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HEALTH BRIEF

ONE PHARMACIST'S VIEW OF CORONARY HEART DISEASE: COMPARING THE "LIPID THEORY" WITH THE "UNIFIED THEORY"

By Mike Ciell, R.Ph.

EXECUTIVE SUMMARY

Conventional wisdom argues that cholesterol, an oily substance produced in the body, is the enemy and must be managed to prevent coronary heart disease. Fighting cholesterol is inherent in the "Lipid Theory" of heart disease. Alternatively, Linus Pauling, two-time Nobel laureate and Ph.D. and Matthias Rath, M.D., created the "Unified Theory" of heart disease, identifying vitamin C, L-lysine, and L-proline as critical nutritional agents that could both improve blood vessel function (flow) *and* reduce cholesterol plaques (blockages). Some researchers have said that vitamin C is the equivalent of "nature's perfect statin."

Most animals produce vitamin C endogenously (within their bodies) and never demonstrate signs of cardiovascular disease. Humans, alternatively, must rely on dietary ascorbate to maintain health, and when insufficient supplies of ascorbate are present, humans suffer from a variety of chronic diseases, including coronary heart disease.

Pauling's and Rath's research provides evidence that cholesterol plaques are actually the body's back-up mechanism for repairing damaged blood vessels, and that if you provide the body with enough free-circulating vitamin C, along with L-lysine and L-proline, the body's primary mechanism for making vascular repairs can be employed and cholesterol numbers can ultimately fix themselves.

CORONARY HEART DISEASE – THE LEADING CAUSE OF DEATH IN AMERICA TODAY

Coronary heart disease (CHD), the most common form of cardiovascular disease, is the leading cause of death in America. Over 13 million Americans suffer from CHD, which results in more than one million heart attacks per year, of which more than one half will be fatal. These statistics are in sharp contrast with the cardiac health of Americans at the beginning of the 20th century.

HEART DISEASE USED TO BE RELATIVELY RARE

In 1900 heart attacks were almost nonexistent, and most cases of heart disease that did occur were usually the result of an infectious disease (rheumatic fever for example) or congenital heart defects. In the ensuing decades, this once rare condition steadily rose in frequency to become *the* leading cause of death in America.

The big question: "What has caused this problem?" Social changes since 1900 are certainly in play, including:

- Widespread use of cigarettes after WWI,
- A huge increase in refined sugar use,
- Sedentary suburban lifestyles, and
- Packaged, chemical-laden food replacing fresh, raw choices

Ask any reasonably-educated person with heart disease what the problem is and you're likely to hear things like, "I have to watch my diet, get more exercise, stop smoking" and so forth. Good starting points, but, as I cover in this article, there's more.

KOREAN WAR AUTOPSY CONNECTION

It was during the Korean War that doctors thought they had discovered the "real" cause of heart disease. Autopsies on young soldiers killed in action showed well-developed atheromas (arteriosclerotic plaques) in their coronary and carotid arteries. Additionally, fatty streaks of the intima of their arteries, arterioles, and heart muscle existed.

While similar fatty streaks were observed in dead Korean and Chinese soldiers, the well-developed atheromas found in American soldiers were conspicuously absent. Analysis of the plaques found showed it was a saturated fat (palmitic acid). The atheromas also contained quite a lot of a familiar waxy substance—better known as cholesterol.

THE "LIPID THEORY OF CARDIOVASCULAR DISEASE"

Thus, out of the battlefields of Korea was born "The Lipid Theory of Cardiovascular Disease." For almost 60 years, this "lipid theory" has been central to medical explanations of and treatment for cardiovascular disease.

Simply put, the Lipid Theory posits that a diet high in cholesterol and saturated fat will cause "gooey" substances (cholesterols) to be deposited in the blood vessels, clogging them up. Clogged blood vessels clearly restrict blood flow to the heart, ultimately causing angina. Eventually a piece or "clot" will break loose, causing a TIA (angina or a mini stroke), a stroke or a full-blown heart attack.

Conventional thinking has centered on removing "causative" agents – cholesterol and bad fats -- to stop coronary heart disease.

For decades we have used diet and drugs to attempt to reach ever-lowering "cholesterol levels" recommended by the American Heart Association.

Tragically, statistics show that heart disease continues to be on the rise, claiming ever more lives.[1]

[1] Recently the mortality rates (deaths) have decreased somewhat —probably due in large part to the new "clot buster" drugs and better emergency treatment and response time. The morbidity (actual cases of disease) is still rising, albeit not quite as fast.

A PLUMBER'S TAKE ON "PLUGS" IN THE SYSTEM

So what's wrong with the Lipid Theory? Any plumber looking at the Lipid Theory model would say, "It simply doesn't make sense."

Let's start by thinking about "sludge" in a plumbing system. Sludge tends to plug up the smallest pipes in the system first—not the largest.

Likewise, if the system is cardiovascular, you would expect sludge (plaques) to build up first in the capillaries and arterioles*, long before* appearing in the carotid and coronary arteries. The first blockages, similarly, you would expect to occur way downstream of the pump, not in close proximity to the heart, where the pressure is the greatest.

Yet, this is not the way cholesterol plugs up arteries. It's the exact reverse. So a plumber's take would be that something else is happening.

THE MYTH ABOUT "GOOD" & "EVIL" (FATS)

When autopsied plaques have been analyzed, they are found to contain cholesterol, but of a very particular type. The offending cholesterol is a highly-oxidized variety of LDL cholesterol attached to a specific protein (Apo A). The whole complex is called Lipoprotein A or Lp(a) – more on Lp(a) further in this article.

The fats found *inside* these plaques, as it turned out, were *un*saturated fats (not the supposedly "evil" saturated fats). Fatty streaks in the intima of the arteries *are* saturated fat, but this appears to be quite normal, since it is the same in many animals.

Avoiding saturated fat, using "healthy" polyunsaturated oils, and building a diet on a base of carbohydrates (grains, breads, and starchy veggies) has been drummed into us by well-meaning authorities, including luminaries such as: the USDA, the American Heart Association, and the American Diabetes Association

Yet, it is a basic physiological fact that *all* carbohydrates are metabolized to glucose. If glucose is not used for fuel, it is automatically converted to and stored as *saturated* fat (only a small part-about 100 grams - will actually ever be stored as glycogen).

Telling people to *avoid* eating saturated fat – while simultaneously telling them to eat food that will be *converted into* saturated fat -- fails a basic logic test. Fat, I will argue, is *not* the real problem.

ANIMAL MODELS, A GENETIC MUTATION, & MODERN DIETS

It helps at this point to explain some genetic history. Only a few animals (the higher apes, the guinea pig, and a species of fruit bat) ever show coronary heart disease. Heart disease, however, appears *only* when these animals are fed a diet that is lacking in adequate amounts of vitamin C.

Zookeepers learned the connection between vitamin C and heart health a long time ago. When their gorillas were fed a diet of early versions of processed "gorilla-chow," instead

of a diet rich in vitamin C from fresh fruits and vegetables, they got sick and developed heart disease.

In contrast, bears -- whose cholesterol levels can be three times as high as man's and whose heart rates slow way down during hibernation – remarkably never show any atherosclerosis.

So what's going on in bears and other animals that is missing in humans, apes, guinea pigs and some fruit bats?

Endogenous Production of Ascorbate and a Genetic Mutation
Other animals produce vitamin C endogenously (which means most animals manufacture ascorbate *inside* their bodies), and this production of vitamin C is essential to maintaining health, including maintaining healthy arteries.

For example, a 150-pound goat has a typical blood concentration of ascorbate equivalent to taking 13,000 mg (13 grams) of vitamin C per day. And, ascorbate concentrations rise much higher in times of stress. Compare this abundance of vitamin C in a goat with the paltry 60mg recommended daily allowance for humans. Consider further the percentage of people who do not get enough vitamin C from their diets, and it's no wonder that heart disease is so prevalent.[2]

Some millions of years ago, a genetic mutation occurred, causing humans to rely on their diets for vitamin C. This mutation was not life-threatening, however, because our early ancestors thrived in the tropics, where vitamin C was in ready supply in fresh fruits and vegetables.

Scurvy (and heart disease) became a real problem for ancestors who settled in other regions of the world, areas with less readily-available dietary ascorbate.

During the Ice Ages, however, many of our ancestors did indeed succumb to scurvy and heart disease, when plant-foods were not as plentiful.[3]

Ancestors who were able to survive possessed a valuable genetic mutation, whereby damaged (leaky) blood vessels could be patched by a "back-up mechanism," an animal food component called cholesterol. Modern humans inherited this ability to use cholesterol to make repairs, which, in other animals, are made through an abundance of freely-circulating vitamin C.

CHOLESTEROL CARRIERS & MECHANISMS OF MOVING CHOLESTEROL IN THE BODY

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[2] According to a 1991 National Survey of Health and Nutrition, half of all American men consume less than 84 mg of vitamin C daily in their food. Half of U.S. women consume 73 mg a day from food. For 20 to 30 percent of U.S. adults, food provides less than the current RDA of 60 mg.
[3] The Eskimos only appear to be a counter-example. However, because the Eskimos consumed *all* organs of the animals they hunted (including the eyes and the surrounding eye tissue, as well as the adrenals, all of which are very high in ascorbate), the Eskimos avoided ascorbate deficiencies and heart disease.

Now let's talk about cholesterols. LDL has been called the bad or "lethal" cholesterol, while HDL is considered the "healthy" cholesterol. I'm afraid this is nothing but nonsense. There is no "good" or "bad" cholesterol. LDL and HDL are just two different types of cholesterol "carriers."

When it comes to how cholesterol moves in the body, I believe it was Dr. Andrew Saul who used the analogy of a bus line, which is an apt one. Since cholesterol is an *oily* substance that travels through a *watery* bloodstream, it must be *carried* (similar to passengers and cargo on a bus) to various destinations in the body.

The HDL "Bus Line"
The HDL cholesterol molecule has a higher protein-to-lipid ratio (contains more protein than lipid material). Protein is denser than lipid, hence the name "high density lipoprotein".

The HDL "bus line" thus carries a single "bag" of this dense cholesterol as cargo in each of its buses. In this way, the HDL cholesterol gets a ride straight to the liver and is eliminated as bile acids via the gall bladder and intestines.[4]

The LDL "Bus Line"
The LDL cholesterol molecule, in contrast, has more lipid or "oil" content, thus a lower protein-to-lipid ratio, and hence the name "low-density lipoprotein." The LDL "bus line," in contrast, carries two "bags" of cholesterol as cargo.

The LDL bus line transports cholesterol to a variety of sites in the body, where it is used to repair and/or protect tissues or to be used in the synthesis of many vital compounds.

LDL cholesterol's role is so important that nature lets the LDL bus line transport twice the number of cholesterol "passengers" as the HDL bus line does. LDL cholesterol is thus taken to important destinations in the body such as:

- Skin, where cholesterol reacts with sunlight to produce the "best kind" of Vitamin D (think healthy bones),
- Sex hormones (the estrogens, testosterone, and progesterone) and their "precursor" molecules (DHEA, DHEA-S and pregnenolone, as well as adrenal stress hormones like cortisol),
- Nerve cells so they are well-insulated and don't short circuit,
- Scar tissue to repair tissue injuries,
- Cell walls to "waterproof" us so we don't melt in the rain or when we take a bath, and
- Blood vessels for vital cardiovascular repairs, as well!

The body was designed pretty exquisitely, and clearly cholesterol's real mission to save our lives, not to kill us.

[4] From Mary Enig, PhD, and Sally Fallon in *The Oiling of America*, printed in The Weston A. Price Foundation website. When you get bored sometime, check out pictures of cholesterol "stones" on a liver-cleanse website and you'll see that the liver processes *a lot* of cholesterol.

CHOLESTEROL READINGS & HEALTH – THE HIGHS & LOWS

Now, without looking at specific numbers (like we medical professionals love to do), let's consider three cases and determine what a person's cholesterol readings suggest about health:

1. ***High LDL*** - A relatively high LDL reading may indicate that the body needs to repair a lot of things and transport cholesterol to areas of stress or disrepair.
2. ***Low HDL -*** A low HDL reading may mean the body needs to hold on to cholesterol to both make repairs and to synthesize molecules that are scarce.
3. ***Lower LDL & High HDL --*** A lower LDL and high HDL reading is likely to indicate the body's systems are pretty well maintained. With no need for a lot of cholesterol, excess cholesterol is regularly eliminated.

As a goal, we want to strive for the third case, with a high percentage of the total cholesterol being of the HDL variety. In mathematical terms, total cholesterol over HDL should be less than 3.5 or:

$$TC/HDL < 3.5$$

However, when we "artificially" lower our cholesterol through pharmacological inhibitors (like "statin" drugs), we really cannot infer anything about our state of health with regards to cholesterol levels.

With drugs, we can make less cholesterol available to block our blood vessels, but at the same time, we will make less cholesterol available to perform vital functions (like converting sunlight to vitamin D, insulating nerve cells, healing scars, etc.).

Total cholesterol, as it turns out, has *never* been an indicator of who is more likely to suffer a heart attack. In fact, Dr. William Castelli, director of the prestigious Framingham Study, said:

> "The more saturated fat one ate, the more cholesterol one ate, the more calories one ate, the lower peoples' serum cholesterol…we found that the people who ate the most cholesterol, ate the most saturated fat, ate the most calories, weighed the least and were the most physically active." [5] [6]

The ongoing Framingham population study also found that there was virtually no difference in coronary heart disease events for individuals with cholesterol levels between 205 mg/dl and 294 mg/dl – where the vast majority of the U.S. population lands.

Even for those with extremely high cholesterol levels, up to almost 1200 mg/dl, the difference in CHD events compared to those in the normal range was trivial. Now that

[5] Castelli, William, "Concerning the Possibility of a Nat…" Archives of Internal Medicine, July 1992, 152: (7): 1371-1372
[6] Smith, R. and E.R. Pickney, Diet, Blood, Cholesterol and Coronary Heart Disease: A Critical Review of the Literature, Vol 2, 1991, Vector Enterprises, Sherman Oaks, CA

being said, please *do not* take this as carte blanche to consume as much saturated fat and calories as you want.

Castelli's comments were made decades ago, when much of our livestock was still grass-fed and the adding of hormones and growth enhancers (both of which become concentrated in animal fat) was just beginning.

Diets high in *domestic* animal fat and partially *hydrogenated poly-unsaturates* (metabolic poisons) have their own set of health risks. As an aside, this is my main concern with Dr. Atkins' dietary guidelines. Without emphasis on the *quality of fats and proteins* and balancing complex carbohydrates into the equation, we may be trading short-term health benefits for long term health risks.

Anyway, because cholesterol supports so many essential physiological processes, it doesn't make a lot of sense to pharmacologically inhibit cholesterol production to "get our numbers right." In fact, in study after study, the group with the lowest cholesterol levels had the *highest* mortality (death due to all causes).

I find the mortality rates for those with the lowest cholesterol readings particularly troubling. I believe it's far better to help our bodies make necessary repairs and let the numbers "fix" themselves. Nature ultimately does not waste energy, so when less cholesterol is needed, less cholesterol will be produced.

LINUS PAULING'S UNIFIED THEORY OF CARDIOVASCULAR DISEASE -- MAKING REPAIRS NATURALLY

A lot of people have heard that Linus Pauling had some theory about vitamin C and heart disease treatment. It's true and it's called the "unified theory of human cardiovascular disease," which posits that ascorbate deficiency is one of the primary causes of cardiovascular disease.

Using data gleaned from literally hundreds of published research papers by world-class scientists (MDs and PhDs), Pauling and his research partner, Matthias Rath, MD, described the link between cardiovascular disease and vitamin C.

In their "Unified Theory of Human Cardiovascular Disease," genetic differences, in species which are susceptible to CVD, are taken into account. The Unified Theory explains how the human body precisely regulates blood concentrations of cholesterol and provides compelling evidence that, with proper nutrition (and not drugs) cardiovascular disease can be prevented and even reversed.

THE COMMON GROUND AND DIFFERENCES OF THE TWO THEORIES

Both the Lipid Theory and the Unified Theory agree that atherosclerotic plaques are deposited in response to injury of the blood vessel wall. Drs. Brown and Goldstein were awarded the Nobel Prize in Medicine in 1985 for this discovery.

Pauling and Rath, however, had a different concept of cause and effect, proposing that the genetic weakness of certain species (that *do not* produce ascorbate endogenously) must be addressed nutritionally, to promote healthy blood vessels.

While Pauling and Rath saw cholesterol as clearly *correlated* with cardiovascular disease, they did *not* consider cholesterol as the enemy.

The Lipid Theory, on the other hand, argues that cholesterol *is* the enemy. Consequently, those who embrace the Lipid Theory emphasize:

- Decreasing the amount of cholesterol and lipids ("patch material") in the body through diet and drugs
- Making the blood itself *less sticky* (by means of blood thinners such as Coumadin, Plavix, aspirin, etc.), to ensure adequate blood flow and prevent heart attacks.

To help put these theories into context better, let's turn to one of my favorite topics: biochemistry.

THE BIOCHEMISTRY OF IT ALL

Biochemistry is literally the "chemistry of life," and, as a pharmacist, this is one of my specialties. Biochemistry helps us understand the structure and function of cellular components, including proteins, carbohydrates, lipids, nucleic acids, and other biomolecules, as well as enzyme-mediated reactions.

When I'm researching something new, I like to understand what is going on at a molecular level. To make the case for the Unified Theory, it's valuable first to understand the roles of lipoprotein A, collagen, and vitamin C.

Lipoprotein A or Lp(a)
Lipoprotein A, otherwise known as Lp(a), is another special cholesterol carrier (bus line) found *only* in species that do not produce their own ascorbate (Vitamin C).

Like LDL, the Lp(a) bus carries two "bags" of cholesterol, which are covered with a protein coat called apo(a). This coat allows cholesterol to move through the watery bloodstream. However, unlike LDL, Lp(a)'s protein coat is *very sticky* -- think of the "a" as meaning "adhesive."

In a non-ascorbate producing animal, the amount of Lp(a) is *inversely proportional* to the amount of circulating ascorbate. That means that higher vitamin C concentrations lead to less production of the sticky Lp(a) particles.

Under stress and when insufficient vitamin C is in circulation, the ability to produce Lp(a) allows the body to patch damaged blood vessels and prevent death by hemorrhage.

Thus, the "sticky" Lp(a) particle circulates through the vessels and adheres to spots where a blood vessel wall is damaged. Due to the Velcro-like surface of Lp(a), circulating LDL particles also will adhere where Lp(a) is busy patching damaged blood vessels, escalating the process of atherosclerosis formation.

Interesting Facts About Lp(a)

- Lp(a) levels *are* influenced by genetics (inheritance)[7]
- Diet *does not* influence Lp(a) levels
- Cholesterol-lowering drugs *have not* been shown to lower Lp(a) levels
- Both ascorbate (vitamin C) and niacin (vitamin B-3) *have been* shown to lower Lp(a) levels
- Natural amino acids, L-lysine and L-proline, prevent the outer coat (apo-a) of a Lipoprotein A carrier from being sticky. These amino acids convert apo-a from a "Velcro" to a "Teflon" quality. L-lysine and L-proline also help remove plaque that is already present in blood vessels (by preferentially binding with receptors on Lp(a) and, thus, displacing Lp(a) cholesterol from artery walls).
- Lp(a) is the *single greatest risk factor* predicting restenosis of blood vessels (the narrowing of blood vessels after widening in bypass surgery)

Collagen
Collagen is by far the most abundant protein in the body. While literally a fiber, collagen acts like a "glue," which holds our cells together. Collagen is actually the body's *preferred repair substance*, whether for closing wounds, healing blood vessels, or helping the skin remain wrinkle-free.[8]

The collagen fiber looks like a 3-strand rope. The "rope" consists of a strand of L-glycine molecules, a strand of L-proline molecules, and a strand of L-lysine molecules. These strands of amino acid chains are twisted around each other in a helical fashion and, in fact, do look like a rope.

When an injury occurs and the collagen fiber breaks, the *frayed* ends dangle just as if a rope were cut.

If adequate ascorbate is present, the amino acids at the broken ends are hydroxylated. That means the "end" molecules of L-glycine, lysine and proline are chemically changed to *L-hydroxyglycine, L-hydroxylysine and L-hydroxyproline.* This allows them to be *spliced* back together (much like a sailor splicing a rope together). This simple chemical change also explains why vitamin C has the ability, not only to repair the damage, but *also to start breaking up existing plaques*, as will soon become apparent.

Of all the amino acids, L-glycine is the simplest one chemically and, in general, is always in ample supply in the body. L-proline and L-lysine, the other two amino acids in the

[7] Kennedy Ron, MD. Lipoprotein(a), Vascular Disease, and Vitamin C http://www.medical-library.net/sites/_lipoprotein(a)_vascular_disease_and_vitamin_c.html
[8] As an aside, I recommend that you don't get collagen injections, but instead help your body make its own collagen through better diet and supplementation.

collagen fiber, however, are not always in ample supply, and the body benefits from supplementation to ensure good collagen synthesis.

Vitamin C -- Ascorbate or Ascorbic Acid

By now, you may have surmised that vitamin C is the lynchpin of the Unified Theory. Vitamin C, or rather the lack of sufficient ascorbate, has implications in practically *every chronic disease* -- osteoporosis, diabetes, arthritis, cancer, macular degeneration, allergies, and chronic or re-occurring infections are just a few examples.

Unfortunately, most people, including doctors, think of vitamin C as the substance that prevents scurvy. A small daily dose of 60 mg is sufficient to prevent scurvy, and conventional wisdom has been that additional vitamin C will just create expensive urine.

Pauling and Rath, however, hypothesized that most humans suffer from *chronic, sub-acute scurvy and CVD is merely one of the symptoms of the underlying disease.* Have your gums ever bled when you flossed your teeth? Have you ever had a nosebleed for no apparent reason? Have you ever had a wound that was slow to heal? If so, you may have (had) a deficiency in ascorbate in your system.

When a person develops a chronic condition (which adds stress to the body and further depletes already inadequate ascorbate stores), many tell-tale symptoms scurvy often appear. The correct diagnosis is typically missed and scurvy sequelae are instead called symptoms of some chronic disease (i.e., poor wound healing in diabetics, hemorrhages in diseases like Crohn's and ulcerative colitis, etc.).

Some salient facts about vitamin C and cardiovascular disease

- During the 1950's, a brilliant Canadian physician, Dr. G. C. Willis, demonstrated that vitamin C was indeed related to cholesterol metabolism. A deficiency in ascorbate caused increased cholesterol synthesis (production). Feeding animals increased amounts of cholesterol reduced their vitamin C levels, and, conversely, vitamin C supplementation decreased cholesterol levels. Dr. Willis also showed that vitamin C could reverse atherosclerosis in guinea pigs, a species that does not produce ascorbate endogenously.[9] [10] [11] [12] [13]

- In 1971, British physician, Dr. Constance Spittle, demonstrated that patients with existing CVD exhibited a transitory rise in blood cholesterol when given vitamin C therapy, while patients with no CVD showed the reverse, namely, lower blood cholesterol levels. Spittle's explanation: the vitamin C therapy was actually healing

[9] Willis GC. 1953. An Experimental Study of the Intimal Hemorrhages and in the Precipitation of Coronary Thrombi. Canadian Medical Association Journal, vol.69:pp.17-22.
[10] Willis et al GC. 1954. Serial Arteriography in Atherosclerosis. Canadian Medical Association Journal, vol.71: pp.562-568.
[11] Willis GC, Fishman S. 1955. Ascorbic Acid Content of Human Arterial Tissue. Canadian Medical Association Journal, vol.72:pp.500-503.
[12] Willis GC. 1957. The Influence of Ascorbic Acid upon the Liver. Canadian Medical Association Journal, vol.76:pp.1044-1048.
[13] Willis GC. 1957. The Reversibility of Atherosclerosis. Canadian Medical Association Journal of Nutrition, vol.77:pp.106-109.

the vessel walls, thus releasing the cholesterol from the existing plaques. By the way, this research was published in the prestigious British medical journal, *The Lancet.*[14]

- Finally, in 1985, when Mevacor (the first statin drug) was the hot new pharmaceutical, Dr. H. J. Harwood, Jr. showed that vitamin C was in fact *"nature's perfect statin."* Low vitamin C levels trigger the enzyme HMG-CoA Reductase to increase its activity and catalyze the synthesis of more cholesterol to ensure an adequate supply of "patch material." Alternatively, high vitamin C levels were shown to inhibit the enzyme activity and cause cholesterol levels to fall. [15] Dr. Harwood's research shows a fundamental difference between drugs and nutrients: drugs can only inhibit or accelerate a biochemical process whereas *nutriceuticals* allow the body to *modulate* (i.e. up regulate or down regulate) enzymatic activity based on the body's current physiological needs.

- There is another very important difference in the mechanism of action of the statins compared with that of vitamin C. The statins, by their mechanism of inhibition of HMG-CoA reductase, also inhibit the production of enzyme CoQ-10. Vitamin C on the other hand actually *increases* the production of this important enzyme. CoQ-10, which incidentally is transported in the bloodstream by lipoproteins also, is thought to be the first antioxidant depleted when LDL is subjected to oxidation thus furthering the plaque forming process. The importance of this is illustrated by the fact that in 1989 the pharmaceutical giant,*Merck,* received a US patent permitting them to add CoQ-10 to their "statins" *Mevacor* and *Zocor*.[16] However, to date, they have seen no financial need to do so.

PAULING'S FINAL PIECES OF THE PUZZLE – L-LYSINE & L-PROLINE

Pauling repeated many of the experiments previously cited, and he found that vitamin C did in fact help reverse some of the plaque in heart disease (remember, vitamin C chemically changes the end amino acid residues). However, there were still blockages in the blood vessels of the experimental animals.

Remembering that plaques formed *only in the damaged areas of the vessels* -- and that damaged collagen "looked like a frayed 3 strand rope" -- he theorized there would be bonding sites (receptors) on the Lp(a) that would be specific to the amino acid fragments of collagen (glycine, lysine, and proline).

Being ubiquitous in the body, Pauling ruled L-glycine out, reasoning that Lp(a) would not stick anywhere there is a glycine moiety (glycine particle).

L-Lysine Binds with Lp(a) Receptors

Pauling then turned his attention to L-lysine, hypothesizing that lysine receptors on the Lp(a) may account for why Lp(a) sticks exclusively to the damaged collagen fibers.

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[14] Spittle CR. 1971. Atherosclerosis and Vitamin C. Lancet, Dec 11;(18):pp.1280-1.
[15] Harwood HJ Jr, Greene YJ, Stacpoole PW. Inhibition of Human Leukocyte HMG-CoA Reductase Activity by Ascorbic Acid. J Biol Chem. 1986 Jun 5; 261 (16):pp7127-35.
[16] US Patent No. 4933165

To understand what Pauling was up to with lysine, it's useful to imagine the way the body uses antihistamines. An antihistamine binds to histamine receptors (steals their parking places if you will) and thus preventing allergens from attaching and causing an allergic response.

Similarly, Pauling added L-lysine to the vitamin C he gave his test animals. Sure enough, the Lp(a) became *way less sticky* and more of the plaques were removed. The L-lysine essentially acts as a "male" end of a plug to the "female" receptors in Lp(a). This is how Lp(a) attaches to the broken strands -- just like a plug in a wall socket. When there is extra L-lysine circulating in the bloodstream, the L-lysine "plugs in" to and seals Lp(a)'s "sockets", thus creating a smooth, inert Lp(a) particle, which can no longer adhere to the body. It is exactly like a parent putting a childproof plug into an empty outlet -- no other plugs *or little fingers* are able to attach.

While the extra lysine is "sealing the sockets" on L(p)a, remember that vitamin C has changed the "plug" itself. By converting the end amino acids to "hydroxyaminos", it essentially replaced the "standard plug" with a "European" type plug. Now there is no way at all for the L(p)a to "make the connection."

In chemistry we say different reactants (here vitamin C and L(p)a) have different *affinities* to the same *substrate* (the dangling amino acids of the broken collagen fiber). The reactant with the greatest *affinity* will preferentially bind to the substrate and displace reactants already bound but having a weaker affinity to the substrate (in this case, the "good guys" have the greatest affinity and the "bad guys" fall off). For all of these reasons, one can readily see that the combination of L-lysine and vitamin C is indeed a very powerful "plaque-buster". The combination is, in fact, so powerful that Pauling and Rath were awarded a U.S. patent for a solution containing ascorbate and L-lysine to remove plaques from donor organs prior to transplant surgery.[17]

You see, once a transplanted organ is in place, blood must quickly perfuse through the new organ or areas of tissue will necrose (die). Bathing transplanted organs in their vitamin C-lysine solution prior to implantation quickly removed any plaques in the major vessels and greatly enhanced transplantation outcomes. Pretty impressive for vitamin C and a lowly amino acid, don't you think?

L-Proline binds with Additional Lp(a) Receptors

Encouraged by their results with L-lysine, Pauling and Rath began to look at L-proline. L-proline is a unique amino acid, with a five-member ring structure, which contains the amine portion of the molecule (it's the only *imino* amino acid).

The biochemical significance of this is that L-proline *prefers to be in oil* rather than water. L-proline is thus *lipophilic* as opposed to the *hydrophilic* L-lysine.

Since Lp(a) is a combination of a water-loving protein (apo a) and the oily cholesterol, Pauling and Rath hypothesized that lipophilic proline would block any receptors that might

[17] US Patent No. 5230996 Pauling/Rath (1993). A Proceedure for the Cleansing/Removal of Atherosclerotic Plaque from Human Organs During Transplant Surgery.

exist on the oily portion of Lp(a). When they added L-proline to their vitamin C-lysine solution, the effects were astonishing. Blockages completely cleared.

By having extra L-proline in the bloodstream (in addition to the supplemented L-lysine and vitamin C) all of the receptor sites on the L(p)a are "sealed" and the molecule does ,in essence, become "Teflon coated".

With sufficient supply, vitamin C preferentially binds to and *hydroxylates* (chemically alters) dangling lysine and proline ends (in areas where the artery was damaged). After hydroxylation, the lysine and proline strands in vessel walls no longer "fit" the Lp(a)'s receptor sites, and some of the Lp(a) particles (or plaque patches) start to strip away from the vessel walls. The experiments of Willis and Spittle previously cited confirm this process.

SOME FINAL THOUGHTS

The conundrum of "causation" versus "correlation" – it's an age-old question and is an important question when it comes to cardiovascular disease.

Think about a child who has seen a number of house fires. He correctly observes that firemen are always present at house fires and concludes, erroneously, that *firemen must cause these fires.* The child does not yet understand that the firemen are actually there to save the day.

It's the same thing with the Lipid Theory, where cholesterol is seen as an evil cause of cardiovascular disease, simply because it is highly correlated with the disease.

With the Unified Theory, we instead view cholesterol, homocysteine, C-reactive protein, and Lp(a) for what they really are: the body's dire attempt to save itself. These so-called "bad guys" are really just markers of malnutrition and proliferate when the body is under stress.

Treating the symptoms of nutritional deficiency with drugs becomes nothing more than an experiment, where we get to observe the toxic effects on a malnourished body. Unfortunately, it has now become standard to treat side effects with other drugs. And, in my profession, this is called *polypharmacy.*

As a first-year pharmacy student, I was told over and over again: "*Never* practice polypharmacy!" Instead, we were taught to replace the offending therapy to get rid of unwelcome or dangerous side effects. Not so today, where prescriptions are layered on top of one another.

As a *cautionary note*, while polypharmacy is generally considered a bad professional practice, I am not advocating that anyone reading this article drop their prescriptions (there may be some extenuatuing circumstances - i.e., an allergy or intolerance to an alternative therapy). I am advocating for informed discussions with medical practitioners, as well as the addition of a nutritional approach to *supplement* conventional approaches.

Meanwhile, I often have wondered what it would be like if first-year medical, nursing, and pharmacy students were introduced to the Unified Theory? I'm not naïve enough to expect this any time soon, but part of writing this paper was about documenting good science that merits more attention by mainstream medicine.

I find it practically criminal that, despite overwhelming scientific evidence, the Center for the Study of Alternative and Complementary Medicine of the NIH has *not done one clinical trial* to test Pauling's and Rath's work. The Center is funded by tax dollars, so you would think that an incredibly affordable solution to the number one cause of death in this country would get some attention, but, alas, not so far.

Meanwhile, I feel privileged to get information on the Unified Theory in front of so many people through Our Health Co-op. May my article be read widely and contribute not only to better awareness, but also to making the case for formal clinical trial research -- someday!

Until then, here's to your health and to our community!

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About Our Health Co-op

Founded in February 2002, Our Health Co-op is dedicated to healthy aging through scientifically-promising health products made affordable.

Our Health Co-op specializes in serving those with limited means, particularly fixed-income seniors. The company emphasizes both independent laboratory testing to ensure quality and chatty humor as part of its community orientation to well-being.

Headquartered in Riveria, FL, Our Health Co-op has an office in Salt Lake City, UT. For more information, see www.ourhealthcoop.com.

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