Foreword and First Chapter reprinted from …..

The Dark Side of Statins

By Dr Duane Graveline MD MPH

Former US Air Force and US Army Flight Surgeon, NASA Astronaut and Family Doctor
www.spacedoc.com

"The first chapter is by my fellow THINCS (The International Network of Cholesterol Skeptics) member **Glyn Wainwright**. I first wanted to cover the critical role that cholesterol plays in human health. Glyn's excellent illustrated summary – The Wonder of Cholesterol – was perfect for this and he kindly allowed me to use it in this book."

Duane Graveline MD MPH (from the Preface)

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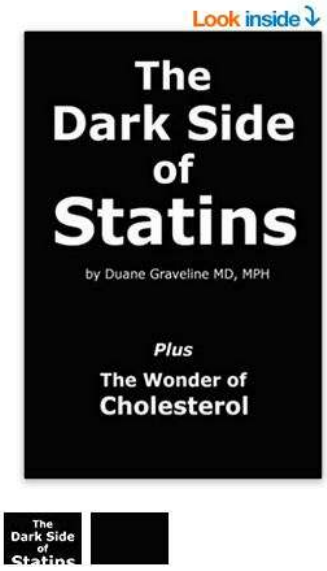

The Dark Side of Statins: Plus: The Wonder of Cholesterol Paperback – 16 Aug 2017

by Duane Graveline MD (Author)

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This is Dr. Duane Graveline's fourth and final book on statin drugs.

This new (August 2017) and expanded edition of The Dark Side of Statins completes the series.

All four of his statin books are equally informative, but he considered this to be both the best and the most important of his works.

Foreword

by Glyn Wainwright MSc MBCS CEng CITP

This book is an exposé of the current, powerfully marketed myth that we can fix, or even prevent, certain of our health problems by consuming a reductase inhibitor – better known to all as a statin.

Statins are 'toxic medicines' that claim to save us by attacking our ability to synthesize those very things that sustain and repair us in our daily lives, the products of our naturally evolved metabolism. Dr. Graveline describes a celebrated metabolic system known to biochemists as our 'Mevalonate Metabolic Pathway', and the problems that arise from attempting to blockade this vital process from taking place in every living cell in our bodies.

Everyone who has experience with statins or cares for someone who has been damaged by statins needs to understand the issues and testimony documented within. Dr. Graveline uses a powerful mix of descriptive explanations, case reports and scientific research references to inform everyone from lay-person to expert.

He exposes a grave historic error in our interpretation of the role of fats and lipids in heart disease. This failed statin 'miracle cure' was based upon the false premise that fats and cholesterol, a major factor in the evolution of complex cells and animals, suddenly became a bad thing in the late 20th Century.

Having something for everyone, this book will inform those who have experienced adverse reactions to statin drugs and those involved as family, friends and caregivers. There is discussion and guidance of dietary changes and supplements, which may help to mitigate the more extreme damage done by statins. Understanding the damage to mitochondria, coenzyme Q10, cholesterol, dolichols and the protective role of antioxidants is an important step in addressing the damage.

Guidance to medical doctors regarding statin adverse effects encourages the sympathetic discussion of these adverse reactions. The case reports in this book show the many ways in which statins can impact the lives of patients. The extensive personal testimony collected by Dr. Graveline is a measured and emotionally powerful perspective on the public's response to the stain crisis.

These patients' accounts of adverse effects should be taken very seriously by health professionals. They should be used to confront the powerful corporations who have misguidedly promoted ever-wider use of statins, and to expose the often devastating consequences of long-term mevalonate blockade

In this fourth book in the series, Dr. Graveline pays particular attention to the damage done by statins to our mitochondrial DNA, the parts of each cell which converts metabolic energy into cellular power known as adenosine triphosphate (ATP). Our mitochondrial DNA is normally protected by the products of the mevalonate metabolic pathway.

It is poignant and disturbing that, having discovered the devastation of cholesterol lowering drugs, many patients continue to seek other ways of reducing their cholesterol levels.

It is important to combat the corporate promotion of the erroneous and harmful myth of 'bad' cholesterol.
The consequence of this error is extensive statin generated damage to all tissues and mitochondrial function in particular, as explored here by Dr. Graveline.

THE WONDER OF CHOLESTEROL

Chapter Summary and Key Points.

- Our cells need cholesterol to function properly.
- High cholesterol is more often associated with good health and longevity.
- The rising obesity rates observed by the 1980s coincided with the widespread adoption of high-sugar, low-fat formulations in processed foods.
- Fats do not cause obesity or disease. It is the excess sugars which create abdominal obesity.
- Cholesterol levels are not measured, merely estimated from the quantity of very useful 'lipid droplets' circulating in your blood.
- LDL cholesterol does many jobs, like transporting vital fat soluble vitamins, lipids and proteins. Sugar-damaged LDL is not recognized by the organs that need these essential nutrients.
- It doesn't matter how high your total blood serum cholesterol is. What really counts is the damaged condition of the LDL lipids.
- As we age, excessive amounts of free sugars in the blood may eventually cause damage quicker than the body can repair it.
- HbA1c is a widely available blood test to check for sugar damage.
- Cholesterol lowering drugs can cause diabetes.
- Cholesterol is vital for brain and nervous system functions.
- Behavioral changes, personality changes and dementias have all been associated with low levels of cholesterol.
- Cholesterol lowering drugs are known to cause adverse side effects in anywhere from 10% to 30% of patients.

When you are asked to reduce your ‘cholesterol level’, why not ask a question which is fundamental to your health: ‘Why?’

Ask “What does cholesterol do and why do we make and store so much of it in all the cells in our bodies?”
The answer is astonishingly simple: all our cells need cholesterol to function properly.

The High Cholesterol Paradox

Being told you have ‘high cholesterol’ is commonly taken as a sign of an unhealthy destiny. However, for many elderly people, the news that they have ‘high cholesterol’ is more often associated with good health and longevity. [1]
For over 50 years this has been a paradox, the ‘High-Cholesterol Paradox’.
What is really going on? Let us look at the scientific facts.

Hypothesis becomes Dogma

In the 1950s the prestigious American medical doctor, Dr Ancel Keys, supported a popular theory that heart disease was caused by dietary Fats and Cholesterol (Lipids) circulating in the blood.[2] In 1972 a British Professor, Dr John Yudkin, published a book called ‘Pure, White and Deadly’ which proposed over-consumption of refined sugar as the leading cause of diabetes and heart disease.[3]

The science was contested by ‘interested parties’, and the matter was resolved by ‘government decree’ in a US Senate report.

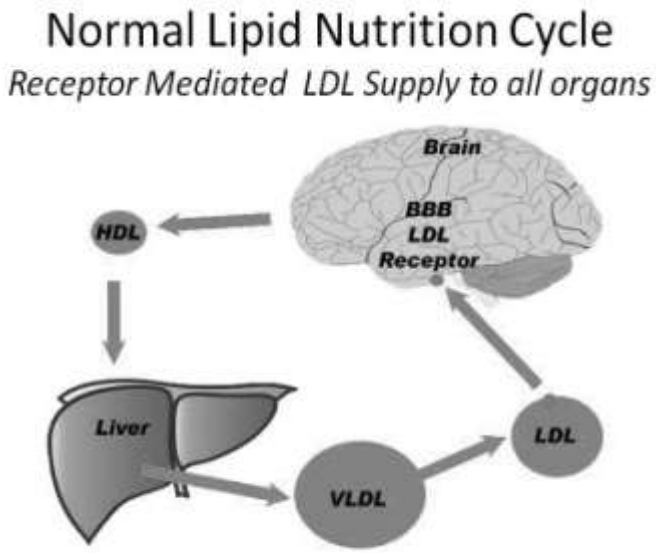
On Friday January 14th 1977, Senator George McGovern’s Senate Select Committee on Nutrition and Human Needs published ‘*Dietary Goals for the United States*’.

This document sided heavily with Dr Keys’ lipid theory. Thus ‘hypothesis became dogma’, without the benefit of scientific proof. The McGovern report recommended that we consume more carbohydrates (sugar generating foods) with more limited amounts of fats, meat and dairy. Since the 1970s there has been a rise in the use of High-Fructose Corn Syrups in processed food, and the introduction of low-fat foods which tend to have added sugar to make them attractive to eat.

Until the 1970s there had been a small but consistent percentage of overweight and obese people in the population. By the 1980s obesity rates had begun to climb significantly. This sudden acceleration of obesity is very closely associated with the adoption of new high-sugar, low-fat formulations in processed foods - the consequences of the McGovern report recommendations being adopted around the world.

Advice to reduce our intake of saturated fats, obtained from meat and dairy, caused a rise in the use of plant based oils and so-called ‘vegetable fats’. This was misleadingly promoted as healthy. The biochemical destiny of dietary saturated fat is not the same as that of excess carbohydrates and sugars’.

Figure 1.



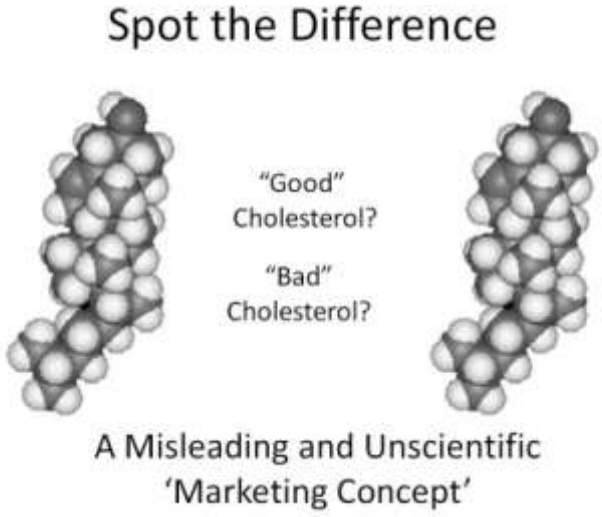
Fats do not cause obesity or disease. It is the excess sugars (glucose and fructose and refined fructose from High Fructose Corn Syrup HFCS) which create abdominal obesity.[4]

The erroneous idea, and fear, of artery blocking fats was exploited to market fat substitutes. Invite anyone talking about 'artery blocking fats' to hold a pat of butter in a closed fist. As the butter melts and runs out between their fingers, ask 'How do fats, which are evolved to be fluids at body temperature, block the vascular 'pipes' in our bodies?'

Plant oils are not the natural lipids for maintaining healthy human or animal cell membranes. Animal sourced fats, and essential fatty acids (EFA), are identical to those we require for the maintenance of the healthy human body.

Good Cholesterol? Bad Cholesterol?

Figure 2.



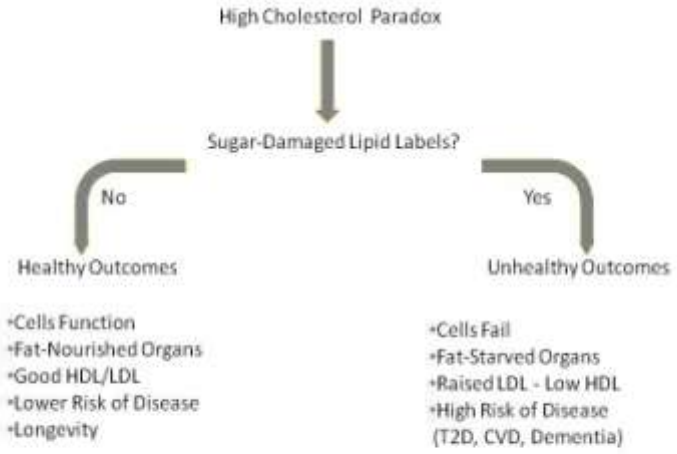
All biochemists can confirm that all cholesterol molecules throughout the known universe are identical in every respect. So how can there be ‘good’ or ‘bad’ cholesterol. It is now possible to frighten people with unscientific descriptions like ‘Good’ and ‘Bad’ when talking about cholesterol.

This single misleading description may have prevented a whole generation from knowing the true causes of the very real disturbance in the levels of fatty nutrients (Lipids) circulating in our blood.[4]

Healthy Lipid Nutrition Cycle

The delivery of fatty nutrients is a very well regulated cycle, a ‘parcel service’ of ‘lipid wraps’ (LDL and HDL lipid particles) circulating in the blood. These lipid wraps, HDLs and LDLs, complete with apo-protein 'labels' assist the delivery (by receptor mediated endocytosis) of fat soluble materials and proteins from where they are sourced (the liver) to where they are needed (the organs).[7]

The measuring of these lipid wraps in your blood is used to estimate the numbers which you are given by your doctor as the ‘cholesterol’ level. No-one really measures the cholesterol; it is merely estimated from the quantity of very useful ‘lipid droplets’ circulating in your blood.



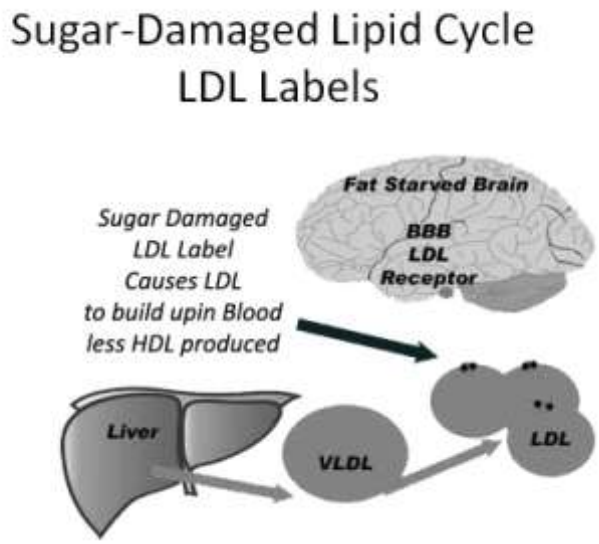
These large ‘lipid wraps’ (LDL) have evolved to do lots of jobs, like transporting vital resources such as fat soluble vitamins, lipids and proteins. The cholesterol, in our lipid wraps, forms an essential part of our fatty nutrition and immune systems.

If the total blood serum cholesterol (TBSC) is high and the organs are getting enough lipids, the blood lipid circulation is healthy. The large parcels of fatty nutrients (LDL lipids) sent by the liver are consumed by our organs (receptor-mediated endocytosis) and the smaller fatty wrappers and left-over lipids (HDL Lipids) return to the liver. The Fatty Nutrients (LDL) and the recycled lipids (HDL) are in balance. Such a healthy-lipid ‘high-cholesterol’ person is well nourished and likely to have a long and healthy life.

Sugar-Damaged Lipid Nutrition – Broken Lipid Cycles

If the total blood serum cholesterol is high but the fatty nutrient droplets (LDLs) have sugar-damaged protein labels, the organs are unable to recognize and feed on them. The supply of fatty nutrients to organs is broken. This can happen in the decades leading up to the onset of mature-onset (type 2) diabetes.

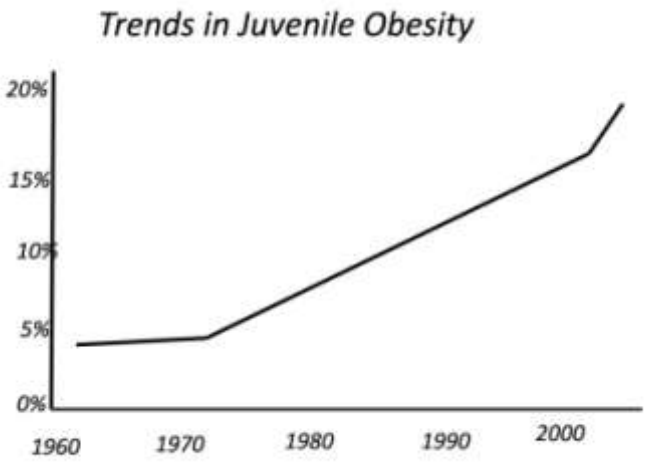
The liver continues to supply fatty nutrients (albeit with damaged LDL labels), but the organs' receptors are unable to recognize them. The organs thus become starved of their fatty nutrients. Like badly labelled parcels in a postal service, the sugar-damaged lipids build up in the blood (raised LDL) and fewer empty wrappers are returned to the liver (low HDL).



The sugar damaged LDL (erroneously called 'bad' cholesterol) in the bloodstream appears to be high. It has not been recognized by the organs that need it. It circulates in the blood, awaiting clearance by the liver. Consequently, there is less HDL (erroneously called 'good' cholesterol) to be returned by the organs.

High Cholesterol (high levels of total blood serum cholesterol TBSC) when caused by damage to the LDL lipid parcels is a sign that lipid circulation is broken. These fats (LDL) will be scavenged to become visceral fats, deposited around the abdomen. This type of damage is associated with poor health. Our organs, such as the brain and heart, would be in a sorry state if the LDL circulating in our blood stream was depleted or damaged.
This is where it all went wrong. In diabetics and pre-diabetics, the raised LDL was seen as overproduction and mislabeled as 'bad' cholesterol. We now know that it was a lack of consumption of LDL that caused its elevated levels in the blood, caused by Glycation (sugar attachment to lipo-proteins)

So it really doesn't matter how high your total blood serum cholesterol (TBSC) is. What really counts is the damaged condition of the blood's fatty nutrient parcels (LDL lipids). In a research review of metabolic syndromes (e.g. diabetes, heart disease, obesity, arthritis and dementia) our associates explained that the major cause of lipid damage was sugar-related.[4]



Sugar Damage (AGEs)

The abbreviation AGE (**A**dvanced **G**lycation **E**nd-product) is used to describe any sugar-damaged protein. As we age, excessive amounts of free sugars in the blood may eventually cause damage quicker than the body can repair it.[5] The sugars attach by a chemical reaction and the sugar called fructose is known to be 10 times more reactive, and therefore more dangerous than our normal blood sugar (glucose).

Since the 1970s we have been using increasing quantities of refined fructose (from high-fructose corn syrup). Its appealing sweetness, and ability to suppress the 'hunger controls' (ghrelin/leptin receptors) is driving excessive food intake.[6] Its ability to damage our fatty nutrients and lipid circulation is also driving waist-line obesity and its associated health problems.[4,7]

Checking for Damage in our Lipids

There is a 'simple to administer' commonly available blood test used to check for sugar damage. It is used to check the proteins in the blood of people who are diabetic or at risk of becoming diabetic. It tests for Glycated Hemoglobin (HbA1c) by counting the proportion of damaged molecules (per 1000) of Hemoglobin protein in the blood (mmol/mol).

Researchers looking at ways of testing for damage to lipids, have found that the sugar-damaged blood protein test (HbA1c), presents a very reasonable approximation of the state of sugar-damage in the blood lipids. Until there is a good general test for sugar-damage in blood lipids, this test (HbA1c) could be a sensible surrogate. This is a better way of assessing health than a simple cholesterol test (TBSC).
Improved sugar-damaged blood protein (HbA1c) scores in diabetic patients is accompanied by improvements in their lipid profiles. This could be very useful to anyone wanting to improve health outcomes by managing lifestyle and nutrition.

Clinical Consequences of Lowering Cholesterol

In 2008 Dr Luca Mascitelli asked me to examine a paper by Xia et al.[8] It was very interesting to note that lowering cholesterol by as little as 10% (molecular in cell walls) in the pancreas (pancreatic beta-cells) prevented the release of insulin (cholesterol-mediated exocytosis).

This paper described a mechanism by which 'cholesterol lowering drugs' directly cause diabetes. It was known that in statin drug trials which looked at glucose (blood sugar) control there was poor

blood-sugar control in the statin user groups. Since 2011 the USA government (FDA) required statins to carry a warning about the risk of causing diabetes.[9]

Memories are made of this – Cholesterol

The healthy human brain may only be 5% of body weight but it requires over 25% of the body's cholesterol. The nervous system uses huge quantities of cholesterol for insulation, protection and structure (myelin). F.W. Pfrieger et al. have shown that the formation of the memory (synapses) is dependent on good supplies of cholesterol.[10] Dr. Duane Graveline has researched and written extensively on the huge impact of statins on brain function.[11]

Post-mortem studies show that depleted cholesterol levels in the cerebrospinal fluids are a key feature of dementias. It was also reported that behavioral changes and personality changes are associated with low levels of cerebrospinal cholesterol.

In another review paper on dementia, our associates commented extensively on the damage done by fructose and the depletion of cholesterol availability.[7] Low cholesterol levels in the nervous system are not conducive to good mental health.

Consequences of Lowering Cholesterol

Drug treatments which lower cholesterol are acknowledged to cause adverse side-effects (ADRs) in at least 10% of statin users.[12] This figure may be as high as 30%.

Conservative estimates indicate that in at least 1% of patients the side-effects are serious enough to be life threatening (e.g. rhabdomyelitis, dementia, behavioral disorders and violence).

Our review[13] found that cholesterol lowering therapies were implicated in:

- Damage to muscles (including the heart) and exercise intolerance.[14]
- Increased risk of dementias (impaired synaptogenesis and neuro-transmission).[15]
- Failure of myelin maintenance (multiple sclerosis risks).[16]
- Neuro-muscular problems, aches and pains (amyotrophic lateral sclerosis - ALS).[17]
- Diabetes (insulin release inhibited).[8]
- Poor Maintenance of bones and joints.
- Suppression of protective skin secretions (Apo-B) and increased MRSA infection.[18]
- Raised incidence of cancers.[19]

Again - Why would anyone want to lower cholesterol?

The Vital Role of Cholesterol

In history, cholesterol became an important evolutionary player when simple cellular organisms began evolving into the complex eukaryotic cells that gave rise to the animals.

The complexity of animal cells would not be possible without all the membrane structures that define the components of our cells. The nucleus, the mitochondria, the cell walls the golgi apparatus, the endoplasmic reticulum–all parts of our cells are contained by bi-lipid membranes. These bi-lipid membranes are a double layer of molecules with a fatty middle (lipophilic) and a water-attracting outer layer (hydrophilic).

While these membranes are basically the wrappers, they are also very dynamic functionally active structures. They support and wrap the cell's contents. They support lipid traffic into and out of the cell and its organelles (internal cell parts).

To do this they need to be flexible, strong and changeable. A molecule is needed to move around the cell membrane, organizing and changing the bi-lipid layers in support of all its cellular functions. Only one molecule is uniquely suitable for this role – we know it as cholesterol.[20]

Many of the personal accounts quoted by Dr. Graveline in this book and in his other statin books, arise from the realization that it is cholesterol-reducing statins which are responsible for the many adverse health outcomes reported.

Therefore it is a puzzle that even after this experience, some people still feel compelled to reduce their cholesterol levels. The problem is no one seems to question the food and drug companies' advertising campaigns, which relentlessly depict our hero 'Mr Cholesterol' as one of the bad guys!

Over the last 40 years, even our doctors have been convinced by this incessant propaganda campaign against cholesterol. There has never been a scientific study that has been able to demonstrate a causal link between cholesterol and heart disease. The reality is that cholesterol, in its natural form, cannot be harmful.[21]

This has not stopped the marketing divisions of the pharmaceutical and food industries from creating an amazingly powerful and valuable myth, about an infamous household dietary and medical obsession, 'Bad Cholesterol'.

This has led to the shunning of natural cholesterol-rich foods, like butter, full-fat cheese, whole milk and eggs, in favor of processed vegetable trans-fats and even sugar-rich low-fat foods.[19]

With doctors convinced that cholesterol was the cause of heart attacks and strokes, we switched to statins and unnatural fats like margarines. The harmful effects of trans-fats were exposed in the 1950s by Professor Fred Kummerow and most recently in his 2014 book *Cholesterol is Not the Culprit*: *A Guide to Preventing Heart Disease.*

The result of these 'commercial myths' is that the word cholesterol frightens people away from eggs and milk and butter and cheese — long established as the most natural foods for creating and rearing healthy offspring.

There is no evil in a cholesterol molecule. Cholesterol is just an incredibly fortunate breakthrough in the evolution of our cellular membrane chemistry.[20]

Almost all the cholesterol in our bodies is found in the cell membranes. An average of 20% of all the molecules in these cellular membranes is cholesterol. Over the past decade there has been a large increase in research publications documenting the action of cholesterol-rich lipid rafts within these membranes. Lipid research now describes an amazingly busy schedule for our membrane-cholesterol molecules which support everything we do.

With the emergence of the cholesterol-rich lipid raft hypothesis, the role of cholesterol in membrane function has become a focus of new research into exocytosis and endocytosis. Exocytosis is the process by which a cell guides the contents of secretory vesicles out of the cell membrane as lipid wraps.

These lipid wraps (membrane-bound vesicles) contain soluble proteins and lipids destined for functions outside of the originating cell. Endocytosis is the process by which cells absorb these lipid

wraps and the molecules they carry (such as proteins) from outside the cell by engulfing it within their cell membrane, amoeba fashion.[22]

Lipid rafts are very rich in cholesterol, which organizes the other biochemicals in support of a variety of trans-membrane functions vital to cell recognition and communication.[23] This cholesterol has been shown to strengthen and stabilize the functional areas of the cell membrane. The physical consequences of cholesterol enrichment on the strength and thickness of membrane lipid-rafts were modeled and demonstrated by de Meyer, et al.[24]

The mediation of lipid membrane form and function by cholesterol affects the ability of a cell to perform exocytosis and endocytosis. The current trend in cardiovascular medicine to promote cholesterol reduction has caused concern in other non-cardiovascular branches of medicine. A growing number of researchers have identified the effects of cholesterol depletion on exocytosis and endocytosis with adverse consequences for our health.

Cholesterol depletion occurs when cholesterol levels in the body have been artificially lowered too far, as through the use of statins — reductase inhibitors. The inhibition of cholesterol synthesis is associated with functional failure of cholesterol-rich lipid rafts in processes such as exocytosis and endocytosis.

Cholesterol-rich lipid rafts support the receptor used to capture (SNARE) the lipid particles required by the cell.[25] These lipid particles transport the specific larger proteins and fat-soluble molecules that cannot simply pass by random diffusion through the cell membrane. This is a highly organized and vital lipid transport system mediated by cholesterol.

The cholesterol in the cell membrane also protects the cells' contents from leakage and loss. This is important when the cell stores energy, ready for some activity like thought (neuron) or exercise (muscle cell). The process of creating energy in all cells is dependent upon what is called electrochemical gradients, the accumulation of sodium ions or protons on one side of the membrane compared to the other, by pumping action through specialized membrane pores.

All animal plasma membranes use sodium for this purpose (members of the plant community use protons.) If adenosine triphosphate (ATP) is the gasoline that fuels our cells, sodium electrochemical gradients are the basis of the process that makes the ATP. The accumulation of sodium ions is accompanied by a natural leakage rate.

The reason for the cholesterol-rich layer of lipids in these plasma membranes is to prevent this leakage. Haines proposed in 2001 that cholesterol is a key inhibitor of sodium ion leakage.[26] Low cholesterol or any process that artificially lowers membrane cholesterol below natural limits must interfere with ATP production.

Of particular interest is Haines' discussion of comparable roles for both coenzyme Q10 and dolichols. Haines maintains that dolichols are responsible for leakage control of lipid for lysosomes (our intracellular disposal units), and ubiquinone (CoQ10) serves as control for our mitochondrial sodium ion leakage. Since statin drugs inhibit the synthesis of all three of these lipids (cholesterol, dolichol and ubiquinone) by the mevalonate blockade common to all reductase inhibitors, the work of Haines and other biochemists serves to document the inevitability of both mitochondrial damage and energy loss associated with statin use.

Haines has also studied the evolution of this mechanism from single cell through multi-cellular organisms, finding that the use of cholesterol for the purpose of inhibiting sodium leakage appeared

very early, and has persisted for billions of years as the mainstay of ATP synthesis, demonstrating the persistence of successful mechanisms.

You can imagine that in the case of neurons and nerves, all the electrical activity transmitted down the connecting axions requires an elaborate containment of impulse and defense against burn out (oxidation).[16] Our neurons and nerves have evolved a protective wrapping of myelin. This myelin is continuously maintained by specialist cells. Oligodendrocytes maintain the myelin wrapping for the neurons, and Schwann cells maintain the myelin sheathing for our nerves.

Myelin is made up of about 40% cholesterol molecules and cholesterol is a very good insulator and antioxidant. Such large quantities of cholesterol, required by the oligodendrocytes, are supplied to the brain as an LDL lipid wrap called alipoprotein E. The detail of the association of cholesterol depletion with dementias is investigated in the book by Dr Henry Lorin, *Alzheimer's Solved*.[27]

Pfrieger's landmark publication in 2003 of the vital role of cholesterol in the formation and function of memory synapses, has been followed by one research report after another documenting the importance of cholesterol and the wide ranging demand for cholesterol in so many of our vital bodily functions, including nerve, muscle and even personality.[10]

When looking at cholesterol research, I asked the basic question: 'What happens if you lower cholesterol levels in all tissues and organs throughout the body?' In answer, a catalogue of serious problems became evident and was already investigated and described in the literature.
A review paper, *Cholesterol-lowering therapy and cell membranes. Stable plaque at the expense of unstable membranes?* that I co-authored with L Mascitelli & M Goldstein, is now regarded as seminal and has been widely cited in other works.[13]

A retrospective analysis of a five-year trial showed a 30% increase in the incidence of diabetes associated with a cholesterol reduction therapy. More recently Xia et al demonstrated a causal link between membrane cholesterol lowering and the impairment of insulin granule release.[8] The effect of statins on glucose levels is well documented in a retrospective analysis of the JUPITER trial. Ridker discusses the effects of statin therapy on incident diabetes, having presented data showing that statins significantly promote diabetes in 6 out of the 7 trials listed.[28], [31] Our internal insulin supply is facilitated by cholesterol.

The impairment of the exocytosis of myelin has been cited as an explanation of the reduced myelination of our glial cells and Schwann cells (a form of glial cell responsible for the myelination of nerve fibers) critical for neural maintenance[16]. The maintenance and repair of our neurons and nerves requires huge amounts of cholesterol.

Inconclusive results in the use of statins for the reduction of bone loss and statin-associated fracture have implications for both the osteocyte (bone cell) and osteoblast (bone formation) action in bone remodeling.[29] The function of osteocytes and osteoblasts are mediated by cholesterol-rich lipid raft through exocytosis and endocytosis. Bone repair and maintenance cells need cholesterol to function.

The loss of exocytotic secretions of Apolipoprotein B, our major lipoprotein cholesterol carrier (LDL), and its role in immunosuppression, has been cited with regard to invasive skin infection.[18] Cholesterol-lipid particles are a big part of our ability to control and stop infections.
The diminished exocytosis of neuromuscular junction (Agrin, LRP4 and MuSK) enzyme secretions has implications for associated neuromuscular junction disease symptoms (ALS), similar to myasthenia gravis, observed in long term statin use.[30] This is another instance where low levels of cholesterol can cause neuro-muscular problems.

The neurological effects of cholesterol depletion can produce a wide range of mental conditions. Commonly, severe anger and irritability may occur in many statin users. But depression, violent behavior, homicidal behavior and suicide are also known to be associated with cholesterol depletion.[13]

As Dr. Graveline explains later, neural systems have significant vulnerability to cholesterol depletion through the reduction in the synaptic function, using lipoprotein neuro-transmitters. Another greater vulnerability may be due to the loss of myelination in the protection of neurons from damage.[16] The protection of our brains and the creation of our every thought requires significant amounts of cholesterol.

Cholesterol is not only the most common organic molecule in our brains, it is also distributed intimately throughout the entire body. Additionally, cholesterol is the precursor for a whole class of hormones known as the steroid hormones that are absolutely critical for life as we know it. Such hormones include estrogen, progesterone, testosterone, aldosterone, cortisol and calcitrol (vitamin D).

These hormones determine our sexuality, control the reproductive process, and regulate blood sugar levels and mineral metabolism. And beyond this, there is yet another class of cholesterol's steroid offspring without which our metabolic well-being might be in serious jeopardy: the production of bile acids. Bile makes it possible for us to emulsify fats and other nutrients. Without bile, we could not digest and absorb the fats in our diet and must slowly starve.

We must also note that HMG-CoA reductase, the key enzyme target for statin use, is found in the membrane walls of the endoplasmic reticulum and the mitochondrial wall. These cell membranes contain between 20% and 50% cholesterol molecules. Our membrane-cholesterol is the very biochemical essence of our existence. How could anyone contemplate getting rid of it? What is needed is a lowering of damage to lipids — caused by sugar.

The large amount of cholesterol required for both the formation and function of these basic structures argues strongly against the rationality of excessively liberal use of drugs such as statins that inhibit cholesterol synthesis.

Based upon a presentation to the European Conference Weston A. Price Foundation, London 2014

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