The Track Your Plaque

Guide to At-Home Laboratory Testing

The Track Your Plaque Guide to At-Home Laboratory Testing is meant help the user understand the significance of the blood and salivary testing provided through the Track Your Plaque program. It is not meant to diagnose any condition, nor is it intended to provide a treatment program; this can only be provided by a healthcare provider familiar with your health situation.

For “normal” or “reference range” values for each test, please refer to the “Reference Range” cited with the return of your test results.

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Cardiometabolic Profile (Total cholesterol, LDL cholesterol, triglycerides, c-reactive protein, HbA1C)
Blood spot only

LDL cholesterol

LDL cholesterol is meant to reflect the amount of cholesterol present in the low-density fraction of lipoprotein blood particles (as opposed to those in the high-density fraction, or HDL). It is the basis for most conventional predictions of heart disease risk, since it has been statistically connected to occurrence of heart attack and is recoverable from atherosclerotic plaque when examined.

LDL is often called "bad" cholesterol, since the higher the LDL, the greater the likelihood of cardiovascular events like heart attack, an observation documented repeatedly from the Framingham Study to other populations (Kannel WB et al 1979; Kannel WB 1995; ATP-III 2001). Despite the controversies the drug industry has created by its overenthusiastic marketing of the LDL-reducing statin drugs, reduction of LDL cholesterol, whether with statin drugs, diet, fibers like oat bran or ground flaxseed, or other strategies has been confidently tied to reduction in heart attack (Selwyn AP 2007).

In the Track Your Plaque program, we aim for an LDL of 60 mg/dl or less, a level consistent with maximal reduction of cardiovascular risk and coronary plaque (Ballantyne CM et al 2008).

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Triglycerides

The liver produces a class of particles called “very low-density lipoproteins,” or VLDL, comprised principally of triglycerides. VLDL production is fueled by poor insulin responses triggered by sedentary life, obesity, and over-reliance on foods that increase blood sugar (Adiels M et al 2008). Increased triglycerides (and VLDL) signal increased risk for heart disease (McBride P 2008).

Some people have high triglycerides due to genetic factors (such as a deficient form of the enzyme, lipoprotein lipase, that clears triglycerides from the blood). In this situation, triglycerides can range as high as *several thousand* mg/dl. Far more commonly, triglycerides are high (100 mg/dl to 500 mg/dl) due to excess weight, indulgence in processed carbohydrates, and resistance to insulin (metabolic syndrome), the very same triggers for VLDL.

When VLDL particles in the blood come into contact with LDL and HDL particles, triglycerides from VLDL are shared with LDL and HDL particles, which also become bloated with triglycerides as a result. Triglyceride-loaded LDL and HDL are a ready target for enzymes in the blood and liver that reconfigure them into smaller versions, small LDL and small HDL. Small LDL and HDL are undesirable particles that powerfully stimulate growth of atherosclerotic plaque in the heart’s arteries and elsewhere. Thus, excess triglycerides and VLDL lurk behind the creation of small LDL and small HDL (Berneis KK, Krauss RM 2002). This process *begins* at a triglyceride level as low as 45 mg/dl, becomes progressively worse with increasing levels of triglycerides, substantial with triglycerides >150 mg/dl.

Increased triglycerides are also a feature of the metabolic syndrome, or pre-diabetes, and diabetes (Adiels M et al 2008). Increased triglycerides from VLDL, along with low HDL and small LDL are very common. This is because the poor insulin responses in these conditions cause the liver to produce VLDL particles without restraint (Verges B 2005). The result: a doubling or tripling of the risk for heart attack (Depres JP et al 2008).

In the Track Your Plaque program, we aim to maintain triglycerides at 60 mg/dl or less, a level that is associated with maximal suppression of triglyceride-containing lipoproteins (Otvos J 1999).

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C-reactive protein

C-reactive protein (CRP) has emerged as a practical measure of low-grade, hidden inflammation. The new “high-sensitivity” method of measure permits detection of inflammation at levels below the threshold of perception. Studies have shown that the higher your CRP, the brighter the flame of inflammation is burning in the body, and the greater your risk for various illnesses, including cancer, diabetes, glaucoma, macular degeneration, carotid disease, depression, and dementia (Il’yasova D et al 2005; Bluher M et al 2005; Toker S 2005).

Active inflammatory blood cells anywhere in the body generate increased levels of the signaling protein, IL-6. Excess fat cells also cause increased IL-6. IL-6, in turn, activates liver production of CRP.

If there’s an obvious source of inflammation like pneumonia, flu, or a healing wound, then CRP can be dramatically elevated, often 100 mg/l or higher. However, in the absence of active, overt inflammation, levels from 0.5 to 10 mg/l can indicate that inflammation is smoldering somewhere in the body and risk for heart attack increases two- or three-fold, regardless of LDL cholesterol levels (Pearson TS et al 2003). When high CRP occurs in the company of small LDL particle size, heart attack risk is *7-fold* greater (St-Pierre AC et al 2003). The Track Your Plaque program is designed to keep CRP at low levels below 0.5 mg/L.

References:

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Hemoglobin A1C (HbA1C)

Blood sugar (glucose) modifies proteins in the bloodstream, a process called "glycosylation." One of the proteins susceptible to glycosylation is hemoglobin contained in red blood cells. If blood sugar is increased, there will be an increased percentage of glycosylated hemoglobin within red blood cells, also known as hemoglobin A1C, or HbA1C.

Because the lifespan of a red blood cell is approximately 120 days, HbA1c provides an assessment of blood glucose over the same period. (Any condition that modifies the lifespan of red blood cells, such as some forms of anemia, can therefore alter the HbA1C; HBA1C must then be interpreted with caution.)

HbA1C can be used to calculate an "Average Glucose" (AG) for the preceding 120-day period (Nathan DM et al 2008):

$$\mathbf{AG_{mg/dl} = 28.7 \times A1C - 46.7}$$

The Track Your Plaque nutritional program is designed to help maintain HbA1C at 5.0% or less, corresponding to an average glucose of 96.8 mg/dl or less.

References:

Nathan DM, Kuenen J, Borg R et al. Translating the A1C assay into estimated average glucose values. Diabetes Care 2008 August;31(8):1473-78.

Cortisol
Blood spot or salivary

The human adrenal gland produces approximately 10 mg of cortisol per day with a predictable circadian pattern. The first daily surge occurs during the first hour of wakefulness, followed by several lesser surges during the course of the day; lowest levels occur in early sleep at 12 am to 3 am. Stressful situations can also trigger cortisol surges, regardless of time of day. Depression, anxiety, caregiver stress, unemployment and other stress life situations can chronically increase cortisol levels. Increased levels of evening cortisol can be associated with unhappy marriage, depression, and social isolation (McEwen BS 1998).

Cortisol influences a multitude of body processes, though its major effect is to maintain fluid and blood pressure. Severe deficiency of cortisol is called Addison's disease and is marked by severe dehydration and drops in blood pressure (Debono M et al 2009).

Cortisol is also at the center of a heated debate. At one end are proponents of the idea that stress, sleep deprivation, and unhealthy diets all lead to increased levels of cortisol, the "hormone of stress." Others argue that the cortisol stress response is a relatively short-lived phenomenon followed by a chronic lack of cortisol, called "adrenal fatigue." Proponents of this concept site the following symptoms as potentially due to adrenal fatigue:

1. Brain fog, mental cloudiness, depression
2. Low thyroid function
3. Low blood sugar
4. Morning and mid-afternoon fatigue
5. Fragmented sleep
6. Low blood pressure
7. Impaired immune function
8. Salt cravings

Both situations can be explored through assessment of cortisol levels. Because of the circadian variation in cortisol levels, 2-4 cortisol values at different times of the day are advised by authorities.

Because obtaining several blood measurements through the course of the day is difficult, salivary cortisol assessment has proven to be a useful means to assess repeated levels (Castro M et al 2000). Salivary cortisol levels provide a measure that correlates best with free, or unbound, cortisol blood levels.

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DHEA-S

Blood spot or salivary

Dehydroepiandrosterone, or DHEA, is a hormone secreted by the adrenal gland in large quantities during our 20s and 30s, followed by a decline of approximately 20% per decade. By age 70, DHEA blood levels in both men and women are at <25% of youthful levels (Kroboth PD et al 1999).

DHEA occurs principally as the sulfated derivative, DHEA-S, in the blood. DHEA-S is the form usually measured to assess DHEA status.

DHEA administration to adults over age 40 has been shown to enhance both physical vigor and emotional well-being (Wolkowitz OM 1999). Beyond the increase in DHEA blood levels, DHEA supplementation increases testosterone and triggers a modest rise in effective growth hormone levels (Morales 1997). Most studies documenting physical and emotional benefits of DHEA have examined doses between 10 and 50 mg per day, the quantity required to restore youthful DHEA-S blood levels in the majority of people.

Over the past 40 years, DHEA has been studied in a number of applications, some successful, some not. Among the successes DHEA has yielded:

- DHEA may slow progression of osteoporosis in postmenopausal women (Baulieu EE et al 2000), and increase bone density (Labrie F et al 1997).
- DHEA improves sexual dysfunction and libido in women over age 60 (Baulieu EE et al 2000).
- DHEA may improve erectile dysfunction in men who have low DHEA levels (Reiter WJ et al 2001).
- DHEA alleviates depressive symptoms such as inability to cope, worrying, lack of motivation, and sadness (Bloch M et al 1999).
- DHEA improves symptoms of chronic fatigue syndrome (Cleare AJ 2003).
- In people with adrenal failure whose adrenal glands produce little or no DHEA, DHEA replacement (50 mg per day) improves mood, physical energy and performance, and bone density when used along with standard hormone replacement therapy (e.g., cortisol; Arlt W et al 1999).

Interestingly, most studies of DHEA in athletic performance have raised questions over its benefits for this purpose, but DHEA remains banned in Olympic athletes.

Beyond increasing DHEA and DHEA-S levels, DHEA supplementation increases testosterone and estradiol modestly in females, with little to no change in males. DHEA has little effect on progesterone (Stomati 2000).

Though difficult to quantify, DHEA replacement helps many people feel better: greater physical stamina, brighter outlook, more “get up and go.” Problems seem less overwhelming and “lows” tend not to be as low. Though there are clinical data to support these “soft” effects, they are inconsistent. In our experience, people who start out with sluggishness, low energy, and a negative outlook, accompanied by a low DHEA-S level, are the most likely to experience positive results with DHEA replacement.

Occasionally, DHEA supplementation will bring out aggressive behavior, such as short-temperedness, intolerance, and impatience. A reduction in dose usually resolves this issue (e.g., reduce from 50 mg to 25 mg per day).

Beyond enhanced energy and stamina, in the Track Your Plaque program we monitor DHEA levels when DHEA supplementation is used to reduce lipoprotein(a).

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Estrogens

Blood spot or salivary

There are three principal human estrogens: estradiol, estrone, and estriol. In addition to being responsible for female characteristics such as breast development, female brain development, and maturation of the female reproductive organs, estrogens also accelerate metabolism, increase bone density, and affect vascular health.

Assessment of estrogen levels become most important in the perimenopausal and menopausal periods in females, when estrogen levels fall substantially, resulting in many (though not all; see Progesterone) of the phenomena of menopause, including weight gain, hot flashes, vaginal dryness, and bone thinning (demineralization; NIH Consensus Statement 2005).

Estrogen assessment can be important in males, also, especially during the period during and after the “andropause,” the period in males after age 40, when testosterone levels begin to decline; increased estrogens can therefore exert outsized effects as testosterone effects recede (Amore M et al 2009).

Estrogen replacement in menopausal females is at the center of a controversy. Large-scale clinical studies, such as HERS and the Women’s Health Initiative, employed *non-human* mixtures of estrogens (ACOG Committee Opinion 2008); large studies using human estrogens (and progesterone) have not been performed, though a vocal and anecdotal grassroots effort have made a strong case for the superiority of so-called “bio-identical” hormone use (Holtorf K 2009).

References

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Insulin

Blood spot only

The fundamental defect in metabolic syndrome, pre-diabetes, and type II diabetes is poor responsiveness to insulin, often called “insulin resistance.” Because the pancreas attempts to maintain blood sugars within a narrow normal range, insulin output increases to overcome the “resistance” to insulin’s effects. Blood level of insulin therefore increases (Lann D et al 2008).

Fasting insulin provides a means to assess the body’s responsiveness or resistance to insulin. Low levels signal normal insulin responsiveness; high levels signal reduced insulin responsiveness. However, if insulin resistance has been present for an extended period, usually several years, then the pancreas loses its capacity to manufacture insulin and insulin blood levels will drop (and may be indistinguishable from normal); at this point, blood sugars will increase above the normal range. Pre-diabetes or diabetes then develops.

Higher insulin levels have been shown to correlate with greater likelihood of progression of heart scan (coronary calcium) scores (Lee KK et al 2009). Increased blood levels of insulin have also been associated with 12% greater risk for cardiovascular events (Sarwar N et al 2007).

In the Track Your Plaque program, we maintain fasting insulin levels at 10 mIU or less.

References

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Progesterone
Blood spot or salivary

Progesterone is the hormone responsible for many uniquely female characteristics. Some call progesterone the “forgotten female hormone,” lost in the shadow of better-known estrogen. As you age, like estrogen, progesterone levels decrease.

Progesterone levels are 10–100 fold greater during pregnancy. Progesterone is the *progestational* hormone, so named because it helps prepare the uterus and associated organs for pregnancy. The “blue” feeling that follows delivery that many women experience, sometimes severe enough to be labeled “postpartum depression,” is suspected to be due to the abrupt 95% drop in progesterone after delivery.

Progesterone levels begin to decline as early as the late 30s with an abrupt drop through the menopause. Symptoms associated with declining progesterone include difficulty sleeping through the night, poor energy, foggy thinking, and irritability. Many women also experience bloating and weight gain. As a rule, all women in the menopausal period have very low levels of progesterone; levels during the transitional years towards menopause, “perimenopause,” can vary from woman to woman.

As with estrogens, clinical studies have employed synthetic forms of progesterone, “progestins,” which have been associated with *increased* cardiovascular risk. Many authorities speculate that natural human, “bio-identical,” progesterone does not share the adverse effects of progestin, though outcome data are lacking (Hermsmeyer RK et al 2008; L’hermite M et al 2008). Anecdotal experience with progesterone has suggested that the natural human form is superior for providing relief of symptoms of progesterone deficiency.

References

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Prostate specific antigen (PSA)

Blood spot only

PSA is a protein produced by the prostate gland in men and detectable in the blood. It is typically measured as a screening test for prostate cancer. It is also used to track results after prostate removal for cancer.

One limitation of PSA testing is that other conditions can influence PSA levels, including factors that reduce PSA, such as obesity and anti-inflammatory agents (aspirin, ibuprofen, etc.), and those that increase PSA, such as prostate inflammation or infection (prostatitis) and benign enlargement of the prostate ("benign prostatic hypertrophy," or BPH). For this reason, most authorities recommend repeating a suspicious level to confirm before action is taken.

References

Fleshner NE, Lawrentschuk N. Risk of developing prostate cancer in the future: overview of prognostic biomarkers. Urology 2009 May;73(5 Suppl):S21-7.

Sex Hormone-Binding Globulin (SHBG)

Blood spot only

SHBG is the blood protein carrier that transports sex hormones, estrogen and testosterone. The fraction of estrogen or testosterone carried by SHBG is called the "bound" fraction and serves as a repository for hormone storage. The fraction of estrogen or testosterone not carried on SHBG is called "unbound" or "free;" this represents the active fraction and is generally no more than 1-2% of total hormone present.

An increase in the pool of SHBG can effectively decrease the free fraction of hormone; conversely, a decrease in SHBG frees up more hormone. Thus, SHBG levels influence the amount of free hormone available. SHBG levels are increased (thereby reducing available hormone) in situations including hyperthyroidism (overactive thyroid), liver disease, and calorie deprivation. SHBG levels are decreased (thereby increasing available hormone) in hypothyroidism (underactive thyroid) and obesity.

However, fluctuations in SHBG levels are not the only determinant of free hormone levels. For this reason, a gauge of free or "bioavailable" testosterone can be obtained by calculating the Free Androgen Index (FAI). The Free Androgen Index (FAI) is calculated as follows:

$$FAI = \text{Total Testosterone} / SHBG$$

(FAI is sometimes modified by a “correction factor,” e.g., multiply by 100.)

Lower SHBG and higher FAI values in females may serve as markers for increased coronary risk (Lambrinoudaki I et al 2006;Sutton-Tyrrell K et al 2005). In both males and females, higher FAI correlates with features of the metabolic syndrome (Pugeat M et al 1995).

References:

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Testosterone

Blood spot or salivary

Testosterone levels peak in a male’s teens and twenties during their reproductive prime. That’s also the period of a man’s greatest physical capacity, muscle mass, physical energy, libido, and stamina.

Starting at age 30, testosterone levels diminish gradually. By the time a man reaches his 70s, testosterone has dropped to low levels. Diminishing testosterone levels lead to loss of muscle mass, increased body fat, and reduced libido. Mood disruptions are prominent, with deeper swings into blue, depressed feelings, struggles with feeling beaten and overwhelmed, and fatigue. Reduced concentration, irritability, passivity, loss of interest in activities, and even hypochondria can also result.

These changes become perceptible after a man passes beyond his mid-40’s. Some call this time the “male menopause” or “andropause.” Though not as visible as a woman’s transition to menopause since there’s no particular external cue like cessation of menses, most men simply dismiss the changes as “getting old.”

Studies have shown that the lower the starting blood testosterone level, the greater the benefits of testosterone replacement. With rare exceptions, few men before age 40 will benefit from testosterone, as they maintain healthy levels.

The rate of testosterone decline varies from one male to another. One 50-year old man, for instance, might have a blood level of 390 pg/ml, and another 50-year old could have a level of 50 pg/ml. The second man enjoys greater benefits because of the lower starting value.

From a heart health standpoint, potential benefits of testosterone replacement in men with lower starting levels include:

- Reduction in vascular tone and endothelial dysfunction—Testosterone increases production of the natural arterial dilator, nitric oxide, and suppresses growth of smooth muscle cells in arteries (a constituent of plaque; Khalil RA 2005).
- Improvement in abnormal resistance to insulin—The essential phenomenon behind pre-diabetes and metabolic syndrome (Marin P et al 1992; Simon D et al 2001).
- Reduction in inflammatory proteins—Levels of tumor necrosis factor and interleukins, in particular, are considerably reduced (Malkin CJ et al 2004).

Men with coronary disease have been shown to be more likely to have low testosterone levels. In one study, a marked deficiency of testosterone was found in 25% of men with overt coronary disease (e.g., history of heart attack or procedures; Malkin CJ et al 2004).

Most males obtain optimal results with testosterone replacement—enhanced well-being, physical stamina, and libido—with restoration of blood testosterone to levels that reproduce the level most men have in their mid- to late-30s.

An assessment of total testosterone blood levels provides a starting point for assessing testosterone status. A more in-depth assessment can be made by adding free testosterone, bioavailable testosterone, and dihydrotestosterone (not yet available through blood spot testing). A Free Androgen index can be calculated if a SHBG level is available (see SHBG, above).

If feelings of sadness, bloating, and weight gain are present, an estradiol level might considered. Estradiol is the form of estrogen that can be increased, particularly in overweight men and increases risk for heart disease. Weight loss can correct elevated estradiol, as can prescription “aromatase inhibitors,” such as Arimidex®. A nutritional supplement, chrysin (usual dose, 1000 mg per day), has been shown to reduce estradiol levels; however, there’s little supportive data documenting its effectiveness and safety.

References

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Thyroid testing:
TSH, free T3, free T4, antithyroglobulin antibody
Blood spot only

The thyroid gland is a butterfly-shaped gland located on the front of the neck just beneath the surface of the skin. The thyroid produces thyroid hormones:

T3 (triiodothyronine), the most active hormone. The thyroid produces no more than 20% of bodily T3 requirements; most T3 develops from conversion of T4 to T3 that occurs in other tissues. The preferred method to assess T3 status is to measure the free, unbound fraction, called *free T3*.

T4 (tetraiodothyronine or thyroxine)—80% of hormone produced by the thyroid is T4. T4 is converted to the active form, T3, via the action of deiodinase enzymes that remove one iodine atom. The preferred method to assess T4 status is to measure the free, unbound fraction, called *free T4*.

TSH (Thyroid Stimulating Hormone or thyrotropin) is the pituitary gland hormone that signals the thyroid to produce thyroid hormones and maintain T4 and T3 levels. If thyroid hormone levels are low, TSH will *increase* in an effort to increase thyroid hormone production; if thyroid hormone levels are high, TSH will *decrease*. In the 25,000-participant HUNT Study, cardiovascular mortality began to increase with TSH of 1.4 mIU, with 70% increased (relative) risk with TSH of 2.5-3.5 mIU (Åsvold BO et al 2008).

Thyroid Peroxidase Antibody (TPO Ab): Thyroid peroxidase (TPO) is an enzyme involved in production of thyroid hormones. Autoimmune conditions result in antibodies targeting TPO. Approximately 90% of sufferers of Hashimoto's thyroiditis will test positive for elevated TPO antibodies (Carlé A et al 2006).

Free T3, free T4, and TSH are the basic lab tests usually obtained when an assessment of thyroid function is required. Antibody assessments, such as thyroid peroxidase antibody, can be added if the question of an inflammatory condition of the thyroid is suspected.

Hypothyroidism, in which thyroid production of T3 and T4 falls below the body's needs, is by far the most common abnormal condition affecting the thyroid. Hypothyroidism can trigger numerous symptoms:

- Reduced energy, fatigue, increased sleep
- Feeling inappropriately cold; reduced sweating
- Dry, itchy skin

- Dry, brittle, thinning hair
- Weight gain without apparent cause or more-than-usual difficulty losing weight
- Impaired short-term memory, slower thinking
- Muscle cramps, joint aches
- Constipation
- Puffiness or swelling around the eyes, hands, ankles, and feet
- Heavier and/or more frequent menstrual periods, worse premenstrual symptoms
- Depression, sadness, apathy
- Abnormally slow heart rate (<60 beats per minute)
- Iron deficiency anemia, low ferritin (an iron storage protein)

Symptoms can be vague, often not present at all. Levels of thyroid hormones, free T3 and T4, along with pituitary hormone, TSH, are therefore used to confirm the diagnosis. Low body temperature (oral temperature below 97.3° F immediately upon arising) can also suggest low thyroid.

Excessive levels of thyroid hormone, hyperthyroidism, can also occur, though much less commonly. Hyperthyroidism can account for anxiousness, unexplained weight loss, muscle weakness, and fast heart rate. Hyperthyroidism is generally caused by inflammation of the thyroid gland, such as Hashimoto's thyroiditis or Grave's autoimmune thyroiditis.

While most laboratories cite a TSH range of 0.5-5.5 mIU as normal, in the Track Your Plaque program, we aim for TSH less than 1.0 mIU. We also aim to keep free T4 and free T3 in the upper half of the reference range.

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Vitamin D: 25-hydroxy vitamin D
Blood spot only

Vitamin D is proving to be among the most exciting phenomena in health uncovered in the past 50 years. This everyday vitamin, ignored for years as just something we got from milk, packs health benefits that have far exceeded anyone's expectations.

Among the fascinating effects recently identified:

- Blood pressure—People deficient in vitamin D are more likely to have high blood pressure; vitamin D supplementation reduces blood pressure with the same effectiveness as prescription medication. One study demonstrated 10–20 mmHg drop in blood pressure in men with pre-diabetes when vitamin D was supplemented (Pfeifer M et al 2001; Lind L et al 1995).
- Anti-cancer effects—Epidemiologic studies demonstrate reduced likelihood of cancer, particularly breast, prostate and colorectal cancer, in people with higher blood levels of vitamin D (Vieth R 1999).
- Anti-inflammatory effects—Recent studies demonstrate reduced inflammation (C-reactive protein, CRP, and matrix metalloproteinase, MMP). Reductions in CRP of 60% or more have been documented (Timms PM et al 2002).
- Anti-diabetic effects—Several studies have shown that vitamin D administration reduces blood sugar and increases sensitivity to insulin. Improvement in insulin sensitivity triggers a cascade of benefits, including beneficial effects on lipoproteins (reduced triglycerides, increased HDL) (Zitterman A 2006).
- Osteoporosis prevention—Vitamin D is sorely neglected in this area. Replacement to healthy levels substantially increases bone density more effectively than calcium supplementation. Intestinal absorption of calcium more than doubles when sufficient vitamin D is present (Holick MF 2006).

New studies are showing that the dose required to achieve a healthy blood level of vitamin D in most adults is around 4000 units per day in the absence of sun exposure (Vieth R et al 2001). That's *10 times* the recommended Institute of Medicine's Adequate Intake.

People from the northern U.S. (Massachusetts, New York, Pennsylvania, Wisconsin, Michigan, the Dakotas, etc.), Canada, or northern Europe, are likely to be deficient, though many people from southern climates are also deficient. If you're like most Americans, you get sun sporadically in summer (weekends) and virtually none from September to April. Dark-skinned races are at greater risk of vitamin D deficiency, since melanin pigment in skin acts as a natural sun screen.

Dark-skinned individuals require five times longer sun exposure to obtain the same amount of vitamin D as a fair-skinned person. African-Americans, for this reason, are among the most vitamin D deficient of all.

The only way to know your vitamin D status is to measure the blood level of **25-hydroxy vitamin D** (not to be confused with 1,25-di-OH-vitamin D, a related metabolite that reflects kidney function). While some authorities argue that a minimum 25-OH-vitamin D3 level of 30 ng/ml, or 75 nmol/l, be achieved (Vieth R et al 2001), in the Track Your Plaque program we aim for a blood level of 60-70 ng/ml (150-175 nmol/L).

In young people, 15 minutes of sun exposure in midday, wearing shorts and t-shirt to expose skin surface area, may provide sufficient vitamin D. The ability to activate vitamin D in the skin is lost as we age, such that a dark tan in our 60s and 70s can conceal severe vitamin D deficiency. Vitamin D supplementation is therefore required as we age.

Personally, I take 5,000 units in the late fall, winter, and early spring, and then I vary doses the rest of the time depending on sun exposure. I also have my 25(OH)D level checked twice a year, once in the early spring and again in the early fall. My 10 year-old daughter takes 2,000 units a day in the winter months and my three-year-old takes 1,000 units a day in the winter.

Dr. John Cannell
The Vitamin D Council
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Anyone with kidney disease, cancer, glandular disorders, or a history of high calcium blood levels should only take vitamin D with medical supervision.

References:

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