Outlive

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Chapter 1: The Long Game

Odds are overwhelming that you will die as a result of one of the chronic diseases of aging that I call the Four Horsemen: heart disease, cancer, neurodegenerative disease, or type 2 diabetes and related metabolic dysfunction. To achieve longevity—to live longer and live better for longer—we must understand and confront these causes of slow death

Chapter 2: Medicine 3.0

"The time to repair the roof is when the sun is shining." – John F. Kennedy

Chapter 3: Objective, Strategy, Tactics:

I ask all my patients to sketch out an alternative future for themselves. What do you want to be doing in your later decades? What is your plan for the rest of your life?
Note: Begin with the end in mind.
Action: Sketch out my life at 80

Our tactics in Medicine 3.0 fall into five broad domains: exercise, nutrition, sleep, emotional health, and exogenous molecules, meaning drugs, hormones, or supplements.

We will break down this thing called exercise into its most important components: strength, stability, aerobic efficiency, and peak aerobic capacity.

I now consider exercise to be the most potent longevity "drug" in our arsenal, in terms of lifespan and healthspan. The data are unambiguous: exercise not only delays actual death but also prevents both cognitive and physical decline, better than any other intervention. We also tend to feel better when we exercise, so it probably has some harder-to-measure effect on emotional health as well.

Good sleep is critical to our innate physiological repair processes, especially in the brain, while poor sleep triggers a cascade of negative downstream consequences, from insulin resistance to cognitive decline, as well as mental health issues.
Note: Starts with sleep preparation, dark cool room, regular hours.

Chapter 4: Centenarians

This is part of why I place so much importance on taking a detailed family history from my patients: I need to know when your relatives died and why. What are your likely "icebergs," genetically speaking?

Action: Get details on health “icebergs” from family

The short answer is that evolution doesn’t really care if we live that long. Natural selection has endowed us with genes that work beautifully to help us develop, reproduce, and then raise our offspring, and perhaps help raise our offspring’s offspring. Thus, most of us can coast into our fifth decade in relatively good shape. After that, however, things start to go sideways. The evolutionary reason for this is that after the age of reproduction, natural selection loses much of its force. Genes that prove unfavorable or even harmful in midlife and beyond are not weeded out because they have already been passed on. To pick one obvious example: the gene (or genes) responsible for male pattern baldness. When we are young, our hair is full and glorious, helping us attract mates. But natural selection does not really care whether a man in his fifties (or a woman, for that matter) has a full head of hair.
Note: The peacock loses it feathers.

Hair loss is not terribly relevant to longevity, luckily for me. But this general phenomenon also explains why genes that might predispose someone to Alzheimer’s disease or some other illness, later in life, have not vanished from our gene pool. In short, natural selection doesn’t care if we develop Alzheimer’s disease (or baldness) in old age. It doesn’t affect our reproductive fitness. By the time dementia would appear, we have likely already handed down our genes. The same is true of genes that would accelerate our risk of heart disease or cancer in midlife.

While your genome is immutable, at least for the near future, gene expression can be influenced by your environment and your behaviors. For example, a 2007 study found that older people who were put on a regular exercise program shifted to a more youthful pattern of gene expression after six months. This suggests that genetics and environment both play a role in longevity and that it may be possible to implement interventions that replicate at least some of the centenarians’ good genetic luck.
Note: 6 months to change your genes, pretty dope

Chapter 5: Eat Less, Live Longer

The results have been remarkably consistent. Studies dating back to the 1930s have found that limiting caloric intake can lengthen the lifespan of a mouse or a rat by anywhere from 15 to 45 percent, depending on the age of onset and degree of restriction. Not only that, but the underfed animals also seem to be markedly healthier for their age, developing fewer spontaneous tumors than normally fed mice. CR seems to improve their healthspan in addition to their lifespan.
Action: Don’t snack, don’t eat when not hungry.

The life-extending effect of CR seems to be almost universal. Numerous labs have found that restricting caloric intake lengthens lifespan not only in rats and mice (usually) but also in yeast, worms, flies, fish, hamsters, dogs, and even, weirdly, spiders.

Chapter 6: The Crisis of Abundance

According to the Centers for Disease Control (CDC), more than 40 percent of the US population is obese (defined as having a BMI greater than 30), while roughly another third is overweight (BMI of 25 to 30).
Action: Track BMI and get under 25

Today we call this cluster of problems "metabolic syndrome" (or MetSyn), and it is defined in terms of the following five criteria: high blood pressure (>130/85) high triglycerides (>150 mg/dL) low HDL cholesterol (<40 mg/dL in men or <50 mg/dL in women) central adiposity (waist circumference >40 inches in men or >35 in women) elevated fasting glucose (>110 mg/dL) If you meet three or more of these criteria, then you have the metabolic syndrome—along with as many as 120 million other Americans, according to a 2020 article in JAMA. About 90 percent of the US population ticks at least one of these boxes. But notice that obesity is merely one of the criteria; it is not required for the metabolic syndrome to be diagnosed.
Action: Track and hit the following:

1. Blood Pressure under 130/85
2. Triglycerides under 150 mg/dL
3. HDL Cholesterol greater than 40 mg/dL (men) 50 mg/dL (women)
4. Waist circumference under 40 inches in men or 35 in women
5. Fasting Glucose under 110 mg/dL

It doesn't take much visceral fat to cause problems. Let's say you are a forty-year-old man who weighs two hundred pounds. If you have 20 percent body fat, making you more or less average (50th percentile) for your age and sex, that means you are carrying 40 pounds of fat throughout your body. Even if just 4.5 pounds of that is visceral fat, you would be considered at exceptionally high risk for cardiovascular disease and type 2 diabetes, in the top 5 percent of risk for your age and sex. This is why I insist my patients undergo a DEXA scan annually—and I am far more interested in their visceral fat than their total body fat.
Action: Get DEXA scan for accurate body fat measure as well as visceral fat reading

One abundant source of calories in our present diet, fructose, also turns out to be a very powerful driver of metabolic dysfunction if consumed to excess. Fructose is not a novel nutrient, obviously. It's the form of sugar found in nearly all fruits, and as such it is essential in the diets of many species, from bats and hummingbirds up to bears and monkeys and humans. But as it turns out, we humans have a unique capacity for turning calories from fructose into fat.
Action: Cut back on fruit

Fructose isn't the only thing that creates uric acid; foods high in chemicals called purines, such as certain meats, cheeses, anchovies, and beer, also generate uric acid.

In my patients, I monitor several biomarkers related to metabolism, keeping a watchful eye for things like elevated uric acid, elevated homocysteine, chronic inflammation, and even mildly elevated ALT liver enzymes. Lipoproteins, which we will discuss in detail in the next chapter, are also important, especially triglycerides; I watch the ratio of triglycerides to HDL cholesterol (it should be less than 2:1 or better yet, less than 1:1), as well as levels of VLDL, a lipoprotein that carries triglycerides—all of which may show up many years before a patient would meet the textbook definition of metabolic syndrome.

Action: Measure and asses ratio of triglycerides to HDL cholesterol (it should be less than 2:1 or better yet, less than 1:1)

But the first thing I look for, the canary in the coal mine of metabolic disorder, is elevated insulin.
Action: Measure and monitor insulin levels

Chapter 7: The Ticker

What is the most common "presentation" (or symptom) of heart disease? It wasn't chest pain, left arm pain, or shortness of breath, the most common answers; it was sudden death. You know the patient has heart disease because he or she has just died from it.
Note: Frightening

Globally, heart disease and stroke (or cerebrovascular disease), which I lump together under the single heading of atherosclerotic cardiovascular disease, or ASCVD, represent the leading cause of death, killing an estimated 2,300 people every day in the United States, according to the CDC—more than any other cause, including cancer.

American women are up to ten times more likely to die from atherosclerotic disease than from breast cancer (not a typo: one in three versus one in thirty). But pink ribbons for breast cancer far outnumber the American Heart Association's red ribbons for awareness of heart disease among women.

While heart disease is the most prevalent age-related condition, it is also more easily prevented than either cancer or Alzheimer's disease.

Your doctor will probably utter it with a frown, because as everyone knows, cholesterol is evil stuff. Well, some of it is—you know, the LDL or "bad" cholesterol, which is inevitably counterpoised against the HDL, or "good" cholesterol. I practically need to be restrained when I hear these terms, because they're so meaningless. And your "total cholesterol," the first number that people offer up when we're talking about heart disease, is only slightly more relevant to your cardiovascular risk than the color of your eyes.
Note: Need to get doctor to read this, they always offer total calculated number vs measured components.

HDL particles are wrapped in a type of molecule called apolipoprotein A (or apoA), while LDL is encased in apolipoprotein B (or apoB). This distinction may seem trivial, but it goes to the very root cause of atherosclerotic disease: every single lipoprotein that contributes to atherosclerosis—not only LDL but several others—carries this apoB protein signature.
Action: Measure and monitor apoB

Eating lots of saturated fat can increase levels of atherosclerosis-causing lipoproteins in blood, but most of the actual cholesterol that we consume in our food ends up being excreted out our backsides. The vast majority of the cholesterol in our circulation is actually produced by our own cells.
Note: Partially saturated fat in diet, but mostly made in house

"There's no connection whatsoever between cholesterol in food and cholesterol in blood," Keys said in a 1997 interview. "None. And we've known that all along. Cholesterol in the diet doesn't matter at all unless you happen to be a chicken or a rabbit."
Note: Not a cholesterol consumption issue.

Fully half of all major adverse cardiovascular events in men (and a third of those in women), such as heart attack, stroke, or any procedure involving a stent or a graft, occur before the age of sixty-five. In men, one-quarter of all events occur before age fifty-four.

So to gauge the true extent of your risk, we have to know how many of these apoB particles are circulating in your bloodstream. That number is much more relevant than the total quantity of cholesterol that these particles are carrying.

I have all my patients tested for apoB regularly, and you should ask for the same test the next time you see your doctor. (Don't be waved off by nonsensical arguments about "cost": It's about twenty to thirty dollars.)

I take a very hard line on lowering apoB, the particle that causes all this trouble. (In short: get it as low as possible, as early as possible.)

This is not an atypical scenario: when a patient comes to me and says their father or grandfather or aunt, or all three, died of "premature" heart disease, elevated Lp(a) is the first thing I look for. It is the most prevalent hereditary risk factor for heart disease, and its danger is amplified by the fact that it is still largely flying under the radar of Medicine 2.0, although that is beginning to change.

We test every single patient for Lp(a) during their first blood draw. Because elevated Lp(a) is largely genetic, the test need only be done once

If we all maintained the apoB levels we had when we were babies, there wouldn't be enough heart disease on the planet for people to know what it was.

While there are seven statins on the market, I tend to start with rosuvastatin (Crestor) and only pivot from that if there is some negative effect from the drug (e.g., a symptom or biomarker). My goal is aggressive: as rationalized by Peter Libby, I want to knock someone's apoB concentration down to 20 or 30 mg/dL, about where it would be for a child.
Action: Measure and start statin if apoB is higher than 30 mg/dL

For people who can't tolerate statins, I like to use a newer drug, called bempedoic acid (Nexletol),

As a monotherapy they have about the same apoB- or LDL-C-lowering potency as high-dose statins, but their most common use is in addition to statins; the combination of statins plus PCSK9 inhibitors is the most powerful pharmacological tool that we have against apoB. Alas, statins do not reduce Lp(a), but PCSK9 inhibitors do in most patients, typically to the tune of about 30 percent.
Action: Measure Lp(a) and start on PCSK9 as needed

Triglycerides also contribute to the apoB particle burden, because they are largely transported in VLDLs. Our dietary interventions are aimed at reducing triglycerides, but in cases where nutritional changes are insufficient, and in cases where genetics render dietary interventions useless, fibrates are the drug of choice.
Action: If diet does not get triglycerides under 150 mg/dL, look at fibrates

While the CT angiogram costs a bit more, requires IV dye, and exposes the patient to slightly more radiation, I struggle to find credible arguments against its use. Approximately 15 percent of people who have a normal calcium score (0) are still found to have soft plaque or even small calcifications on CT angiograms, and as many as 2 to 3 percent of people with a zero calcium score are found on CT angiogram to have high-risk plaques. For this reason, I almost always prefer my patients to have a CT angiogram over a calcium scan if we opt to look for evidence of disease via imaging studies.
Note: My calcium score was good but it might have been the wrong test.
Action: Schedule CT angiogram

Chapter 8: The Runaway Cell

It is this metastatic cancer that is responsible for most cancer deaths.

With a few exceptions, such as glioblastoma or other aggressive brain tumors, as well as certain lung and liver cancers, solid organ tumors typically kill you only when they spread to other organs. Breast cancer kills only when it becomes metastatic. Prostate cancer kills only when it becomes metastatic. You could live without either of those organs. So when you hear the sad story of someone dying from breast or prostate cancer, or even pancreatic or colon cancer, they died because the cancer spread to other, more critical organs such as the brain, the lungs, the liver, and bones. When cancer reaches those places, survival rates drop precipitously.

Globally, about 12 to 13 percent of all cancer cases are thought to be attributable to obesity. Obesity itself is strongly associated with thirteen different types of cancers, including pancreatic, esophageal, renal, ovarian, and breast cancers, as well as multiple myeloma (see figure 7). Type 2 diabetes also increases the risk of certain cancers, by as much as double in some cases (such as pancreatic and endometrial cancers). And extreme obesity (BMI ≥ 40) is associated with a 52 percent greater risk of death from all cancers in men, and 62 percent in women.
Note: Don't get fat

I suspect that the association between obesity, diabetes, and cancer is primarily driven by inflammation and growth factors such as insulin. Obesity, especially when accompanied by accumulation of visceral fat (and other fat outside of subcutaneous storage depots), helps promote inflammation, as dying fat cells secrete an array of inflammatory cytokines into the circulation (see figure 4 in chapter 6). This chronic inflammation helps create an environment that could induce cells to become cancerous. It also contributes to the development of insulin resistance, causing insulin levels to creep upwards—and, as we'll see shortly, insulin itself is a bad actor in cancer metabolism.

There is already some evidence that tinkering with metabolism can affect cancer rates. As we have seen, laboratory animals on calorically restricted (CR) diets tend to die from cancer at far

lower rates than control animals on an ad libitum (all-they-can-eat) diet. Eating less appears to give them some degree of protection.

What I am saying is that we don't want to be anywhere on that spectrum of insulin resistance to type 2 diabetes, where our cancer risk is clearly elevated. To me, this is the low-hanging fruit of cancer prevention, right up there with quitting smoking. Getting our metabolic health in order is essential to our anticancer strategy.

Work by Valter Longo of the University of Southern California and others has found that fasting, or a fasting-like diet, increases the ability of normal cells to resist chemotherapy, while rendering cancer cells more vulnerable to the treatment. It may seem counterintuitive to recommend fasting to cancer patients, but researchers have found that it caused no major adverse events in chemotherapy patients, and in some cases it may have improved the patients' quality of life. A randomized trial in 131 cancer patients undergoing chemotherapy found that those who were placed on a "fasting-mimicking diet" (basically, a very low-calorie diet designed to provide essential nutrients while reducing feelings of hunger) were more likely to respond to chemotherapy and to feel better physically and emotionally.
Note: Fast if chemo is required

An immunotherapy is any therapy that tries to boost or harness the patient's immune system to fight an infection or other condition (example: vaccines).

Most polyps remain small and harmless and never become cancerous, but some have the potential to become malignant and invade the wall of the colon. Not all polyps become cancer, but all colon cancers came from polyps.

In my practice, we go further, typically encouraging average-risk individuals to get a colonoscopy by age forty—and even sooner if anything in their history suggests they may be at higher risk. We then repeat the procedure as often as every two to three years, depending on the findings from the previous colonoscopy.
Action: Set colonoscopy

It remains one of the top five deadliest cancers in the United States, behind lung (#1) and breast/prostate (#2 for women/men), and just ahead of pancreas (#4) and liver (#5) cancers. Of these five, though, CRC is the one we have the best shot at catching early.

15 percent of lung cancers are diagnosed in people who have never smoked. Lung cancer is the #1 cause of cancer deaths overall, but lung cancer in never-smokers ranks seventh, all by itself.

The only modifiable risks that really stand out in the data are smoking, insulin resistance, and obesity (all to be avoided)—and maybe pollution (air, water, etc.), but the data here are less clear.
Note: Don't smoke, and stay fit.

If the first rule of cancer is "Don't get cancer," the second rule is "Catch it as soon as possible."

Chapter 9: Chasing Memory

The greatest obstacle to discovery is not ignorance—it is the illusion of knowledge. —Daniel J. Boorstin

Although Alzheimer's disease was first named in the early 1900s, the phenomenon of "senility" has been remarked on since ancient times. Plato believed that because advancing age seemingly "gives rise to all manners of forgetfulness as well as stupidity," older men were unsuited for leadership positions requiring acumen or judgment. William Shakespeare gave us an unforgettable portrayal of an old man struggling with his failing mind in King Lear.
Note: Willy Wonka's grandfather too

Typically, what we call Alzheimer's disease (or late-onset Alzheimer's disease) does not present in significant numbers until age sixty-five. But Dr. Alzheimer's own Patient Zero, Auguste Deter, showed severe symptoms by the time she was fifty, a trajectory more in line with early-onset Alzheimer's disease than with the dementia that slowly begins to afflict people in their late sixties, seventies, and eighties.

One important section of the cognitive testing evaluates the patient's sense of smell. Can they correctly identify scents such as coffee, for example? Olfactory neurons are among the first to be affected by Alzheimer's disease.
Note: Daily test, can you smell the coffee?

"The evidence from dozens of rat experiments seemed to be screaming at me," he later wrote. In those experiments, he had restricted the amount of blood flowing to the rats' brains, and over time they had developed symptoms remarkably similar to those of Alzheimer's disease in humans: memory loss and severe atrophy of the cortex and hippocampus. Restoring blood flow could halt or reverse the damage to some extent, but it seemed to be more severe and more lasting in older animals than younger ones. The key insight was that robust blood flow seemed to be critical to maintaining brain health.

The brain is a greedy organ. It makes up just 2 percent of our body weight, yet it accounts for about 20 percent of our total energy expenditure.

The dementia symptoms that we see result from a gradual reduction in blood flow, which eventually creates what he calls a "neuronal energy crisis," which in turn triggers a cascade of unfortunate events that harms the neurons and ultimately causes neurodegeneration. The amyloid plaques and tangles come later, as a consequence rather than a cause. "We believed, and still do, that amyloid-beta is an important pathological product of neurodegeneration," de la Torre wrote recently, "…[but] it is not the cause of Alzheimer's disease."

Scientists and physicians have long noted a connection between Alzheimer's disease and metabolic dysfunction. Having type 2 diabetes doubles or triples your risk of developing Alzheimer's disease, about the same as having one copy of the APOE e4 gene. On a purely mechanistic level, chronically elevated blood glucose, as seen in type 2 diabetes and prediabetes/insulin resistance, can directly damage the vasculature of the brain. But insulin resistance alone is enough to elevate one's risk.
Note: Stay fit, keep blood flow, avoid dementia

Just like reduced blood flow, reduced glucose metabolism essentially starves these neurons of energy, provoking a cascade of responses that include inflammation, increased oxidative stress, mitochondrial dysfunction—and ultimately neurodegeneration itself.

The best interpretation I can draw from the literature suggests that at least four sessions per week, of at least twenty minutes per session, at 179 degrees Fahrenheit (82 degrees Celsius) or hotter seems to be the sweet spot to reduce the risk of Alzheimer's by about 65 percent (and the risk of ASCVD by 50 percent).
Action: Get a sauna (or access) and use it four times per week, 20+ minutes per session, at 179 degrees

Chapter 10: Thinking Tactically

Long ago, when we consumed fructose mainly in the form of fruit and honey, it enabled us to store energy as fat to survive cold winters and periods of scarcity. Fructose was our friend. Now fructose is vastly overabundant in our diet, too much of it in liquid form, which disrupts our metabolism and our overall energy balance.
Action: Stay of the fruit and fructose unless trying to store fat for winter.

Chapter 11: Exercise

Going from zero weekly exercise to just ninety minutes per week can reduce your risk of dying from all causes by 14 percent. It's very hard to find a drug that can do that.

Study after study has found that regular exercisers live as much as a decade longer than sedentary people. Not only do habitual runners and cyclists tend to live longer, but they stay in better health, with less morbidity from causes related to metabolic dysfunction.
Note: How many times have we heard about the exercisers that drop dead on a run or ride? Its just an excuse. Data does not back up sedentary lifestyle.

It turns out that peak aerobic cardiorespiratory fitness, measured in terms of VO2 max, is perhaps the single most powerful marker for longevity. VO2 max represents the maximum rate at which a person can utilize oxygen. This is measured, naturally, while a person is exercising at essentially their upper limit of effort. (If you've ever had this test done, you will know just how unpleasant it is.) The more oxygen your body is able to use, the higher your VO2 max.
Action: Improve VO2 max

Consider this: A person who smokes has a 40 percent greater risk of all-cause mortality (that is, risk of dying at any moment) than someone who does not smoke, representing a hazard ratio or (HR) of 1.40. This study found that someone of below-average VO2 max for their age and sex (that is, between the 25th and 50th percentiles) is at double the risk of all-cause mortality compared to someone in the top quartile (75th to 97.6th percentiles). Thus, poor cardiorespiratory fitness carries a greater relative risk of death than smoking.
Note: Sitting might not be the new smoking but sedentary is.

"Being unfit carried a greater risk than any of the cardiac risk factors examined,"

A ten-year observational study of roughly 4,500 subjects ages fifty and older found that those with low muscle mass were at 40 to 50 percent greater risk of mortality than controls, over the study period. Further analysis revealed that it's not the mere muscle mass that matters but the strength of those muscles, their ability to generate force.
Action: Get jacked.

I will find a way to lift heavy weights in some way, shape, or form four times per week, no matter what else I am doing or where I might be traveling.
Action: Lift four times per week

Chapter 12: Training

The three dimensions in which we want to optimize our fitness are aerobic endurance and efficiency (aka cardio), strength, and stability.

For our purposes, we are interested in two particular regions of this continuum: long, steady endurance work, such as jogging or cycling or swimming, where we are training in what physiologists call zone 2, and maximal aerobic efforts, where VO2 max comes into play.
Note: Zone 2 and VO2 max work are primary aerobic targets

Zone 2 is more or less the same in all training models: going at a speed slow enough that one can still maintain a conversation but fast enough that the conversation might be a little strained.

In technical terms, San Millán describes zone 2 as the maximum level of effort that we can maintain without accumulating lactate. We still produce it, but we're able to match production with clearance. The more efficient our mitochondrial "engine," the more rapidly we can clear lactate, and the greater effort we can sustain while remaining in zone 2. If we are "feeling the burn" in this type of workout, then we are likely going too hard, creating more lactate than we can eliminate.
Note: Should measure but shorthand is MAF method, 180 – Age# = Target Zone 2 HR (138 bpm)

The goal is to keep lactate levels constant, ideally between 1.7 and 2.0 millimoles.

This in turn explains why exercise, especially in zone 2, can be so effective in managing both type 1 and type 2 diabetes: It enables the body to essentially bypass insulin resistance in the muscles to draw down blood glucose levels. I have one patient with type 1 diabetes, meaning he produces zero insulin, who keeps his glucose in check almost entirely by walking briskly for six to ten miles every day, and sometimes more. As he walks, his muscle cells are vacuuming glucose out of his bloodstream via NIMGU.

Based on multiple discussions with San Millán and other exercise physiologists, it seems that about three hours per week of zone 2, or four 45-minute sessions, is the minimum required for most people to derive a benefit and make improvements, once you get over the initial hump of trying it for the first time.
Action: four 45-minute sessions of zone 2 per week

Four times a week, I will spend about an hour riding my stationary bike at my zone 2 threshold.
Note: Always the overachiever

Typically, for patients who are new to exercising, we introduce VO2 max training after about five or six months of steady zone 2 work.

I push my patients to train for as high a VO2 max as possible, so that they can maintain a high level of physical function as they age. Ideally, I want them to target the "elite" range for their age and sex (roughly the top 2 percent). If they achieve that level, I say good job—now let's reach for the elite level for your sex, but two decades younger.

One study found that boosting elderly subjects' VO2 max by 6 ml/kg/min, or about 25 percent, was equivalent to subtracting twelve years from their age.

Even if we are not out to set world records, the way we train VO2 max is pretty similar to the way elite athletes do it: by supplementing our zone 2 work with one or two VO2 max workouts per week.

VO2 max intervals are a bit longer, ranging from three to eight minutes

The tried-and-true formula for these intervals is to go four minutes at the maximum pace you can sustain for this amount of time—not an all-out sprint, but still a very hard effort. Then ride or jog four minutes easy, which should be enough time for your heart rate to come back down to below about one hundred beats per minute. Repeat this four to six times and cool down.

Unless you are training to be competitive in elite endurance sports like cycling, swimming, running, triathlon, or cross-country skiing, a single workout per week in this zone will generally suffice.
Action: 1 VO2 workouts per week, 4 minutes on x 4 minutes off, 4-6 times

We lose muscle strength about two to three times more quickly than we lose muscle mass. And we lose power (strength x speed) two to three times faster than we lose strength.

A study of twelve healthy volunteers with an average age of sixty-seven found that after just ten days of bed rest, which is about what a person would experience from a major illness or orthopedic injury, study participants lost an average of 3.3 pounds of lean mass (muscle). That's substantial, and it shows just how dangerous inactivity can be. If someone is sedentary and consuming excess calories, muscle loss accelerates, because one of the primary destinations of fat spillover is into muscle.

In its most extreme form, this muscle loss is called sarcopenia, as noted in chapter 11. Someone with sarcopenia will have low energy, feelings of weakness, and problems with balance. Sarcopenia is a prime marker for a broader clinical condition called frailty, where a person meets three of these five criteria: unintended weight loss; exhaustion or low energy; low physical activity; slowness in walking; and weak grip strength (about which more soon). It can become difficult to stand or walk, and they are at huge risk of falling and breaking bones.

BMD is important, demanding at least as much attention as muscle mass, so you should at least check your BMD every few years.
Action: Measure and monitor bone mass

This frames how I view strength training in general. It's largely about improving your ability to carry things.

Three or four days a week, I'll spend an hour rucking around my neighborhood, up and down hills, typically climbing and descending several hundred feet over the course of three or four miles. The fifty- to sixty-pound pack on my back makes it quite challenging, so I'm strengthening my legs and my trunk while also getting in a solid cardiovascular session.

(A good goal is to be able to carry one-quarter to one-third of your body weight once you develop enough strength and stamina.)

I structure my training around exercises that improve the following: Grip strength, how hard you can grip with your hands, which involves everything from your hands to your lats (the large muscles on your back). Almost all actions begin with the grip. Attention to both concentric and eccentric loading for all movements, meaning when our muscles are shortening (concentric) and when they are lengthening (eccentric). In other words, we need to be able to lift the weight up and put it back down, slowly and with control. Rucking down hills is a great way to work on eccentric strength, because it forces you to put on the "brakes." Pulling motions, at all angles from overhead to in front of you, which also requires grip strength (e.g., pull-ups and rows). Hip-hinging movements, such as the deadlift and squat, but also step-ups, hip-thrusters, and countless single-leg variants of exercises that strengthen the legs, glutes, and lower back. I focus on these four foundational elements of strength because they are the most relevant to our Centenarian Decathlon—and also to living a fulfilling and active life in our later decades. If you can grip strongly, you can open a jar with ease. If you can pull, you can carry groceries and lift heavy objects. If you can do a hip-hinge correctly, you can get up out of a chair with no problem. You're setting yourself up to age well. It's not about how much weight you can deadlift now, but how well you will function in twenty or thirty or forty years.

Many studies suggest that grip strength—literally, how hard you can squeeze something with one hand—predicts how long you are likely to live, while low grip strength in the elderly is considered to be a symptom of sarcopenia, the age-related muscle atrophy we just discussed. In these studies, grip strength is likely acting as a proxy for overall muscle strength, but it is also a broader indicator of general robustness and the ability to protect yourself if you slip or lose balance. If you have the strength to grab a railing, or a branch, and hold on, you might avoid a fall.

One of the standards we ask of our male patients is that they can carry half their body weight in each hand (so full body weight in total) for at least one minute, and for our female patients we push for 75 percent of that weight.
Action: Measure and monitor ability to farmer carry body weight, about 115 lbs. in each hand.

Here we like to see men hang for at least two minutes and women for at least ninety seconds at the age of forty. (We reduce the goal slightly for each decade past forty.)
Action: Measure and monitor hang time, goal 2 minutes.

This is why we have our patients work up to weighted hip-hinging very slowly, typically beginning with single-leg step-ups (see description below) and split-stance Romanian deadlift, either without weights or with only very light weights held in the hands.

Chapter 13: The Gospel of Stability

That is, older people tend to exercise less, or not at all, because they simply can't. They have hurt themselves in some way, at some point in their lives, and they just never got back on the horse. So they continued to decline.

All of the above, the research and my own experience, support my first commandment of fitness: First, do thyself no harm. How do we do this? I think stability is the key ingredient.

If you are struggling to get through your workout, then you are likely resorting to your body's own "cheats," your ingrained but potentially dangerous movement patterns. Instead, we need to change our approach so that we are focused on doing things right, cultivating safe, ideal movement patterns that allow our bodies to work as designed and reduce our risk of injury.

DNS. Short for dynamic neuromuscular stabilization, DNS sounds complicated, but it is based on the simplest, most natural movements we make: the way we moved when we were babies.

I suggest visiting the websites for DNS (www.rehabps.com) and the Postural Restoration Institute (PRI) (www.posturalrestoration.com), the two leading exponents of what I'm talking about here.

Twice a week, I spend an hour doing dedicated stability training, based on the principles of DNS, PRI, and other practices, with ten to fifteen minutes per day on the other days.
Action: Twice a week stability and mobility training as well as integration into warm up and cool down.

Practice this 360-degree abdominal breathing every day

Now when I squat, or do any standing lift, my first step is to ground my feet, to be aware of all four "corners," and distribute weight equally. (Also important: I prefer to lift barefoot or in minimal shoes, with little to no cushioning in the soles because it allows me feel the full surface of my feet at all times.)

One key test in our movement assessment is to have our patients stand with one foot in front of the other and try to balance. Now close your eyes and see how long you can hold the position. Ten seconds is a respectable time; in fact, the ability to balance on one leg at ages fifty and older has been correlated with future longevity, just like grip strength.

The spine has three parts: lumbar (lower back), thoracic (midback), and cervical (neck) spine. Radiologists see so much degeneration in the cervical spine, brought on by years of hunching forward to look at phones, that they have a name for it: "tech neck."

Chapter 14: Nutrition 3.0

Religion is a culture of faith; science is a culture of doubt. —Richard Feynman

Nutrition is relatively simple, actually. It boils down to a few basic rules: don't eat too many calories, or too few; consume sufficient protein and essential fats; obtain the vitamins and minerals you need; and avoid pathogens like E. coli and toxins like mercury or lead. Beyond that, we know relatively little with complete certainty.
Action: Don't eat too many calories; consume sufficient protein and essential fats; obtain the vitamins and minerals you need; and avoid poison.

The problem is that epidemiology is incapable of distinguishing between correlation and causation. This, aided and abetted by bad journalism, creates confusion. For example, multiple studies have found a strong association between drinking diet sodas and abdominal fat, hyperinsulinemia, and cardiovascular risk. Sounds like diet soda is bad stuff that causes obesity, right? But that is not what those studies actually demonstrate, because they fail to ask an important question: Who drinks diet soda? People who are concerned about their weight or their diabetes risk, that's who.

The point is that humans are terrible study subjects for nutrition (or just about anything else) because we are unruly, disobedient, messy, forgetful, confounding, hungry, and complicated creatures.

This is another problem in the world of nutrition: too many people are majoring in the minor and minoring in the major, focusing too much attention on small questions while all but ignoring the bigger issues.

One classic example of this, I believe, lies in the vast, well-publicized literature correlating "moderate" drinking with improved health outcomes. This notion has become almost an article of faith in the popular media, but these studies are also almost universally tainted by healthy user bias—that is, the people who are still drinking in older age tend to do so because they are healthy, and not the other way around. Similarly, people who drink zero alcohol often have some health-related reason, or addiction-related reason, for avoiding it. And such studies also obviously exclude those who have already died of the consequences of alcoholism. Epidemiology sees only a bunch of seemingly healthy older people who all drink alcohol and concludes that alcohol is the cause of their good health.

Chapter 15: Putting Nutritional Biochemistry …

The leading source of calories that Americans consume is a category called "grain-based desserts," like pies, cakes, and cookies, according to the US Department of Agriculture. That is our number one "food group."

In other words, if you are overnourished, and statistically speaking about two-thirds of us are, you will need to apply at least one of these methods of caloric reduction: deliberately tracking (and reducing) what you eat; cutting out certain foods; and/or giving yourself less time in which to eat. That's it.

In prior chapters, we've seen how excess calories contribute to many chronic diseases, not only metabolic disorders but also heart disease, cancer, and Alzheimer's disease. We also know from decades of experimental data (chapter 5) that eating fewer calories tends to lengthen lifespan, at least in lab animals such as rats and mice—although there is debate over whether this represents true lifespan extension or an elimination of the known hazards of overfeeding, the default state of the control animals in most of these experiments.

One slight advantage is that calorie counting is agnostic to food choices; you can eat whatever you want so long as you stay within your daily allowance.

Avoiding diabetes and related metabolic dysfunction—especially by eliminating or reducing junk food—is very important to longevity. There appears to be a strong link between calories and cancer, the leading cause of death in the control monkeys in both studies. The CR monkeys had a 50 percent lower incidence of cancer. The quality of the food you eat could be as important as the quantity. If you're eating the SAD, then you should eat much less of it. Conversely, if your diet is high quality to begin with, and you are metabolically healthy, then only a slight degree of caloric restriction—or simply not eating to excess—can still be beneficial. I think this last point is key. These two studies suggest that if you are eating a higher-quality diet—and are metabolically healthy to begin with—then severe caloric restriction may not even be necessary. The NIH control monkeys ate as much as they wanted of their better diet and still lived nearly as long as the CR monkeys in both studies.

People tend to (erroneously) assume you can't eat too much if you're just restricting fill-in-the-blank (e.g., carbohydrates). This is incorrect. Even if done correctly and strictly, DR can still result in overnutrition. If you cut out carbohydrates altogether but overdo it on the Wagyu steaks and bacon, you will fairly easily find yourself in a state of caloric excess. The key is to pick a strategy to which you can adhere but that also helps achieve your goals.

The real art to dietary restriction, Nutrition 3.0–style, is not picking which evil foods we're eliminating. Rather, it's finding the best mix of macronutrients for our patient—coming up with an eating pattern that helps them achieve their goals, in a way that they can sustain. This is a tricky balancing act, and it requires us (once again) to forget about labels and viewpoints and drill down into nutritional biochemistry. The way we do this is by manipulating our four macronutrients: alcohol, carbohydrates, protein, and fat. How well do you tolerate carbohydrates? How much protein do you require? What sorts of fats suit you best? How many calories do you require each day? What is the optimal combination for you?

Alcohol It's easy to overlook, but alcohol should be considered as its own category of macronutrient because it is so widely consumed, it has such potent effects on our metabolism, and it is so calorically dense at 7 kcal/g (closer to the 9 kcal/g of fat than the 4 kcal/g of both protein and carbohydrate). Alcohol serves no nutritional or health purpose but is a purely hedonic pleasure that needs to be managed. It's especially disruptive for people who are overnourished, for three reasons: it's an "empty" calorie source that offers zero nutrition value; the oxidation of ethanol delays fat oxidation, which is the exact opposite of what we want if we're trying to lose fat mass; and drinking alcohol very often leads to mindless eating.

Ethanol is a potent carcinogen, and chronic drinking has strong associations with Alzheimer's disease, mainly via its negative effect on sleep, but possibly via additional mechanisms.

My personal bottom line: if you drink, try to be mindful about it. You'll enjoy it more and suffer fewer consequences. Don't just keep drinking because they're serving it on the plane. I strongly urge my patients to limit alcohol to fewer than seven servings per week, and ideally no more than two on any given day, and I manage to do a pretty good job adhering to this rule myself. **Action:** Limit alcohol to fewer than seven servings per week, and ideally no more than two on any given day

The higher their blood glucose, the greater their risk of death—even in the nondiabetic range of blood glucose. Another study in 2019 looked at the degree of variation in subjects' blood glucose levels and found that the people in the highest quartile of glucose variability had a 2.67 times greater risk of mortality than those in the lowest (most stable) quartile. From these studies, it seems quite clear that we want to lower average blood glucose and reduce the amount of variability from day to day and hour to hour.

Overall, I like to keep average glucose at or below 100 mg/dL, with a standard deviation of less than 15 mg/dL. These are aggressive goals: 100 mg/dL corresponds to an HbA1c of 5.1 percent, which is quite low. But I believe that the reward, in terms of lower risk of mortality and disease, is well worth it given the ample evidence in nondiabetics and diabetics alike.

Also, everyone tends to be more insulin sensitive in the morning than in the evening, so it makes sense to front-load our carb consumption earlier in the day.

One thing CGM pretty quickly teaches you is that your carbohydrate tolerance is heavily influenced by other factors, especially your activity level and sleep. An ultraendurance athlete, someone who is training for long rides or swims or runs, can eat many more grams of carbs per day because they are blowing through those carbs every time they train—and they are also vastly increasing their ability to dispose of glucose via the muscles and their more-efficient mitochondria. Also, sleep disruption or reduction dramatically impairs glucose homeostasis over time. From years of experience with my own CGM and that of my patients, it still amazes me how much even one night of horrible sleep cripples our ability to dispose of glucose the next day.

Not all carbs are created equal. The more refined the carb (think dinner roll, potato chips), the faster and higher the glucose spike. Less processed carbohydrates and those with more fiber, on the other hand, blunt the glucose impact. I try to eat more than fifty grams of fiber per day.

Rice and oatmeal are surprisingly glycemic (meaning they cause a sharp rise in glucose levels), despite not being particularly refined; more surprising is that brown rice is only slightly less glycemic than long-grain white rice.

A good versus bad night of sleep makes a world of difference in terms of glucose control. All things equal, it appears that sleeping just five to six hours (versus eight hours) accounts for about a 10 to 20 mg/dL (that's a lot!) jump in peak glucose response, and about 5 to 10 mg/dL in overall levels.

Foods high in protein and fat (e.g., eggs, beef short ribs) have virtually no effect on blood sugar (assuming the short ribs are not coated in sweet sauce), but large amounts of lean protein (e.g., chicken breast) will elevate glucose slightly. Protein shakes, especially if low in fat, have a more pronounced effect (particularly if they contain sugar, obviously).
Action: Measure and monitor glucose, target average at or below 100 mg/dL, with a standard deviation of less than 15 mg/dL. Also, identify foods that cause spikes and avoid them.

How much protein do we actually need? It varies from person to person. In my patients I typically set 1.6 g/kg/day as the minimum, which is twice the RDA. The ideal amount can vary from person to person, but the data suggest that for active people with normal kidney function, one gram per pound of body weight per day (or 2.2 g/kg/day) is a good place to start—nearly triple the minimal recommendation.
Action: Eat 1g of protein per pound of body weight (*I do wonder if that should be ideal body weight for those that are fluffy)

The literature suggests that the ideal way to achieve this is by consuming four servings of protein per day, each at ~0.25 g/lb of body weight. A six-ounce serving of chicken, fish, or meat will provide about 40 to 45 grams (at about 7 grams of actual protein per ounce of meat), so our hypothetical 180-pound person should eat four such servings a day.

For me and my patients, this works out to four servings, as described, with at least one of them being a whey protein shake. (It's very difficult for me to consume four actual meals. Typically, I will consume a protein shake, a high-protein snack, and two protein meals.)

Putting all these changes into practice typically means eating more olive oil and avocados and nuts, cutting back on (but not necessarily eliminating) things like butter and lard, and reducing the omega-6-rich corn, soybean, and sunflower oils—while also looking for ways to increase high-omega-3 marine PUFAs from sources such as salmon and anchovies.

Our per capita consumption of soybean oil, for example, has increased over a thousand-fold since 1909; meanwhile, studies have found that levels of linoleic acid found in human fat tissue have also increased, by 136 percent over the last half century.

Some people (like me) can consume saturated fats with near impunity, while others can hardly even look at a slice of bacon without their apoB number jumping to the 90th percentile.

Finally, unless they are eating a lot of fatty fish, filling their coffers with marine omega-3 PUFA, they almost always need to take EPA and DHA supplements in capsule or oil form.
Action: Get EPA/DHA supplement

The jury is still out on the utility of infrequent (e.g., yearly) prolonged fasts.

My rule of thumb for any eating pattern, in fact, is that you must eat enough to maintain lean mass (muscle) and long-term activity patterns. That is part of what makes any diet sustainable.

(If there is one type of food that I would eliminate from everyone's diet if I could, it would be fructose-sweetened drinks, including both sodas and fruit juices, which deliver too much

fructose, too quickly, to a gut and liver that much prefer to process fructose slowly. Just eat fruit and let nature provide the right amount of fiber and water.)
Action: Don't drink soda or fruit juice

Chapter 16: The Awakening

We now know that even one sleepless night can create a state that is the functional equivalent of being legally drunk. Studies have found that sleep-deprived medical personnel, in particular, commit many more errors and cause many more deaths than those who are well rested.

We now know that chronic sleep debt is a far more insidious killer than the acute sleep deprivation that results in falling asleep at stop signs. Many studies have found powerful associations between insufficient sleep (less than seven hours a night, on average) and adverse health outcomes ranging from increased susceptibility to the common cold to dying of a heart attack. Poor sleep dramatically increases one's propensity for metabolic dysfunction, up to and including type 2 diabetes, and it can wreak havoc with the body's hormonal balance.

As important as sleep is for the body, it may even be more so for the brain. Good sleep, in terms of not only quantity but quality, is critical to our cognitive function, our memory, and even our emotional equilibrium. We feel better, in every way, after a night of good sleep. Even while we are unconscious, our brain is still working, processing thoughts and memories and emotions (hence, dreams). It even cleans itself, in a manner similar to a city sweeping the streets. Relatedly, there is a growing body of evidence that sleeping well is essential to preserving our cognition as we age and staving off Alzheimer's disease.

Even a single night of bad sleep has been found to have deleterious effects on our physical and cognitive performance. Athletes who sleep poorly the night before a race or a match perform markedly worse than when they are well rested. Endurance drops, VO2 max drops, and one-rep-max strength drops. Even our ability to perspire is impaired. And we are more likely to be injured: A 2014 observational study found that young athletes who slept less than six hours per night were more than two and a half times more likely to experience an injury than their peers who slept eight hours or more. Good sleep is like a performance-enhancing drug. In one study, Stanford basketball players were encouraged to strive for ten hours of sleep per day, with or without naps, and to abstain from alcohol or caffeine. After five weeks, their shooting accuracy had improved by 9 percent, and their sprint times had also gotten faster.

Compounding the problem, poor sleep also changes the way we behave around food. Studies by Eve van Cauter's group have found that limiting subjects' sleep to four or five hours a night suppresses their levels of leptin, the hormone that signals to us that we are fed, while increasing levels of ghrelin, the "hunger" hormone. When we sleep poorly, we can be desperately, irrationally hungry the next day, and more likely to reach for high-calorie and sugary foods than their healthy alternatives. Studies show that people who are more sleep deprived tend to have a higher likelihood of indulging in a fourth meal late in the evening.

Deep sleep, on the other hand, seems to be essential to the very health of our brain as an organ. A few years ago, researchers in Rochester discovered that while we are in deep sleep, the brain activates a kind of internal waste disposal system that allows cerebrospinal fluid to flood in

between the neurons and sweep away intercellular junk; while this happens, the neurons themselves pull back to allow this to happen, the way city residents are sometimes required to move their cars to allow street sweepers to pass through. This cleansing process flushes out detritus, including both amyloid-beta and tau, the two proteins linked to neurodegeneration. But if we do not spend enough time in deep sleep, the system cannot work as effectively, and amyloid and tau build up among the neurons.

Clearly, sleep and cognitive health are deeply intertwined; this is why one of the pillars of Alzheimer's disease prevention, particularly for our high-risk patients, is improving their sleep. It is not enough merely to spend time in bed; good-quality sleep is essential to long-term brain health. This is the crucial distinction. Sleep that is irregular, or fragmented, or not deep enough will not allow the brain to enjoy any of these benefits.

If they are using Ambien or Xanax once a month, or only with travel, or to help them sleep during a time of emotional distress, it's not alarming. But if they are using such drugs regularly, it becomes our highest priority to get them off these sleep aids and have them begin to learn to sleep correctly without them.

The first requirement for good sleep is darkness. Light is the enemy of sleep, full stop. Thus, you want to make your bedroom itself as dark as possible—installing room-darkening curtains if you live somewhere with a lot of outdoor evening light, and removing all light sources in the bedroom, even down to electronic equipment like TVs and cable boxes and such. Their little pinpoint LEDs are more than bright enough to keep you from sleeping well. Digital clocks are especially deadly, not only because of their bright numerals but also because if you wake up and see that it's 3:31 a.m., you might start worrying about your 7 a.m. flight and never fall back asleep.
Note: Ditch the clock as soon as I'm done at Apex.
Action: Sleep a lot, set up routine and environment for success, and don't use sleep drugs

Even worse is the relatively recent advent of LED household lighting, which is predominantly on the blue end of the spectrum, meaning it more resembles daylight. When our brain detects that blue light, it thinks it is daytime and that we should be awake, so it tries to block us from falling asleep. Therefore, you should also reduce the amount of bright, LED light that you're exposed to in the evening. A couple of hours before you go to bed, begin turning off unnecessary lights in your house, gradually reducing your light exposure from there. Also, try to swap out blue-intensive LED bulbs for those on the warmer end of the spectrum.

The devices we stare at before bed—phones, laptops, video games—are even worse for our sleep. Not only do they bombard us with more blue light, but they also activate our minds in ways that impede our ability to sleep. One large-scale survey found that the more interactive devices subjects used during the hour before bedtime, the more difficulties they had falling asleep and staying asleep—whereas passive devices such as TV, electronic music players, and, best of all, books were less likely to be associated with poor sleep. This may partially explain why watching TV before bed does not seem to affect sleep quite as negatively as playing video games or scrolling social media does.

Do NOT bring your laptop or phone into bed with you.

Many people associate sleep with warmth, but in fact the opposite is true: One of the signal events as we are falling asleep is that our body temperature drops by about one degree Celsius. To help that happen, try to keep your bedroom cool—around sixty-five degrees Fahrenheit seems to be optimal.

I tell my patients who are having difficulty sleeping is to cut back on alcohol—or better yet, give it up entirely. It's counterintuitive, because alcohol initially acts as a sedative, so it can help us fall asleep more quickly. But as the night wears on, alcohol turns from friend of sleep to foe, as it is metabolized into chemicals that impair our ability to sleep.

The effects of alcohol on memory and cognition are apparent even in moderate drinkers. Studies have found that young people who drink heavily are more likely to forget even basic tasks like locking the door or mailing a letter. Students who averaged nine drinks per week (not much, by collegiate standards) performed worse on a word-based memory test. And, in a finding that will surprise no one, students who drank more slept later and felt sleepier in the daytime, as well as performing worse on tests. More alarming is the finding that students who drank heavily two days after a bout of learning or study forgot or failed to retain most of what they had learned.

Chapter 17: Work in Progress

When I think of suicide, I often think of a man named Ken Baldwin, who leaped off the Golden Gate Bridge in 1985, when he was twenty-eight. Unlike 99 percent of jumpers from that bridge, he survived. As he fell, he later told the author Tad Friend, "I instantly realized that everything in my life that I'd thought was unfixable, was totally fixable—except for having just jumped."

There are many rules at the Bridge, and one of the most important ones is no minimizing. You are not allowed to minimize anything that someone else is saying, and you are especially not allowed to minimize your own experiences. But she didn't flag me for that. Instead, she asked a simple question: "You were five years old when this first happened to you, right?" "That's right," I replied. "And your son Reese is almost five years old now, right?" I nodded. "So you're saying it's okay that this happened to you when you were his age—but would you be okay with people doing that to Reese now?" Another rule at the Bridge is that you're not supposed to hand anyone a Kleenex when they're crying. They're supposed to get up and fetch it themselves. Now it was my turn to stand up and walk over to the Kleenex box.
Note: Everyone with childhood trauma goes through that rationalization, fooling themselves into thinking it was for the good. It made them stronger. And then reframing that, towards their own children at the same age, if it makes you head towards that Kleenex box, maybe pull back on the rationalization just a tad. What do you think now?

Just as an aside, a 2019 study provides an elegant demonstration of the principle that setbacks can be net positive. The researchers looked at junior scientists who had applied for NIH grants and separated them into two groups: One group had scored just above the threshold for funding, while the other had scored just below the funding line, meaning their grants were not funded. While the near-miss group were more likely to drop out of science in the immediate aftermath, those who stuck with it eventually outperformed their peers who had received

funding on their first try. The early setback had not impaired their careers but may have had an opposite effect.

His main thesis is that with women, depression is generally overt, or obvious, but men are socialized to conceal their depression, channeling it inward or into other emotions, such as anger, without ever wanting to discuss it.

Terry helped me continue to connect the dots between my own childhood and the kinds of dysfunction that had marked my adolescence and my life as an adult. Looking back at my teenage self, and the way I was in college, I realize now that I was morbidly depressed—clinically, off-my-rocker depressed. I just didn't know it at the time. I had the classical symptoms of covert male depression, which were a tendency to isolate myself and, above all, a propensity to anger, perhaps my most potent addiction. One of the first things I wrote in my journal, after an early discussion with Terry, still resonates today: "90% of male rage is helplessness masquerading as frustration."

At the Bridge, I learned that children don't respond to a parent's anger in a logical way. If they see me screaming at a driver who just cut me off, they internalize that rage as though it were directed to them. Second, trauma is generational, although not necessarily linear. Children of alcoholics are not inevitably destined to become alcoholics themselves, but one way or another, trauma finds its way down the line. As Terry had written: "Family pathology rolls from generation to generation like a fire in the woods taking down everything in its path until one person, in one generation, has the courage to turn and face the flames. That person brings peace to his ancestors and spares the children that follow." I wanted to be that person.

We covered an enormous amount of ground, but one task absolutely stymied me. On my second day, I was assigned to write out a list of forty-seven affirmations, representing one positive statement about myself for each year of my life. I made it to about five or six before I got completely stuck. For days and days, I couldn't come up with anything good to say about myself. My perfectionism and my shame did not permit me to believe anything nice about myself. I just couldn't do it.
Action: List one positive affirmation statement about myself for each year of my life. If you can't, maybe there is a problem to be addressed.

Another way in which mindfulness helps is by reminding us that when we are suffering, it is rarely because of some direct cause, like a rock that is crushing our leg at this very moment. Much more often, it is because we are thinking about some painful event that occurred in the past or worrying about something bad that may occur in the future. This, too, was an enormous revelation to me. Simply put, I experience less pain because I am able to recognize when the source of that pain is inside my own head. This was not an original insight, but it was nevertheless profound. I was about 2,500 years behind the Buddha, who said that "your worst enemy cannot harm you as much as your own unguarded thoughts." Seneca improved on that in the first century AD, observing that "we suffer more often in imagination than in reality." And later, in the sixteenth century, Shakespeare's Hamlet noted, "There is nothing either good or bad, but thinking makes it so."

One simple tactic that I use to cope with mounting emotional distress is inducing an abrupt sensory change—typically, by throwing ice water on my face or, if I'm really struggling, taking a

cold shower or stepping into an ice bath. This simple intervention stimulates an important cranial nerve, the vagus nerve, which causes our heart rate and respiratory rate to slow and switches us into a calm, parasympathetic mode (and out of our fight-or-flight sympathetic mode).

There's a quote from Paulo Coelho that I think about often: "Maybe the journey isn't so much about becoming anything," he writes. "Maybe it's about unbecoming everything that isn't really you, so you can be who you were meant to be in the first place."

Epilogue:

"I think people get old when they stop thinking about the future," Ric told me. "If you want to find someone's true age, listen to them. If they talk about the past and they talk about all the things that happened that they did, they've gotten old. If they think about their dreams, their aspirations, what they're still looking forward to—they're young." Here's to staying young, even as we grow older.

Actions:

Journal -

1. Sketch out my life at 80
2. Get details on health "icebergs" from family
3. List one positive affirmation statement about myself for each year of my life. If you can't, maybe there is a problem to be addressed.

Eating -

1. Don't snack, don't eat when not hungry.
2. Limit alcohol to fewer than seven servings per week, and ideally no more than two on any given day
3. Get EPA/DHA supplement
4. Don't eat too many calories; consume sufficient protein and essential fats; obtain the vitamins and minerals you need; and avoid poison.
5. Eat 1g of protein per pound of body weight (*I do wonder if that should be ideal body weight for those that are fluffy)
6. Stay of the fruit and fructose unless trying to store fat for winter.

Testing & Tracking –

1. Set colonoscopy
2. Track BMI and get under 25
3. Blood Pressure under 130/85
4. Triglycerides under 150 mg/dL
5. HDL Cholesterol greater than 40 mg/dL (men) 50 mg/dL (women)
6. Waist circumference under 40 inches in men or 35 in women
7. Fasting Glucose under 110 mg/dL
8. Get DEXA scan for accurate body fat measure as well as visceral fat reading
9. Measure and asses ratio of triglycerides to HDL cholesterol (it should be less than 2:1 or better yet, less than 1:1)

10. Measure and monitor insulin levels
11. Measure and monitor apoB
12. Measure and start statin if apoB is higher than 30 mg/dL
13. Measure Lp(a) and start on PCSK9 as needed
14. If diet does not get triglycerides under 150 mg/dL, look at fibrates
15. Schedule CT angiogram
16. Measure and monitor bone mass
17. Measure and monitor glucose, target average at or below 100 mg/dL, with a standard deviation of less than 15 mg/dL. Also, identify foods that cause spikes and avoid them.

Workout –

1. Lift four times per week
2. Zone 2, four time per week, 45 minutes+
3. VO2, 1 time per week, 4 minutes on x 4 minutes off, 4-6 times, Improve VO2 max
4. Measure and monitor ability to farmer carry body weight, about 115 lbs. in each hand.
5. Measure and monitor hang time, goal 2 minutes.
6. DNA, twice a week stability and mobility training as well as integration into warm up and cool down.

Lifestyle -

1. Get a sauna (or access) and use it four times per week, 20+ minutes per session, at 179 degrees
2. Sleep a lot, set up routine and environment for success, and don't use sleep drugs.