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The Great Cholesterol Quandary and Global Consciousness Awakening

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Abstract

Ancestors of human species have been living on the planet earth for about six million years while the modern form of humans only evolved about 200,000 years ago. Civilization is only about 6000 years old, and industrialization started in the 1800s. World population interconnectedness is enhanced with love and mercy but disrupted with dishonest, lies and wars. Human species has been feeding on animal fat as the finest and prime source of energy for all human history. In 1953 Ancel Keys and collaborates illusionized human kind that their historical source of energy is toxic. Massive health, behavioral as well as economic consequences followed. Human hearts known historically to be the seat of spirit, soul and emotions is proven by our group to orchestrate with global energetic fields. Global consciousness as represented by the planetary electromagnetic fields is in critical need to be feed with awakening merciful information. Documents refuting Ancek Keys dogma and reverting humanity to the truth in their energy source is presented in this review. The harmony we expect is not only physiological but will affect our global planetary environment for the wellbeing of human species in earth.

Keywords

Atherosclerosis, Cholesterol, Statin, Nobel Laureates, Hypocholesterolemia, Global Consciousness

1. Introduction

Progress and advances in medical practice and updates in guidelines for cardiovascular diseases and in medicine in general are the role of the medicine profession. Medical professionals occupy higher respect in their communities as the source of health preservation, wellbeing, trust and wisdom. It is always shocking to discover the opposite. Since the dawn of humanity, higher wisdom and raising

global consciousness is a holy mission. The fact that heart disease rank number one killer in most world nations according to World Health Organization (WHO) records in the last decades denotes a very important fact: *the road map needs urgent review*. Cardiovascular diseases risk factors must be exposed to critical investigations. Since 2006, the medical literature witnessed increasing number of respectful reports and reviews questioning the contribution of cholesterol, in specific, Low Density Lipoprotein (LDL), in the process of human atherosclerosis and Coronary Heart Disease (CHD). In this review we present a well balanced evaluation of this critical matter, illustrating the historical roots of the cholesterol since it was described in the early nineteenth century and the journey of its development, in specific its proposed role in the genesis of atherosclerosis. In addition, we will elaborate in the fact of the statistical deception in the major trials where today's practice sanctification protocols derived. Voluminous reviews defeating the cholesterol theory will be reported as well as the cherry picking process of the updated guidelines that are avoiding selectively all the medical literature questioning or rejecting the cholesterol hypothesis. In the era where humanity is crossing 8 billion population, the demand for raising our global consciousness is at its highest priority. Lies and conflicts of interest especially in medicine and the pharmaceutical industry are creating huge crack in the merciful interconnectedness of humanity. We humans are integral sacred part of the astrophysical energetic universe. Our heart rate variability (HRV) frequencies are beating and orchestrating with the intricate great universe. The reform efforts in the human history are the mission of prophets. We, physicians and scientists are the heirs of the prophets in our fields. Strict scientific evidence should not be exposed to any compromise and the total exclusion of the impact of pharmaceutical forces on the exact role of LDL in heart diseases must be implemented. Wisdom must prevail to prevent the chaos in our planet.

2. Historical Perspective

2.1. Atherosclerosis from Pharonic Papyrus Documents until Nikolai Anichkov

Review of human history with eye of wisdom and heart of mind should reveal astonishing facts and events. Medical history is not exemption. The cholesterol hypothesis links cholesterol intake and cholesterol blood levels to cardiovascular disease constitute cornerstone of mainstream medicine in the last seven decades. It has had enormous impact on health care services, health economics and world population well being for decades, but has little or no scientific back ground that is relevant for the human species. Apparently, the hypothesis is false and should be buried. [1] The documents of the Pharaonic civilization, as recorded in the papyrus, prove the existence of atherosclerosis in ancient humans before 5000 years. Human species has been dealing with animal fat as premium source of energy and health. In 1665, Robert Boyle (1627-1691), discovered a fat transport

system in animals. François Poulletier de la Salle (1719-1788) first identified solid cholesterol in gallstones in 1769. He isolated crystals from cholesterol for the first time. As his work was never published, attribution and dating are known only roughly, quoted by Pierre-Joseph Macquer (1718-1784) and Felix Vicq-d'Azyr (1748-1794). It was not until 1815 that Michel Eugène Chevreul (1786-1889) rediscovered it and named the compound "cholesterine". In recognition to his work, the names of 72 famous French scientists were inscribed on the Eiffel Tower in Paris, Chevreul is one of them. Cholesterol in human blood was first reported in 1833 by Félix-Henri Boudet (1806-1878). [2] The historical background of the hypothesis of a causal relationship between the level of serum cholesterol and the development of atherosclerosis began with Rudolf Virchow's (1821-1902) description in 1856 of the atherosclerotic plaque with its cholesterol deposits.

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Friedrich Richard Reinitzer (1857-1927) was an Austrian botanist and chemist. In late 1880s, experimenting with cholesteryl benzoate, he discovered properties of liquid crystals and was able to described the molecular structure of cholesterol ($C_{27}H_{46}O$). Ehrmann, Lehzen and Knauss reported the first cases of familial xanthoma in 1889. Ehrmann's cases occurred in two brothers, aged 7 and 9 years, respectively. In both, the eruption began when the brothers were aged 5 years, and was present over the elbows and buttocks. Lehzen and Knauss reported the case of a girl, aged 11 years, who presented both xanthomas and xanthelasmas.

Friedrich Richard Reinitzer (1857-1927), describes the molecular structure of cholesterol ($C_{27}H_{46}O$).

In the time between 1907 and 1909, Alexander Ignatowski (1875-1955), who was involved in the laboratory of Nobel prize winner Ivan Pavlov, was trying to prove the role of cholestrole in the genesis of atherosclerosis. He fed rabbits large amounts of meat, eggs and milk. It was indeed toxic for young rabbits, while adult rabbits developed atherosclerosis. Nikolai Anichkov's (1885-1964) experiment with rabbits in St Petersburg first demonstrated what thought to be the role of cholesterol in the development of atherosclerosis. In 1912, Nikolai Anichkov (1885-1964) and Semen Chalatov (1884-1951) reproduced Ignatowski's work, by feeding pure cholesterol to rabbits, thus eliminating proteins. He fed rabbits which is herbivores, with cholesterol from egg yolks and found that they developed atherosclerotic plaques containing cholesterol. When he tried with other animals (dogs as carnivores and rats as omnivore) it was not possible to reproduce the results. They didn't develop atherosclerosis. [1] Anichkov, who later became a good friend of Joseph Stalin, laid the basis for the "Lipid Hypothesis" in 1913. As he was not able to reproduce his results in dogs and rats, his findings were rejected. In 1915 Anichkov moved to Freiburg to work under Dr. Aschoff, who at that time was considered the most accomplished of all German pathologists but this did not create any change in the scientific communities toward his assumption. Ignatowski and Nikolai Anichkov's were both overhasty when they concluded the presence of cause effect relationship between high cholestrol ingestion and atherosclerosis in human species. *The basic fundamentals in medical research where absent in their work as they can not reproduce it*. An other major weakness in their assumption was *extrapolating experimental results between different animal species.* The fact that they both missed is that cholesterol transport systems are different in different species. Dogs and rats, unlike rabbits, are quite efficient in converting cholesterol into bile acids.

Nikolai Anichkov's (1885-1964) induced atherosclerosis in herbivores (rabbits) after massive meals of egg yolk. Reproduction of this failed in carnivores dogs and rats as omnivore.

2.2. The Cholestrol War and Nobel Laureates

Because of the crucial role of cholesterol as basic building stone in biology in general and because of its criucial role in human physiology, it was the focus of scientific communities and Nobel Prize competitions, as prime molecule of crucial role in human beings well being. After Ancel keys -as well be discussed the tragic wave of the economy and industry was forcing in all directions to illusinize medical communities as well as the public of the role of cholestrol in atherosclerosis. Nobel prize competition was not an exemption. Although the dominant spirit is scientific discussing human biochemistry and molecular synthesis and metabolism of cholesterol, but the prudent reader can easily perceive the implication of the heart diet dogma as if it is part of the scientific discussion qualified the scholars of Nobel Prize to be laurates. This was too conspicuous after Ancel keys wave of fraud science in 1953 and after. Sobriey verified scientific reading with eye of wisdom will disclose the absence of cause effect relationship between cholesterol and the heart diet dogma. As a matter of fact the epidemics of congestive heart failure, diabetes milletus, obesity, impaired memory at younger age and many others were observed after enforcing the new dogma on the planet populations.

In 1928 Adolf Windaus won Nobel Prize in chemistry for sterols structure and their relation to Vitamins (https://www.nobelprize.org/prizes/chemistry/1928/windaus/facts/). He was involved in the discovery of the transformation of cholesterol through several steps to vitamin D3 (Cholecalciferol). He passed his patents to Merck and Bayer and they brought out the medicine Vigantol in 1927.

Konrad Emil Bloch shared the Nobel Prize in Physiology or Medicine in 1964 with his compatriot Feodor Lynen, for their discoveries concerning the mechanism and regulation of the cholesterol and fatty acid metabolism. Konrad Bloch, showed HMG-CoA reductase was the enzyme in this complex synthesis. His Nobel Lecture was "The Biological Synthesis of Cholesterol". The cholesterol serves as a precursor for bile acids, sex hormones, and cortisol. Both Bloch and Lynen showed that mevalonic acid is converted into chemically active isoprene, the precursor to squalene. The body then converts the squalene to cholesterol. Bloch also discovered that two major physiological systems, bile and a female sex hormone were made from cholesterol, which led to the discovery that all steroids were made from cholesterol.

Konrad Emil Bloch (191-2000) Nobel Prize laureate in Physiology or Medicine in 1964 (joint with Feodor Lynen) for discoveries concerning the mechanism and regulation of the cholesterol and fatty acid metabolism (https://www.nobelprize.org/prizes/medicine/1964/bloch/facts/).

The Nobel Prize in Physiology or Medicine 1985 was awarded jointly to Michael S. Brown and Joseph L. Goldstein "for their discoveries concerning the regulation of cholesterol metabolism". In a press release of Nobe prize organization they stated that:

"Michael S. Brown and Joseph L. Goldstein have through their discoveries revolutionized our knowledge about the regulation of cholesterol metabolism and the treatment of diseases caused by abnormally elevated cholesterol levels in the blood. They found that cells on their surfaces have receptors which mediate the uptake of the cholesterol-containing particles called low-density lipoprotein (LDL) that circulate in the blood stream. Brown and Goldstein have described that the underlying mechanism to the severe hereditary familial hypercholesterolemia is a complete, or partial, lack of functional LDL-receptors. In normal individuals the uptake of dietary cholesterol inhibits the cells own synthesis of

cholesterol. As a consequence the number of LDL-receptors on the cell surface is reduced. This leads to increased levels of cholesterol in the blood which subsequently may accumulate in the wall of arteries causing atherosclerosis and eventually a heart attack or a stroke.

Brown and Goldstein's discoveries have been used as an evidence to new principles for treatment, and prevention, of atherosclerosis" based on unproved assumption considering cholesterol as a cause of atherosclerosis. They ascribed this to an effect of the high levels of cholesterol circulating in the blood. In reality, the accelerated arterial damage is likely to be a consequence of more brittle arterial cell walls, as biochemists know cholesterol to be a component of them which modulates their fluidity, conferring flexibility and hence resistance to damage from the ordinary hydrodynamic blood forces. In the absence of efficient receptors for LDL cholesterol, cells will be unable to use this component adequately for the manufacture of normally resilient arterial cell walls, resulting in accelerated arteriosclerosis. [3] In spite of the high level research which qualifies the two authors for Nobel Prize, the two bolded underlined statements is assumption based on the era dogma about the casual role of cholesterol in atherosclerosis. Increasing number of recent reports strike the Nobel Laureates conclusion unmercifully. As the current era, carries higher respect and more scientific value for the evidence based medicine, we published in the reputable "evidence based medicine BMJ" a striking report which shake the last eight decades practice in the Dietary Recommendations for Familial Hypercholesterolaemia: and describe it as " Evidence-Free Zone". We have challenged the rationale for FH dietary recommendations based on the absence of support for the diet-heart hypothesis, and the lack of evidence that a low saturated fat, low cholesterol diet reduces coronary events in FH individuals. As an alternative approach, we have summarised research which has shown that the subset of FH individuals that develop CHD exhibit risk factors associated with an insulin-resistant phenotype (elevated triglycerides, blood glucose, haemoglobin A1c (HbA1c), obesity, hyperinsulinaemia, high-sensitivity C reactive protein, hypertension) or increased susceptibility to develop coagulopathy. [4] As a matter of fact, there is strong support for the view that LDL-C, in isolation, is a poor marker of risk for CHD in the general population, as well as in FH. ([5] [6] [7] [8]) Bittencourt *et al.*, in a prestigious recent publication in *Atherosclerosis* commented on the finding of a substantial percentage of individuals with very high LDL-C (>190 mg/dL) who also had a zero Coronary Artery Calcium (CAC) score. Despite their high LDL-C levels, these individuals risk for future coronary events, with a zero CAC score is very low. [9] In a comprehensive review concerning mortality rate in relation to LDL-C in people over 60 years of age with the highest LDL-C showed that they lived as long, or even longer, than those with low LDL-C. [10] It was shown that FH individuals responded to the low carbohydrate, high fat, high cholesterol diet in an equivalent manner to non-FH individuals. The effects of a moderately LCD (30%), high fat (55%) diet, supplemented with up to 1800

mg/day of eggs cholesterol, on serum lipids in FH subjects was evaluated by Cole *et al.* These investigators reported that consumption of additional fat and cholesterol in the context of an LCD lowered TGs and raised HDL, and did not affect LDL-C levels in FH individuals. [11] In addition there are recent documents of poly functional capacity of LDL Receptors. In addition to lipid physiology other functions of LDL Receptors (LDLR) are related to the immune system and infectious diseases. 11 LDLR molecular weight is 164,000 daltons, consists of 830 amino acids, distributed in 5 domains and its charge is negative. LDLR is sharing sequences with important biochemical pathways including the complement system (C9), epidermal growth factor, and three proteins of the coagulation system (IX, X, and protein C).This shared sequence is raising the possibility that LDLR belongs to supergene families. LDLR gene is located in the short arm of chromosome 19. [12]

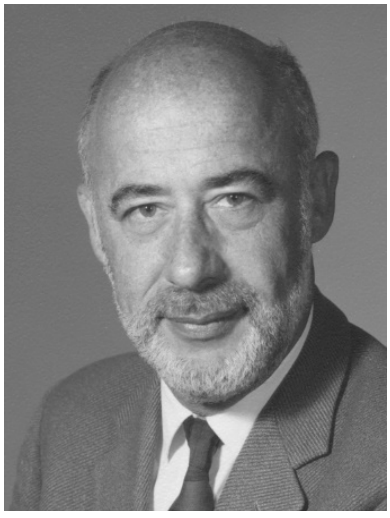
Restricting the LDLR anomalies to abnormal cholesterol metabolism is restriction of the true science.

2.3. John Gofman and II Ward War Era

John Gofman is physicist and chemist with qualification in medicine, with major contribution to radiation safety science. He is best known for his research since the 1960s on the biological effects of low doses of radiation. He and his collaborators investigated the body's lipoproteins, which contain both proteins and fats, and their circulation within the bloodstream. They described low-density and high-density lipoproteins and their roles in metabolic disorders and coronary disease. This work continued throughout the late 1940s and early 1950s. Gofman work, apparently attracted attentions of medical communities and was honoured with the title of "Father of Clinical Lipidology" by the Journal of Clinical Lipidology in 2007. [13] Most of Gofman scientific contribution was in the field of radiation safety science. In medicine his main contribution was to investigate on health studies of the survivors of Hiroshima and Nagasaki, as well as other epidemiological studies, and research on radiation's influences on human chromosomes. *His contribution to lipid science was exaggerated and exploited to extrapolate his reputation in nuclear sciences to support the growing up efforts to link cholesterol blood levels to atherosclerosis in human species. In lipidology, it seems that his discovery of lipoproteins, a molecule in charge of carrying cholesterol, was taken, mistakenly, as a prove of the casual relation of cholesterol to cardiovascular disease.* We investigate this issue critically. The pioneering name of John Gofman in the nuclear era in the 1094s and 1950s was attempted to be exploited to atherosclerosis science. In spite of all distortion efforts, Gofman accurately pointed out in 1955 that total cholesterol is false guide to effect of diet on heart disease and excess carbohydrates especially refined sugar causes liver to overproduce VLDL Triglycerides which in turn reduce HDL. John Gofman with Hardin B Jones and colleagues published a critical paper on the actual culprit in atherosclerosis pathophysiology in human in the year 1951. [14] A critical com-

parison has been made, in which the analysis of total cholesterol and S_f 12 - 20 lipoproteins was done on aliquots of the same serum sample, of both levels in normals vs. atherosclerotics, using myocardial infarctions as the test group. The over-all correlation of S_f 12 - 20 lipoprotein levels with atherosclerosis is two to four times as great as that for serum cholesterol levels with atherosclerosis. The S_f 12 - 20 lipoprotein levels are associated with atherosclerosis, independently of their relationship with serum cholesterol. The S_f 12 - 20 levels account for the bulk of the over-all predictive segregation of atherosclerotics from normals. *The serum cholesterol level is very much less, if at all, associated with atherosclerosis, when considered independently of its association with the S_f 12 - 20 lipoprotien levels*. A bombshell conclusion in the same publication by Gofman and colleagues shorten the history and shake the scientific corruption in the field as if they knew of the upcoming decades scientific deviations. *They stated that: The only pharmacologic agent which rapidly shifts the lipoprotein pattern in humans in the direction of normality is parenteral heparin. The possibility is considered that a deficiency of heparin or a heparin-like substance may be involved in causing the basic lipid metabolic defect in humans*. [13]

In 1966, Gofman, *et al.* published an estimation of Ischemic Heart Disease Risk from Blood Lipid Parameters. *They stated that if for any blood lipid parameter, the difference in value between ischemic heart diseases and the base population approaches zero*, and if the distribution of values of that parameter about the central value is similar in the de novo ischemic heart diseases group and the base population, then it follows that the risk of future ischemic heart disease in such a population is independent of the blood lipid parameter, even for widely differing values of the parameter. *It follows, further, that under such circumstances, there would exist no rationale for the expectation that dietary or pharmacological alteration of such blood lipid parameters might alter the risk of de novo ischemic heart disease in the study population*. [14]



John Gofman (1918-2007) was named father of lipidology in 2007 but with paradox to his honorable scientific heritage.

Little, J, *et al.* in 1965 supported the findings of John Gofman and colleagues when they demonstrated that Survivors of Myocardial Infarction: Serum Lipids carries NO prognostic information. Blood lipid parameters are not likely to provide any prognostic information in already established ischemic heart disease in persons above 50 years of age or probably even in younger groups. Little, J. Alick and associates have already tested this and found it to be the case for subjects who have experienced myocardial infarction. This study examined life expectancy and serum lipids in 120 men with atherosclerotic coronary heart disease. Five-year survival from onset of infarction was 79 per cent. *No relationship could be demonstrated between survival and the level of the total serum cholesterol, Std. Sf 0 - 12, 12 - 20, 20 - 100, and 100 - 400 lipoproteins.* Survival for patients with an infarct less than 6 months before entry into the study was shorter, despite serum lipid levels the same as the remainder of the group. Although the age of onset of coronary disease is influenced by serum lipid levels, survival subsequent to infarction is not. This paradox suggests that serum lipids affect rate of atherogenesis in the long preclinical stage but in the short clinical stage other factors determine survival. [15]

2.4. Earlier Studies of the Relationship of Serum Lipids and Lipoproteins to Degree of Arterial Narrowing Utilizing Autopsy Material

Excellent approach to grasp the fact of the true relationship of Serum Lipids and Lipoproteins to Degree of Arterial Narrowing is the study of atherosclerosis in autopsy material from subjects whose blood lipid and lipoprotein parameters had been determined during their life. The first prospective study in this direction has been carried out by Paterson and his coworkers. ([16] [17]) Multiple determinations of low-density lipoproteins, Atherogenic Index values, and serum cholesterol were performed in a population sample of institutionalized psychotic individuals. The youngest group available in the Paterson study is the 60 - 69 year age category. Blood lipid parameters were measured on a regular basis during life, which provide much more reliable comparisons of indices of blood lipid status to coronary artery narrowing than a single determination. It was demonstrated by Paterson and associates, that the *degree of coronary artery atherosclerosis was unrelated, or at best very weakly related, to the blood levels during life of serum cholesterol, Atherogenic Index or individual lipoprotein classes for the 60 - 69 year group of subjects. This result of such highly confident autopsy findings is actually an earlier announcement in 1966 of another serious nail in the coffin of* the atherogenesis theory. As a matter of fact, Paterson and associates interpreted the absence of relationship between blood lipid parameters and degree of coronary sclerosis as sounding the death knell for the blood lipid theory of atherogenesis. [13]

2.5. Ancel Keys: The Deception of Science in the Awful Era

Two publications by Ancel Keys had a tremendous impact on the general belief of the cholesterol hypothesis. Ancel Keys (1904-2004) was a Harvard-trained

physiologist and epidemiologist. He did some research on human starvation in the mid-20th century before moving on to study heart disease for which he is probably the most famous. In 1953 he reported that the dietary intake of fat was significantly correlated to the serum cholesterol level and the incidence of cardiovascular death in six countries. [18] It appeared very convincing but the problem was that these six countries were selected from all together 22 countries (**Figure 1(a)**). In 1958 he launched the Seven Countries Study, after exploratory research on the relationship between dietary pattern and the prevalence of coronary heart disease in Greece, Italy, Spain, South Africa, Japan, and Finland. There was no correlation whatsoever if all the countries were included. The study was obviously a falsification. With statistical maneuvers he "showed" that saturated fat was the culprit (**Figure 1(b)**). [1] Keys mentions that only a few countries are available for any kind of real comparison at the time of his publication. Some countries he leaves out of the graph because of major population shifts or poorly-maintained vital health statistics. He does mention that there are good quality health statistics available for many other European countries, but since World War 2 had such an effect on diets, the food data of Nazi-occupied territories were left out. What is left appears to be a remarkable relationship between fat intake and death. The other publication came 1986.15 cohorts of the Seven Countries Study, comprising 11,579 men aged 40-years and "healthy" at entry, 2288 died In 15 years. [19] At this time, the conclusion of Keys, *et al.* came with flavor of remorse as they stated: All-cause and coronary heart disease death rates were low In cohorts with olive oil the main fat. Causal relationships are not claimed but consideration of characteristics of populations as well as of Individuals with In populations is urged evaluating risks. Ancel keys publication is an ethical and tragic turning point in the history of the field. It is Keys publication and the forces behind it that create the anticholestrol agents and in particular statin dilemma in the medical communities. The propagation and the unprecedented enthusiasm to his conclusions were abused by the pharmaceutical and food industries since 1953 until the moment.

Ancel Keys (1904-2004).

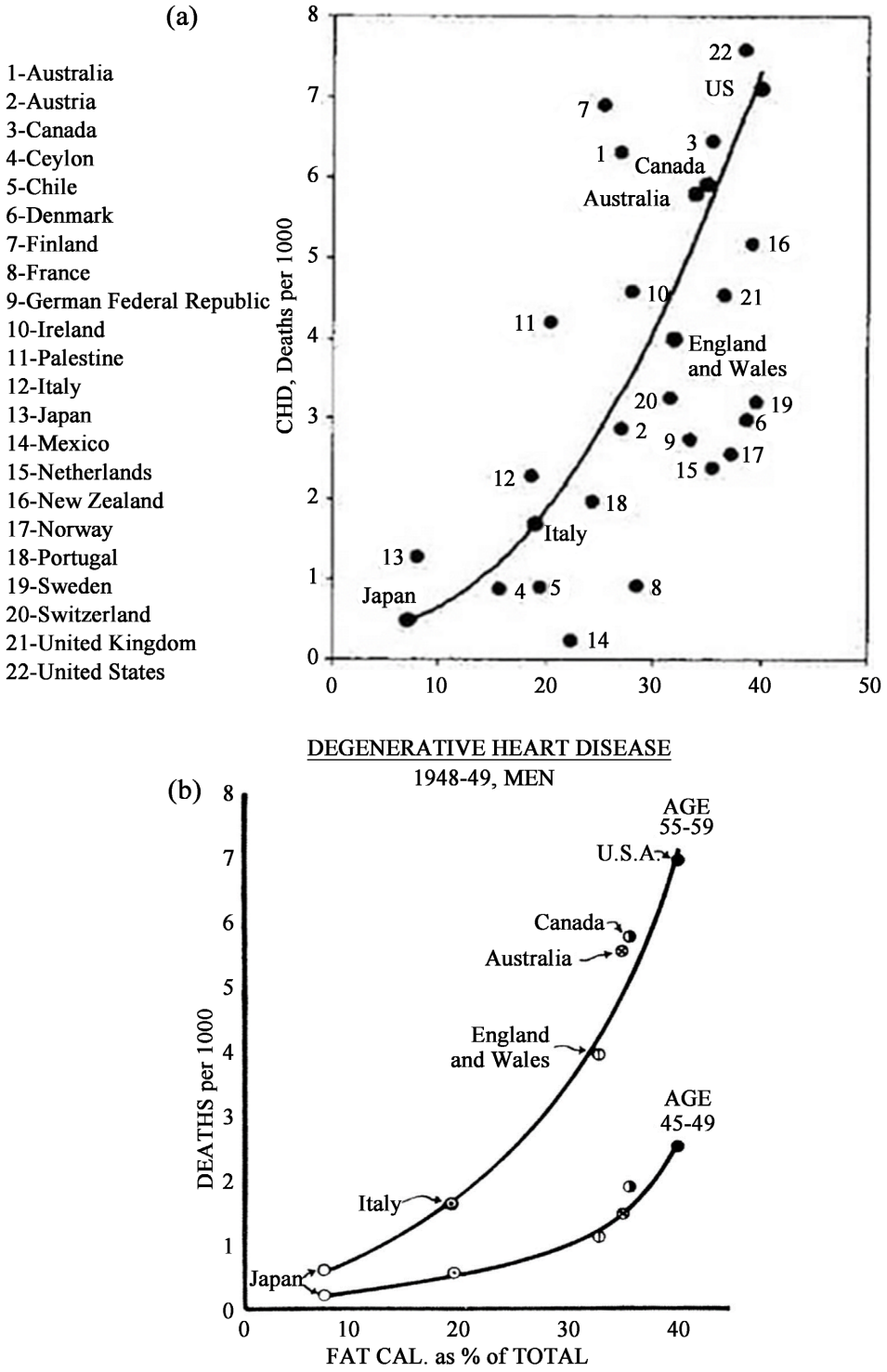


Figure 1. The famous deceptive graphs behind the cholesterol myth. In (a) the true collected data from 22 countries. In (b) The highly selective 7 countries between the 22 creating true historical statistical deception for the human kind.

Massive wave of protest was initiated by Ancel Keys cherry picking findings and conclusions. Jacob Yerushalmy and Herman E. Hilleboe in 1957 pointed out that, for an earlier study demonstrating this association, Keys had selected six

countries out of 22 for which data were available. Analysis of the full dataset made the analysis between fat intake and heart disease less clear. When they published their critique, Yerushalmy and Hilleboe stated: "the association between the percentage of fat calories... and mortality from... heart disease is not valid" and then they call Keys's work a "tenuous association". [20]

Many epidemiological studies on coronarty heart disease with etiological perspective, were carried out. Most reputable is the Framingham Heart Study (began in 1948 and still on going with the third generation). Other similar scope studies are Busselton Health Study, Caerphilly Heart Disease Study and China-Cornell-Oxford Project. Over 1000 medical papers have been published related to the Framingham Heart Study. The main argument is based on the presence of cholesterol in atherosclerotic tissue, and demonstrating the epidemiological association between high levels of serum cholesterol and coronary heart disease.

2.6. Towards the Path of Recalling the Wisdom

Careful contemplation with comprehensive examination of what has been published in the field of lipid hypothesis, and all subsequent medical protocols based on those publications will disclose frightening fact and discrepancy. For the last seven decades cardiologists are instructing their patients with full confidence that cholesterol is the culprit for coronary heart disease. *In fact, there is no proven scientific cause effect relationship to support this hypothesis. It is merely, an epidemiological correlation contaminated by cherry picking, selective data management, skewed with statistical deception.* What is worse: attempts to manage this assumption brought out new epidemics of cardiac and non cardiac diseases for the first time in human history.

A critical assessment of research on the reduction of cholesterol levels by statin treatment to reduce cardiovascular disease was published recently. [21] Although statins are effective at reducing cholesterol levels, they have failed to substantially improve cardiovascular outcomes. The deceptive approach statin advocates have deployed to create the appearance that cholesterol reduction results in an impressive reduction in cardiovascular disease outcomes was described. This historical deception was carried through the incorporation of a statistical tool called relative risk reduction (RRR), a method which amplifies the trivial beneficial effects of statins. David Diamond and Uffe Ravnskov in this prestigious publication described how the directors of the clinical trials have succeeded in minimizing the significance of the numerous adverse effects of statin treatment. [21] The general mainstream medical communities believe in the last 70 years is that "there is no longer any doubt about the benefit and safety' of reducing cholesterol levels". [22] We and others have raised major questions in this direction and acknowledged that statin treatment has been shown to reduce mildly and non impressively coronary events. ([23] [24]) Close inspection reveals that the benefit is much less impressive than clinicians and the general

public have been told and that it must be because of other mechanisms than cholesterol reduction. ([25] [26] [27]) As a matter of fact, an absence of an association between cholesterol levels and the degree of atherosclerosis in unselected people is very early discovery and was originally described in 1936. [28] In spite of the apparent success of lipid hypothesis advocates, the medical history witnessed numerous past and contemporary studies exposing the cholesterol scandal. Today millions of healthy people are on statins, which are drugs that reduce cholesterol levels via inhibition of HMG-CoA reductase. [21] The number of healthy people on statins will increase considerably if the new guidelines from the American College of Cardiology and the American Heart Association are followed. [29] Despite the many contradictory findings, the advocates have praised statins as "miracle drugs" which are "the best antiatherosclerotic insurance" ([23] [30]), as well as "the most powerful inventions to prevent cardiovascular events" ([23] [31]). They have also promoted the view that "there is no longer any doubt about the benefit and safety" of reducing cholesterol levels ([21] [22]) The skeptics have acknowledged that statin treatment has been shown to reduce coronary events, but close inspection reveals that the benefit is much less impressive than clinicians and the general public have been told and that it must be because of other mechanisms than cholesterol reduction ([21] [25] [26] [27]).

Assessments of the association between serum cholesterol and mortality have been studied for decades, and extensive research has shown a weak association between total cholesterol and mortality in the elderly; several studies have even shown an inverse association. It is therefore surprising that there is an absence of a review of the literature on mortality and levels of LDL-C, which is routinely referred to as a causal agent in producing CVD [32] and is a target of pharmacological treatment of CVD.

In a prestigious publication in BMJ Uffe Ravnskov and elite group of investigators published a systematic review on the Lack of an association or an inverse association between low-density lipoprotein cholesterol and mortality in the elderly. [33]

19 cohort studies including 30 cohorts with a total of 68,094 elderly people, where all-cause mortality was recorded in 28 cohorts and CV mortality in 9 cohorts. Considering its scientific content and the prestigious names of coauthors this review is a landmark in the field. The international medical communities were expecting massive reactions from lipid hypothesis and cholesterol advocates. Hunan *et al.* from the Center of Evidence Based Medicine (CEBM), university of oxford statement was the most important.
(http://www.cebm.net/cebm-response-lack-association-inverse-association-low-density-lipoprotein-cholesterol-mortality-elderly-systematic-review-post-publication-pee/) It is serious disappointment to turn the scientific discussion to a true deviation. The truth is most difficult to hide. The whole statement is a weak contin-

uation to the past trials of selective creation of correlation in spite of absence of evidence to confirm causation. The criticism on Uffe Ravnskov and colleagues review about accuracy of data extraction mandate looking at Ancel Keys reputable publication as priority when 15 countries data out of 22 countries were blinded in an attempt to support the lipid hypothesis dogma. ([18] [19]) It is like closing the eyes to avoid seeing the mountain while attempting to negotiate the presence of small stone.

Uffe Ravnskov responded to the statement: it is very wise to ask Hunan *et al.* to show us a study where the authors have found the opposite. What they have shown is that lower genetically determined LDL cholesterol concentrations are associated with lower all-cause mortality, but association does not mean causation. The design of the study satisfies almost all points of reliability and validity according to the Newcastle Ottawa Scale as regards selection, comparability and exposure. [34] Risk of bias across studies and proper logic explanation of outcome differences and confounders was dealt with responsibly in this review which increased the level of confidence toward approaching the truth. In the studies reviewed an explanation for the increased risk of mortality among people with low cholesterol is that serious diseases may lower cholesterol soon before death occurs. Evidence to support this hypothesis may be obtained from 10 of the studies in which no exclusions were made for individuals with terminal illnesses. However, in four of the studies, participants with a terminal illness or who had died during the first observation year were excluded. In one of those studies, [35] LDL-C was not associated with all-cause mortality; in the three others ([36] [37] [38]) which included more than 70% of the total number of participants in Uffe Ravnskov *et al.* review, LDL-C was inversely associated with all-cause mortality and with statistical significance. Thus, there is little support for the hypothesis that the author's analysis is biased by end of life changes in LDL-C levels. Inverse association between all cause mortality and LDL-C was seen in 16 cohorts (in 14 with statistical significance) representing 92% of the number of participants, where this association was recorded. In the rest, no association was found. In two cohorts, CV mortality was highest in the lowest LDL-C quartile and with statistical significance; in seven cohorts, no association was found. They concluded that High LDL-C is inversely associated with mortality in most people over 60 years. This finding is inconsistent with the cholesterol hypothesis (i.e., that cholesterol, particularly LDL-C, is inherently atherogenic). Since elderly people with high LDL-C live as long or longer than those with low LDL-C, this analysis provides reason to question the validity of the cholesterol hypothesis. This literature review has revealed either a lack of an association or an inverse association between LDL-C and mortality among people older than 60 years. In almost 80% of the total number of individuals, LDL-C was inversely associated with all-cause mortality and with statistical significance. [33]

These findings provide a paradoxical contradiction to the cholesterol hypo-

thesis. As atherosclerosis starts mainly in middle-aged people and becomes more pronounced with increasing age, the cholesterol hypothesis would predict that there should be a cumulative atherosclerotic burden, which would be expressed as greater CVD and all-cause mortality, in elderly people with high LDL-C levels. [33]

This study provides a critical rationale for a re-evaluation of guidelines recommending pharmacological reduction of LDL-C in the elderly as a component of cardiovascular disease prevention strategies. Part of the precious targets behind this review is to heighten the international intelligence toward the mercy of human kind. It is our holy mission to convince medical communities to revert back to wisdom in the cholesterol issue. Equally important is to convince health policy makers and politicians. We are stressing always in basic medical fact that we are teaching to our undergraduate and postgraduate students that cholesterol (which became like synonym to heart disease) is a vital component of cell metabolism. ([25] [33] [39]-[45]) Cholesterol is essential for all animal life, each cell is capable of synthesizing it by way of a complex 37-step process, beginning with the mevalonate pathway and ending with a 19-step conversion of lanosterol to cholesterol. The amount absorbed directly from foods is estimated to be only, 15% of the total. The major area of synthesis of cholesterol is the liver but it is produced also in the intestines, adrenal glands, and reproductive organs. In concordance with this basic understanding cholesterol is essential to maintain normal mental functions and neurotransmitters integrity, cognitive functions, immunity, sexual functions, smooth and skeletal muscle functions, as well as all basic cellular and metabolic physiology.

3. Hypocholesterolemia: Therapeutic Target or a Disease?

One hypothesis to address the inverse association between LDL-C and mortality is that low LDL-C increases susceptibility to fatal diseases. Support for this hypothesis is provided by animal and laboratory experiments from more than a dozen research groups which have shown that LDL binds to and inactivates a broad range of microorganisms and their toxic products. ([33] [46]) Diseases caused or aggravated by microorganisms may therefore occur more often in people with low cholesterol, as observed in many studies. ([33] [47]) In a meta-analysis of 19 cohort studies, performed by the National Heart, Lung and Blood Institute and including 68,406 deaths, total cholesterol was inversely associated with mortality from respiratory and gastrointestinal diseases, most of which are of an infectious origin. ([32] [48]) It is unlikely that these diseases caused the low TC, because the associations remained after the exclusion of deaths occurring during the first 5 years. In a study by Iribarren *et al.*, more than 100,000 healthy individuals were followed for 15 years. At follow-up, those whose initial cholesterol level was lowest at the start had been hospitalised significantly more often because of an infectious disease that occurred later during the 15-year follow-up period. ([33] [49]) This study provides strong evidence

that low cholesterol, recorded at a time when these people were healthy, could not have been caused by a disease they had not yet encountered. Another explanation for an inverse association between LDL-C and mortality is that high cholesterol, and therefore high LDL-C, may protect against cancer. The reason may be that many cancer types are caused by viruses. ([33] [50]) Nine cohort studies including more than 140,000 individuals followed for 10 - 30 years have found an inverse association between cancer and total cholesterol measured at the start of the study, even after excluding deaths that occurred during the first 4 years. ([33] [51]) Furthermore, cholesterol lowering experiments on rodents have resulted in cancer, ([33] [52]) and in several case–control studies of patients with cancer and controls matched for age and sex, significantly more patients with cancer have been on cholesterol-lowering treatment. [51] In agreement with these findings, cancer mortality is significantly lower in individuals with familial hypercholesterolaemia. ([33] [53]) That high LDL-C may be protective is in accordance with the finding that LDL-C is lower than normal in patients with acute myocardial infarction. This has been documented repeatedly without a reasonable explanation. ([33] [54] [55] [56]) In one of the studies, the authors concluded that LDL-C evidently should be lowered even more, but at a follow-up 3 years later mortality was twice as high among those whose LDL-C had been lowered the most compared with those whose cholesterol was unchanged or lowered only a little. If high LDL-C were the cause, the effect should have been the opposite. [56]

4. Statistical Exposure of the Conspiracy

Policy makers who are seeking more wisdom and coherence will discover with ease that the major clinical trials upon which cholesterol advocates were considered pillars of the practice are piece of statistical trick. Focusing in statistical terms like relative and absolute risk, relative and absolute risk reduction (ARR) and the number needed to treat (NNT) should revolutionized our cardiovascular practice in terms of the cholesterol calamity. To illustrate the use of these terms in clinical research, David M Diamond and Uffe Ravnskov in expert review solved the puzzle. [21] In simple and clear straight forward scientific language they gave example of a 5-year trial that includes 2000 healthy, middle-aged men. The aim of the trial is to see if a statin can prevent heart disease. Half of the participants are administered the statin and the other half a placebo. In most clinical trials, we find that during a period of 5 years about 2% of all healthy, middle-aged men experience a nonfatal myocardial infarction (MI). Consequently, at the end of our hypothetical trial, 2% of the placebo-treated men and 1% of the statin-treated men suffered an MI. Statin treatment, therefore, has been of benefit to 1% of the treated participants. Thus, the ARR, which quantifies how effective a treatment is on the population at risk, was one percentage point, and the NNT was 100, resulting in only 1 of 100 people benefiting from the treatment. Put another way, the chance of not suffering from an MI during the 5-year pe-

riod without treatment was 98% and by taking a statin drug every day it increased by 1 percentage point to 99%. When it comes to presenting the findings of this hypothetical trial to healthcare workers and the public, the directors of this trial do not think people would be impressed by a mere 1% point improvement. Therefore, instead of using the ARR they present the benefit in terms of relative risk reduction (RRR). The RRR is a derivative of the ARR in which the difference in disease outcomes in two groups is expressed as a ratio. Hence, using RRR, the directors can state that statin treatment reduced the incidence of heart disease by 50%, because 1 is 50% of 2. [21]

The injustice wave of propaganda for cholesterol advocates reach to extreme limits where statins manipulated data end up with recommendations extended to other chronic diseases especially diabetes and systemic hypertension. Statin stands in their literature like the new mercy medicine for human kind. David M Diamond and Uffe Ravnskov reported detailed explanation of the statiatical deception in the major statin tyrials. [21] In The British Heart Protection Study 20,536 UK adults (aged 40 - 80 years) with coronary disease, other occlusive arterial disease, or diabetes were randomly allocated to receive 40 mg simvastatin daily (average compliance: 85%) or matching placebo (average non-study statin use: 17%). Analyses are of the first occurrence of particular events, and compare all simvastatin-allocated versus all placebo-allocated participants. The author's final statement in their results interpretation was as follows: The size of the 5-year benefit depends chiefly on such individuals' overall risk of major vascular events, rather than on their blood lipid concentrations alone. [57] The findings were discussed in an accompanying editorial which praised the effects of cholesterol lowering in this trial, as well as in a press release with the headline: "LIFE-SAVER: World's largest cholesterol-lowering trial reveals massive benefits for high-risk patients." The Lancet editorial stated that "the implications of these findings are profound". ([21] [58]) We will now look at the ARR instead of the RRR. In the simvastatin group, 781 (7.6%) had died because of CVD, in the placebo group the number was 937 (9.1%). Thus, the ARR was only 1.5 percentage points (9.1 - 7.6) and the NNT was 67. [21] In addition 26% of all eligible subjects withdrew, most likely because of simvastatin intolerance. The Anglo-Scandinavian Cardiac Outcomes Trial-Lipid Lowering Arm (ASCOT-LLA) [59] is considered by lipid hypothesis advocates as one of the major pillars in the field. This trial included 10,305 individuals with hypertension. In addition, all of them had at least three of the following risk factors: Type 2 diabetes, left ventricular hypertrophy, peripheral arterial disease, previous stroke or transient ischemic attack, or smoking. Half of them received 10 mg atorvastatin, half of them a placebo and the primary endpoint was nonfatal and fatal CHD. The trial was planned to continue for 5 years, but the authors found the preliminary findings so impressive that the study was terminated at 3.3 years. The reason was that at that time "cholesterol lowering with atorvastatin 10 mg conferred a 36% reduction in fatal CHD and nonfatal MI compared with placebo". However, the bene-

fit was actually unimpressive. In the placebo group, 3% suffered a heart attack vs 1.9% in the atorvastatin group. Thus, the ARR was only 1.1 percentage points, which is 36% of 3. Moreover, there was no significant benefit in subgroups of patients at high risk of CHD, including those with diabetes, left-ventricular hypertrophy and previous vascular disease or for patients aged 60 years or younger, for those without renal dysfunction and for individuals with metabolic syndrome. For women there were no benefits at all. Indeed, there was a trend for worse, albeit non-significant, effects. Finally, there was no effect on either cardiovascular or noncardiovascular mortality. [21]

One of the relatively new and reputable trials is JUPITER trial. [60] It was founded to find away to save the losing merchandise of the cholesterol myth. Simply, it says: if it is not cholesterol then it is inflammation. *The project theorizes that: Inflammation rather than cholesterol is the culprit of coronary heart disease*. People with elevated high-sensitivity C-reactive protein (HS-CRP) levels but without hyperlipidemia were targeted. It is in our perspective that this trial is a bold attempt to create confusion and misleading by twisting the numbers in favor of the lipid hypothesis in, surprisingly, normal lipid profile individuals. The goal is clear: endless number of world populations to be encouraged to be statin users. In JUPITER, rosuvastatin (Crestor) or placebo was administered to 17,802 healthy people with elevated C-reactive protein, but with no prior history of CHD or elevated cholesterol levels. The primary outcome was the occurrence of a major cardiovascular event, defined as nonfatal MI, nonfatal stroke, hospitalization for unstable angina, arterial revascularization or death from cardiovascular causes. The trial was stopped after a median follow-up of 1.9 years. The number of subjects with a primary endpoint was 251 (2.8%) in the control group and 142 (1.6%) in the rosuvastatin group. The disappointing analysis for JUPITER advocates is as follows: the difference in endpoint rate of 2.8% vs 1.6% yields an ARR of 1.2 percentage points and an NNT of 83. The benefit with regards to the number of fatal and nonfatal heart attacks was even smaller. There were only 68 (0.76%) vs 31 (0.35%) events, respectively, resulting in, an ARR of 0.41 percentage points and an NNT of 244. This means that regarding fatal and nonfatal CHD, less than one-half of 1% of the treated population (0.41%) benefited from rosuvastatin treatment, and 244 people needed to be treated to prevent a single fatal or nonfatal heart attack. Despite this meager effect, in the media the benefit was stated as "more than 50% avoided a fatal heart attack", because 0.41 is 54% of 0.76. [21] Lancent publication as part of the smooth hidden forces claimed that statins reduce the risk of stroke, but LDL is not an important risk factor for stroke. [61]

Even in studies designed to prove statin benefit there were conflicting data. In a study where B-mode ultrasound assessment of pravastatin treatment was carried out to evaluate the effect on carotid and femoral artery walls and its correlations with coronary arteriographic findings, there were no individual correlations between carotid intima media thickness (IMT) and coronary lumen va-

riables ($p > 0.30$) and the improvements observed in on carotid and femoral artery walls cannot at all be contributed to anticholestrol effect. *As a matter of fact the cause effect relationship which is critically needed in scientific arena is totally abscent*. [62]

Philips S Mullinex *et al.* in Annals of Vascular Surgery in 2005, published on going believe that systemic and local inflammatory events mediate all phases of plaque development, progression, and degeneration. No longer regarded as a bland, mechanical process, plaque evolution is now best understood as a pitched battle between proinflammatory and anti-inflammatory cellular and molecular elements. [63]

It is the anti inflammatory properties of statin that result in the minute change in ARR and in the reported other benefits. Inflammatory processes, in this regard, play a pivotal role in the pathogenesis of atherosclerosis, and elevated plasma levels of markers of inflammation such as high sensitivity C-reactive protein (hs-CRP), serum amyloid A, IL-6 and soluble intercellular adhesion molecule-1 have been shown to predict cardiovascular events ([64]-[70]) Laboratory evidence for anti-inflammatory effects of statins can be seen in voluminous published literature in prestigious books and journals but not the scope of this review. Since the year 2000, the PRINCE trial provide evidence supporting anti-inflammatory effects of statins and suggest that further laboratory investigations regarding the effects of these agents on adhesion molecules, cytokine function, metalloproteinases, and tissue factor may be fruitful. [71]

Recently published analysis of data from clinical trials have also provided further evidence that statin therapy has anti-inflammatory effects and benefits independent of lowering cholesterol. [72]

5. Statin and the Era of New Human Kind Epidemics

Further regrettable evidence of the great health disasters created by statins came from the science of the magnificent truth disclosure, namely biostatistics and epidemiology. Epidemics of new diseases are evident for the first time in human history. In our perspective those epidemics are secondary to human kind new health believes, values and attitudes. Statin massive spread is in the heart of the matter.

Whereas the benefits of statins are routinely reported as relative risk, adverse effects are always expressed in terms of absolute risk. [21] Adverse effects of using statin are many. Cancer, myopathy and disorders of the CNS are the most common. Alawi A. Alsheikh-Ali *et al.*, in their elegant paper representing insights from large randomized statin trials found there was a highly significant inverse relationship between achieved LDL-C levels and rates of newly diagnosed cancer. They raised the question: what is the relationship between LDL-C lowering in statin-treated patients and incident cancer? They stated: this question is particularly relevant in the present-day era of "lower is better." They rightly addressed the limitations in the published meta-analyses in addressing

this question particularly because the 4 randomized trials of intensive LDL-C lowering (PROVE-IT– TIMI-22, A to Z, TNT [Treating to New Targets study] and IDEAL [Incremental Decrease in Endpoints through Aggressive Lipid Lowering study]) were not placebo controlled and hence were not included in the meta-analyses. [73] Early in the last century humanity recognized the catastrophic health consequences in the state of hypocholesterolemia. Apart from the iatrogenic effect of statins, other disease entities which are associated with hypocholestrolemia are: hyperthyroidism, adrenal insufficiency, liver disease malabsorption syndromes, abetalipoproteinemia, hypobetalipoproteinemia, manganese deficiency, Smith-Lemli-Opitz syndrome, Marfan syndrome, and leukemias. *Those established association made it easier for investigators to expose the devastations of intentionally reducing cholesterol by statins and other drugs*. Nowadays research indicates that lipoproteins actively participate in immune system functioning by binding to and inactivating all kinds of microorganisms and their toxic products. [74] There is a well-established role of viruses in cancer development [54] and it is well-known that reduced levels of cholesterol are associated with a greater incidence of viral infection and cancer, for instance hepatitis B and liver cancer. [75] Furthermore, at least nine cohort studies have shown that low cholesterol measured 10 - 30 years previously is a risk factor for cancer later in life [76] Cancer is unique by itself due to the poor out come and the high mortality as well as the significant lack of knowledge in the etiology and possible preventive measures. The CARE trial was a secondary-preventive trial including 4159 patients (576 women and 3583 men) with MI and average cholesterol levels. [77] Half of the patients were administered 40 mg pravastatin and half of them received placebo. After 5 years treatment, 24 (1.15%) had died because of CHD in the treatment group and 38 (1.83%) in the placebo group, resulting in an ARR of 0.68 percentage points. The most serious adverse event was breast cancer, which occurred in 12 of the women (4.2%) in the pravastatin group but in only one of the women (0.34%) in the placebo group. Although the difference in the incidence between the groups was statistically significant ($p = 0.002$), the authors chose to neglect the numbers and responded by claiming absence of known potential biologic basis. [21]

Most of the large prospective randomized statin trials have shown no difference in the risk of incident cancer in statin-treated patients compared with placebo. *It is critical for those who read the large clinical trials in the field of statins and cancer to note that the primary end point utilized in the large-scale statin trials demonstrating benefit is typically a combined cardiovascular end point and NOT total mortality. This fatal statistical direction may improve the outcome of the culprit agent, masking extracardiac draw backs and converting killer to mercy provider.*

Another devastating effect of statins is the epidemic of myopathy, ranging from benign myalgias to rare cases of fatal rhabdomyolysis. Reviewing the literature of statins and myopathy disclose the fact it is under reported and drasti-

cally under measured. Most of the studied were guided by high creatinine kinase level. To reach that number, the authors have only recorded muscular damage in patients with high creatine kinase (CK), and high CK is defined as a value that is 10-times higher than the normal upper limit at two successive determinations. [21] Another way to minimize the muscular symptoms is to separate them into numerous categories. According to the FDA Adverse Event Reporting System, adverse muscular symptoms are recorded in 11 categories (muscle disorder, myopathy, muscle tightness, musculoskeletal stiffness, myalgia, muscular weakness, muscle cramp, muscle enzyme, muscle fatigue, muscle necrosis and muscle spasm). In most of them, a low incidence of adverse effects is reported, which disperses the total number of adverse event reports across many subtypes of muscular pathologies. It is widely accepted that myopathy is the commonest adverse effect from statin treatment and it is seen most often in women and elderly people. ([78] [79] [80] [81]) The mechanism of statin myopathy is unknown, but possible mechanisms include decreased sarcolemmal cholesterol, [82] reduction in small guanosine triphosphate-binding proteins, [82] increased intracellular lipid producing a lipid myopathy, ([83] [84]) increased myocellular phytosterols, [85] and mitochondrial dysfunction possibly from reduced intramuscular coenzyme Q10 (CoQ10). ([82] [83]) Coenzyme Q10 was discovered by Crane *et al.* in 1957. [86] The importance of coenzyme Q10 in myocardial functioning is occupying significant position in the scientific communities since its discovery but mostly in the last two decades. It is is a naturally occurring, fat-soluble quinone that is localized in hydrophobic portions of cellular membranes. Approximately half of the body's CoQ10 is obtained through dietary fat ingestion, whereas the remainder results from endogenous synthesis. [87] Coenzyme Q10 participates in electron transport during oxidative phosphorylation in mitochondria, protects against oxidative stress produced by free radicals [88] and regenerates active forms of the antioxidants ascorbic acid and tocopherol (vitamin E). ([75] [79] [90]) *Statins block production of farnesyl pyrophosphate, an intermediate in the production of CoQ10*. [91]

Neurological functioning and psychological deteriorations are now well known drawbacks of statins. In a meta-analysis of cholesterol-lowering trials, Muldoon *et al.* [92] found a statistically significant increase in the number of deaths from accidents, suicide or violence in the treatment groups. In addition they found that low blood cholesterol levels are seen more often in criminals, in people with diagnoses of violent or aggressive-conduct disorders, in homicidal offenders with histories of violence and suicide attempts related to alcohol, and in people with poorly internalized social norms and low self-control. [92] Muldoon *et al.* finding has been confirmed by other authors. In a review published 4 years later, Boston *et al.* [93] concluded that lowering cholesterol levels have been associated with an increase in violent deaths in cardiovascular primary prevention studies, and that altered cholesterol levels had been reported in relation to other psychiatric disorders. Finally, Asellus *et al.* [94] found that in pa-

tients with serum cholesterol below the median, the correlation between exposure to violence as a child and adult violence was significant. A low serum cholesterol level has also been found to serve as a biological marker of major depression and suicidal behavior, whereas high cholesterol is protective. ([95] [96] [97] [98]) In a study by Davison and Kaplan, the incidence of suicidal ideation among adults with mood disorders was more than 2.5-times greater in those taking statins. [99] Moreover, several studies have shown that low cholesterol is associated with lower cognition and Alzheimer's disease and that high cholesterol is protective. ([100] [101]) Those observations of reduced brain functioning with statins have been supported by Evans and Golomb. In a study of 143 patients with memory loss or other cognitive problems associated with statin therapy, they reported that 90% of them improved, sometimes within days, of statin discontinuation. [102]

6. Module of Hypocholesteremia and Future Research

Massive scientific striking evidence of the devastating consequences of hypocholesterolemia emerged from the field of molecular biology. Natural call for this understanding rose from the new perspective of Smith-Lemli-Opitz syndrome (SLOS) which can act as natural biological module to study the devastating effects and pathways of cholesterol deficiency in human species. The syndrome was first described by Smith, Lemli and Opitz in 1964. [103] Elucidation of the biochemical and genetic basis for SLOS, specifically understanding SLOS as a cholesterol deficiency syndrome caused by mutation in DHCR7, opened up enormous possibilities for therapeutic intervention. [104] Low cholesterol levels produced by treating cholesterol deficient mutant mice with a cholesterol synthesis inhibitor (BM 15.766) between days 4 to 7 of pregnancy resulted in malformations consistent with those in the Smith-Lemli-Opitz syndrome (SLOS). [105] Cholesterol is required for normal development of the placenta and fetus, therefore major concerns of teratogenic effects exist for any drug that inhibits endogenous cholesterol production. Cholesterol plays a crucial role in specific processes during embryonic development, including the covalent modification of Hedgehog proteins. [106] The medical literature is still awaiting for brave statement in statins use during pregnancy, rather than the humble and soft indications apparently suppressing the facts by pharmaceutical industry. *In our perspective, decision on the teratogenicity of hypocholesterolemia of statins needs urgent call from World Health Organization (WHO) and all world health policy makers.* Recently, cellular biochemistry research creates another massive strike to the lipid hypothesis. In recent publication in the journal of cellular biochemistry Shuan Shian Huang and colleagues opened new era of respectful scientific understanding about the true mechanism of the plaque formation. The authors were activated by the new epidemiological evidence documenting absence of support of the claimed relationship between dietary cholesterol and/or blood cholesterol and atherosclerosis. These results suggest that 7-dehydrocholesterol (7-DHC;

the biosynthetic precursor of cholesterol), but not cholesterol, promotes lipid raft/caveolae formation, leading to suppression of canonical TGF-β signaling and atherogenesis. [107]

7. Economic Burden of Treating Healthy Human Beings

In the era of economic crises striking all planets nations after Covid 19, the world governments are in the stage of prioritizing their nations outlay. The wisdom dictate immediate cessation of any questionable spends and the demand of re-evaluating the intended issue. The evidence against using statins in secondary prevention seems to be in the phase of consolidated confidence. Furthermore, there is rising skepticism against statin treatment in primary prevention in today literature that can be reviewed by the reader. ([108] [109] [110]) One out of every four Americans over the age of 45 takes a statin drug. Grants or consulting payments from statin drug manufacturers paid to guidelines panelists to promote statin industry is well known. The vast majority of journalists and news publishers never even questioned the bias of the guidelines. It sounded scientific to them, so they published it as fact, even while remaining ignorant to the fact that this advice came from a group of people who are essentially on the payroll of these pharmaceutical companies. Annually 29 Billion $ is the minimum expense that goes to statin companies accounts. Cholesterol guidelines published in 2012 by the Cholesterol Treatment Trialists' (CTT) Collaborators. [111]

8. Humanity Transcendence to Raise the Global Consciousness

The planet nations are facing serious natural and societal cataclysm and tragedies which poses a serious threat to the stable progress of civilization and may cause a huge death toll and economic damage all over the world in the next few years. [112] The nowadays nuclear threat in the Russian war against Ukraine is a living example. The recent Covid 19 global crisis is a miniature for the potential impact of such natural and societal cataclysm on world populations. Major international debate is on going about the possibility that Covid 19 epidemic is due to weaponized virus made intentionally in laboratories. [113] It is well perceived that we are not willing to sustain additional human made disasters and epidemics of diseases such as those created by statins. The future therapies will be based on electromagnetics and physical therapies rather than chemicals and its intoxications consequences.

Planetary electromagnetics is integral part of biology in earth. Alexander Chizhevsky work in 1920 followed by Franz Halberg (1897-2013) and our team in the HeartMath Institute (HMI) and the Global Coherence Initiative (GCI) established the scientific prove of earth magnetic field correlation to biology in earth. [114] Frans Halber, the founder of chronobiology in modern science and his group as well as the heartMath Institute (California, USA) and other investigators established the science of the heart as emotional organ orchestrating deli-

cately with universal energetic levels. [115]

Natural as well as societal cataclysm induced by massive historical lies, has its massive interplay between biology and cosmos. Humanity global consciousness can be raised to angles levels with wisdom and truth. Likewise it can descend to the level of evil with unfaithfulness and injustice. The mechanism of interplay between biology and cosmos involves human heart, central nervous system (CNS) and autonomic nervous system (ANS).The longest investigation of synchronized monitoring and data recorded from human rate variability (HRV) at one hand and Schumann Resonances (SR), solar winds indices as well as Galactic Cosmic Rays (GCR) was done by our group (Alabdulgader, McCraty and colleagues) in the Saudi HRV study. In this regard, it appears that an increase in solar radio flux, cosmic rays and Schumann resonance power are all associated with increased HRV and increased parasympathetic activity, although the ANS responds more quickly to cosmic rays and SRP than the solar radio flux. [116] Hear Rate Variability science and innovations stand in a critical intersection between cardiac sciences, psychology, cosmology, quantum physics and consciousness research. This variation in human heart beats seems to be in a highly sophisticated and delicate connection to the global commander from genes to galaxies and to carry the secrets of life and beyond. [114] Tuning our HRV to coherent pattern is beyond the scope of this review, but the relationship of it to psychophysiological health and its resonance with the planet is well established. ([117]-[122]) The most likely mechanism for explaining how solar and geomagnetic influences affect human health and behavior are a coupling between the human nervous tissue in brain and heart as reflected by Heart Rate Variability (HRV) and resonating geomagnetic frequencies, called Schumann resonances, which occur in the earth-ionosphere resonant cavity and Alfvn waves. [123]

The secrets of relations between consciousness as we perceive it in our current life and Heart Rate Variability (HRV) and human emotions is mysterious direction in science that should evolve to raise our global consciousness. [114] One of the pillar hypothesis in the Global Coherence Initiative (GCI) is that when enough individuals and social groups increase their coherence (where HRV is seen as regular sine wave) and utilize it to intentionally create a more coherent standing reference wave in the global field, it will help to lift global consciousness. This can be achieved when an increasing ratio of people move toward more balanced and self-regulated emotions and responses with dominance of wisdom. The concept of resonance and its implementations in physiological as well as astrophysical rhythms is of critical significance for life on earth and to human consciousness experience. [124] In consciousness science new scientific findings of the elegant role of the afferent vagal nerve has been discussed in many publications by us and others in the last 4 decades. The majority of higher brain centers, as well as emotional experience and cognitive processes are operated by Cardiovascular related afferent neural traffic. [125] An integral scientific perspective incorporating the beating heart frequencies and its orchestration with

universal frequencies has been conceptualized in the Heart Based Resonant Fields (HBRF) theory. ([126] [127]) It has been found that individuals have different degrees of sensitivity to the Earth's magnetic fields, and can even respond in opposite ways to changes in the same environmental variable. ([128] [129]) Earth's magnetic fields can act as a carrier of and encoded by physiologically patterned and relevant information. [130] The state of humanity internal peace improves our synchrony with the universal energetic fields. In contrast the chiastic state converts us to poor responders. As a matter of fact our intuitive capabilities are gauged by the level of our synchrony to the energetic fields of the universe. This in turn can help promote cooperation and collaboration in innovative problem-solving and intuitive discernment for addressing society's significant social, environmental and economic problems. In time, as more individuals stabilize the global field and families, workplaces and communities, achieve increased social coherence, global coherence will increase. This will be indicated by countries adopting a more coherent planetary view that will lead them to address social and economic oppression, warfare, cultural intolerance, crime and disregard for the environment in more meaningful and successful ways. [123] After the demise of the League of Nations, the Health Organization became the World Health Organization (WHO), founded on 7 April 1948 and based in Geneva. World Health Organization (WHO) collaborates with the UN system to position health in the debates and decisions of UN intergovernmental bodies; contribute to a coherent and effective United Nation (UN) system at global, regional and country levels. [131] Since its conception, WHO goal is to build a better, healthier future for people all over the world. Working through offices in more than 150 countries, WHO staff work side by side with governments and other partners to ensure the highest attainable level of health for all world populations. We scrutinized The European Society of Cardiology and European Atherosclerosis Society (ESC/EAS) guidelines for the management of chronic heart disease, blood lipids, and diabetes. [132] Many studies that are in conflict with the authors conclusions and recommendations were simply ignored. They ignored that LDL-cholesterol (LDL-C) of patients with acute myocardial infarction is lower than normal; that high cholesterol is not a risk factor for diabetics; that the degree of coronary artery calcification is not associated with LDL-C; and that 27 follow-up studies have shown that people with high total cholesterol or LDL-C live just as long or longer than people with low cholesterol. [132] Exposure-response in the statin trials was lacking but related studies were included as references for the guidelines. Several of these trials have been unable to lower CVD or total mortality. Statin trial has failed to lower mortality in women, elderly people, or diabetics. In top of all cholesterol-lowering with statins, this type of practice has been associated with many serious side effects and new epidemics. Recent review of a total of 46,728,889 statin users suggest that the use of statins is associated with a decrease in insulin sensitivity and insulin resistance. These findings deserve critical awareness in the whole world population but

more in countries where diabetes is endemic. [133]

Our consciousness dictate that the weight of the evidence against statin therapy for cholesterol reduction and its wide spread use in all world nations, deserve massive global move initiated by WHO through all world governments to announce the official obituary and condolence of cholesterol theory. It is the time where wisdom should prevail and for our hearts to orchestrate with the cosmos in planetary resonance satisfactory to raise our global consciousness.

9. Conclusion

Human beings have been living on planet earth for the last 200,000 years with their premium nutrient being animal fat. Only in 1953 (which is equivalent to the last station of the previous 2500 stations of human history) and as part of human being riot, Ancel Keys and collaborates illusionized human kind that their historical source of energy is toxic. Papyrus documents from Pharaonic civilization prove the existence of atherosclerosis in ancient humans before 5000 years. Michel Eugène Chevreul (1786-1889) named the compound "cholesterine". The historical background of the hypothesis of a causal relationship between the level of serum cholesterol and the development of atherosclerosis began with Rudolf Virchow's (1821-1902) description in 1856 of the atherosclerotic plaque. Alexander Ignatowski (1875-1955) tried to prove the role of cholesterol in the genesis of atherosclerosis by feeding rabbits large amounts of meat, eggs and milk. In 1912, Nikolai Anichkov (1885-1964) and Semen Chalatov (1884-1951) reproduced Ignatowski's work in herbivores (rabbit), but failed to reproduce it in carnivores (dogs) and omnivores (rats). Nobel Prize competitions witnessed three episodes of winning in 1928, 1964 and 1985, related to cholesterol research and understanding of its role in vit D synthesis, and regulation of the cholesterol and fatty acid metabolism. What is aggressively shocking the history of cardiovascular medicine in the era is the fact that all those developments and discoveries are based on wrong assumption, based on the assumed presence of cause effect relationship between cholesterol levels and atherosclerosis in human. Brown and Goldstein, in 1985 have described that the underlying mechanism to the severe hereditary familial hypercholesterolemia is a complete, or partial, lack of functional LDL-receptors but we in 2021 challenged this assumed victory in the evidence based medicine journal of BMJ, documenting that Dietary Recommendations for Familial Hypercholesterolaemia are "Evidence Free Zone". In addition, we were able to expose the conspicuous bias in the European guidelines for heart disease treatment and reveal its misleading protocols. John Gofman was a physicist and chemist, with major contribution to radiation safety science during World War II era. *Gofman contribution to lipid science was exaggerated and exploited to extrapolate his reputation in nuclear sciences to support the growing up efforts to link cholesterol blood levels to atherosclerosis in human species. In lipidology, it seems that his discovery of lipoproteins, was taken, mistakenly, as a proof of the casual relation of cholesterol to cardi-*

ovascular disease. In 1966, Gofman, *et al.* published an estimation of Ischemic Heart Disease Risk from Blood Lipid Parameters and found *no rationale for the expectation that dietary or pharmacological alteration of blood lipid parameters might alter the risk of de novo ischemic heart disease.* Study of the relationship of serum lipids and lipoproteins to degree of arterial narrowing utilizing autopsy material in the elderly revealed that the *degree of coronary artery atherosclerosis was unrelated, or at best very weakly related, to the blood levels during life of serum cholesterol, atherogenic Index or individual lipoprotein classes.* Two publications, in 1953 and 1958 by Ancel Keys (1904-2004) who was a Harvard-trained physiologist and epidemiologist, had a tremendous impact on the general belief of the cholesterol hypothesis. Ancel Keys, six countries study in 1953 is actually true scandal of science in the era as he boldly selected six countries from 22 countries to justify his assumption. Keys' other conquest was in 1958 where he launched the Seven Countries Study. With statistical maneuvers he "showed" that saturated fat was the culprit. Another publication by Keys *et al.* appeared in 1986 for 15 cohorts of the Seven Countries Study, came with flavor of remorse where causal relationships were not claimed. In spite of the apparent success of lipid hypothesis advocates, the medical history witnessed numerous past and contemporary studies exposing the cholesterol scandal. Comprehensive systematic reviews published, demonstrating the lack of an association or an inverse association between low-density lipoprotein cholesterol and mortality in the elderly. The new epidemics of human made diseases parallel to the statin era were the subject of large number of publications especially after 2006. The deceptive statistical tools that were utilized in the cholesterol myth campaigns and research were exposed. The dual contradicting incorporation of relative risk reduction (RRR) and absolute risk reduction (ARR) and the negligence of the number needed to treat (NNT) were discussed. When amplification is needed, like in case of impact of treatment in survival (RRR) it is used but not the (ARR) which is becoming favorable tool when complications are in board. The significant adverse effects of using statin are many but the most important are cancer, myopathy and disorders of the CNS. The deceptive methodologies in statin research attempting to subcategorize side effects in order to minimize or dissolve them was discussed. Emphasis has been made to describe hypocholesterolemia as a pathologic status not indicator of well being in human species. Lies and conflicts of interest especially in medicine and the pharmaceutical industry are creating huge crack in the merciful interconnectedness of humanity. In an era of human history characterized by true threat with natural and political cataclysm, we in medical arena considered by world nations as the angels of humanity must contribute to planet populations, mercy and love. The astronomical budgets paid by world countries to provide statin must be redirected to humanity well being. Our beating hearts are part of the universal orchestration that must emanate peace and tranquility information to the surrounding universe. Raising planetary global consciousness is our duty.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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