Cholesterol & Statins: Myths & Madness Exposed

If you have been put on a cholesterol-lowering drug (also called a 'statin') by your doctor, the information you are about to read is critical to your long-term health.

In this paper, we will explain what cholesterol really is, why statins don't benefit 99% of the people taking them, and the long-term dangers you expose yourself to by taking them. We will also show you the real cause behind rising cholesterol levels and a way of managing your cholesterol that improves your health instead of destroying it.

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What exactly is Cholesterol?

Cholesterol is a unique molecule. There's really no other way to describe it. It is somewhat like a lipid (fat), a steroid, or an alcohol, but it is none of these things.

What most people have been manipulated to believe at this point is that cholesterol is "The Enemy." By now, everyone has seen a thousand commercials on TV for various cholesterol-lowering medications. Cholesterol has become the focus of huge advertising campaigns to make us all "Ask your Doctor if _____ might be right for you." The aisles of our grocery stores are now filled with products proclaiming themselves to be "Fat Free! - Cholesterol Free!" So, obviously, cholesterol is the enemy, right?

Wrong. Without cholesterol, we would not be alive - period. **It is part of the structure of every cell in our bodies**. Without it, we would literally fall apart. Cholesterol is one of the elements used by our body's miraculous ability to repair itself. Without cholesterol, every injury would be permanent.
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It is also major component of the myelin sheath around every neuron in our nervous systems. Without this 'insulation' of our nervous system's wiring, our brains and bodies couldn't function.

So, how did "Cholesterol is the Enemy" thinking get started? Let's take a look. In 1908, Russian scientist M.A. Ignatovsky fed high fat, meat-based food to a group of rabbits. He soon discovered that the rabbits developed arterial plaques and cardiovascular disease. Researchers discovered that the same thing happens when chickens, guinea pigs and goats eat a high-fat diet. These studies were later cited as evidence that a high-fat diet causes heart disease. **Never mind that all of the animals tested were strict herbivores (plant-eaters).**

In the 1950s, physiologist Ancel Keys, Ph.D., published what became known as the Seven Countries Study. Keys presented a comparison of heart disease mortality and fat intake across seven different countries. His comparison showed a "remarkable relationship." The countries with the highest fat intake had the highest levels of heart disease. The countries with the lowest fat intake had the lowest levels of heart disease. Those in the middle fell conveniently in between. That makes it obvious, right?

At the time, Jacob Yerushalmy, a PhD statistician at the University of California at Berkeley pointed out that we had fat consumption data on **22** countries. So why wasn't it called the 22 Country Study? It wasn't called that because it would seem that Dr. Keys started with the conclusion, included the countries that matched his pre-conceived notion and threw out the ones that contradicted it. **When all 22 countries were analyzed, the "remarkable relationship" remarkably disappeared**. And yet somehow, Dr. Keys' theories became the commonly held belief.

Why? I can only assume that Dr. Keys' paper got the most press. It fits perfectly into social critic H.L. Mencken's observation that **"For every complicated problem there is a solution that is simple, direct, understandable and *wrong*."**

It was right about this time (the 1950's) that the assumption that "eating fats raises your cholesterol levels" got added to the equation. The hypothesis became "Fat Consumption = High Cholesterol = Heart Disease." That turned out to be a false conclusion as well.

Surely more recent studies have proven the connection between fats, cholesterol and heart disease, right? Judge for yourself. The Women's Health Initiative of the 90's was a huge government study, costing almost three quarters of a billion dollars. **Among 20,000**

women in the study who adhered to a low saturated fat diet for eight years, there was no reduction in the rates of heart disease or stroke. Just recently, the *American Journal of Clinical Nutrition* published a review of 21 studies. The studies ranged from 5 to 23 years in length and encompassed 347,747 subjects. In the authors' own words: **"Intake of saturated fat was not associated with an increased risk of coronary heart disease, stroke, or cardiovascular disease."**

So if cholesterol isn't "The Enemy", what is it? Cholesterol is, in fact, a very versatile raw material that your body uses to create many different things. In addition to building every cell wall and the 'insulation' on your nervous systems, cholesterol is the compound that most of your hormones are made from.

Your body produces 85-90% of the cholesterol you need through its own processes in your liver and you get the rest from what you eat. **Your body makes its own cholesterol because it needs it to survive**.

While we're at it, let's take a look at the current way we measure cholesterol. **The number you've been given as your cholesterol level is a guess – no more.**

Cholesterol tests don't actually measure cholesterol. I'll bet that's a bit of a surprise to everyone outside of the medical business, isn't it? The number is created by measuring HDL (high density lipoproteins) and LDL (low density lipoproteins) and triglycerides. (circulating fats n your bloodstream) You'll notice that none of these elements are actually cholesterol! They are fats and proteins.

Lipoproteins are simply the cholesterol "carriers" that your body uses to move cholesterol around to where it is needed (LDL) and to bring it back to the liver for processing (HDL) when it is unneeded. But as Dr. Dzugan says in his lectures, these "carriers" are like UPS and USPS trucks. Counting the number of brown trucks and white trucks on the highway doesn't give you any details about what they have in the back. That makes the whole "good cholesterol/bad cholesterol" conversation a little silly, doesn't it? There is no good or bad cholesterol – there is simply cholesterol.

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What are statins and what do they do to you?
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Statins are pharmaceutical company inventions that interfere with the body's natural production of cholesterol in the liver. As such, these chemicals can be very effective at lowering a person's overall cholesterol level by cutting off the source of supply.
Statins actually interfere with quite a few other processes in your body as well - as the side effects will show.

The question you really want answered is **"What do they do to me?"** The answer to that is **"a lot"** and **none of it good**. Whenever you disrupt your body's core natural processes, things rarely go well.

It is estimated that about 60% of the people prescribed statins quit taking them because of the side effects. Some investigators have listed as many as **300 negative side effects for statins**, but here are just a few:

Diabetes
Kidney Failure
Erectile Dysfunction
Neuropathy (nervous system breakdown)
Cataracts
Pancreatic Dysfunction
Osteoporosis
Liver Dysfunction
Cognitive Impairment/Memory Loss
Muscle weakness and destruction (your heart is a muscle, right?)
Anemia
Immune System Suppression
Cancer
None of these side effects are well publicized, but they are very well documented.

For example: A team of six doctors from multiple universities in Scandinavia published the final results of their six year, 8749 person detailed study March, 2015 in the medical journal *Diabetologia*. Their conclusion: **"Statin treatment increased the risk of type 2 diabetes by *46%*, attributable to decreases in insulin sensitivity and insulin secretion."** (Cederberg, Stančáková and Yaluri)

There has been evidence in the past of statins increasing adult-onset Type 2 diabetes, but it has always been estimated to be in the 10-20% range. Those studies, however, were designed to observe the effects of statins on cholesterol. The diabetes risk information was simply an interesting by-product involving only the obvious cases.

This study, on the other hand, was specifically designed to observe **the long-term effects of statin use with regard to causing otherwise healthy people to develop type 2 diabetes**. The worst results were found to be caused by two of the most common statins in use worldwide: atorvastatin (aka Lipitor) and simvastatin (aka Zocor).

Given that the net positive effect on primary prevention of heart attacks for statins across the board is roughly 10% (and only in those with pre-existing heart disease) it would seem to be a really bad deal to trade a 10% risk improvement for a 46% risk increase! For people without 'pre-existing heart disease', it is estimated that statin use can only prevent one heart attack in one person out of every 100.

Some say that's being generous.

One set of researchers from Ireland found "a categorical lack of clinical evidence to support the use of statin therapy in primary prevention." (Sultan and Hynes) They also found that **statins actually increase cardiovascular risk** in women, the young and people with diabetes.

Another international study published in the medical journal *Atherosclerosis* showed that **statin use is associated with a 52 percent increase in the prevalence and extent of calcified coronary plaque compared to non-users**. (Nakazato, Gransar, and Berman) Coronary artery calcification is the hallmark of potentially lethal heart disease!

With risk/reward ratios like this, you are probably wondering why so many physicians are prescribing statins to so many patients. The answer is simple and frightening in and of itself. They've been intentionally misled by drug manufacturers through statistical manipulations and lies of omission. It's not your doctor's fault.

Statins are the most profitable drugs the Big Pharma companies have ever found to sell. Worldwide sales are estimated at $30 Billion Dollars per year. With that much profit, Big Pharma companies have no problem spending tens of millions of dollars (of your money) promoting their products. You've seen it on TV for the last 20 years.

What you haven't seen is the total domination of the information your doctor gets in the form of medical journal "studies" and medical conference presentations. Big Pharma only lets your doctor hear what they want them to hear. And the FDA lets them get away with it.

Example: One report published in the *Expert Review of Clinical Pharmacology* concluded that **"Big Pharma statin promoters use a statistical tool called relative risk reduction (RRR) to amplify statin's trivial beneficial effects."** (Diamond and Ravnskov) Just by using this statistical sleight of hand, statins suddenly become beneficial for 30 to 50 percent of the population. As STATS.org at George Mason University explained, "**An important feature of relative risk is that it tells you nothing about the actual risk**."

Let me show you another perfect example - the PROSPER study, published in the prestigious British medical journal, *The Lancet*. In it, the authors declare "Pravastatin given for 3 years reduced the risk of coronary disease in elderly individuals. PROSPER therefore extends to elderly individuals the treatment strategy currently used in middle aged people." (Shepherd, Blauw and Murphy) What they don't mention in the conclusions (and your doctor will never see unless they comb through the study data themselves) is that while the risk of heart disease in this hand-selected group dropped by 25%, **the incidence of cancer went UP for them by 25% as well!**

This brings me to one of my favorite parts of Dr. Dzugan's lectures:

> "Taking potent cholesterol-lowering medications has never been shown in clinical research to actually improve mortality. **In fact, in the biggest trials, significantly more people who took the drugs died than those who did not**. They didn't die of a heart attack, but dead is dead - whatever the cause." *(Dzugan)*
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> ***Sergey Dzugan, MD, PhD.***

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What is the real truth about Cholesterol, and how do you get yours down?

Elevated cholesterol is related to heart disease, just not in the way you've been sold. High cholesterol is merely a warning signal that your body chemistry is off and your body is trying to fix it. Cholesterol is the raw material from which your body makes the hormones that control your body chemistry and nearly all of its functions. When your body's hormone levels drop, your body puts more cholesterol into the system to compensate.

Thus, hormone deficiency would seem to be the primary cause behind rising cholesterol levels, and in turn, one of the main causes of the imbalances in body chemistry that lead to heart disease.

The Dzugan Institute has proven this concept with two separate studies. (Paper attached) **In both studies, 100% of patients responded to hormone restoration therapy with reductions in their total cholesterol levels**. Serum cholesterol figures post-treatment averaged 188 in the first study and had a mean of 191 in the second. None of the patients reported any negative side effects and most felt that their quality of life had significantly improved.

Restoring hormone levels to that of someone in their mid-twenties has been shown in thousands of cases to optimize overall body chemistry and the operation of the body's systems. This allows the body to heal itself of many of the disease states associated with hormonal decline. Those same disease states are the ones commonly associated with aging: heart disease, menopause, erectile dysfunction, fatigue, metabolic syndrome, diabetes, depression, arthritis, migraine, cancer and many more.

If you are over 30 and your cholesterol is going up, it is likely to be the impact of naturally declining hormone levels. If you take a statin to lower your cholesterol levels, you need to realize that you will be further lowering your body's ability to create the hormones you need for the proper functioning of your body. As you now know, that can have dire consequences.

We suggest that you have your overall blood chemistry checked – including all of your major hormones. If you don't know what tests you'll need, contact DzLogic and we'll be happy to send you a copy of our standard lab panel and the ranges you should be in for optimal health. "Normal" ranges and "Optimal" ranges are very different. "Normal" ranges include those of 80 year olds. What's normal for most 80 year olds is far from optimal.

Visit us at dzlogic.com or give us a call at 866-225-4877

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Correction of steroidopenia as a new method of hypercholesterolemia treatment

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Abstract

OBJECTIVE: In 2002 we proposed a new hypothesis of the etiology and pathogenesis of hypercholesterolemia. There is paucity of information in the literature regarding the association of steroidopenia and hypercholesterolemia. Our goal is to determine if the treatment of steroidopenia with hormonorestorative therapy (HT) to youthful levels will normalize total cholesterol (TC) levels.

MATERIAL AND METHODS: We retrospectively analyzed 43 hypercholesterolemic patients treated with HT. Laboratory workup included lipid profile, serum pregnenolone, dehydroepiandrosterone sulfate (DHEA-S), progesterone, total estrogen, cortisol, total testosterone, and vitamin D-3 levels at presentation with follow up ranging from 3 to 9 months. HT therapy included a combination of several agents such as pregnenolone, dehydroepiandrosterone (DHEA), triestrogen, progesterone, testosterone, hydrocortisone, and vitamin D-3.

RESULTS: HT lowered mean TC from 228.8 mg/dL to 183.7 mg/dL (19.7%) ($p<0.05$) in all patients. In 12 men of mean age 58, HT statistically significantly lowered TC from 227.9 mg/dL to 177.1 mg/dL (22.3%) ($p<0.05$). Apparently it did so mostly by lowering LDL and triglycerides (TRG) while HDL did not appreciably change. In 31women, mean age 57, TC declined from 229.2 mg/dL to 186.3 mg/dL (19%) ($p<0.05$). HDL, LDL, and TRG are also decreased to a statistically significant degree. These results were associated with statistically significant elevations in pregnenolone, DHEA Sulfate, testosterone, progesterone but not total estrogen, cortisol or vitamin D-3 changes in both men and women.

CONCLUSIONS: We conclude that correction of steroidopenia with the use of hormonorestorative therapy is an effective strategy for normalizing and maintaining cholesterol homeostasis.

ORIGINAL ARTICLE

INTRODUCTION

Hypercholesterolemia is a very widely debated and frequently discussed subject in medical literature. There are multiple methods to treat this affliction ranging from diet and exercise to pharmacological intervention with various medications (Bhatnagar *et al.* 2008; Schuff-Werner & Kohlschein, 2002; Manzoli *et al.* 2001). However, the problem remains because the etiology and pathogenesis of hypercholesterolemia remains unclear.

There is no information in the medical literature regarding the relationship between low levels of steroid hormones and the development of high cholesterol except for a few studies (Dzugan & Smith, 2002 a,b; Dzugan, 2004; Dzugan *et al.* 2004; Dzugan, 2007; Dzugan *et al.* 2009). In 2002 a ground-breaking study of hormonodeficit hypothesis of hypercholesterolemia was described that showed the association between steroidopenia and hypercholesterolemia (Dzugan & Smith, 2002 a,b). This novel study proposed that hypercholesterolemia develops as a compensatory response caused by declining levels of steroid hormones.

The goal of this study is to test a hypothesis concerning the association of steroidopenia and hypercholesterolemia by evaluating the impact of the restoration of multiple steroidal hormone deficiencies to youthful levels in hypercholesterolemia treatment.

MATERIAL AND METHODS

This study is a retrospective chart review of 43 patients treated for hypercholesterolemia with HT after they failed the conventional treatment of high cholesterol or had side-effects of cholesterol lowering drugs. There were 12 male and 31 female patients. The mean overall age was 58.4 years, mean female age was 57.0 years, and mean male age was 62.3 years. HT was utilized in all of the patients. In 1996 we employed the term hormonorestorative therapy into our practice for the regimen that was used for our patients. HT is defined as a multi-hormonal therapy with the use of chemically identical formulas to human hormones (anthropo-identical) and is administered in physiologic ratios with dose schedules intended to simulate the natural human production cycle at optimal levels.

Hormonorestoration includes a combination of several agents such as pregnenolone, DHEA, triestrogen, progesterone, testosterone, hydrocortisone, and vitamin D-3.

Lipid profile, serum pregnenolone, DHEAS, progesterone, total estrogen, cortisol, total testosterone, and vitamin D-3 levels were checked at presentation and at 3 months intervals. The follow up period was from 3 to 9 months.

One of the most significant age-related events is an alteration in amplitude and pulsatile pattern of hormone release (Smith *et al.* 2005). Hormone restoration should provide a serum hormone profile similar to that found in normal physiology. For restoration of estrogen, progesterone, and testosterone we utilize topical gels because they contain highly lipophilic molecules with low molecular weight, are very well absorbed through the skin, may use adipose tissue as a reservoir, and facilitate individualized dose of prescription. The typical formulation for Triest gel (E3:E2:E1 – 90:7:3) was 1.25–2.5 mg/mL, progesterone 5–10% – 50–100 mg/mL, and testosterone 5–10% – 50–100 mg/mL. In cases of insufficient absorption with gel we utilized drops of Triest (E3:E2:E1 – 80:10:10) at 5 mg/ml, progesterone at 50 mg/mL, and testosterone at 50 mg/mL. Pregnenolone, DHEA, and hydrocortisone were used in oral form (capsules or tablets). The pregnenolone dose ranged from 15 mg to 300 mg, DHEA from 15 mg to 200 mg, and hydrocortisone from 2.5 mg to 10 mg. Vitamin D-3 was used in the doses that ranged from 1000 IU to 5000 IU.

The following factors were included in our decision making in the dosing of our patients during the HT:

1. the recommended dosages for different patients during HT varied significantly and were determined by clinical data and serum hormonal levels during serial testing
2. dosages were individually selected during HT to produce physiologic serum levels typical for healthy individuals between the age of 20 and 30 years for both genders
3. we administered hormones in doses sufficient to restore the optimal level that was defined as the level of hormones in the upper one third of normal range from the testing laboratory.

We employed the following rules in the use of our hormones:

- anthropo-identical structure of hormones
- individually modified doses
- cyclical manner
- larger doses in the morning
- treatment control by serum hormonal level
- mono- or bi-hormonal therapy is usually inadequate
- multi-hormonal therapy is optimal.

RESULTS

All of the patients responded favorably to HT (Figure 1). There were no adverse effects. The student T Test was chosen to evaluate the results. The mean TC dropped from 228.8 mg/dL to 183.7 mg/dL (19.7%). Seven patients still had cholesterol levels ranging from 202 mg/dL to 211 mg/dL but all of these patients had a beneficial drop in TC. These patients still required additional treatment and optimization of their steroid levels. The mean TC in women declined from 229.2 mg/dL to 186.3 mg/dL (18.7%). The mean TC in men decreased from 227.9 mg/dL to 177.1 mg/dL (22.3%). Total cholesterol declined an average of 51 points in men and 43 points in women and 45 points in all patients. This

decline in total cholesterol was found to be statistically significant ($p<0.05$) in men despite the small sample size and significant ($p<0.05$) in female as well as the entire population of patients.

The mean HDL dropped from 65.0 mg/dL to 53.8 mg/dL. The mean HDL in women declined from 73.3 mg/dL to 59.5 mg/dL ($p<0.05$). The mean HDL in men declined from 43.6 mg/dL to 38.8 mg/dL, but was not statistically significant. The mean LDL dropped from 137.4 mg/dL to 110.2 mg/dL ($p<0.05$). The mean LDL in women decreased from 132.8 mg/dL to 109.3 mg/dL ($p<0.05$). The mean LDL in men decreased from 149.3 mg/dL to 112.5 mg/dL ($p<0.05$). It must be remembered that total cholesterol is a calculation of HDL plus LDL plus triglycerides divided by 5. Therefore, it was expected that LDL levels were found to decline in males, females, and in all patients at highly statistical ($p<0.05$) levels consistent with the change in total cholesterol. HDL on the other hand, being a carrier bringing substances back to the liver was found to not have changed in men at a statistically significant ($p=0.09$) level but did decline in women ($p<0.05$) and was statistically significantly ($p<0.05$) declined in all patients. The mean triglycerides dropped from 132.7 mg/dL to 100.3 mg/dL (24%). The mean triglycerides in women declined from 115.0 mg/dL to 86.7 mg/dL ($p<0.05$). The mean triglycerides in men declined from 178.7 mg/dL to 135.6 mg/dL ($p<0.05$). Triglyceride levels were found to be statistically significantly declined in men and women in all patients.

Figures 2 and 3 portray the most significant steroid hormone level change in males and females after HT. The mean pregnenolone was elevated from 53.9 ng/dL to 172.1 ng/dL. The mean pregnenolone in women increased 61.2 ng/dL to 193.5 ng/dL. The mean pregnenolone in men increased from 35.0 ng/dL to 112.0 ng/dL. Pregnenolone levels were found to be significantly elevated ($p<0.05$) in men and women in all patients. Mean DHEA-S was elevated from 92.8 µg/dL to 457.0 µg/dL. Mean DHEA-S in women increased from 88.3 µg/dL to 408.5 µg/dL. Mean DHEA-S in men increased from 104.4 µg/dL to 597.3 µg/dL. DHEA-S levels were also found to be elevated in a statistically significant ($p<0.05$) manner for men and women and all patients. In men, testosterone level was elevated on the program from 424.0 ng/dL up to 625.1 ng/dL on average and this was statistically significant ($p<0.05$). The mean testosterone in women increased from 37.6 ng/dL to 63.2 ng/dL ($p<0.05$). Total estrogen decreased in men from 124.0 pg/mL to 112.5 pg/mL, but did not change statistically significant. In women significant changes in total estrogen also were not observed. The mean progesterone level increased in women from 3.2 ng/mL to 7.5 ng/mL. Progesterone levels increased in women in a statistically significant ($p<0.05$) manner. The same result was observed for men – progesterone was elevated from 0.4 ng/mL to 2.5 ng/mL. The mean cortisol level in all patients increased from 13.8 µg/dL

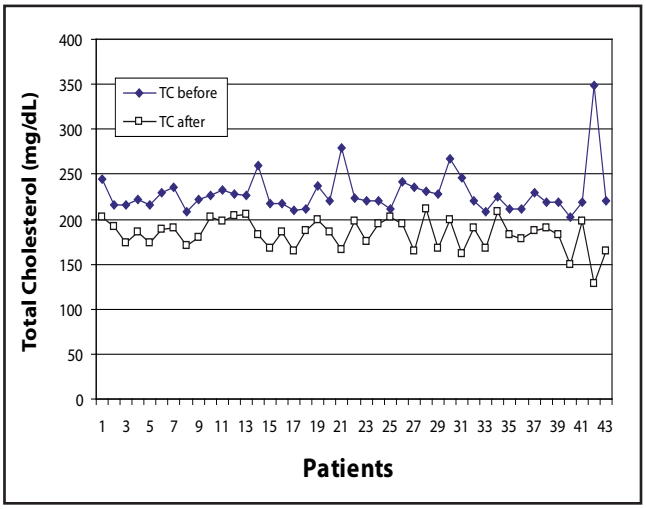

Fig. 1. Total Cholesterol Before and After Hormonorestorative Therapy.

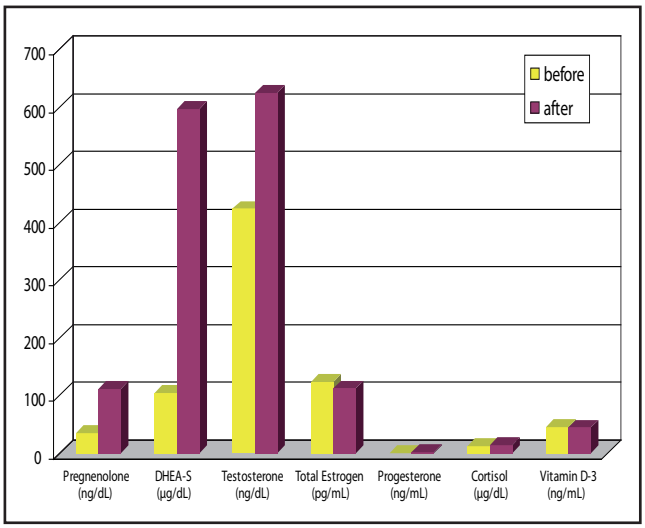

Fig. 2. Steroid hormone levels in males before and after Hormonorestorative Therapy.

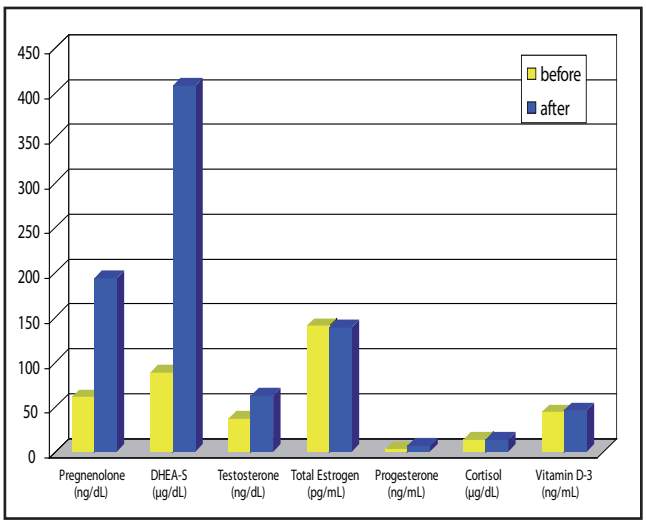

Fig. 3. Steroid hormone levels in females before and after Hormonorestorative Therapy.

to 14.1 μg/dL. Cortisol levels were not elevated in men or women to a statistically significant degree. The mean vitamin D-3 level for all patients increased from 44.8 ng/mL to 46.3 ng/mL. Vitamin D-3 levels were also not elevated in a statistically significant manner in men and women.

DISCUSSION

In men, HT lowered total cholesterol. Apparently it did so mostly by lowering LDL and triglycerides. However, HDL did not appreciably change. Based on this analysis, the decline of cholesterol and LDL levels was related to the elevation in pregnenolone, DHEA Sulfate, testosterone, and progesterone. No significant change had occurred in vitamin D-3 levels, cortisol or total estrogen as explained in the results.

In women, total cholesterol declined after HT as did HDL, LDL and triglycerides to a statistically significant degree. This is associated with statistically significant elevations in pregnenolone, DHEA Sulfate, testosterone, progesterone but not total estrogen, cortisol or vitamin D-3 changes.

We believe that decreasing the level of HDL during HT is a good sign of our intervention if HDL was not very low initially, because if we normalize the level of TC, what reason is there for extra production of HDL? If there is nothing to transport back to the liver, why produce the extra carrier? HDL, by this logic, should decrease! The absence of significant changes in the levels of total estrogen in females can be explained by the fact that total estrogen was measured as a sum of estrone and estradiol only. Even though the laboratory values of the total estrogen did not change, clinically all of the patients experienced resolution of menopausal symptoms. The mean vitamin D-3 level was not elevated in a statistically significant manner because more than eighty percent of the patients were taking certain doses of vitamin D-3 supplement prior to the initiation of our HT, and this can explain small changes in the levels of vitamin D-3 in our study.

Cholesterol is the precursor or the building block for the basic steroid hormones such as pregnenolone, DHEA, progesterone, cortisol, aldosterone, estrogen, testosterone, and others (Figure 4). As the human body ages there is a natural decline in the level of these steroid hormones. We proposed that, since cholesterol is responsible for the production of the steroid hormones, the human physiology is designed to increase the production of cholesterol to balance or attempt to reverse declining hormones. As a result, the cholesterol level rises in a way negative feedback loops work to compensate for the low steroid hormones. Unfortunately, in the aging body the enzymatic system is less efficient and, therefore, these hormones never quite reach the "normal" youthful level. This is how we arrive at the picture of hypercholesterolemia and steroidopenia. Cholesterol elevation should, therefore, be seen as a marker for steroid hormone deficiency.

The administration of steroid hormones restores their optimal levels in the body and, thus, alleviates the need to over-produce of cholesterol. As the feedback loop is complete and steroid hormone levels are normalized, then cholesterol level normalizes as well, since the body is now in equilibrium.

This study reconfirms our hypothesis that steroidopenia and hypercholesterolemia are closely interrelated strongly suggesting that one drives the other. The con-

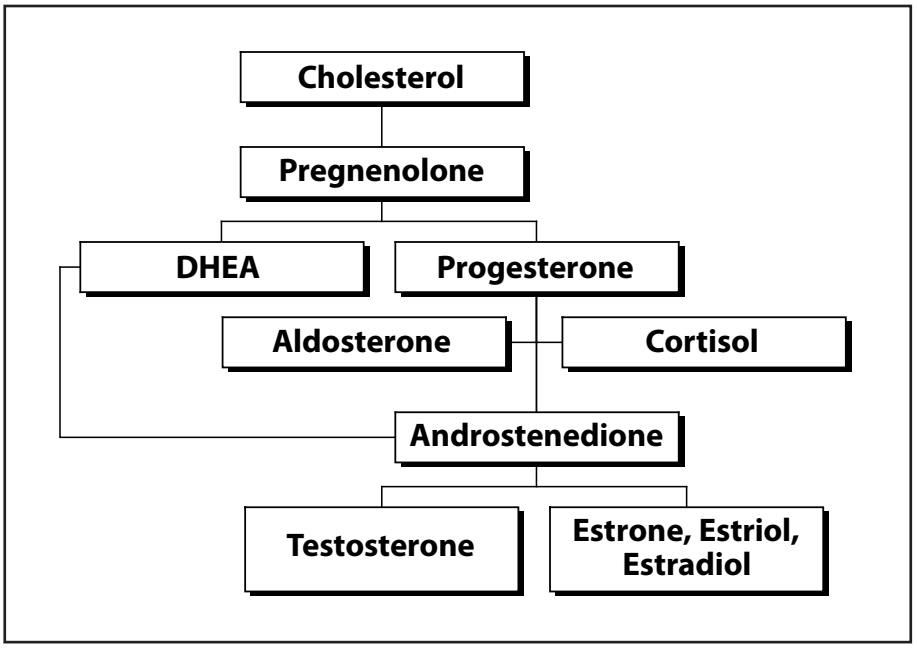

Fig. 4. Simplified version of cholesterol metabolism.

sequence and ramification of these results argue that the optimization of physiology or physiologic medicine should precede pharmacologic intervention. Physiologic medicine treats the symptoms of deficiency and imbalance. Pharmacologic medicine of cholesterol elevation interferes with normal physiologic mechanisms and is associated with numerous side effects. The mindset of our methodology is based on the optimization of human physiology with gentle assistance, whereas the root of the conventional treatment of hypercholesterolemia is based on "fighting" the body with drugs.

CONCLUSION

Our study confirms that there is indeed a valid connection between steroidopenia and hypercholesterolemia. We believe that correction of steroidopenia with anthropo-identical hormones could serve as an inexpensive and effective method of treatment of hypercholesterolemia in healthcare.

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