of dropping dead within the next three years.

Is this a crystal ball you really want to peer into? Blackburn's own experiences suggest that we do. "People seem to respond," she says. "Every party I go to, every person I talk to, they want their telomere length." This is a good thing, she believes, because telomere length is a test capable of delivering bad news that you can *do* something about. So, for that matter, is a cholesterol reading—and here, the comparison is instructive. Both are numbers that reflect your particular genetic makeup—in one case, how much cholesterol your liver has been programmed to make, and in the other, the length of your telomeres (at birth, you have approximately 10,000 DNA base pairs, about a third of which you'll lose by age 20). And both tests reflect your environment and lifestyle: diet, exercise, stress, the works. How well you maintain your cholesterol readings or your telomere length for the rest of your life is very much a function of how you live it.

The differences between the tests are telling too. An LDL cholesterol reading only tells you about your risk of heart disease, and while a high number is certainly a risk factor, it's hardly the kiss of death—most people with high readings don't die from atherosclerosis. But telomere length reflects any number of underlying things going on with your metabolism, and if you have a number that's significantly low for your age, especially one dropping faster than average, you'll ignore it at your own peril.

This is typical new science, in which the line between causation and correlation can blur. For instance, stress causes disease. As we shall see below, stress causes telomere loss. Does the telomere loss cause the disease? "Partially," Blackburn says with a smile. In other words, there are lots of gaps in our understanding of telomeres and the aging process; Blackburn aims to fill in quite a few of them with a massive new study she's undertaking with the giant California-based HMO Kaiser Permanente, which over the past few months has supplied her lab with saliva samples from 100,000 patients to assay for telomere length. That data will then be analyzed against detailed patient records (medical histories, work environments, etc.), and meaningful patterns in the results should emerge and become clearer over time. Some patients are being reanalyzed so telomere length and health outcomes can be tracked in sync. Kaiser also has genetic information on some of these patients, so Blackburn will be able to trace links between subtle but common mutations that affect telomere length (as opposed to the freakish but rare mutations that account for diseases such as Werner syndrome), as well as what's going on in people's lives that might help explain, for instance, why one person is at risk for heart disease and another for cancer. "With this number of patients, we'll be able to 'see' these genetic/nongenetic interactions," she says. "Because I think that's where the money is. That's a terrible phrase—but I think this may be *big*."

To handle the magnitude of this job, Blackburn oversaw installation of a custom-built robotic system that sends thousands of samples daily through a centrifuge and a PCR (polymerase chain reaction) machine to obtain telomere data with minimal human involvement. The system is humming away in a room the size of a large storage closet a few yards down the hall from her office. "This was our first foray into really big automation," she says. With the telomere numbers made more reliable via this sort of deep epidemiological drilling, Blackburn foresees that five or 10 years from now you might take a monthly reading with something akin to today's blood-sugar monitors, input it into your telomere iPhone app, "and say, 'Oh, here's my trend—it's lookin' okay.'"

In the meantime, Blackburn has taken what she has learned from her research with Epel on the effects of exercise and meditation to keep her telomeres in good shape. "Do you have to pop a pill, or do you tweak your physiology, which has been evolving for millions of years?" she asks rhetorically. "I really think about this stuff. For Christmas, my husband [UCSF biochemistry professor John Sedat] and I gave each other an elliptical trainer, and this time I was really smart and put it right in front of the TV. So you do your half hour, and you're watching something interesting. And I've learned to meditate. My life doesn't allow me four hours a day of meditation, but I do short bursts, on the [campus] shuttle bus or when you're not allowed to use the computer on the plane. It's not unpleasant."

(CONTINUED ON *page* 222)

HEY—WANNA LIVE FOREVER?

(CONTINUED FROM *page 156*)

"I'm bored with stress," Elissa Epel confesses when I visit her UCSF office. Whereas Blackburn radiates a kind of matriarchal confidence—it would take a lot to throw her off her game, though the last Bush administration did manage to force her off the President's Council on Bioethics in 2004 for refusing to toe the party line against stem-cell research—the petite, sharp-featured Epel gives off a whirlwind vibe. "I've read every article on stress. I've written lots about it. And yet we still don't know very well how to measure or to reduce it, partly because it's such an individual thing."

Epel latched on to telomeres (and to Blackburn) not to measure stress, per se (because telomere length is dependent on all sorts of factors), but to enable her to calibrate the toll that stress can take on health. In their first and still most famous collaboration, Epel and Blackburn studied women who were primary caregivers to ill and dependent family members. These women were all in similar high-stress situations, but the group that reported feeling most overwhelmed had telomeres approximately 10 years shorter than those in the better-coping group (that is, their telomeres were shorter by about the length a middle-aged person might lose over 10 years).

In lectures, Epel often uses a quote from the father of stress theory, physiologist Hans Selye: "Every stress leaves an indelible scar, and the organism pays for its survival after a stressful situation by becoming a little older." With telomere measurements, Epel can gauge, in a rough way, biological aging and how stress can accelerate it. "We can see stress-induced damage at the level of DNA decades before people actually develop disease," she says. "We're hoping this motivates people to take control of their health by making lifestyle changes—not just pounds on the scale, which are very hard to lose, but by doing things that promote healthier immune cells."

How, exactly? Research is yielding some preliminary answers, in some cases providing hard-science backup for things we already knew. Consuming a plentiful amount of omega-3 fatty acids correlates with having nice, long telomeres. For the caregivers in the highest-reported-stress group, regular daily exercise was a major buffer against telomere loss (but interestingly, in the women who were coping relatively well, exercise didn't alter the state of their telomeres much). In a new study, Epel has found that the psychological measure most predictive of telomere length was how the women answered questions about whether they were focused in the present moment or were wishing they were somewhere else: The latter group had shorter telomeres.

"It does suggest that there's a lot we can do with our state of mind," Epel says, "not by *trying* to be happy or to reduce stress, but by being present. I personally don't have an affinity for meditation, but I do yoga, which brings you right there to the moment."

If measuring the effect of mindfulness on your DNA sounds pretty abstract, Epel notes that knowing your telomere length can have some very practical benefits. Thanks to a major study in Scotland that tracked telomeres and heart disease, we know that statins, the most commonly prescribed drugs in America, probably have no effect on people with high cholesterol who also have long telomeres. Similar work on telomeres and depression may help settle another hugely vexing medical question: who is a good candidate for antidepressant drugs, and who is not?

Measuring telomere length is still mostly a research tool, but to speed up the transit from the lab to your doctor's office, Blackburn and Epel have joined forces with Calvin Harley, a leading telomere biologist turned Geron chief scientist turned entrepreneur who last year founded Telome Health, Inc. "We're going to commercialize this technology," Harley tells me when I visit his start-up's digs an hour south of UCSF, in Menlo Park. "In the next five to 10 years, we'd like to have our test done more frequently than cholesterol readings. And there are 150 million cholesterol tests done every year."

It turns out that our extended-play telomere- and telomerase-driven future is being worked out not only in Bay Area labs but also in my own backyard. I pay a visit to Joe Raffaele, MD, a former clinical assistant professor of medicine at Dartmouth who runs an anti-aging—"age-management" is the term of art—practice on Manhattan's Central Park South. Raffaele is himself a walking advertisement for his services, a lean, handsome devil who looks at least a decade younger than his 51 years—the result, in part, he says, of taking the hormone supplements (testosterone and human growth hormone) that he often prescribes to his patients. Hormone supplementation, which often raises troubled eyebrows in mainstream medical circles, has been anti-aging medicine's most potent armamentarium, but that could be about to change. Raffaele tells me he has 25 female patients—several of whom are rich and famous, and whose names he declines to divulge—taking Noel Patton's TA-65 supplement. In sum, Raffaele says, "We've got a perfect storm. We have a way to measure telomere lengths, we have a massive amount of data about their importance in chronic diseases of aging, and now we have interventional studies."

Actually, as of this writing, we only have one published clinical study, which Raffaele himself helped shepherd into existence, looking at telomerase activation in humans. By 2007, Patton had spent some of the millions he'd made in his family appliance-manufacturing business to create TA Sciences, acquire the license to Geron's TA formula, and manufacture the supplement he labeled TA-65. (The number is a throwaway, a nod to the traditional age of retirement.) Patton could have simply sold the product on the Internet or tried to get it in health food stores, but he wanted to take what, by supplement-industry standards, is the high road: to persuade reputable doctors to sell it to their patients—no easy feat, given the known fact that metastasizing cancers are fueled by telomerase. (Geron is in clinical trials with a breast-cancer drug, Imetelstat, that works by *inhibiting* telomerase.) Even if virtually all the scientific evidence suggests that telomerase by itself can't induce a good cell to go bad, there remains at least a theoretical risk that upping telomerase might help a clump of premalignant nonproliferating cells to cross the fatal divide.

Patton eventually convinced Raffaele to put together, with TA Sciences consultant Harley, an observational study that would monitor and evaluate the almost 100 people who had paid $25,000 to take TA-65 along with an accompanying package of standard nutritional supplements. (These folks weren't about to stop taking their supplements, so they were included in the protocol.) This was a cross-your-fingers human experiment made possible only by our schizophrenic regulatory policies: While the FDA puts pharmaceutical drugs through an enormously expensive safety-and-efficacy-testing wringer, supplements get the FDA's "buyer beware" treatment as long as their active ingredients have a history of human use, though it's doubtful that anyone had ever before consumed such enormous concentrations of one fairly obscure molecule found in the root of the astragalus plant. "Whenever you're doing an intervention that's related to a basic biological process, you need to be very cautious that you do no harm," says Jeffrey Bland, PhD, a supplement-industry savant and the head of a research nonprofit, The Institute for Functional Medicine.

The experiment has paid off, Raffaele and Harley believe. They saw improvements in a number of health measurements Raffaele uses in his clinical practice (skin elasticity, arterial stiffness, cognitive function, and others)—and, thankfully, no cancer. A year into his study, Raffaele concluded that TA-65 was the real deal and began recommending it, cautiously, to other patients over 40 whose lab work suggested that their immune systems could use a boost. (Forty is the minimum age recommended by TA Sciences; before that age, short telomeres aren't much of a health concern, and starting on a telomerase activator at a younger age does increase doctors' anxiety about lifetime cancer risk.) "If a patient asks, I won't tell them that I'm sure this won't have an adverse effect," Raffaele says. "Because we really don't have good long-term safety data." What he does have are a lot of glowing anecdotes from TA-65 clients. "But that's the problem with anecdotal responses," he says. "If you pony up $25,000, you're going to want to feel good." Nevertheless, Raffaele was struck that most of the study subjects and his patients on TA-65 reported feeling renewed energy, without jitters; sexual performance got high marks (from the men, at least); and there were some unexpected reports of improvement in eyesight. (Former Yale associate clinical professor of medicine Florence Comite, who has her own age-management practice on Manhattan's Upper East Side, says she was initially baffled when her longtime eyeglass prescription came down in strength and can only assume that taking the supplement herself for the past year is the reason.)

"Intriguing" might be a fair description for what was, after all, only a pilot study with no control group that focused on subtle and open-to-interpretation improvements in immune-system response. (A proper placebo-controlled study tracking elderly TA-65 clients is on TA Sciences' drawing board, and a recent controlled study showed mice doing nicely on the compound, with no statistically significant added cancer risk.) Skeptics such as DePinho and Greider see the results as flimsy at best, offering no hard evidence of telomere lengthening or any other improvement. ("Oh, we've got this magic elixir," mocks DePinho.) But for those ready to be persuaded that life extension or, at any rate, extra protection against the diseases of aging, might be found in a single supplement, there's enough there. Media attention perked up, demand increased, and Patton signed up a slew of new doctors to sell his product, some of whom do so on their websites.

The ideal of tracking and testing every single TA-65 patient went by the wayside as the once-atmospheric price dropped. "We still encourage people to do the testing," Patton says. "But the market is the ultimate decider, and a lot of people don't want the expense and the burden of all the blood draws." Or, as Ed Park, a Harvard-trained

ob-gyn, puts it, "At this point, there is a critical mass of people addicted to it." Park fell so in love with his own experience on the supplement (he says he lost his midlife spare tire without even trying) that he turned his San Fernando Valley practice into a kind of TA-65 dispensary, with appropriate testing when patients are willing. He also does weekly webcasts interviewing patients, doctors, and scientists about the wonders of telomeres and TA-65. (Look him up on YouTube.) "The more you talk about it, the more it sounds like bullshit," he admits. "It's most unbelievable to the people actually taking it. That's why I decided to do the webcasts. We have to talk to each other and say, 'Is this really happening?' "

The bubble could burst. The FDA might decide to swoop in for a closer look. Or a formerly healthy TA-65 client could be diagnosed with cancer. Given the incidence of cancer and TA Sciences' expanding sales, that seems an inevitable eventuality. But to the best of the company's knowledge, it hasn't happened yet—a testament to luck, or to the modest potency of the formula, or to the paradoxical relationship between telomerase and cancer. As Blackburn wrote back in the '90s, telomerase is a Dr. Jekyll and Mr. Hyde sort of enzyme. The brutish Mr. Hyde fuels runaway cancer growth, but the civilized Dr. Jekyll has a couple of different talents: Telomerase keeps the immune system restocked with new cells responsible for keeping small cancers in check; and protecting telomeres maintains the genetic stability of cells and prevents chromosomes from fraying at the ends (remember the shoelaces!), reducing the likelihood of a cancerous mutation. Dr. Jekyll has been much more in evidence in the research literature these past few years—witness a *Journal of the American Medical Association* study from last summer that found people with short telomeres were three times more likely to develop cancer and 11 times more likely to die from it.

I visited Johns Hopkins University in Baltimore to let Carol Greider have the last word—after all, it was she who found telomerase in that single-celled pond-dweller. Like Blackburn, Greider and her lab are still busy pulling apart the basic molecular machinery of the telomere. And she, too, is excited about the human dimension of telomere biology—although the UCSF studies on exercise and meditation are way too squishy for her. "I think you should exercise whether or not it elongates your telomeres," the former competitive triathlete says with a laugh. "We're focused on diseases and the drugs you could treat them with."

She and a colleague, Mary Armanios, are bearing down on diseases such as pulmonary fibrosis, in which there's a demonstrable connection between a mutation affecting telomeres and what goes fatally wrong in the lungs. Fifty thousand people a year die from pulmonary fibrosis in the U.S., Greider says. For her, regarding efforts to bring a telomerase-enhancing compound to the general public as a good-for-what-ails-you supplement, the less said the better. "I don't care about the supplement people," she fairly shouts. "That's not real science!" Still, she allows that the list of major diseases caused, at least in part, by telomere dysfunction is bound to increase. That very day, Armanios publishes a paper linking diabetes to telomere-related genetic factors. Can their connection to heart disease be far behind? The ripples from those tetrahymena zipping around the pond keep expanding ever outward. ●

SOUL PROVIDER

(CONTINUED FROM *page 168*)

these women can transcend their suffering and go on to serve the greater good, then surely I can stop spewing mind venom at the person who stole my laptop.

At the lecture, Bernstein leads us in a forgiveness meditation. (You can download it, free, on Gabbyb.tv.) We close our eyes and breathe, and Bernstein tells us to invite into the room the image of a person we resent, who we need to forgive. I suddenly see Chris standing before me. I'd broken up with him a few days earlier—two trite e-mails were exchanged, and *le fin*—but now, here he is, wearing a private-equity-perfect button-down and that wide Franco smile. Bernstein's voice is low and steady. "Take a deep breath in: I call on the willingness to forgive you. Breathe out: I choose to release you." The room has fallen into deep silence; Radiohead plays from Bernstein's iPod. "Envision a black cord between you and the other person; this cord represents your anger and resentment. See in your mind an image of the angel Raphael [a biblical healer and patron saint of travelers] flying in with a golden pair of scissors. He stands over you and gently cuts the cord. On your exhale, watch the cord fall to the ground." When I look up, Chris isn't there anymore; I am, in my new black dress on New Year's Day, having just left his apartment to catch the waning dawn. My heavy eye makeup seems to have migrated to my forehead. I look disheveled but happy, trying to piece together the night before. Yes, I should have known: It's me I've needed to forgive. Some thoughtless misdeeds by a guy I never even liked, and really never knew, don't come close to the damage I've done myself, clinging to a perception that I'm not worth more than the way he, and other men before him, treated me. My New Year's Day self and I stare each other down. "With each inhalation, breathe in white light," Bernstein says softly. She tells us to envision the light pouring through our body, down our head, face, arms, stomach, pelvis, through our legs, extending from our feet, and down into the earth. "On the exhalation, extend this light to the person who has caused you pain." There I am with my plastic silver tiara. I open my eyes, and the tears that have been welling up fall down my cheeks. I look around and am met by a roomful of red, wet faces. It's heavy—the energy of a hundred women who feel, at once, weightless.

What I learned from Bernstein is that inviting so-called spirit into your life isn't about drum circles and rain dances, or about looking outward, to some ethereal cosmos, for answers. It's about looking inward—and being honest about what you see. Working to perceive the world with compassion—not just for your in-laws but, on some level, for everyone, including the guy who gave you wrong directions on the turnpike—can simply make us happier. And if the world doesn't seem deserving of your kindness, then, hey, get new glasses.

It sounds as pat as a greeting card, but think about that moment you leave a funeral, having faced the fragility of life; everything looks different. Babies seem smilier, the cashier at the drugstore seems kinder, that bridesmaid dress doesn't bother you as much. It's not because anything's changed, but because *you* have, if only for a day or two—simply because you've brought more compassion to the world around you.

The thing about embarking on any spiritual journey is that it doesn't ever really end. Besides a few Zen masters in the world, throw in Gandhi and probably Jesus, too, we're all, for the most part, perennial newcomers. The practice—the meditations, prayers, and struggle to see differently—is a constant, daily thing; what's more, as Bernstein teaches, once you start the "work," the universe (just go with me here) essentially conspires to throw situations in your path that make you confront all the dreck you've yet to clear. I left that first lecture on a pink cloud; the feeling didn't last. What does endure is the desire to shift my perspective. I can't get into the slightest altercation, feel the tiniest bit annoyed, without hearing myself wonder how I'm contributing to the problem and what I can change. I'm a pain in the ass to talk trash with these days. My friends can barely get through a single string of disgruntled what-a-bitches without me calmly inquiring, "Okay, but what's your part in this?" One especially fed-up coworker gave it to me straight: "Your hippie moon shit is killing my bitch buzz." This stuff may be working for me, but I'm not about to mess with *anyone*'s bitch buzz.

It's Monday, and I'm back on the ratty cushions in the Hare Krishna temple. I'm addicted to the energy in this room; Bernstein's like a campfire, crackling and warm, around which secrets can safely be spilled. Tonight we're doing an "inner child" meditation. "There's literally a little girl in each of us who needs to be held," she says. She tells us to close our eyes and picture our childhood selves, at any age, in a moment when we felt sad, alone, unloved. I'm 14. I've just started a new public school, where I don't know a soul; my older brother, Brad, whom I've never liked and swear I never will, attends a private school along with two of my closest friends; they've invited me to a party thrown by someone I've never met. My dad has driven me to this stranger's backyard on a warm Saturday night. I'm wearing cutoffs and my favorite T-shirt, with a picture of sunbathers from the '40s (and a conspicuous Armani Exchange logo). I get out of the car, and before I can find my friends, I catch my brother's eye. "What the hell is my sister doing here?" he yells to the crowd. "Who invited my sister?!" The strangers laugh as they turn to stare. I run back to the driveway, and, by some miracle, my dad hasn't yet driven away. I jump in the front seat, but I'm crying too hard to explain. "I want you to go to this girl, in the depths of her sadness, and comfort her," Bernstein says. I do, I walk right up to her, this me at 14, freckles still dark and peppering my nose. I take her hand. Suddenly, I see us levitating clear off the ground, floating, now flying, now soaring above the trees, above all that throbbing hurt down below; it can't touch us.

I leave Bernstein's class overwhelmed by memories of Brad. We were always fighting; he never liked having me around. Somewhere in our twenties we'd finally landed on the same page and could share music and literature and genuine laughter—I even started looking up to him with earnest admiration—yet he hadn't been a good brother when it counted. And I had every right to hold on to that! It was *I* who'd suffered! But tonight, I can't fight it: I feel different. What about his own adolescent feelings and apprehensions that I was always too "right" to consider? Why not focus on the relationship Brad and I managed to build as adults? Am I going to let it be obscured, for the rest of my life, by the kids we used to be?

I call my brother. We talk as I walk the 10 blocks home. "The Braves need a freaking bullpen, that's the problem," he says. "But did you see Jason Heyward's leadoff homer? *Perfection.*" He tells me he hates law school, but I know he'll grow to love it, because his passion for public policy is everything to him; it often leaves me in awe. It's really, really nice to hear his voice. ●