Biomarkers of Aging

Follow-Up 6 Report

prepared for
Male Sample

by physician
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based on tests performed
06/17/2009

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Introduction

Congratulations on completing your follow-up PhysioAge biomarkers. The information we have gathered will enable you to better control your health. By measuring how well you're aging in important body systems, we give you a personalized view of your overall health and identify your weakest and strongest systems.

We compare your measurements to our database of hundreds of patients, each with multiple visits. We use this data, along with statistical modeling, to calculate your physiological age. Three main factors affect your physiological age: actual age, rate of aging, and functional capacity. On average, the aging process diminishes functional capacity by 1 to 3 percent per year. If you start off at a higher capacity, you start falling from a higher point (for instance, if you're genetically endowed, or if your lifestyle, diet or medication improves it.) The rate at which you fall depends on similar factors. In order to understand what is happening at any particular point or over time, you need to measure that system's function objectively.

Does that mean that I'm aging like a 57-year-old even though I'm 45?

You can't determine your rate of aging from one test, which is just a snapshot. In order to know your rate of aging, you must have at least two separate time points, generally a year apart, to calculate the rate of change in that system.

Understanding follow-up biomarker diagrams

Now that you have more than one set of biomarkers, we can begin to capture your rate of aging and understand how your body is changing over time. Each subsequent set of biomarkers increases our ability to accurately assess your rate of aging. The diagrams that follow are a variation of the chart that appeared in your baseline report. The vertical axis represents the physiological age of each system, while the horizontal axis shows your measurements over time. Each dot marks the physiological age during that biomarker session, while the slope of the line shows your rate of aging. The thicker shaded gray line represents your actual age and will always have an upward slope because it increases with time.

If the slope of your physiological age line is steeper than that of your actual age line, your system is aging more rapidly than average. In contrast, if the slope is less steep, it's aging more slowly than average; if it's running parallel, it's not aging at all. Finally, if the slope is declining, this system is actually functioning better - it's getting more youthful! As in the baseline diagrams, any point above the actual age line indicates an older physiological age, and anything below indicates a younger physiological age.

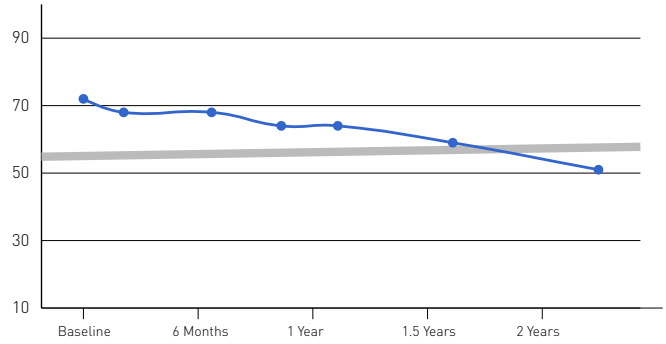
You may notice that for some of your biomarkers, the line is wavy, with fluctuations up and down. This occurs because there is an inherent variability in these measurements that can sometimes be greater than the actual change in the body system. Your physician will also point out changes in medications that can cause more extreme changes, such as the dramatic change certain blood pressure medications can cause to CardioAge. Over time, however, these fluctuations become less important, and the trend in your physiological age becomes more solid.

Biomarkers of Aging

CardioAge

"A man is as old as his arteries."

— Thomas Sydenham, 1600s
known as the English Hypocrates



Your CardioAge is 51, which is 6 years younger than average for your age.

It's all in the pulse. Today we know that what Mr. Sydenham said holds true for women as well. Even before him, Chinese doctors knew that you could tell a lot about the age of a person's cardiovascular system just by feeling the pulse at the wrist. The SphygmoCor device updates this hallowed practice by replacing the doctor's fingers with a highly sensitive pressure probe to record the shape of the radial artery pulse wave. The human pulse is a product of the cardiac cycle - the rhythmical filling of the chambers of the heart with blood from the veins, called diastole, and the subsequent ejection of the blood into the arteries, called systole. Similar to the way an ECG tracing gives us information about the health of the heart by displaying a summary of its electrical activity as it travels throughout the contracting heart muscle, the arterial pulse wave shape offers information about the health of the arterial system as blood moves through it during the pulsatile cardiac cycle.

Central blood pressure is what matters. The SphygmoCor software analyses the shape of the pressure wave at the wrist and then determines the pressure in the aorta as it comes off the heart. The pressure in the aorta is different from that in the arm (where blood pressure is traditionally measured) because of the effect of what is called the "reflected wave". As the heart pumps out a large amount of blood, the elastic aorta expands to accept the increased volume causing the pressure to increase less than if it were a rigid tube. The pressure wave then travels down the aorta to the legs and arms where it meets the smaller arteries feeding the capillary beds of your organs. The drop in pressure at these resistance arteries causes a reflected wave to return to the aorta.

Arteries stiffen and constrict with age. The aorta and other large arteries stiffen from a loss of elastin and cross-linking of collagen. This stiffening causes the reflected wave to travel back to the heart faster so that it rushes into the last bit of blood coming out of the heart in the aorta at the end of systole. The increased central pressure as a result of the collision of the forward and reflected waves occurs in the aorta but not in the arms, and is called the augmentation pressure (AP). The amount of augmentation pressure is dependent not only on the speed of the reflected wave, but also on how much resistance the forward wave meets when it hits the smaller vessels. Thus, the AP combines the two major facets of arterial aging - stiffening of the large arteries and constriction of the resistance arteries - into a single parameter that increases linearly with age. Your CardioAge is created by comparing your AP with that of 4001 healthy (no other cardiac risk factors), men and women aged 18 to 90 years old.

Why is my CardioAge important?

» It increases linearly with age and tells you how well your arteries are aging starting at a very young age - the time to start acting

» It reflects the pressure your brain, kidneys, and heart experience and therefore better predicts disease of these organs than arm blood pressure

» It can tell you better than arm blood pressure if you need to be on anti-hypertensive medication or, if you are on therapy, how well it's working.

What factors affect my CardioAge?

Age: It takes time for all of the following risk factors to affect your arteries.

Sedentary vs. active lifestyle: Activity, in particular vigorous exercise, increases the production of nitric oxide in your small arteries, which decreases AP. Chronic aerobic exercise lowers your resting heart rate, which decreases the total number of times your heart beats in a day.

Height: Taller people have lower central pressures because the reflected wave takes longer to travel back up the aorta to the heart.

Gender: Women have slightly stiffer arteries than men, even after adjusting for height. Your CardioAge is gender-adjusted.

Smoking: After having a cigarette, even in young people, AP is increased because it causes constriction of the resistance arteries. Despite this increase in central pressure, arm blood pressure often remains deceptively low in young smokers. With years of smoking, the large arteries stiffen more rapidly and the smaller arteries become clogged - both of these processes increase your CardioAge.

Obesity: Increased abdominal fat (central obesity) has been associated with increased arterial stiffness independent of arm blood pressure, age, and ethnic group.

Cholesterol: High total and LDL cholesterol levels have been associated with increased arterial stiffness. Thus, cholesterol lowering medications can lower your CardioAge.

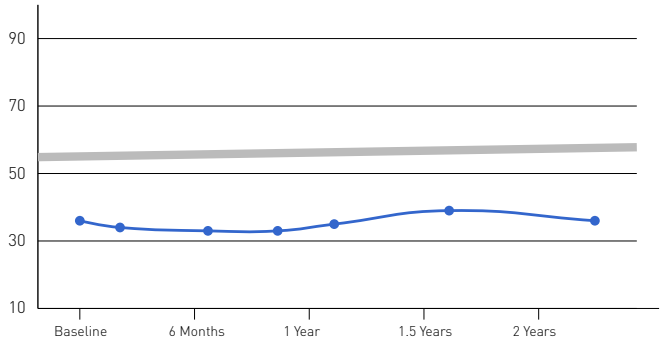
Caffeine: Consumption of caffeinated coffee has been associated with increased AP, even after one cup, without a similar increase in arm blood pressure. If you had a cup of a caffeinated drink within 2 hours of your test, your CardioAge could be somewhat higher.

Hormones: Low testosterone increases AP in men undergoing androgen deprivation therapy. Growth hormone deficiency is associated with increased arterial stiffness.

Blood pressure medications: The more recent blood pressure medications, such ACE inhibitiors, angiotensin receptor blockers, and calcium channel blockers, lower your augmentation pressure and therefore your CardioAge. Beta blockers, such as atenolol, propranolol, and metoprolol can actually increase your CardioAge. Diuretics often have a neutral effect. If you are on any of these medications at the time of your test, you must tell you doctor so that your results can be interpreted correctly.

CardioAge is determined with a proprietary algorithm using certain aspects of the output from the SphygmoCor device.

PulmoAge



Your PulmoAge is 36, which is 21 years younger than average for your age.

Most of us are familiar with the "breathing tests" used to assess and monitor symptoms of asthma and emphysema patients. These symptoms include coughing, wheezing, and shortness of breath. Yet if you've never had any of these symptoms, you are unlikely ever to have taken such a test. We, and many prominent pulmonary specialists, think this is an unfortunate shortcoming of our healthcare system. Everyone over age 40 should be screened for lung disease because of the existence of asymptomatic (no symptoms) lung disease that can gradually develop in those exposed to passive smoke, asbestos and other environmental toxins.

From screening to lung age. Spirometry (from the Latin word spirare, to breathe) turns out to be an informative biomarker of aging hiding in the guise of a pulmonary specialist's test. While it certainly functions as an excellent screen for early lung disease in symptomatic, undiagnosed asthmatics and smokers, it also measures the lung age of healthy individuals. In fact, in order to determine if the result of a spirometry test is abnormal, it must be compared to a large database of healthy adults adjusted for height, gender, ethnicity, and age. Spirometry performed by tens of thousands of individuals has resulted in large databases which can be used to determine lung age.

Vital Capacity. The two main results of spirometry are:

» Forced vital capacity (FVC): the total amount of air you can force out after a maximal inhalation

» Forced expiratory volume in the first second (FEV1): the maximum amount of air you can force out in the first second of your forced vital capacity exhalation.

FVC is determined by the size of your chest, the number and health of the air sacs (alveoli) where gas exchange takes place, the elasticity of your large airways, and the strength of your breathing muscles. The FEV1 is largely determined by the elastic recoil of the air the small airways.

Why is my PulmoAge important?

» Many large studies, including those that tracked people over long periods, have shown that both the FVC and FEV1 decline with age - about one percent a year.

» Screen for pulmonary disease. If your FEV1/FVC < 0.72, then you could have obstructive pulmonary disease.

» There is a correlation between these biomarkers and mortality. One investigation, the Buffalo Health Study, followed nearly 1,200 men and women between the ages of 20 and 89 for twenty-seven years, and found that lower lung function predicted earlier death.

» While spirometry results can be predictive of mortality risk, the correlation is not solely between respiratory function and death caused by respiratory disease. This is where the picture becomes a bit ambiguous, and where it deviates from other biomarkers of aging. Arterial stiffness, for instance, correlates directly with life-threatening cardiovascular disease. But while the average person's FEV1 can be expected to decline about one percent a year - and that can add up to a significant percentage of functional loss over the course of a lifetime - such normal lung aging is not going to cause early death from lung disease in most people. As the studies cited above indicate, the more significant association is between lower spirometry readings and increased mortality of all kinds. These findings bolster the idea that respiratory function over time reflects how well the body as a whole is aging, making spirometry one of the most valuable biomarkers of aging.

"We consider FEV1 as a surrogate for a number of unmeasured aging processes ... and not as a specific measure of lung function."

— Dr. Milton Hollenberg
University of California, San Francisco Medical Center

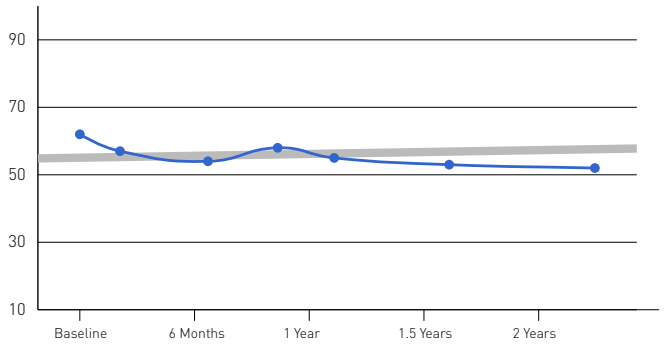
What factors affect my PulmoAge?

Many of the same factors that affect your CardioAge also affect your PulmoAge. Your PulmoAge is adjusted for height and gender, but differences in the structure of your chest, barrel vs. narrow, can also affect it. Smoking affects your lungs directly and indirectly, by affecting the arteries supplying your lungs with oxygen.

Asthma/emphysema inhaled medications such as bronchodilators and anti-inflammatories can improve your FEV1 and FVC, so your physician needs to know if you are taking one in order to accurately interpret your results.

Taking a baseline PulmoAge test and repeating it annually is an effective way to see how well your anti-aging efforts are working. If there is no significant increase over time, you.re doing something right. In addition, a baseline test can tell you if you are at significant increased risk for lung disease and can motivate a smoker to quit.

NeuroAge™



Your NeuroAge is 52, which is 5 years younger than average for your age.

The brain ages. Neuroscientists who study the aging brain agree that your brain ages much like your arteries and lungs.

Just as there are tests to detect changes in arterial stiffness and lung elasticity that occur decades before the onset of significant disease, there are tests of cognitive function sensitive to the gradual decline preceding clinically obvious neurologic impairment. This gradual decline is part of normal brain aging and can be experienced as a decreased ability to play video games and "brain fog," that feeling that you just are not thinking up to par.

Brain aging is a slow process. As early as your mid-twenties, certain aspects of your cognitive function begin to decline in a linear fashion. You don't notice the decline unless you tax the system, e.g., play a video game, do long division in your head, or take this test. If that decline is steeper than the average person, you are more likely to have significant neurologic disease decades later than someone whose decline is average, and now is the time to act.

Screen for early cognitive impairment. The CNS Vital Signs battery of neuropsychological tests assesses the main areas of cognitive function, by taxing them more than your daily activities (Most people find the 20-minute battery to be quite challenging). It is used to screen for significant neurologic impairment from dementia, ADHD, or medications and is a screening test that everyone should take periodically. Just as we saw with spirometry, however, in order to know if there is a significant impairment, the results of the test must be age-adjusted. While most domains of cognitive function are affected by normal brain aging, two areas are particularly sensitive to age and start showing changes in early adulthood.

The most sensitive measures of normal brain aging are:

» **Reaction time.** The Stroop Test measures how quickly and accurately you can apply a rule to a stimulus and then inhibit the application of that rule (press the space bar when the word spells the color of its font, then reverse the rule).

» **Processing speed.** The Symbol Digit Coding test measures how many paired sets of symbols and digits you can process on a computer screen with your eyes and then press the corresponding key on the keyboard. This test involved the grid with corresponding symbols and numbers.

Your NeuroAge is a weighted composite of the scores on these two tests.

Why is my NeuroAge important?

» A NeuroAge significantly higher than your age may mean that your brain is aging more rapidly than it should and could be indicative of an adverse affect of a medication you are taking or early brain disease. This can occur even when your memory is unaffected.

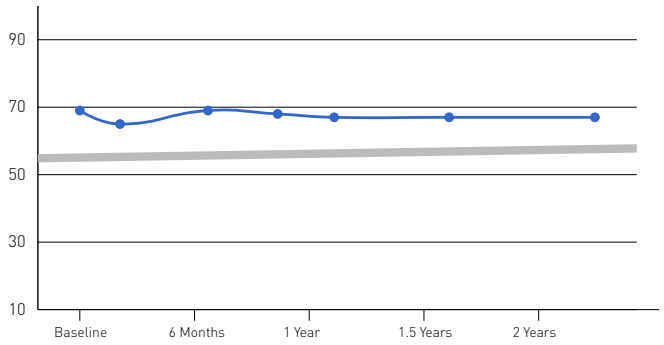
» Mild concussions can transiently raise your NeuroAge, as can moderate to severe depression.

What factors affect my NeuroAge?

Sleep deprivation and alcohol intoxication can hurt your performance, and caffeine can improve your performance. Cognitive neuroscientists have shown that the decrease in performance on tests like these is correlated with the loss of dopamine activity in the frontal lobes of the brain.

CutoAge

Why do I need a fancy instrument to tell me how well my skin is aging?



Your CutoAge is 67, which is 10 years older than average for your age.

Intrinsic vs. extrinsic skin aging. Most people think that just looking in the mirror ought to suffice, yet studies have demonstrated that facial skin appearance is more affected by the amount of sun exposure (photoaging) than it is by the passage of time. As such, your skin's appearance it is not a great biomarker of aging. However, intrinsic skin aging (the loss of elasticity and fine wrinkling that occur in areas of your body that receive relatively little sun exposure), correlates very closely with age. This linear change in

elasticity is hard to appreciate with the naked eye until it is relatively advanced.

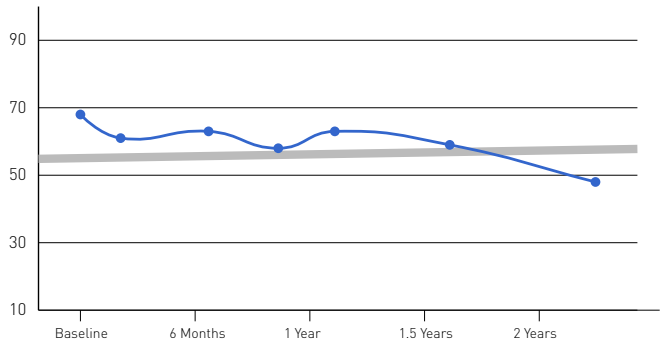
More than just skin deep. To measure intrinsic skin aging we use the Cutometer, an instrument that has been validated in hundreds of studies of skin aging over the past 25 years. It works by applying a sequence of precise and gentle suctions to a small area of skin and then measuring with an optical sensor how much the top two layers of your skin move with each suction. The movement is very slight - only 0.2-0.5 mm - much less than the amount it moves when you do a "pinch test" to see how fast your skin returns to normal after pinching it between your two fingers. Yet by involving only the top two layers, the Cutometer can non-invasively assess the amount and structure of the collagen and elastins in your skin. The result is reported as skin elasticity percent: the extent to which your skin returns to its original position after being stretched and released. When you finish adolescence, the average skin elasticity is almost 90%; with each passing year the average person loses about 1%, leaving the average 80-year-old with about 35% elasticity.

Why is my CutoAge important?

» Skin elasticity as measured by the Cutometer has been correlated with bone density.

» It has been shown to be improved by HRT in women.

ImmunoAge™



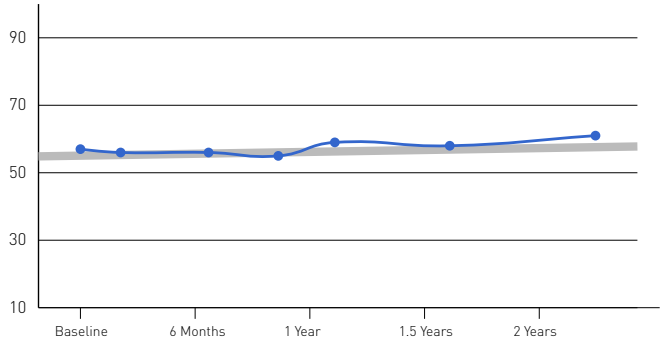
Your ImmunoAge is 48, which is 9 years younger than average for your age.

Not surprisingly, as we age our immune systems falter. Older adults are more likely to succumb to infections their younger selves blithely warded off with barely a sniffle. Cancer rates increase exponentially with age, partly because of less effective tumor surveillance from an aging immune system. Dysfunctional immune cells cause chronic inflammation, which increases the likelihood of degenerative diseases such as osteoporosis, atherosclerosis, and dementia. This condition, called "immunosenescence", results from changes in the relative proportion and function of certain white blood cells, the thymus and the bone marrow.

One of the most prominent changes of immunosenescence is the decrease in white blood cells that are able to fight off new infections or tumors. Called naive suppressor T-cells, they are designated by the lack of the CD95 and presence of the CD28 molecules protruding from the membranes of the cell's surface. The presence of these "CD95-CD28+" T-cells can be detected at specialized labs from the same of tube of blood used to do the routine CBC (complete blood count). Cells that do not have CD95 sticking out of them have not yet encountered the foreign substance (the molecular snippet of an infectious bug or tumor), called "antigen", they are uniquely created to recognize. The CD28 molecule enables these cells to divide rapidly and mount a brisk response to their designated antigen, which results in clearance of the infection/tumor.

The linear decline in the number of CD95-CD28+ T-cells is so highly correlated with age that it has been called the best biomarker of immune aging discovered to date. When we are young adults, we have over 250 cells/μL of blood but start to lose about 3 cells/μL per year from then on. If you do the math, you can see that this leads to complete absence of the ability to fight off new infections and tumors by the ninth decade, which is just about the average life expectancy. If you add B-lymphocytes (CD19+) to the model with naive T-cells, your understanding of age-related immune system changes increases even more, and we are able to explain up to 60% of the variation among individual immune system aging.

TelomerAge



Your TelomerAge is 61, which is 4 years older than average for your age.

Your molecular "ends" of time. If there is a candidate to be considered the human body's molecular clock, then one of the front runners must certainly be the length of our telomeres. From the Greek, telos, meaning 'end,' and mere, meaning 'part,' telomeres are the caps on the ends of each of our chromosomes that protect them from being mistaken for damaged DNA. They are composed of thousands of repetitions of the same sequence of 6 base pairs (the letters of DNA, TTAGGG). With each cell division, they shorten by about 50-100 base pairs because of the difficulty DNA polymerase has replicating one of the strands. At young adulthood, the mean lymphocyte telomere length (MTL) is about 8 kb (8,000 kilo base pairs). Once it reaches 4 kb, the cell no longer is able to divide and enters what is called 'replicative senescence,' in which it fails to perform its function and produces detrimental inflammatory molecules. The molecular clock stops ticking.

How are telomeres measured? The subset of your white blood cells used to calculate your ImmunoAge, the lymphocytes, is the cell type whose telomere length has been most studied. This is mostly because of its easy access through a routine blood draw, as opposed to a biopsy of solid tissue such as your lungs or arteries. Hundreds of studies have linked the shortening of lymphocyte MTL not only to the aging process, but also to cardiovascular disease, smoking, various cancers, and even psychological stress. (A few studies have measured the correlations between MTL and the telomere lengths of other tissues in the body and have found general agreement.)

The average person's lymphocyte MTL decreases about 30-50 base pairs per year; you are more likely to be in the higher end of this range if you smoke, don't exercise, have a lot of stress, or have a chronic disease. Thus, if you start out with a lymphocyte MTL of 8 kb and lose 0.04 kb/yr (40 base pairs), you will get to the critical length of 4 kb in 66 years, again, very close to the average human lifespan.

Why is my TelomerAge important?

» Telomere length is predictive of mortality after controlling for age

» Telomere length is associated with risk for osteoporosis, diabetes, cardiovascular disease, dementia and cancer.

What factors affect my TelomerAge?

» Genetic inheritance. In addition to the rate of loss, each of us inherits a particular average telomere length from our parents.

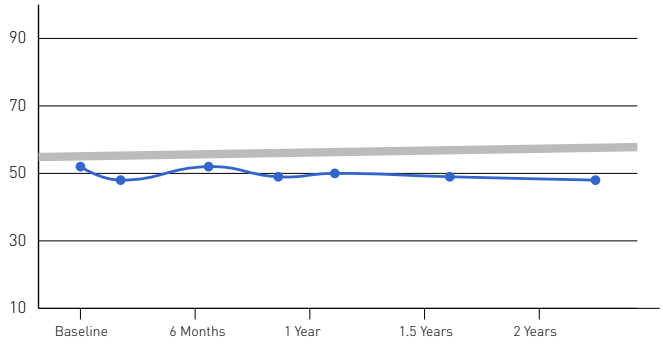
» Psychological stress. Studies have shown that chronic emotional stress, such as being the caregiver to an Alzheimer.s patient, can increase one.s rate of telomere loss.

» Oxidative stress. Chronic increased inflammation can shorten telomeres, in addition to the effect of continuous cell division.

» Antigenic stress. When your lymphocytes are chronically stimulated to divide in response to latent infections (particularly viral infections such as HIV or CMV) or tumors, their telomeres shorten more rapidly.

You may be thinking, "If you telomere length functions as such a great biomarker of aging and disease, why don't we just measure it instead of bothering with all these other tests?" We wondered about that too as we were searching for the best biomarkers of aging. To our amazement, we found that while telomere length correlates with age very well, the combination of biomarkers correlates much better, confirming the notion that the aging process is best understood by looking at combinations of biomarkers of important physiological systems.

PhysioAge™ Composite



Your PhysioAge is 48, which is 9 years younger than average for your age.

By analyzing these individual Ages together, we can calculate an overall PhysioAge Composite, which tells you how well you are aging as a whole. This composite is not a simple average of the individual Ages. Simply averaging the results of each Age would be like sewing together a human from the parts of different cadavers (like Dr. Frankenstein did). It's a flawed approach because they don't naturally go together. The result would be a composite picture that doesn't necessarily reflect the way the aging of these body systems interact with one another in actual people.

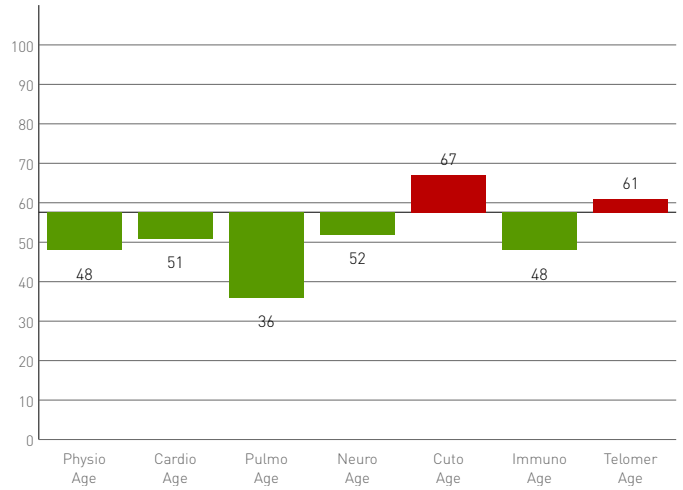
To avoid this pitfall, we measured over 150 biological parameters in 113 men and women between the ages of 30 and 87 during the course of a 3-year study. We then utilized the multiple linear regression technique to see how the parameters that most highly correlate with age interact within the study subjects to explain the aging process. The result is a PhysioAge Composite that tells you the chronological age at which the average person's physiological systems function as well as yours at the time you took the test.

Explanatory power. The advantage of this method of calculating Physiological Age is the statistical power it provides. When you measure the first four biomarkers on the chart after the PhysioAge, you can explain 70% of the variation in aging among individuals. If you add ImmunoAge to the first four, it increases to 75%, and topping it off with TelomerAge takes you up to an unprecedented 84%. This gives you a single barometer of how well you are aging.

Measuring effectiveness of therapy. With this level of explanatory power, we are able to tell you how an intervention you are utilizing - such as hormone therapy, exercise, antioxidant supplementation, or any prescription meds you are taking for other conditions - is affecting your overall aging process. This is important because some interventions have beneficial effects in one system, but may be causing adverse effects in another. For example, high intensity exercise can improve your arterial and pulmonary systems, but if too intense it can age your immune system. Moreover, some therapies, such as telomerase activators, subtly but profoundly affect all organ systems, so you need a measure of the aggregate effect in order to appreciate how well they are working.

Comparing Systems

Physiological Ages for Your Current Visit

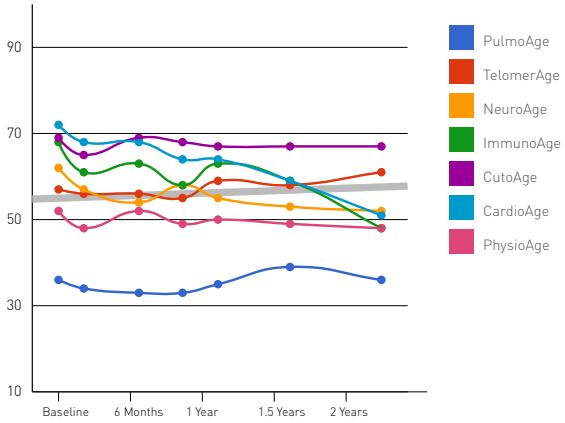


Comparing Your Calculated Ages This bar chart shows the most current ages of your overall PhysioAge Composite and the physiological systems we measured to calculate it. Your PhysioAge Composite is a weighted average of the other underlying ages.

A bar pointing up indicates that this particular physiological system is older, or weaker than average for your age. A bar pointing down indicates it is younger, or stronger than average for patients your age. Each of us has a different combination of weaker and stronger systems depending on genetic inheritance and lifestyle factors/therapeutic interventions.

Comparative Chart for All Visits

This chart shows how your measurements have changed over time in comparison with your actual age (the solid grey line).



Health Status Indicators

Vital Signs

Biomarker	Result	Units	Baseline	Change
Height	74.0	inches	74.5	-1 %
Weight	201.0	lbs	201.0	0 %

Arterial Stiffness

Biomarker	Result	Units	Normal Range	Baseline	Change
SBP	130.0	mm Hg	90.0 - 120.0	126.0	3 %
DBP	76.0	mm Hg	60.0 - 80.0	72.0	5 %
Augmentation Pressure	8.0	mm Hg	0.0 - 8.0	13.0	-39 %
Augmentation Index@75	12.0	mm Hg	0.0 - 23.0	16.0	-25 %
Ejection Duration	357.0	mSec		322.0	10 %
Ejection Duration %	28.0	%	26.0 - 32.0	32.0	-13 %
SEVR	203.0	%	186.0 - 240.0	172.0	18 %

As discussed in the CardioAge section, the SphygmoCor parameter, Augmentation Pressure (AP), is used to calculate the age of your arterial system. There is additional information supplied by the SphygmoCor device that can help to further assess your cardiovascular health.

Heart Rate is the number of times your heart beats per minute. The normal range is less than 100, and by definition, a level above 100 is called tachycardia. Heart rate has been shown to be a good low-tech marker for cardiovascular disease risk. Several studies have shown that resting heart rate predicts cardiovascular mortality, with the risk decreasing in line with heart rate.

Systolic Blood Pressure (SBP) is the peak blood pressure reached during systole, the part of the cardiac cycle during which the heart contracts and squeezes blood into the peripheral circulation. SBP increases with age and predicts strokes and heart attacks in many studies. However, it is not as good a predictor of CVD or biomarker of aging as AP.

Diastolic Blood Pressure (DBP) is the time during the cardiac cycle that the heart muscle is relaxing and filling up with blood. It is often misunderstood to continue to rise with age, when in fact, it reaches a peak in mid-adulthood and then starts to decline because of the decrease in elastic recoil of the aorta and muscular arteries. A decline in DBP in an older adult accompanied by an increase in SBP increases rather than decreases his CVD risk.

Augmentation Index (AI) is the AP corrected for the difference between systolic and diastolic blood pressure, called the 'Pulse Pressure,' and heart rate. In younger patients, it may be a slightly better indicator of cardiovascular age than AP. However, in older patients, it is less useful because it plateaus by age 65.

Ejection Duration (ED) is the length of systole in milliseconds (msec), i.e., the time it takes the heart to pump out the blood it filled up with during diastole. This number changes as the heart rate goes up and down, but the ratio between the time in systole and diastole is a measure of the amount of work the heart has to do.

Subendocardial Viability Ratio (SEVR) is a measure of the ability of the heart to fill the coronary arteries (at a given heart rate) so that sufficient nutrients and oxygen can get to the inner lining of the heart muscle (the subendocardium). When this ratio drops below 130, angina can occur. A level above 220 is a measure of a healthy heart and vascular system.

Cardiovascular Risk

The first five cardiovascular risk factors (CRF) are from the traditional lipid ('fats' in Greek) panel. The risk of these was measured in the famous Framingham heart study and it is well-established that abnormal values can contribute to the risk for CVD (cardiovascular disease).

Biomarker	Result	Units	Normal Range	Baseline	Change
Total Cholesterol	153.0	mg/dL	125.0 - 200.0	134.0	14 %
LDL Cholesterol	54.0	mg/dL	0.0 - 129.0	69.0	-22 %
HDL Cholesterol	92.0	mg/dL	> 40.0	57.0	61 %
Cholesterol/HDL Ratio	1.7		0.0 - 5.0	2.35	-28 %
Triglycerides	37.0	mg/dL	0.0 - 149.0	43.0	-14 %
C-Reactive Protein	0.9	mg/L	0.0 - 2.9	2.5	-64 %
Homocysteine	7.0	µmol/L	0.0 - 10.3	10.07	-31 %
Coenzyme Q10	1.0	mg/L	0.44 - 1.64	1.0	0 %

Low density lipoprotein (LDL) cholesterol is known as the "bad" cholesterol because it can become oxidized by free radicals and initiate atherosclerosis as discussed above. All things being equal, the lower the LDL the better. A level below 130 mg/dL is normal, and above 160 mg/dL confers a significant increase risk of CVD. When the total cholesterol is 150 mg/dL, the LDL is usually about 80 mg/dL. Nutritional anthropologists have speculated that this is the average human level of LDL when we consumed the natural diet of our ancestral environment.

High density lipoprotein (HDL) cholesterol is protects your arteries from damage because it transports lipids back to the liver and keeps them from getting oxidized. A level below 50 mg/dL in women and 40 mg/dL in men confers increased CVD risk. In fact, it is an independent and potent risk factor irrespective of total cholesterol. It was thought that a very high HDL could help to offset a high LDL, but it is now appreciated that in most cases the extra HDL above 60 mg/dL is a slightly different type and is not as protective.

Triglycerides (TG) are the three- (hence the 'tri') chain fatty acid molecules which (unlike HDL and LDL) are unattached to a lipoprotein. They are increased significantly immediately after a meal, but circulate in lower levels in the fasting state. Levels above 150 mg/dL are associated with an increased risk of CVD, but can also be the result of inflammation of the pancreas. You must be fasting for 8 hours for the TG level to be interpretable by the table above. This because the type of food you ate just prior to a test can have a great impact on the level and not be representative of the average TG level. For example, if you had high-fat meal prior to the test, your TG could be twice as high as if you had an average or low-fat meal.

Total cholesterol is made up of the LDL, HDL, and TG. A normal cholesterol level is below 200 mg/dL, but ideally it should be below 150. However, the relative amounts of the LDL and HDL affect how "dangerous" a given total cholesterol level is. For example, if your total cholesterol is 230, and your HDL is 70, you are in a less risky range than if your total cholesterol is 190 and your HDL is 30. This is because the LDL accompanying the first of these profiles would likely be significantly lower than that of the second.

C-reactive protein is called an 'acute phase reactant' because it is released from the liver during an acute infection to help fight off microbial invaders. In the absence of infection, however, a C-reactive protein level greater than 3 mg/L has been associated with an increased risk of CVD in a large number of studies. This is thought to be a result of the low level of inflammation (immune system activation) produced by atherosclerotic plaques. A level of 1-3 mg/L is normal, but ideally it should be less than 1 (the lower the better).

Homocysteine is an intermediary metabolite produced during the conversion of the amino acid methionine (commonly found in dietary meat protein) into cysteine. This conversion requires adequate levels of vitamin B12 and folic acid. High levels of homocysteine act like free radicals and can damage arteries in the same way as oxidized LDL. Studies have associated levels of homocysteine greater than 9 micromol/L with an increase risk of CVD. While more recent studies have called this association into question, the preponderance of evidence suggests that maintaining a normal to low homocysteine level by ensuring adequate intake of B-vitamins is likely to benefit your cardiovascular health.

Lung Function

As mentioned in the PulmoAge section, FEV1 is a powerful biomarker of aging hiding in the guise of a lung disease test.

But the forced expiratory volume test you took is also an important screening test for lung disease.

Biomarker	Result	Units	Baseline	Change
FVC	5.6	L	5.38	4 %
FEV1	4.38	L	4.41	-1 %
FEV1/FVC	74.0	%	81.97	-10 %
FEF25-75%	3.0	%	3.2	-7 %
PEF	9.1	L/sec	8.2	10 %

Forced Vital Capacity (FVC) is the total amount of air you can exhale after a maximal inhalation. Many factors affect FVC, including height, gender, race, and age, as well as the health of your lung tissue and chest muscles. FVC is almost as good a biomarker of aging as is FEV1. The predicted range is adjusted for these factors. Obesity and an extremely muscular chest can reduce your FVC because it makes it more difficult to completely expand your lungs.

Forced Expiratory Volume in the first second (FEV1) is the amount of air in liters you can expel in 1 second. It is determined by multiple factors and is expected to decline 20-40 ml each year after age 25. Asthma and chronic obstructive pulmonary disease (COPD) reduce your ability to rapidly expel air from your lungs because of constriction and inflammation of the smaller airways.

Screen for potential lung disease. If your FEV1/FVC is less than 0.72, then you may have some element of chronic obstructive pulmonary disease like asthma or bronchitis. Your doctor will ask you to repeat the test and if it is the same will likely have you do more extensive tests or refer you to a pulmonary specialist. If your FVC is below the normal range for your age, then you may have some element of restrictive lung disease such as emphysema or pulmonary fibrosis.

Cognitive Function

Biomarker	Result	Normal Range	Baseline	Change
Composite Memory	66.0	90.0 - 109.0	75.0	-12 %
Verbal Memory	110.0	90.0 - 109.0	102.0	7 %
Visual Memory	75.0	90.0 - 109.0	106.0	-30 %
Psychomotor Speed	97.0	90.0 - 109.0	61.0	59 %
Processing Speed	92.0	90.0 - 109.0	81.0	13 %
Reaction Time	100.0	90.0 - 109.0	52.0	92 %
Cognitive Flexibility	98.0	90.0 - 109.0	77.0	27 %
Executive Functioning	75.0	90.0 - 109.0	75.0	0 %
Subject Composite Memory	100.0		102.0	-2 %
Subject Verbal Memory	60.0		100.0	-40 %
Subject Visual Memory	45.0		102.0	-56 %
Subject Psychomotor Speed	197.0		168.0	17 %
Subject Processing Speed	62.0		50.0	24 %
Subject Reaction Time	695.0		800.0	-14 %
Subject Cognitive Flexibility	52.0		52.0	0 %
Subject Executive Functioning	52.0		52.0	0 %

There are six core tests you are asked to complete on the PhysioAge version of the CNS Vital Signs computerized neuropsychological battery. As we discussed in the NeuroAge section above, two of them - Stroop and Symbol Digit Coding - are the most age-sensitive and are used to calculate your brain's processing speed and reaction time. The other four tests - finger tapping, shifting attention, verbal and visual memory - are used to calculate the cognitive domains of psychomotor speed, verbal and visual memory, cognitive flexibility, and executive functioning. The everyday relevance of each domain follows:

Memory: ability to retain verbal and visual information, e.g., grocery lists and remembering a person's name upon seeing his face again.

Psychomotor Speed: ability to move your limbs or fingers quickly when completing simple task such as typing.

Processing Speed: ability to rapidly complete a series of tasks, e.g., scanning a table of contents, inputting numbers into keyboard, or playing a video game.

Reaction Time: ability to make a complex decision quickly when presented with a stimulus, e.g., putting on the brakes when a yellow light appears or swerving to avoid hitting a cyclist suddenly appearing in your lane.

Executive Function: ability to shift focus from one task quickly to another and back, e.g., multitasking at work, and applying different rules to a changing situation.

Cognitive Flexibility: ability to adapt to a changing environment and not be stuck applying old rules to new situations.

For each of these domains, the software calculates a raw subject score listed in the results table below that is not adjusted for age, gender, or education. To follow your change with time in each these tests, we used this raw subject score. However, in order to interpret the clinical meaning of these tests, the scores must be normalized for age and gender, similar to the concept of "grading on a curve" or the results of a standardized IQ test. By definition, the average IQ is 100. Similarly, the Standard score in the table below indicates that an average score range is 90 -109, with 100 being the average score.

Percentile Range	Standard Score	Interpretation	Clinical Severity
> 74%	> 109	High	High Function
25% - 74%	90 - 109	Average	Normal
9% - 24%	80 - 89	Low Average	Slight Deficit
2% - 8%	70 - 79	Low	Impairment Possible
< 2%	< 70	Very Low	Impairment Likely

If you score low on any two domains, or very low on any single domain, the first thing your doctor will do is have you repeat the test battery to make sure that the low scores are not the result of your misunderstanding the instructions or getting off to a bad start. If your results are confirmed, you should be evaluated for the possibility of a neuropsychological disorder, adverse medication reaction, or accelerated brain aging. The sensitivity of the individual domains for specific disorders is shown in the table below.

	MCI	Dementia	Brain Injury	Depression	Medication Effect
Memory	●	●	○	○	◐
Psychomotor Speed	◐	○	●	◐	●
Reaction Time	◐	○	◐	◐	●
Processing Speed	●	○	●	◐	●
Cognitive Flexibility	●	●	●	●	●

● Most Sensitive ◐ Somewhat Sensitive ○ Least Sensitive

Immune Function

As discussed in the ImmunoAge section, there is no better biomarker of immune system aging than the decline in CD28+CD95- suppressor T-cells. The level of these virgin immune cells declines linearly with age. However, there are other aspects of the change in immune system health with age that are more complicated. To understand them, a brief overview of the players involved in protecting your body from invaders - both from the inside and outside - is necessary. The table below depicts the main cell types of the immune system found in the bloodstream.

Biomarker	Result	Units	Normal Range	Baseline	Change
WBC	3956.0	cells/ µL	3500 - 9500	4123.0	-5 %
Neutrophils Percent	47.5	%	38.0 - 80.0	64.0	-26 %
Neutrophils Count	1568.0	cells/ µL	1500.0 - 7800.0	1400.0	12 %
Monocytes Percent	7.0	%	0.0 - 13.0	6.0	16 %
Monocytes Count	290.0	cells/ µL	200.0 - 950.0	256.0	13 %
Eosinophils Percent	1.8	%	0.0 - 8.0	3.0	-40 %
Eosinophils Count	59.0	cells/ µL	15.0 - 550.0	60.0	-2 %
Basophils Percent	0.9	%	0.0 - 2.0	1.0	-10 %
Basophils Count	30.0	cells/ µL	0.0 - 200.0	21.0	42 %
Lymphocyte Percent	41.0	%	20 - 48	25.0	64 %
Lymphocyte Count	1353.0	cells/ µL	1078 - 2828	800.0	69 %

White Bloodcell Count (WBC) is the total number of white blood cells per microliter of blood circulating in your vascular system at the time of the blood draw. This number can increase significantly when you have an infection, but under normal conditions it represents only about 2% of the total number of white blood cells in your body. The rest are in your lymph glands, gastrointestinal tract, spleen, and other tissues. However, the relative proportions (percentages) of the different types in the bloodstream are usually representative of the proportions in the rest of the body. While there are numerous sophisticated immune

function tests, much can be learned about the state of the immune system simply by measuring the number and relative proportions of WBC subsets.

The next three cell types in the table above (neutrophils, eosinophils, and basophils) are called 'granulocytes' because of fine granules in their cytoplasm that appear after being stained for microscopic examination. These cells are part of the innate immune system, the earliest responders to microbial and parasitic infections. They fight infection by releasing their granules of enzymes and free radical generators which destroy the invading organisms. They recognize the invaders as foreign by molecular structures that appear on most infectious agents. They also release the mediators of inflammation which cause blood to flow to the site of infection.

Neutrophils are the most abundant immune cell in the bloodstream, accounting for 50-65% of the total WBC; they do the majority of the work of the innate immune system. Their main function is phagocytosis (eating cells) and releasing their granules to destroy the engulfed bacteria and parasites. The ability of neutrophils to secrete bactericidal enzymes declines with age, increasing the severity of common bacterial infections in older adults. At the same time, their ability to turn off the inflammatory signals after the infection clears also declines, leaving the body in a chronic inflammatory state. Interestingly, their number increases with age in healthy adults, probably as a compensatory mechanism to offset the decline in per-cell functional activity.

Eosinophils circulate in lower numbers, 0-8% of WBC, and their main function is to defend against infection by parasites. An increase in the number of eosinophils above 550 is often indicative of a parasitic infection or an allergic reaction to drugs, pollen or other allergen. There is some evidence that eosinophil function decreases with age, but the data is currently limited. There is no evidence of a change with age.

Basophils are found in very low numbers in the bloodstream and body (less than 2%). They are recruited to a site of infection and release histamine, a substance that causes capillaries to dilate and become permeable. This allows other important infection-fighting substances to move from the bloodstream into the site of infection. Currently available data indicates that there is no change in basophil number with age.

The last two main categories of white blood cells generally do not have granules.

Monocytes are released from the bone marrow, circulate in the blood for about 8 hours, and then enter the bone, brain, liver, lung, and skin to differentiate into specialized cells called macrophages. There they function as phagocytes which break down and process invading cells so that parts of them can be "presented" to the adaptive immune system. These cells are very important in controlling inflammation and the function of the other main white blood cell type, lymphocytes.

Sometimes infections are wiped out by this immediate innate immune system response. But if the infection persists, the body recruits the next wave of defense called the adaptive immune system which can recognize the particular molecular structure of the invader to more effectively target it.

Lymphocytes

Lymphocytes are the next most abundant white blood cell in the bloodstream and can be divided into subsets that have specific functions and characteristic changes with age and disease states. The table below depicts the important lymphocyte subsets that we assessed.

Many cells of the body are identified by molecule markers that protrude out of (are expressed on) their cell membranes. These molecules enable the cell to communicate with other cells and receive instructions from signaling molecules, such as those that direct them to a site of infection. Lymphocytes can be subdivided by these 'cluster designation' or 'CD' markers into Natural Killer (NK) cells, B-cells, and T-cells.

Biomarker	Result	Units	Normal Range	Baseline	Change
NK Cell %	15.0	%	3 - 26	13.0	15 %
NK Cell Count	190.0	cells/µL	51 - 543	200.0	-5 %
B-cells %	22.0	%	5 - 22	9.0	144 %
B-cell Count	200.0	cells/µL	74 - 447	200.0	0 %
T-cells %	68.0	%	57.0 - 85.0	71.0	-5 %
T-Cell Count	1032.0	cells/µL	767 - 2318	981.0	5 %
Helper T-Cells %	41.0	%	32 - 59	45.0	-9 %

Biomarker	Result	Units	Normal Range	Baseline	Change
Suppressor T-Cells %	27.0	%	13 - 38	23.0	17 %
Suppressor T-Cell Count	369.0	cells/µL	201 - 868	259.0	42 %
Helper/Suppressor T-Cell Ratio	1.5		0.96 - 3.93	1.95	-24 %
Healthy Suppressor T-Cells %	39.0	%	49 - 96	44.0	-12 %
Healthy Suppressor T-Cell Count	180.0	cells/µL	100 - 850	61.0	195 %
Senescent Suppressor Cells %	61.0	%	4.0 - 51.0	56.0	8 %
Senescent Suppressor Cell Count	189.0	cells/µL	17.0 - 364.0	198.0	-5 %
Naive Suppressor Cells %	19.0	%	11.0 - 57.0	6.0	216 %
Naive Suppressor Cell Count	100.0	cells/µL	32.0 - 347.0	48.0	108 %
CMV AB (IGG)	0.24		0.0 - 1.0	0.14	71 %

NK-cells carry the CD56 and CD16 proteins on their surface. They are part of the innate immune system because they do not have a T-cell receptor and can kill virally infected and certain tumor cells. Recent research has demonstrated that they are deeply involved with the adaptive immune system. In healthy adults, the function of individual NK-cells decreases with age, but as for neutrophils, their number increases to compensate.

B-cells are designated by expression of the CD19 marker and are derived from the bone marrow. They are the part of the adaptive immune system that produces antibodies that travel in the bloodstream looking for the antigens found on the surface of pathogens. Through a complex process of DNA rearrangement during maturation, each B-cell produces only one type of antibody. However, the large number of B-cells produced by a healthy young immune system enables it to recognize virtually any new pathogen that may invade the body. When an antibody encounters its unique antigen, it initiates a process that results in the invader's destruction. Unfortunately, the number of B-cells decreases linearly with age, which may be one of the reasons older adults are more susceptible to bacterial infections and cancer.

T-cells express the CD3 marker and constitute over 60% of circulating lymphocytes. They are derived from the thymus gland, a small structure sitting just behind the sternum. They perform the other type of adaptive immune system function called 'cell-mediated immunity.' In contrast to B-cells, which recognize pathogens themselves, T-cells recognize host cells that have been infected with pathogens. Similar to an antibody but remaining attached to the cell, the T-cell receptor (TCR) undergoes a rearrangement that allows it to recognize a particular antigen sticking out of an infected cell. The cytotoxic T-cell then attaches to the infected cell and releases substances (cytokines) that destroy the infected host cell for the good of the body. This type of immunity is necessary for combating intracellular pathogens, such as viruses, and tumor cells.

After a T-cell is released from the thymus, it is considered virgin until it encounters the marker it is uniquely designed to recognize. A young, healthy thymus releases myriad T-cells with different TCRs. However, the thymus starts to shrink (the technical term is 'involutes') shortly after birth and is virtually non-existent by the 5th decade of adulthood. Thus, the range of different molecular targets, i.e., invaders or tumors, against which the adaptive immune system can defend, decreases with age.

Helper T-cells express the CD4 marker and help to orchestrate function of other WBCs by releasing cytokines (attracting and stimulating molecules) or by binding to them. They don't actually kill infectious agents or tumor cells (they are not cytotoxic) by themselves but rather recruit other cell to do so. They do not significantly decrease in number with age.

Suppressor/Cytotoxic T-cells express the CD8 marker and have three functions. They can function to suppress the activity of other WBC to tone down the immune system. After dividing rapidly to fight an infection, the cytotoxic T-cells disappear through apoptosis (programmed cell death). A few of them, however, turn into memory cells so that if the antigen appears again, a more rapid and effective response can be launched. This is the mechanism through which vaccinations work.

Helper/Suppressor T-cell Ratio (CD4/CD8) has been shown to be a powerful predictor of death from infection in elderly adults. It normally ranges between 1.5 and 4.0, but when it goes below 1.0, it is defined as the 'immune risk ratio' which a number of studies have shown increases 2-, 4-, and 6-year mortality rates in older individuals.

Healthy Suppressor T-cells express the CD28 marker which is necessary for these cells to rapidly divide when their TCRs engage pathogenic antigens. When you are born, 99% of your

suppressor T-cells express CD28. There is a gradual loss of healthy suppressor cells beginning in early adulthood.

Senescent Suppressor T-cells are defined as cells that have lost their ability to express the CD28 costimulatory molecule because they have undergone multiple rounds of cell division, often in response to chronic viral infections. They are no longer able to divide but do not die; far from being inert, they secrete inflammatory cytokines that can damage tissues. In older adults, they can comprise over 50% of the circulating suppressor cells. It is the increase in their number that is usually the cause of the decrease in the CD4/CD8 ratio which defines the major component of the immune risk profile.

Naive Suppressor T-cells are designated by lack of expression of the CD95 molecule with is involved in apoptosis. They are known as "virgin T-cells" because they have not encountered the antigen for their TCR and can be thought of as the reservoir of cells able to fight off new infections and tumors. They reach a peak of up to 50% of suppressor cells in young adulthood, but gradually decline as the thymus involutes. By the ninth decade, they can circulate in the single digits.

Telomere Length

As discussed in the TelomerAge section, the mean lymphocyte telomere length is a potent biomarker of aging. Although it is measured in lymphocytes, is reflects telomere attrition in other tissues as well.

Biomarker	Result	Units	Normal Range	Baseline	Change
Lymphocyte Length	5.9	kb	4.5 - 9.0	6.4	-8 %
Granulocyte Length	6.5	kb	5.5 - 10.0	7.2	-10 %

Lymphocyte Telomere Length (LTL) is measured in all the circulating lymphocytes, including B-cells, T-cells, and NK-cells. The majority consists of T-cells which can exist in the peripheral circulation and lymph nodes for years, dividing when necessary to combat infection. Therefore, the lymphocyte telomere length is a good marker for the amount of time and degree of chronic stimulation to which your immune system has been subjected from pathogens.

Granulocyte Telomere Length (GTL) is measured in the very short-lived neutrophils, eosinophils, and basophils (hours to a few days lifespan). Because these cells do not continuously divide after released into the bloodstream, the GTL reflects very well the telomere length of the hematopoietic progenitor cell residing in the bone marrow. This reflects the genetically determined component of your telomere length. As a result, the GTL is almost always longer than the LTL. The GTL-LTL gap (the difference between the lengths of each) is an even better (than LTL) measure of the chronic stress affecting your immune system from latent infections and also inflammatory diseases such as atherosclerosis.

Hormones

Biomarker	Result	Units	Normal Range	Baseline	Change
IGF-1	248.0	ng/mL	90.0 - 360.0	229.0	8 %
IGF Binding Protein-3	4.6	mg/L	3.0 - 6.6	4.0	14 %
Sex Hormone Binding Globulin	25.0	nmol/L	17.0 - 54.0	20.6	21 %
DHEA Sulfate	138.0	µg/dL	75.0 - 510.0	113.0	22 %
Estrone Sulfate	412.0			522.0	-22 %
Total Testosterone	1314.0	ng/dL	260.0 - 1000.0	1374.0	-5 %
Free Testosterone	351.5	pg/mL	50.0 - 250.0	42.4	729 %
Free Testosterone %	2.7	%	1.0 - 2.7	1.7	58 %
Estradiol	32.0	pg/mL	< 30	65.63	-52 %
LH	1.1	mIU/mL	1.5 - 9.3	0.1	1000 %
TSH	0.89	mIU/L	0.4 - 4.5	1.6	-45 %
T4	7.2	ug/dL	4.5 - 12.0	7.0	2 %
Free T3	347.0	pg/dL	230.0 - 420.0	340.0	2 %
Total T3	114.0	ng/dL	60.0 - 181.0	117.0	-3 %

Insulin-like growth factor-1 (IGF-1) is a large, protein hormone that has a similar structure to insulin. It is the hormone used to assess your level of growth hormone (GH) secretion. Because GH is secreted from the pituitary gland in short bursts throughout the day (particularly during the deep stages of sleep, fasting, and after intense exercise), a random GH blood level doesn't impart much information

about your body's total GH production. In contrast, IGF-1, which is produced in most tissues after stimulation by GH, circulates in a relatively steady state throughout the day. Therefore, it is a good measure of your total 24 hour GH production. IGF-1 has potent anabolic (muscle and bone building), immune-enhancing, and cardiovascular health-promoting effects. It is a good biomarker of aging because the average IGF-1 declines 15% per decade (in both men and women) starting in the mid-twenties.

Dehydroepiandrosterone sulfate (DHEAS) is a weak androgen produced by the adrenal glands, which sit on top of the kidneys and also produce adrenaline. DHEA is the actual hormone produced in the adrenals; it then circulates to the liver where it is sulfated so it can stay in the blood longer. DHEA is often called the "Mother Steroid" because it is a precursor to many sex hormones, including testosterone, estradiol, and estrone. In women is it the source of up to 75% of the circulating testosterone level (through the intermediary androstenedione). In men, testicular production of testosterone dwarfs the amount produced by conversion of DHEA into testosterone. In addition to being a precursor hormone, it has direct effects on the health of your arteries, bone, and immune system. The level of DHEAS is a well-documented biomarker of aging as its level reaches a peak in the mid-twenties and declines 10-20% per decade thereafter.

Luteinizing hormone (LH) is a protein hormone secreted from the pituitary gland in pulses and stimulates the production of testosterone from the Leydig cells of the testicles.

Testosterone (men) is the principal sex steroid hormone produced in the testicles under stimulation by LH. It has potent anabolic (muscle- and bone-building) effects directly, and androgenic (libido and mood enhancement/skin oil and hair production) effects through its immediate metabolite, dihydrotestosterone (DHT). Studies have shown that the total testosterone level declines modestly with age in men, but that the free testosterone level declines 10-20% per decade starting in the mid-twenties. This discrepancy is explained by the fact that the total testosterone level includes SHBG and the decrease in testosterone production is offset by the increase in SHBG. Declining free testosterone levels have been associated with the increase in low libido, depressed mood, loss of muscle and bone, and CAD risk in aging men

Testosterone (women) is important for libido/sexual function, mood, cognitive function, bone health, and muscle mass in women, but circulates in the blood at about 5% the level of men. During a woman's early reproductive years, 75% of testosterone comes from the conversion of DHEA/DHEA-S to testosterone and only 25% directly from the ovaries. In the post-menopause, about 50% of the testosterone comes from the ovaries because they continue to produce testosterone while the level of DHEAS continues to decline.

Follicle stimulating hormone (FSH) is a protein hormone secreted from the pituitary gland whose function is to recruit one of the follicles in the ovaries to undergo the final stages of maturation before becoming an egg and being ovulated. At the beginning of a new menstrual cycle, the FSH level begins to rise. Once a follicle is selected, the hormone inhibin is released from the surrounding cells into the circulation and inhibits further secretion of FSH from the pituitary gland. As a woman approaches menopause, the number of follicles declines and it becomes more difficult to find one to recruit. This causes the FSH level to rise (decreased inhibin production) and can help to indicate how far into the perimenopause a woman is. The best time to draw the FSH level is the third to fifth day after the first day of the period. If the FSH is above 10-15, then she is most likely in early perimenopause. An FSH level of 15-25 is usually found in the mid-perimenopause, and late-perimenopause and post-menopause usually are associated with levels above 25. Once a woman has not had a period for 12 months, the FSH is usually in the 60-100 range.

Estradiol (women) is the most potent estrogen of the ovaries. It is metabolized into estrone and estriol, both active, but weaker estrogens. In addition to causing the build-up of the lining of the uterus (endometrium) characteristic of a menstrual cycle, estradiol is important for maintaining the health of the arteries, bone, brain, skin, and immune system. Similar to FSH, the estradiol level needs to be interpreted in the context of the timing of the menstrual cycle. It is lowest in the first few days of the period (30-50 pg/mL) and gradually rises (100-150 pg/mL) during the 12 or so days prior to the pre-ovulatory spike (350-700 pg/mL). In the second half of the menstrual cycle (days 15-28), it averages between 150-200 and then declines just prior to the onset of the next cycle. In the perimenopause, the levels can fluctuate wildly from very low (less than 20 pg/mL) to very high (greater than 700 pg/mL), depending on whether a healthy follicle is recruited. In the post-menopause, the level is less than 20, but can be almost undetectable (less than 5 pg/mL). A minimum level of 30 pg/mL is about what is needed to eradicate hot flushes and maintain healthy bones

and skin. A somewhat higher level of 50-100 pg/mL is needed to maintain arterial and brain health.

Estradiol (men) is just as important in men as it is in women, but circulates in a more steady range and at about one-fifth the level of a premenopausal woman. It is important for maintaining the health of the arteries, bone, brain, skin, and immune system. The bulk of the estradiol circulating in a man is derived from the direct conversion of testosterone into estradiol by the enzyme called aromatase. The average estradiol level of a man runs between 20 and 50 pg/mL, but gradually increases from the low to the high end of this range as men get older. DHEA also serves as a source of estradiol. Estradiol is metabolized into estrone and then sulfated by the liver into estrone sulfate.

Estrone sulfate is a circulating storage form of estrogens. After estradiol is metabolized to estrone, it can be converted back to estradiol or a sulfate group can be added to enable it to circulate in the blood for a longer period of time. If a tissue has the enzyme to take the sulfate group off, then it can extract it from the circulation and convert it back into estradiol. Thus, the estrone sulfate level is measure of the total reservoir of estrogens available. Estrone sulfate levels vary between 230-2200 in men and 100-3600 in women. Women who take oral estrogen therapy (as part of their HRT) can raise the estrone sulfate to over 10,000 pg/mL because of the first pass effect in the liver; this can increase the effective estradiol exposure of the body which might not be apparent when only the estradiol level is measured.

Progesterone is the "pro-gestational" hormone because it aids in maintaining a pregnancy (gestation). There is essentially no progesterone (< 0.7 ng/mL) circulating in the first half of the menstrual cycle. Once an egg is ovulated, the part of the ovary that released it (called the corpus luteum) starts to produce progesterone for about two weeks. A healthy luteal-phase progesterone level runs between 13 and 25 ng/mL. Progesterone transforms and stabilizes the lining of the uterus that has built up in the previous two weeks so that it is ready for a fertilized egg to implant. If no egg is implanted, then the corpus luteum shrivels up and the progesterone declines after 14 days causing the lining to destabilize and shed off. One of the first signs of perimenopause is the decrease in the amount of progesterone the corpus luteum produces (< 9 ng/mL). These lower levels cannot adequately stabilize the lining and can cause the shorter cycle lengths characteristic of perimenopause. Progesterone receptors occur in the breast as well as in the uterus. There they can modulate the effect of estrogens. Progesterone also has bone and central nervous system enhancing effects.

Tri-iodothyronine (T3) is critical for development of the brain and body of infants and control of metabolic activity in adults. Receptors for thyroid hormones are found in virtually all tissues and can critically affect the function of all organ systems. The heart, bone, and subcutaneous fat are major targets of thyroid hormone action. T3 constitutes 20% of circulating thyroid hormone and is the more biologically active form.

Thyroxine (T4) essentially functions as a precursor hormone reservoir (80% of circulating thyroid hormones) of T3. It is released into the circulation and then is converted into T3, either in the liver or target cells such as the heart and subcutaneous fat. IGF-1 is important for the conversion of T4 into T3.

Thyroid stimulating hormone (TSH) is produced in the pituitary gland and controls the production and release of thyroid hormones from the thyroid gland. T4, and to a greater degree, T3, inhibit the release of TSH in a negative feedback fashion. Therefore, a higher TSH indicates that the pituitary gland senses there is inadequate circulating thyroid hormone and tries to stimulate further production and release by secreting more TSH. In contrast, a TSH below the normal range - particularly if it is undetectable - indicates the state of hyperthyroidism where excess thyroid hormone is causing the suppression TSH.

Blood

There are three components of the common Complete Blood Count (CBC): the WBC, RBC, and platelets. As discussed above, the WBC tells us about the state of the immune system. The RBC tells us about the state of the oxygen carrying capacity of our blood. The platelet count is a measure of our ability to form a 'plug' when a blood vessel has been damaged.

Biomarker	Result	Units	Normal Range	Baseline	Change
RBC	4.73	million/µL	4.2 - 5.8	5.8	-19 %
Hematocrit	42.9	%	38.5 - 50.0	52.4	-19 %
Hemoglobin	14.8	g/dL	13.2 - 17.1	17.0	-13 %
MCH	31.3	pg	27 - 33	29.2	7 %
MCHC	34.5	g/dL	32 - 36	32.4	6 %

Biomarker	Result	Units	Normal Range	Baseline	Change
MCV	90.8	fL	80 - 100	90.0	0 %
RDW	13.8	%	11 - 15	14.0	-2 %
Platelet Count	172.0	thousand/ µL	140 - 400	191.0	-10 %
MPV	8.0	fL	7.0 - 11.0	8.0	0 %

RBC is a measure of the total number of red blood cells found in a microliter of blood. Anemia is defined as an RBC below the normal range. It can be caused by acute blood loss from trauma or bleeding of an internal organ. If the blood loss is more gradual, then it can go on undetected until the body's stores of the nutrients necessary to make red blood cells are depleted. An RBC above the normal range is called 'erythrocytosis.'

Hematocrit is a term similar to the RBC but is the concentration of red blood cells in the blood rather than the number. These two measures of the oxygen carrying capacity of the blood usually track together. They can diverge when you are in a state of dehydration (in which case the number of cells stays the same, but the concentration increases). The hematocrit is the more often used parameter.

Hemoglobin is the oxygen carrying protein found in the red blood cells of all humans and other vertebrate organisms. Low hemoglobin is most often indicative of blood loss and/ or iron deficiency, but can also result from genetic disorder of hemoglobin production such as thalassemia. An elevated hemoglobin level occurs with erythrocytosis.

Mean Corpuscular Hemoglobin (MCH) is the average amount of hemoglobin in each red blood cell (aka 'corpuscle') and can help your doctor understand the type and cause of anemia your may be exhibiting. It is calculated by dividing the total amount of hemoglobin by the RBC. It is reduced in iron-deficiency anemia.

Mean Corpuscular Hemoglobin Concentration (MCHC) is similar to the MCH, but is calculated by dividing the hemoglobin by the hematocrit. It is the most sensitive indicator of iron deficiency anemia.

Mean Corpuscular Volume (MCV) This is a measure of the average size of your red blood cells and when it is above or below the normal range can be indicative of particular disease states or nutrient deficiencies. For example, in vitamin B12 and/or folate deficiency, the size of the red blood cells increases. In iron deficiency anemia, the size of the red blood cell decreases.

Red cell distribution width (RDW) is a measure of the variation in the size (MCV) of the red blood cells. B12/folate deficiency causes red blood cells to be larger than normal, while iron deficiency causes them to be smaller. Thus, if there is a mixed iron and B12/folate deficiency, there will both and increased number of small and large cells and the RDW increases. During active blood loss, the bone marrow steps up the production of red blood cells to replace them and maintain oxygen carrying capacity. These new red blood cells (called reticulocytes), are larger than mature red blood cells and can increase the RDW. The RDW has been shown to be a predictor of mortality in older populations, possibly because it is a marker for vitamin/nutrient deficiencies.

Platelets are fragments of bone marrow precursor cells called 'megakaryocytes' and are the critical component in thrombus (clot) formation. In addition to maintaining hemostasis (controlling bleeding), platelets release growth factors that play significant roles wound healing and repair/ regeneration of connective tissue. There are disorders of abnormally low and high platelet count called thrombocytopenia and thrombocytosis, respectively. Ninety five percent of people have a platelet count between 150,000 and 450,000 per microliter. A level 100,000-150,000 may be physiologically insignificant in a small percentage of people, but is more likely to be the early stages of a gradually worsening thrombocytopenia. Platelet levels below 25,000 can lead to an increase in life-threatening hemorrhages from minor trauma. Thrombocytosis above 500,000 can lead to an increase risk of strokes and heart attacks.

Mean platelet volume (MPV) is a measure of the average size of your platelets. It is used to determine if a low platelet count is the result of clumping together of individual platelets and not an actual decrease in platelet number.

Vitamins

Biomarker	Result	Units	Normal Range	Baseline	Change
Iron	129.0	µg/ dL	45.0 - 170.0	85.0	51 %
TIBC	359.0	µg/ dL	250.0 - 425.0	290.0	23 %
Transferrin Saturation	36.0	%	20.0 - 50.0	29.0	24 %

Biomarker	Result	Units	Normal Range	Baseline	Change
Ferritin	60.0	ng/mL	20 - 250	52.0	15 %
Folate	24.0	ng/mL	> 5.5	8.5	182 %
Vitamin B12	1298.0	pg/mL	200 - 1100	1889.0	-32 %
Vitamin D	30.0	ng/mL	20 - 100	23.5	27 %
Vitamin D2	0.0	ng/mL	0 - 75	3.5	-100 %
Vitamin D3	30.0	ng/mL	20 - 100	20.0	50 %

Iron is an essential nutrient for the production of red blood cells and many enzyme-dependent reactions. While iron deficiency can cause anemia and fatigue, an excess of iron can increase free-radical production and increase the risk of cancer and cardiovascular disease. As with many nutrients, an optimal level is needed for good health - too much or too little can cause harm. The serum iron level can fluctuate quite significantly depending on recent dietary iron intake. Iron levels less than 45 µg/dL are indicative of iron deficiency, but a better indicator is the transferrin saturation.

Total iron binding capacity (TIBC) is the amount of the iron binding protein called "transferrin" in the blood stream. It is usually increased in iron deficiency and decreased in conditions of excess iron.

Transferrin saturation is the percentage of TIBC that has iron attached to it. Iron deficiency is diagnosed when the level drops below 20%. If the saturation is above 50%, then an iron overload condition should be considered.

Ferritin is the intracellular storage form of iron and is the best indicator of total body iron stores. While the serum iron, TIBC, and transferrin saturation (collectively known as 'iron studies') can be good screening tests for iron deficiency and overload disorders, they should be confirmed with a ferritin test. A level below 10 is diagnostic of iron deficiency. If it is accompanied by anemia, then it is called iron deficiency anemia. Serum ferritin levels above 500 ng/mL should raise suspicion of an iron overload disorder, the most common of which is hereditary hemochromatosis. This genetic disorder causes the gradual accumulation of iron in many tissues of the body because of a defect in the natural block in the stomach to absorbing more dietary iron than necessary. However, ferritin is also an acute phase reactant (like CRP) and in the presence of an acute infection, a high level cannot be used to diagnose iron overload. We believe the "normal" range is too wide, and consider an optimal level of ferritin to be 25-100. This avoids the possibilities of inadequate iron for enzyme function, and too much iron and consequent increased free-radical production.

Folate is the form of the member of the B-vitamin family (B9) that is naturally found in the body and foods. Folic acid, the synthetic form of the vitamin found in most supplements and fortified foods, needs to be reduced to folate before it can be combined into its active form tetrahydrofolate. Its name is derived from the Latin for leaf, folium, because leafy vegetables are a rich source of folate. Folate is important for rapidly-growing tissues because it is needed for DNA and RNA synthesis. Folate deficiency during pregnancy can result in neural tube defects. It is also critical for red blood cell production, and folate deficiency results in a form of anemia characterized by enlarged red blood cells (megaloblastic anemia). The metabolism of homocysteine requires adequate levels of folate; in fact, an increase in serum homocysteine level is an early and sensitive indicator of folate deficiency.

Vitamin B12 is a water-soluble vitamin found in animal products such as fish, meat, eggs, fowl, and dairy but not generally in plant foods. As a result, vegetarians are at risk for B12 deficiency unless they eat B12-fortified cereals or supplements. There are several forms of B12, but all contain the mineral cobalt and are called cobalamins. In food, B12 is bound to proteins and must be released by stomach acid, whereas the form in supplements and fortified foods is free and doesn't require acid for release. Therefore, people on stomach acid-suppressing medications (proton pump inhibitors, antacids) can have low B12 levels. B12 is important for red blood cell production, nerve cell health, and DNA synthesis. The anemia of B12 deficiency can be corrected or avoided by a high folate level, but this is a dangerous situation because folate will not prevent the neuropathy that results from B12 deficiency. Therefore, these levels should always be assessed together. People in the lower end (200-400) of the "normal" range can be effectively suffering from B12 deficiency. Pernicious anemia is the autoimmune disorder in which the lining of the stomach does not produce intrinsic factor, the molecule necessary for absorption of B12 from the small intestine once it is released in the stomach. B12 deficiency can also lead to an elevated homocysteine level.

Vitamin D can be synthesized in the skin from a precursor molecule upon exposure to ultraviolet B light. This is an important source because there is not much vitamin D in the

typical diet, although it is found in some fish and eggs as well as vitamin D-fortified foods (e.g., dairy products). As a result, vitamin D deficiency is common in Northern latitudes and in the elderly who often get little sun exposure and have poor diets. Vitamin D is critical for maintaining normal calcium metabolism, bone health, and immune system function. Severe vitamin D deficiency in childhood causes rickets (malformed long bones), but lesser levels of deficiency have been demonstrated to adversely affect the cardiovascular and immune systems and cause osteoporosis. The normal vitamin D range is 20-100 ng/mL, but a level between 20-33 ng/mL is considered insufficient for normal bone and calcium physiology. An optimal level is generally considered to be above 40 ng/mL. Levels below 15 ng/mL are associated with an increase in hypertension, general joint and muscle aches, and poor immune system function. Vitamin D exists in two forms, D2 and D3. Some studies have suggested that repletion with vitamin D3 more efficiently raises vitamin D levels than repletion with D2. To raise the level of a person with vitamin D insufficiency (20-32 ng/mL) to sufficiency (>33 ng/mL), it takes a three-month course of 800-1000 IU of vitamin D3. A number of studies have suggested that higher levels of serum vitamin D are associated with lower rates of common cancers.

Kidney Function

Electrolytes are the positively and negatively charged small molecules (called 'ions') found in your cells, blood stream, and extracellular fluids. They are maintained in a delicate balance by your kidney, lungs, and endocrine system. They are critical in the electrical signaling between and within cells, the acid-base balance (pH), and the maintenance of the fluid balance between different body compartments. Small fluctuations in their relative levels can be clues to serious disease processes. Cations and anions (negatively and positively charged ions) travel together as salts to balance the overall charge of a fluid. They are usually measured together in a serum sample because interpretation of one requires knowledge of the levels of most of the others.

Biomarker	Result	Units	Normal Range	Baseline	Change
Sodium	141.0	mmol/L	135 - 146	139.0	1 %
Potassium	4.3	mmol/L	3.5 - 5.3	3.9	10 %
Chloride	102.0	mmol/L	98 - 110	104.0	-2 %
CO2 Carbon Dioxide	27.0	mmol/L	21 - 33	24.0	12 %
Calcium	9.6	mg/dL	8.6 - 10.2	9.1	5 %
Phosphorus	3.7	mg/dL	2.5 - 4.5	2.9	27 %
Uric Acid	5.2	mg/dL	4.0 - 8.0	4.5	15 %
Creatinine	1.03	mg/dL	0.5 - 1.3	1.0	3 %
Urea Nitrogen	17.0	mg/dL	7 - 25	22.0	-23 %
BUN/Creatinine Ratio	16.5		6 - 22	22.0	-25 %

Sodium is the principal positively charged ion (called a 'cation') of the extracellular space. It is the same as the sodium found in most foods and table salt (sodium chloride). The body responds to changes in the serum sodium level in three main ways: (1) Modulating thirst: as little as a 1% increase in serum sodium can make you thirsty so you consume water to decrease the level to normal. (2) Producing sodium-regulating hormones: certain hormones (natriuretic peptide) cause the kidneys to lose sodium while others (aldosterone) cause them to retain sodium. (3) Producing water-regulating hormones: antidiuretic hormone (ADH) causes the kidneys to hold onto free water. Water follows sodium. When you eat a salty meal, you become thirsty and drink water. The extra fluid is retained (causing the characteristic post-Chinese food bloating and edema) until it can be excreted as the hormonal sodium excretion pathways kick in. ADH is inhibited by alcohol causing the excessive urination of clear water noted after drinking a lot of beer or other alcoholic beverages. When these mechanisms are not functioning well or are overwhelmed, a state of hypernatremia (high serum sodium) or hyponatremia (low serum sodium) can ensue. The most common cause of hypernatremia is dehydration from decreased water intake. Hyponatremia is most commonly from sodium loss through sweat that is replaced only with water. Other causes include diuretics, Addison's disease, diarrhea, and kidney disease.

Potassium is the principal intracellular cation, and only about 2% of your total body potassium is located in your body fluids and blood stream. Increased serum potassium (hyperkalemia) is most commonly caused by kidney disease, but other medications, such as ACE inhibitors, potassium-sparing diuretics, and NSAIDs, can cause it. Hyperkalemia can cause abnormal heart rhythms and respiratory failure. Low serum potassium (hypokalemia) can be caused by dehydration, vomiting, diarrhea, and inadequate repletion when taking diuretics.

Chloride is the anion that travels with sodium in and out of cells to help regulate body fluids and acid-base balance. When a problem arises with the serum sodium level, the chloride can diverge from sodium to buffer the pH of the blood temporarily. Chloride is ingested as sodium chloride in food and table salt. Increased chloride levels most commonly indicate dehydration, and a decreased level can be caused by vomiting, chronic lung disease or with a loss of acid from the body.

Carbon dioxide (CO2) should really be called bicarbonate or HCO3 because CO2 is actually the gas that your lungs exhale. When it is dissolved in water, CO2 is associates with a hydrogen ion to become HCO3 and acts as a buffer for acid in the blood. A low serum bicarbonate level indicates that your body is in an acidic state and the bicarbonate is being used up to buffer it. This can be caused by diabetes, kidney disease, and chronic diarrhea. A high bicarbonate level indicates alkaline pH of the blood due to acid loss from vomiting, lung disease, or Cushing's syndrome.

Calcium is a mineral cation in your blood that is essential for the healthy functioning of your muscles, nervous system, and heart. Its serum concentration is very tightly regulated by your kidneys and endocrine system because deviations from the normal level can have serious consequences. If there is a mild elevation (less than 10.5), the first thing to do is to repeat the blood test to make sure it is not a lab error. Persistently high serum calcium (hypercalcemia) is commonly caused by either hyperparathyroidism (benign tumors of the parathyroid gland secreting too much parathyroid hormone) or cancer that has spread to the bones. Low serum calcium is most commonly caused by a low serum protein level (from malnutrition) because the calcium is bound to protein. A follow-up ionized serum calcium level will be normal if this is the only cause. Other causes of hypocalcemia are low vitamin D level, underactive parathyroids (hypoparathyroidism), magnesium deficiency, and kidney failure.

Phosphorus is a mineral which forms phosphates when combined with oxygen. The bonds between the phosphorus and oxygen in phosphates contain energy which is used in many chemical reactions in the body. Only a very small amount (1%) of your total body phosphate circulates in your blood. The rest is incorporated into bones, teeth, and muscle or is found in the rest of the cells of body in energy storage molecules. An abnormal level of phosphorus normally does not cause any symptoms. However, it can indicate a problem with parathyroid hormone or vitamin D and can help to interpret the cause of low or high calcium. Low phosphorus can be associated with hypercalcemia, hypothyroidism, out-of-control diabetes, and diuretic abuse. High phosphorus can be associated with kidney disease and excess phosphate intake.

Uric Acid is a product of the breakdown of one type of nucleic acid (a component of DNA/RNA) called a purine. The serum uric acid level can rise when there is excessive production or decreased excretion via the kidneys and feces. Excess production can occur during chemotherapy/ radiation (breakdown of cells with release of DNA) and decreased excretion because of kidney disease. Some people have an inherited condition in which they produce a higher level of uric acid and are at risk for kidney stones and gout (the painful inflammation of joints caused by the presence of uric acid crystals in the joint space). Like bilirubin, uric acid is also a potent antioxidant and people with a level in the upper range of normal may have better protection from free-radical damage.

Creatinine is a product of the breakdown of creatine, compound produced by your muscles when they are actively contracting. Because creatinine is produced at a relatively constant rate and is excreted almost exclusively by your kidneys, its serum level is a good indicator of kidney filtration rate (health). However, an increased serum level can be found in people with higher muscle mass or who have been exercising vigorously prior to the test. Cystatin C is a newer measure of kidney function that is not affected by these factors. Low levels of creatinine are usually found in people with relatively low muscle mass and higher levels can indicate kidney function decline. A level above 2 is usually an indication that some impairment of kidney function is present. In most individuals, creatinine slowly increases (0.5-1% per year) with age, making it a biomarker of kidney function aging.

Blood urea nitrogen (BUN) is produced when your body breaks nitrogen-containing protein down into its constituent amino acids and the liver combines the nitrogen into the waste product urea. There is a relatively constant production of BUN, which the kidneys then filter out and excrete into the urine. Diseases that affect the kidneys or the liver can raise BUN. Other causes include heart failure, dehydration, and or gastrointestinal bleeding. Low BUN is not very common and if present is usually not a cause for concern. The most common cause of an elevated BUN in an otherwise healthy individual is mild dehydration.

The BUN/Creatinine ratio is used to differentiate dehydration or excess BUN production from kidney

problems. In kidney failure, both creatinine and BUN rise, so the ratio will not increase. When there is excess BUN production only (from, e.g., gastrointestinal bleeding) you will see an increased BUN/Creatinine. A mildly elevated ratio (22-25) can occur in people with low muscle mass (which causes a low creatinine level) and slight dehydration, but is usually not a cause for concern.

Liver Function

The tests usually grouped under the heading of 'liver function tests' impart a variety of information, not all about just the liver. They can be divided into 3 categories: synthetic function, liver/bile duct damage, and immune system function.

Biomarker	Result	Units	Normal Range	Baseline	Change
A/G Ratio	1.6		1.0 - 1.2	1.3	23 %
Albumin	4.5	g/dL	3.6 - 5.1	3.8	18 %
Globulin	2.8	g/dL	2.1 - 3.7	2.9	-4 %
Total Protein	7.3	g/dL	6.2 - 8.3	6.7	8 %
GGT	17.0	U/L	3 - 70	22.0	-23 %
ALT	37.0	U/L	9.0 - 60.0	42.0	-12 %
AST	33.0	U/L	10 - 40	31.0	6 %
Alkaline Phosphatase	64.0	U/L	40.0 - 115.0	67.0	-5 %
Bilirubin,direct	0.2	mg/dL	0.0 - 0.2	0.3	-34 %
Bilirubin,total	0.9	mg/dL	0.2 - 1.2	0.5	80 %
LD	158.0	U/L	120 - 250	152.0	3 %

Albumin is the most abundant protein in human serum. It is important for maintaining normal osmotic pressure (the force keeping the fluid in the blood vessels), carrying certain hormones, and neutralizing free radicals. It is produced in the liver, and a decreased level can indicate reduced liver function or liver disease. The serum level of albumin decreases with age even in the absence of disease. An increased level is generally the result of dehydration. In the absence of dehydration, a higher serum level is generally a sign of good health.

Globulin is the term used for the non-albumin proteins circulating in the blood. These include many proteins but can roughly be divided into two groups. The gamma globulins are mostly composed of circulating antibodies made by mature B-cells called plasma cells. The other group contains SHBG, transferrin, ferritin, thyroid binding globulin, etc. Elevation of the serum globulins of the gamma variety can occur in lymphoma, multiple myeloma, and monoclonal gammopathy of undetermined significance.

Albumin/Globulin (A/G Ratio) can serve as a more sensitive flag for disorders of high or low production of albumin or globulins because the individual levels can fluctuate according to hydration status. A low ratio can be caused by increased production of gamma globulins, as occurs in multiple myeloma. A high ratio is usually a sign of good health unless it is associated with a very low globulin, in which case it may signal hypogammaglobulinemia, which can be caused by kidney disease.

Protein (total) is the sum of the albumin and globulin proteins in the serum.

Gamma glutamyl transpeptidase (GGT) is an enzyme that transfers a gamma glutamyl group to other molecules. It is found in the liver, prostate, kidney, intestines, and pancreas. Often it is the earliest liver function test to be elevated in the event of bile duct blockage. It is most useful in determining if an elevation of alkaline phosphatase is from bone disease because if the GGT is elevated, liver disease is the cause of the elevation. Uncomplicated diabetes, acute pancreatitis, myocardial infarction, and certain liver-damaging drugs can also raise GGT.

Alanine aminotransferase (ALT) is an enzyme that transfers the amino group from alanine during certain energy producing reactions. It is found in liver tissue, but also in skeletal and heart muscle. It is often used as an indicator of liver inflammation because its serum level can increase markedly when liver cells are damaged and leak the enzyme into the circulation, as occurs during hepatitis. Minor elevations can be seen after intense exercise, alcohol consumption, or when taking certain drugs, particularly cholesterol-lowering drugs of the statin family such as Lipitor.

Aspartate amino transferase (AST) is an enzyme similar to ALT except it transfers the amino group of aspartate. It is used as an indicator of liver and cardiac muscle damage. When both ALT and AST are elevated, there is an increased risk of liver damage. The ratio of AST to ALT can be used to distinguish among causes of aminotransferase elevations. An AST/ALT < 2 is often indicative of chronic liver disease from

Wilson's disease and alcoholic liver disease. An AST/ALT < 1 is indicative of acute liver disease or that caused by fatty deposits in the liver, as occur in obesity and diabetes.

Alkaline phosphatase is an enzyme that takes phosphate groups off of molecules and works best in an alkaline (high pH) environment. It is present throughout the body, but particularly in liver, bile duct, and bones. The level of alkaline phosphatase is measured as a biomarker for damage to one of the organ system where it is produced. Alkaline phosphatase serum level can be elevated when the bile duct is blocked or inflamed. A healing fracture can cause an increase in alkaline phosphatase. Low levels of alkaline phosphatase can be associated with low thyroid function, but for the most part, a level lower than the low end of normal is of no clinical significance.

Bilirubin (total) is produced from the breakdown of hemoglobin when red blood cells are destroyed. It is relatively abundant in the blood stream of mammals and may have evolved as a potent antioxidant system to protect against the oxidation of lipids (cholesterol) in cell membranes. Increased levels of bilirubin are an indication of blockage of the bile duct (by gallstones or inflammation), or increased destruction of red blood cells that overwhelms the ability of the liver to process the bilirubin into its water-soluble form for elimination in the urine. Interestingly, levels in the higher end of the normal range are associated with a decreased incidence of heart attack. Chronic slightly elevated levels of bilirubin, in the absence of bile duct blockage, are most often caused by a benign genetic disorder called Gilbert's syndrome (found in about 5% of the population). When total bilirubin levels rise above 3 mg/dl, a yellowing of the skin called 'jaundice' can occur.

Bilirubin (direct) is the fraction of the total bilirubin that has been attached to a molecule (glucuronide) to prepare it for excretion. The indirect bilirubin is that fraction of the total that is free and unattached. The total bilirubin is the sum of the indirect and the direct bilirubin. In Gilbert's syndrome, it is the indirect bilirubin that is increased because of a low level of the enzyme that attaches the glucuronide molecule to bilirubin to prepare it for excretion. Gilbert's syndrome causes indirect bilirubin to rise during illness and can lead to a mild case of jaundice.

Cancer Screening

Biomarker	Result	Units	Normal Range	Baseline	Change
PSA	0.92	ng/mL	0.0 - 4.0	1.36	-33 %

Prostate-specific antigen (PSA) is an imperfect prostate cancer-screening test; it can be above the normal range when you don't have prostate cancer and can be in the normal range when you do (although this is very unlikely if the level is < 1). PSA is a protein that is made within the prostate gland (though contrary to its name, it can be made in small amounts by a few other tissues) which leaks out into the blood stream when a prostate tumor is growing or if the prostate is inflamed (prostatitis) or enlarged (benign prostatic hyperplasia aka "BPH"). The PSA velocity (determined through repeated measurements over time) is a more informative metric of the risk of prostate cancer. If the PSA level is rapidly rising, it is more likely to be a result of prostate cancer than BPH. While PSA is not a very good cancer screening tool, it is a very good indicator of prostate health. If you have a PSA less than 1, then you have a healthy prostate - the lower the number, the healthier the gland. This is demonstrated by the fact that most men under 30 have a PSA less than 1.

Abnormal Results

Name	Result	Units	Normal Range	Optimal Range	Baseline	Change
Arterial Stiffness						
SBP	130.0	mm Hg	90.0 - 120.0		126.0	3 %
Cognitive Function						
Composite Memory	66.0		90.0 - 109.0		75.0	-12 %
Verbal Memory	110.0		90.0 - 109.0		102.0	7 %
Visual Memory	75.0		90.0 - 109.0		106.0	-30 %
Executive Functioning	75.0		90.0 - 109.0		75.0	0 %
Lymphocytes						
Healthy Suppressor T-Cells %	39.0	%	49 - 96	> 80	44.0	-12 %
Senescent Suppressor Cells %	61.0	%	4.0 - 51.0	< 20	56.0	8 %
Hormones						
Total Testosterone	1314.0	ng/dL	260.0 - 1000.0	600 - 1000	1374.0	-5 %
Free Testosterone	351.5	pg/mL	50.0 - 250.0	150 - 250	42.4	729 %
Estradiol	32.0	pg/mL	< 30		65.63	-52 %
LH	1.1	mIU/mL	1.5 - 9.3	1.5 - 5	0.1	1000 %
Vitamins						
Vitamin B12	1298.0	pg/mL	200 - 1100	500 - 1100	1889.0	-32 %
Liver Function						
A/G Ratio	1.6		1.0 - 1.2		1.3	23 %

Complete Data

Name	Result	Units	Normal Range	Optimal Range	Baseline	Change
Vital Signs						
Height	74.0	inches	‡		74.5	-1 %
Weight	201.0	lbs	‡		201.0	0 %
Arterial Stiffness						
SBP	130.0	mm Hg	90.0 - 120.0		126.0	3 %
DBP	76.0	mm Hg	60.0 - 80.0		72.0	5 %
Augmentation Pressure	8.0	mm Hg	0.0 - 8.0		13.0	-39 %
Augmentation Index@75	12.0	mm Hg	0.0 - 23.0		16.0	-25 %
Ejection Duration	357.0	mSec			322.0	10 %
Ejection Duration %	28.0	%	26.0 - 32.0		32.0	-13 %
SEVR	203.0	%	186.0 - 240.0		172.0	18 %
Cardiovascular Risk						
Total Cholesterol	153.0	mg/dL	125.0 - 200.0	< 150.0	134.0	14 %
LDL Cholesterol	54.0	mg/dL	0.0 - 129.0	< 100.0	69.0	-22 %
HDL Cholesterol	92.0	mg/dL	> 40.0	> 50.0	57.0	61 %
Cholesterol/HDL Ratio	1.7		0.0 - 5.0	< 3.0	2.35	-28 %
Triglycerides	37.0	mg/dL	0.0 - 149.0	< 75	43.0	-14 %
C-Reactive Protein	0.9	mg/L	0.0 - 2.9	< 1.0	2.5	-64 %
Homocysteine	7.0	µmol/L	0.0 - 10.3	< 9.0	10.07	-31 %
Coenzyme Q10	1.0	mg/L	0.44 - 1.64	> 1.5	1.0	0 %
Diabetes & Glucose						
Glucose	81.0	mg/dL	0.0 - 99.0	70 - 90	90.0	-10 %
Insulin,serum	2.0	uIU/mL	0.0 - 16.0	< 9	4.6	-57 %
Hemoglobin A1C	5.5	%	0.0 - 6.0	< 5	5.5	0 %
Lung Function						
FVC	5.6	L			5.38	4 %
FEV1	4.38	L			4.41	-1 %
FEV1/FVC	74.0	%	‡		81.97	-10 %
FEF25-75%	3.0	%			3.2	-7 %
PEF	9.1	L/sec			8.2	10 %
Cognitive Function						
Composite Memory	66.0		90.0 - 109.0		75.0	-12 %

‡ - range depends on a variety of factors

Name	Result	Units	Normal Range	Optimal Range	Baseline	Change
Verbal Memory	110.0		90.0 - 109.0		102.0	7 %
Visual Memory	75.0		90.0 - 109.0		106.0	-30 %
Psychomotor Speed	97.0		90.0 - 109.0		61.0	59 %
Processing Speed	92.0		90.0 - 109.0		81.0	13 %
Reaction Time	100.0		90.0 - 109.0		52.0	92 %
Cognitive Flexibility	98.0		90.0 - 109.0		77.0	27 %
Executive Functioning	75.0		90.0 - 109.0		75.0	0 %
Subject Composite Memory	100.0				102.0	-2 %
Subject Verbal Memory	60.0				100.0	-40 %
Subject Visual Memory	45.0				102.0	-56 %
Subject Psychomotor Speed	197.0				168.0	17 %
Subject Processing Speed	62.0				50.0	24 %
Subject Reaction Time	695.0				800.0	-14 %
Subject Cognitive Flexibility	52.0				52.0	0 %
Subject Executive Functioning	52.0				52.0	0 %
Immune Function						
WBC	3956.0	cells/µL	3500 - 9500		4123.0	-5 %
Neutrophils Percent	47.5	%	38.0 - 80.0		64.0	-26 %
Neutrophils Count	1568.0	cells/µL	1500.0 - 7800.0		1400.0	12 %
Monocytes Percent	7.0	%	0.0 - 13.0		6.0	16 %
Monocytes Count	290.0	cells/µL	200.0 - 950.0		256.0	13 %
Eosinophils Percent	1.8	%	0.0 - 8.0		3.0	-40 %
Eosinophils Count	59.0	cells/µL	15.0 - 550.0		60.0	-2 %
Basophils Percent	0.9	%	0.0 - 2.0		1.0	-10 %
Basophils Count	30.0	cells/µL	0.0 - 200.0		21.0	42 %
Lymphocyte Percent	41.0	%	20 - 48		25.0	64 %
Lymphocyte Count	1353.0	cells/µL	1078 - 2828		800.0	69 %
Lymphocytes						
NK Cell %	15.0	%	3 - 26		13.0	15 %
NK Cell Count	190.0	cells/µL	51 - 543		200.0	-5 %
B-cells %	22.0	%	5 - 22		9.0	144 %
B-cell Count	200.0	cells/µL	74 - 447		200.0	0 %
T-cells %	68.0	%	57.0 - 85.0		71.0	-5 %
T-Cell Count	1032.0	cells/µL	767 - 2318		981.0	5 %
Helper T-Cells %	41.0	%	32 - 59		45.0	-9 %
Suppressor T-Cells %	27.0	%	13 - 38		23.0	17 %

‡ - range depends on a variety of factors

Name	Result	Units	Normal Range	Optimal Range	Baseline	Change
Suppressor T-Cell Count	369.0	cells/µL	201 - 868	‡	259.0	42 %
Helper/Suppressor T-Cell Ratio	1.5		0.96 - 3.93	1.5 - 2.5	1.95	-24 %
Healthy Suppressor T-Cells %	39.0	%	49 - 96	> 80	44.0	-12 %
Healthy Suppressor T-Cell Count	180.0	cells/µL	100 - 850		61.0	195 %
Senescent Suppressor Cells %	61.0	%	4.0 - 51.0	< 20	56.0	8 %
Senescent Suppressor Cell Count	189.0	cells/µL	17.0 - 364.0	< 50	198.0	-5 %
Naive Suppressor Cells %	19.0	%	11.0 - 57.0	> 30	6.0	216 %
Naive Suppressor Cell Count	100.0	cells/µL	32.0 - 347.0		48.0	108 %
CMV AB (IGG)	0.24		0.0 - 1.0		0.14	71 %
Hormones						
IGF-1	248.0	ng/mL	90.0 - 360.0	250 - 360	229.0	8 %
IGF Binding Protein-3	4.6	mg/L	3.0 - 6.6	4 - 6.6	4.0	14 %
Sex Hormone Binding Globulin	25.0	nmol/L	17.0 - 54.0		20.6	21 %
DHEA Sulfate	138.0	µg/dL	75.0 - 510.0	350 - 550	113.0	22 %
Estrone Sulfate	412.0				522.0	-22 %
Total Testosterone	1314.0	ng/dL	260.0 - 1000.0	600 - 1000	1374.0	-5 %
Free Testosterone	351.5	pg/mL	50.0 - 250.0	150 - 250	42.4	729 %
Free Testosterone %	2.7	%	1.0 - 2.7	1.0 - 2.0	1.7	58 %
Estradiol	32.0	pg/mL	< 30		65.63	-52 %
LH	1.1	mIU/mL	1.5 - 9.3	1.5 - 5	0.1	1000 %
TSH	0.89	mIU/L	0.4 - 4.5	0.5 - 1.5	1.6	-45 %
T4	7.2	ug/dL	4.5 - 12.0	6 - 9	7.0	2 %
Free T3	347.0	pg/dL	230.0 - 420.0	300 - 420	340.0	2 %
Total T3	114.0	ng/dL	60.0 - 181.0		117.0	-3 %
Blood						
RBC	4.73	million/µL	4.2 - 5.8		5.8	-19 %
Hematocrit	42.9	%	38.5 - 50.0	‡	52.4	-19 %
Hemoglobin	14.8	g/dL	13.2 - 17.1	‡	17.0	-13 %
MCH	31.3	pg	27 - 33		29.2	7 %
MCHC	34.5	g/dL	32 - 36		32.4	6 %
MCV	90.8	fL	80 - 100		90.0	0 %
RDW	13.8	%	11 - 15		14.0	-2 %
Platelet Count	172.0	thousand/µL	140 - 400	200 - 400	191.0	-10 %
MPV	8.0	fL	7.0 - 11.0		8.0	0 %
Vitamins						
Iron	129.0	µg/dL	45.0 - 170.0	45 - 75	85.0	51 %

‡ - range depends on a variety of factors

Name	Result	Units	Normal Range	Optimal Range	Baseline	Change
TIBC	359.0	µg/dL	250.0 - 425.0	250 - 350	290.0	23 %
Transferrin Saturation	36.0	%	20.0 - 50.0	20 - 30	29.0	24 %
Ferritin	60.0	ng/mL	20 - 250	25 - 100	52.0	15 %
Folate	24.0	ng/mL	> 5.5	15 - 24	8.5	182 %
Vitamin B12	1298.0	pg/mL	200 - 1100	500 - 1100	1889.0	-32 %
Vitamin D	30.0	ng/mL	20 - 100	45 - 100	23.5	27 %
Vitamin D2	0.0	ng/mL	0 - 75	‡	3.5	-100 %
Vitamin D3	30.0	ng/mL	20 - 100	45 - 100	20.0	50 %
Kidney Function						
Sodium	141.0	mmol/L	135 - 146		139.0	1 %
Potassium	4.3	mmol/L	3.5 - 5.3		3.9	10 %
Chloride	102.0	mmol/L	98 - 110		104.0	-2 %
CO2 Carbon Dioxide	27.0	mmol/L	21 - 33		24.0	12 %
Calcium	9.6	mg/dL	8.6 - 10.2		9.1	5 %
Phosphorus	3.7	mg/dL	2.5 - 4.5		2.9	27 %
Uric Acid	5.2	mg/dL	4.0 - 8.0		4.5	15 %
Creatinine	1.03	mg/dL	0.5 - 1.3		1.0	3 %
Urea Nitrogen	17.0	mg/dL	7 - 25		22.0	-23 %
BUN/Creatinine Ratio	16.5		6 - 22		22.0	-25 %
Liver Function						
A/G Ratio	1.6		1.0 - 1.2		1.3	23 %
Albumin	4.5	g/dL	3.6 - 5.1	> 4.5	3.8	18 %
Globulin	2.8	g/dL	2.1 - 3.7		2.9	-4 %
Total Protein	7.3	g/dL	6.2 - 8.3		6.7	8 %
GGT	17.0	U/L	3 - 70		22.0	-23 %
ALT	37.0	U/L	9.0 - 60.0		42.0	-12 %
AST	33.0	U/L	10 - 40		31.0	6 %
Alkaline Phosphatase	64.0	U/L	40.0 - 115.0		67.0	-5 %
Bilirubin,direct	0.2	mg/dL	0.0 - 0.2		0.3	-34 %
Bilirubin,total	0.9	mg/dL	0.2 - 1.2		0.5	80 %
LD	158.0	U/L	120 - 250		152.0	3 %
Cancer Screening						
PSA	0.92	ng/mL	0.0 - 4.0	< 1.0	1.36	-33 %
Telomere Length						
Lymphocyte Length	5.9	kb	4.5 - 9.0	> 8	6.4	-8 %
Granulocyte Length	6.5	kb	5.5 - 10.0	> 9	7.2	-10 %

‡ - range depends on a variety of factors

Name	Result	Units	Normal Range	Optimal Range	Baseline	Change
Skin Elasticity						
Skin Elasticity	53.38		30 - 95	> 80	51.64	3 %

‡ - range depends on a variety of factors

Historical Data

Visit Date	03 / 20 2007	05 / 23 2007	10 / 10 2007	01 / 29 2008	04 / 28 2008	10 / 28 2008	06 / 17 2009
Vital Signs							
Height	74.5	74.5	74.5	74.5	74.0	74.0	74.0
Weight	201.0	198.0	197.8	196.6	184.7	199.0	201.0
Arterial Stiffness							
SBP	126.0	135.0	109.0	110.0	127.0	109.0	130.0
DBP	72.0	86.0	68.0	66.0	67.0	68.0	76.0
Augmentation Pressure	13.0	12.0	12.0	11.0	11.0	10.0	8.0
Augmentation Index@75	16.0	19.0	16.0	15.0	17.0	19.0	12.0
Ejection Duration	322.0	313.0	301.0	285.0	373.0	348.0	357.0
Ejection Duration %	32.0	28.0	43.0	29.0	28.0	30.0	28.0
SEVR	172.0	215.0	198.0	207.0	202.0	196.0	203.0
Cardiovascular Risk							
Total Cholesterol	134.0	149.0	125.0	152.0	128.0	142.0	153.0
LDL Cholesterol	69.0	81.0	65.0	82.0	50.0	53.0	54.0
HDL Cholesterol	57.0	60.0	54.0	56.0	68.0	84.0	92.0
Cholesterol/HDL Ratio	2.35	2.48	2.31	2.7	1.9	1.7	1.7
Triglycerides	43.0	42.0	33.0	71.0	48.0	25.0	37.0
C-Reactive Protein	2.5	9.1	3.1	8.5	1.7	0.7	0.9
Homocysteine	10.07	8.88	7.97	6.8	7.8	7.8	7.0
Coenzyme Q10	1.0	2.0	1.0	2.0	2.0	1.0	1.0
Diabetes & Glucose							
Glucose	90.0	94.0	87.0	78.0	86.0	77.0	81.0
Insulin,serum	4.6	3.1	6.7	7.0	2.0	2.0	2.0
Hemoglobin A1C	5.5	5.5	5.4	5.5	5.4	5.5	5.5
Lung Function							
FVC	5.38	6.02	5.71	5.85	6.13	5.45	5.6
FEV1	4.41	4.48	4.5	4.5	4.4	4.27	4.38
FEV1/FVC	81.97	74.41	78.8	76.92	81.0	78.0	74.0
FEF25-75%	3.2	3.5	4.0	2.3	5.0	4.0	3.0
PEF	8.2	9.1	9.0	8.0	11.0	12.0	9.1
Cognitive Function							
Composite Memory	75.0	79.0	86.0	86.0	97.0	86.0	66.0
Verbal Memory	102.0	102.0	104.0	108.0	101.0	101.0	110.0
Visual Memory	106.0	100.0	103.0	120.0	100.0	100.0	75.0
Psychomotor Speed	61.0	91.0	101.0	81.0	73.0	82.0	97.0
Processing Speed	81.0	98.0	82.0	100.0	93.0	101.0	92.0

Visit Date	03 / 20 2007	05 / 23 2007	10 / 10 2007	01 / 29 2008	04 / 28 2008	10 / 28 2008	06 / 17 2009
Reaction Time	52.0	49.0	100.0	92.0	85.0	88.0	100.0
Cognitive Flexibility	77.0	92.0	89.0	85.0	99.0	92.0	98.0
Executive Functioning	75.0	89.0	102.0	93.0	101.0	81.0	75.0
Subject Composite Memory	102.0	103.0	105.0	105.0	111.0	105.0	100.0
Subject Verbal Memory	100.0	104.0	102.0	101.0	98.0	98.0	60.0
Subject Visual Memory	102.0	102.0	110.0	110.0	92.0	102.0	45.0
Subject Psychomotor Speed	168.0	186.0	168.0	178.0	174.0	179.0	197.0
Subject Processing Speed	50.0	55.0	55.0	48.0	51.0	57.0	62.0
Subject Reaction Time	800.0	750.0	679.0	695.0	678.0	686.0	695.0
Subject Cognitive Flexibility	52.0	45.0	52.0	47.0	50.0	49.0	52.0
Subject Executive Functioning	52.0	60.0	68.0	66.0	72.0	66.0	52.0
Immune Function							
WBC	4123.0	5154.0	3912.0	4521.0	4000.0	3289.0	3956.0
Neutrophils Percent	64.0	61.0	54.0	48.4	63.1	53.7	47.5
Neutrophils Count	1400.0	2230.0	2660.0	1694.0	2840.0	2041.0	1568.0
Monocytes Percent	6.0	9.0	8.0	9.0	6.0	8.0	7.0
Monocytes Count	256.0	250.0	300.0	291.0	284.0	289.0	290.0
Eosinophils Percent	3.0	4.0	4.0	3.9	1.4	2.3	1.8
Eosinophils Count	60.0	160.0	136.0	137.0	63.0	87.0	59.0
Basophils Percent	1.0	1.0	1.0	0.6	0.5	0.7	0.9
Basophils Count	21.0	19.0	22.0	21.0	23.0	27.0	30.0
Lymphocyte Percent	25.0	27.0	30.0	38.8	28.7	35.7	41.0
Lymphocyte Count	800.0	1400.0	1220.0	1358.0	1292.0	1357.0	1353.0
Lymphocytes							
NK Cell %	13.0	11.0	16.0	14.0	16.0	14.0	15.0
NK Cell Count	200.0	154.0	180.0	242.0	256.0	182.0	190.0
B-cells %	9.0	20.0	16.1	17.0	15.0	20.0	22.0
B-cell Count	200.0	281.0	206.0	130.0	207.0	286.0	200.0
T-cells %	71.0	69.0	70.0	75.0	69.0	69.0	68.0
T-Cell Count	981.0	1126.0	1441.0	1320.0	1098.0	945.0	1032.0
Helper T-Cells %	45.0	42.0	40.0	40.0	38.0	43.0	41.0
Suppressor T-Cells %	23.0	26.0	25.3	36.0	29.0	24.0	27.0
Suppressor T-Cell Count	259.0	365.0	324.0	468.0	382.0	298.0	369.0
Helper/Suppressor T-Cell Ratio	1.95	1.61	1.57	1.1	1.31	1.81	1.5
Healthy Suppressor T-Cells %	44.0	42.0	41.0	41.0	42.0	48.0	39.0
Healthy Suppressor T-Cell Count	61.0	109.0	-16.0	115.0	96.0	113.0	180.0
Senescent Suppressor Cells %	56.0	58.0	59.0	59.0	58.0	52.0	61.0
Senescent Suppressor Cell Count	198.0	256.0	340.0	353.0	286.0	185.0	189.0
Naive Suppressor Cells %	6.0	11.0	14.0	18.0	10.0	14.0	19.0
Naive Suppressor Cell Count	48.0	42.0	46.0	66.0	49.0	50.0	100.0
CMV AB (IGG)	0.14	0.18	0.25	0.16	0.12	0.15	0.24

Visit Date	03 / 20 2007	05 / 23 2007	10 / 10 2007	01 / 29 2008	04 / 28 2008	10 / 28 2008	06 / 17 2009
Hormones							
IGF-1	229.0	221.0	267.0	295.0	156.0	274.0	248.0
IGF Binding Protein-3	4.0	4.66	4.51	5.0	4.4	4.7	4.6
Sex Hormone Binding Globulin	20.6	20.0	21.7	26.0	32.0	21.0	25.0
DHEA Sulfate	113.0	70.0	136.0	103.0	124.0	126.0	138.0
Estrone Sulfate	522.0	321.0	442.0	333.0	425.0	460.0	412.0
Total Testosterone	1374.0	1185.0	952.0	235.0	248.0	865.0	1314.0
Free Testosterone	42.4	34.4	251.0	34.7	29.8	235.3	351.5
Free Testosterone %	1.7	2.1	2.2	1.5	1.2	2.7	2.7
Estradiol	65.63	84.94	34.59	32.0	50.0	32.0	32.0
LH	0.1	0.2	0.1	2.1	1.7	1.6	1.1
TSH	1.6	1.44	1.3	1.84	0.66	1.13	0.89
T4	7.0	7.6	8.3	8.0	7.5	6.6	7.2
Free T3	340.0	320.0	320.0	330.0	260.0	348.0	347.0
Total T3	117.0	97.0	105.0	154.0	108.0	132.0	114.0
Blood							
RBC	5.8	5.7	5.6	5.03	4.6	4.75	4.73
Hematocrit	52.4	52.2	49.1	45.3	42.0	42.7	42.9
Hemoglobin	17.0	16.8	17.0	15.5	14.5	14.7	14.8
MCH	29.2	29.7	30.5	30.8	31.5	30.9	31.3
MCHC	32.4	32.2	34.6	34.2	34.4	34.4	34.5
MCV	90.0	92.4	88.2	90.2	91.4	89.8	90.8
RDW	14.0	14.1	13.1	12.9	13.2	13.2	13.8
Platelet Count	191.0	198.0	185.0	161.0	197.0	161.0	172.0
MPV	8.0	7.0	7.0	7.6	8.6	8.5	8.0
Vitamins							
Iron	85.0	115.0	114.0	113.0	109.0	170.0	129.0
TIBC	290.0	296.0	285.0	331.0	277.0	332.0	359.0
Transferrin Saturation	29.0	39.0	40.0	34.0	39.0	51.0	36.0
Ferritin	52.0	60.0	60.0	76.0	28.0	57.0	60.0
Folate	8.5	20.0	17.4	23.3	24.0	18.7	24.0
Vitamin B12	1889.0	1358.0	1445.0	1527.0	1539.0	1141.0	1298.0
Vitamin D	23.5	56.1	49.2	44.0	40.0	37.0	30.0
Vitamin D2	3.5	5.1	3.2	2.0	0.0	0.0	0.0
Vitamin D3	20.0	51.0	46.0	42.0	40.0	37.0	30.0
Kidney Function							
Sodium	139.0	139.0	138.0	139.0	141.0	143.0	141.0
Potassium	3.9	4.2	4.0	3.9	4.8	4.3	4.3
Chloride	104.0	101.0	102.0	103.0	103.0	107.0	102.0
CO2 Carbon Dioxide	24.0	28.0	25.0	21.0	23.0	25.0	27.0
Calcium	9.1	9.4	9.3	9.1	9.8	9.5	9.6

Visit Date	03 / 20 2007	05 / 23 2007	10 / 10 2007	01 / 29 2008	04 / 28 2008	10 / 28 2008	06 / 17 2009
Phosphorus	2.9	3.4	3.6	3.1	2.9	3.7	3.7
Uric Acid	4.5	4.2	4.5	5.3	4.2	5.4	5.2
Creatinine	1.0	1.1	1.1	0.9	1.18	1.0	1.03
Urea Nitrogen	22.0	22.0	18.0	18.0	32.0	24.0	17.0
BUN/Creatinine Ratio	22.0	20.0	16.36	20.0	27.11	24.0	16.5
Liver Function							
A/G Ratio	1.3	1.3	1.9	1.3	1.8	1.8	1.6
Albumin	3.8	4.0	4.5	4.4	4.6	4.3	4.5
Globulin	2.9	3.0	2.4	3.4	2.6	2.4	2.8
Total Protein	6.7	7.0	6.9	7.8	7.2	6.7	7.3
GGT	22.0	22.0	17.0	28.0	19.0	19.0	17.0
ALT	42.0	97.0	26.0	44.0	28.0	36.0	37.0
AST	31.0	66.0	24.0	28.0	30.0	34.0	33.0
Alkaline Phosphatase	67.0	74.0	71.0	97.0	65.0	60.0	64.0
Bilirubin,direct	0.3	0.2	0.3	0.1	0.2	0.2	0.2
Bilirubin,total	0.5	0.6	0.8	0.6	0.6	0.9	0.9
LD	152.0	228.0	130.0	148.0	156.0	160.0	158.0
Cancer Screening							
PSA	1.36	1.81	1.48	0.69	0.88	0.89	0.92
Telomere Length							
Lymphocyte Length	6.4	6.5	6.5	6.6	6.2	6.3	5.9
Granulocyte Length	7.2	7.3	7.3	7.2	6.7	6.8	6.5
Skin Elasticity							
Skin Elasticity	51.64	54.76	51.46	52.57	53.46	53.04	53.38

Disclaimer

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