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William R. Ware, PhD - Editor

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Readers have perhaps wondered why two important and highly controversial subjects seem to be avoided in IHN. They are vaccination and GMOs. These are highly charged subjects with immense political content and industrial influence. Attempts to control governments, regulatory agencies, the media, academic scientists and mainstream medicine in connection with the debatable aspects of these two areas appear to have been phenomenally successful and the scientific debate has shifted from the peer-reviewed medical and scientific literature to the minor journals, the internet, the courts and in the US, to the legislatures. Some secondary sources such as blogs on the internet and books have problems with credibility, others definitely do not, but it is an uphill battle to establish positions and opinions that are truly evidence-based or to read of debates where both sides have full access to the data and are unbiased The proponents have made sure that this rarely exists.

The debate currently taking place in vaccines and GMO is rendered ineffective by the absence of studies related to the con side. They are hard to finance, impossible to publish in high impact journals, and this represents an area of research that may have a very negative impact on issues of career and reputation. Some negative papers peer reviewed and published are subsequently withdrawn and unavailable after direct or indirect industrial pressure. Negative comments regarding these two subjects are strictly taboo for major media sources. Thus many academics are simply being realistic in avoiding these research areas altogether, even if they could find funding. However, there is lots of money for the pro side. Academics are fully aware of examples of reputation destruction engineered by industry or government departments. The very few exceptions where more freedom and less fear exists appears to be mostly in Europe with only a relatively few qualified researchers in North America pursuing what to most reasonable individuals appear to be valid, important questions.

Vaccination is unique in science in that decisions by individuals regarding accepting therapy or even the freedom to do this has shifted from the consideration of scientific arguments and freedom to make a decision and are now in some jurisdictions limited to having to deal with departments of justice and law passed by state legislatures. Government enforced therapy is here and growing and one by one, US states remove the right of individuals to make up their own minds and say yes or no. The pro side is deemed by

authorities and opinion leaders to be 100% correct and evidence-based, the con side of little or no scientific merit at best, and at worst, made up of incompetents, crackpots, and idiots. The same applies to food choices and the issue of labeling for GMO content. We saw this in the climate change debate, although there the con side was able to execute and publish a substantial body of research and establish on the their side a rational basis for debate, but the positions of many on the pro side did not enhanced their reputations for absence of intellectual and emotional bias. The vaccine and GMO issues are much worse in this context. Thus they will continue to be avoided in IHN in favor of exposing on occasion what appear to be serious if not illegal shortcomings in drug research, marketing and safety information.

Finally, the physical and biological sciences would not have evolved to achieve their current highly respected status and immense achievements if the influences outlined above had been operating.

Wishing you and your family good health,

William R. Ware, PhD, Editor

Highlights

POLYUNSATURATED FAT 101

In the context of a healthy diet, conventional wisdom has, for a couple of decades, been to decrease intake of saturated fat (SFA) and replace it with polyunsaturated fatty acids (PUFA). Two US organizations have recently taken positions concerning saturated fat. They are The American Heart Association in conjunction with the American College of Cardiology (AHA/ACC) and the Dietary Guidelines Advisory Committee (DGAC). The AHA/ACC recommends limiting intake to 5-6% of calories whereas the DGAC suggest less than 10%. Prior to this, 7% was a popular number.

Thus it was surprising, even shocking to read a commentary in the April 2014 Mayo Clinic Proceedings which went into considerable detail concerning a contrary point of view.[1] The six authors were from the US, Sweden, Scotland, Japan and Germany. The authors summarize the objections to this hypothesis as follows (see the free paper for references):

- Ten randomized controlled or cross-over trials found that a high intake of SFA, even up to 50% of total energy intake, had little effect or none at all on either LDL or total cholesterol.
- Two meta-analyses of follow-up studies found no association between CVD mortality and SFA intake.
- In 10 cohort studies of patients who had experienced a stroke, 3 found no difference in SFA consumption between patients and controls and in addition for 7 of the studies, patients had eaten *significantly less* as compared to controls.
- In a meta-analysis of 16 long-term follow-up cohort studies, when the highest dairy consumption was compared to the lowest, the highest consumption was significantly *protective* for all-cause mortality, atherosclerotic heart disease, stroke and diabetes.
- Studies conducted in 1998 found increasing the intake of SFAs changed the LDL particle size in in a favorable direction as judged by the increased prevalence of large buoyant LDL particles.
- Three studies of patients with heart attacks were compared with healthy controls as regards the concentration of short-chain fats (considered bad) in their fat cells because this reflects recent dietary intake. No difference of levels was found.

The advice that accompanies the current recommendations to limit saturated fat is to replace it with unsaturated fat, also a common recommendation for decades. In particular we are encouraged to eat vegetable oils which included corn, soybean, canola, safflower, and sunflower oil, although mention is frequently made of nuts and fish. Nevertheless, we are encouraged to believe that our search for healthy fat can end in the vegetable oil section of the market. Most of the PUFAs consumed in the US are derived from soybean oil at a rate of 18 billion pounds per year making it a source of about 20% of caloric intake. Soybean oil is used extensively in prepared foods. Today, almost all soybean oil is made from genetically modified plants which are so-called Roundup-Ready and thus the oil is potentially contaminated with traces of the active ingredient glyphosate found to be carcinogenic. The extent of contamination is a dark deep secret since there are no regulations requiring that levels be determined or reported.

Dietary guidelines regarding PUFAs frequently do not distinguish between the two classes, omega-3 and omega-6, and are thus meaningless with one, the omega-3s being in general considered health promoting and the other, the omega-6s potentially harmful unless consumed in moderation and balanced by omega-3s. One of the classical and significant failures over many years of nutritional research into fats has involved lumping either all fats or all PUFAs together in observational studies.

In a systematic review, Ramsden *et al* examined the effects of both omega-6 alone and mixed omega-6 and omega-3 interventions when used to replace saturated and trans-fats.[2] They found in randomized trials:

- Mixed omega-3 and omega-6 in dietary intervention trials as replacement for saturated fat and trans-fat yielded a 27% reduction in heart attacks and death.
- When only omega-6 was used as the replacement there was a 13% increase in heart attacks.
- Omega-6 alone, while reducing saturated fat and trans-fat, resulted in an increase in risk of death.
- For women only, substitution with omega-6 resulted in short term harm and a non-significant indication for long term harm. There was only one trial.

The authors concluded that advice to maintain or increase omega-6 PUFAs should be reconsidered since there was no evidence of benefit and the possibility of harm. Many trials they examined were confounded by the presence of omega-3 PUFAs in the intervention but interpreted as omega-6 interventions.

The American Heart Association in 2015 presented on an internet site a list of healthy cooking oils. Let's look at the omega-6 to omega-3 rations in the recommended oils. Olive oil had the lowest omega-6/3 ratio of about 10. Peanut was high in omega 6 with no omega-3. Sesame oil had a 6/3 ratio of 41/0.3, corn oil 54/1.2, soybean 50/7, and sunflower 65/0. The ratios depend on the source and were not included in the AHA list, but even with variations, the picture presented does not change. Except for olive oil, the AHA is recommending oils high in omega-6 PUFAs and there is one school of thought that considers this far from "healthy."

Judging by the guidelines, mainstream medicine appears to be in denial concerning the dangers of consuming large amounts of omega-6 PUFAs. Ignored is the fact that prior to the industrialization of food, omega-6 fats were a minor part of human diet and the ratio of omega-6 to omega-3 intake was 1:1 to 4:1. Current estimates give anywhere from 10:1 to 25:1. Also, meat and dairy products now have high levels of omega-6 PUFAs due to the change in diet resulting from the dramatic decline of free-range poultry and grass-fed beef and pork and milk producing animals. Obviously, there would be intense pressure from the involved industries if any attempt was made by governments to demonize omega-6 PUFAs. It has taken over a decade for trans-fats to finally be viewed as a significant health hazard, but nevertheless we still have the curious definition of zero trans-fats as being less than a gram per serving.

The principal justification for favoring a low 6/3 ratio is that, not only are omega-6 PUFAs inflammatory, but also act as an antagonist for the beneficial effects of omega-3 PUFAs. Omega-6 acids are also thought to increase the risk of blood clot formation and convert LDL cholesterol into an oxidized form which, according to one theory, is what makes it active in atherosclerosis. In more general terms, excess omega-6 fats impact health by an adverse effect on cardiovascular disease, type 2 diabetes, obesity, the metabolic syndrome and prediabetes, irritable bowel syndrome and inflammatory bowel disease, macular degeneration, rheumatoid arthritis, asthma, cancer, psychiatric disorders and autoimmune diseases in general.[3-6]

The conclusion is that vegetable and even some nut oils are poor sources of omega-3 acids and very significant sources of what one should neither want nor need in large amounts, the omega-6 acids. Diets low in omega-6 fats will still generally be at levels more than adequate. Among the best omega-3 sources, as most readers already know, are fish and shellfish where the omega-3 PUFAs have been changed into more desirable forms such as EPA and DHA. But life is never simple. Large ocean fish such as tuna are contaminated with mercury. Significant amounts of shellfish, for example shrimp ready to eat, are from Asia and are farmed in water suspected of being rather less than pure and probably in fact loaded with toxins. The same problem, no doubt to a lesser degree, exists with farmed fish in North America. Fresh wild fish is hard to come by in most places. Farmed fish are also fed unnatural food. Plant sources of the omega-3 acid contain alpha-linoleic acid which is converted, albeit with variable efficiency, to EPA and DHA in the body. Thus there is an advantage in letting the fish do the job. Fish oils are readily available as supplements, and those derived from small fish generally have much lower levels of mercury. Some suppliers take great pains to eliminate mercury from fish oil supplements and some are described as pharmaceutical grade.

A diet modified to enhance omega-3 intake and minimize omega-6 intake might include grass-fed meat and omega-3 eggs, ideally from free-range chickens. Frying would be done with coconut oil or ghee, ideally organic and the latter derived from grass-fed milk. Butter would also be both organic and from grass-fed cows. Salad dressing would be made with organic extra virgin olive oil, hopefully from a source that does not secretly dilute it with cheap vegetable oils, a growing problem today. Flaxseed products are high in alpha-linoleic acid, the metabolic precursor for the long-chain omega-3 fats in fish; nuts have poor 6/3 ratios but are still valuable nutrients. Green vegetables such as spinach, watercress, brussel sprouts, bok choy, lettuce, and broccoli have a 6/3 ratio close to 1:1 and some have more omega 3 than 6. The challenge here has to do with finding organic sources. In fact, a major problem in adopting a healthy diet is finding enough organic foods and free range or grass fed sources of foods, not only if the intent is minimizing omega 6-intake but also in general simply to avoid eating too much poison. The GMO industries are working with great skill, effort and success to prevent consumers from avoiding GMO containing foods or foods that contain dangerous chemical residues attributable to the cultivation of GMO crops. However, the rapidly growing popularity of organic and wild food will make this easier in the future. Note that even Amazon carries organic coffee, ghee and coconut oil. Labels such as *grass-fed*, *free-range* and *organic* are probably going to become huge marketing tools in the near future just as low fat or fat free has been for decades.

Aside from the approaches outlined above, the most important action is to avoid as much as possible processed foods that come in cans or frozen or from bakeries. These can be an important omega-6 source as well as a source of hidden sugar and toxins. Prepare meals from scratch rather than microwaving prepared dishes. Reflect on the changes that have taken place since the late 1800s as industrialized food industry developed until today it dominates the stock of the supermarkets and has dramatically changed the nature of the fat intake of most humans. The “modern” diet has become rich in omega-6 fats, highly processed carbohydrates such as flour and sugar and high

fructose corn syrup to all of which are added dozens of chemicals, mostly synthetic to improve texture, flavor, color and sweetness. Governments have worked closely with the food industry to make sure that the real dangers of these added chemicals are either not studied properly or simply suppressed in the interest of economic health. Tell people something is safe and even if they don't believe it, it is frequently difficult to do much about the problem. Consider Flint Michigan and its lead problem.

DOES A VEGETARIAN DIET LEAD TO LOWER ALL-CAUSE MORTALITY AND CANCER RISK?

There is ongoing interest in vegetarian and plant-based diets with some regarding this dietary approach the ideal human diet. It is even claimed incorrectly that this is the diet we evolved to eat. Readers may recall that when Dr. Weston Price searched the world for populations isolated from all the dietary and lifestyle aspects that had become common in his time, i.e. the 1930s, while he found totally isolated societies and observed excellent health and almost total absence of the chronic diseases he observed in his patients, much to his surprise he found no vegetarian societies (see the September 2015 Research Review and Commentary for the story of his study and the amazing results). Related to this observation is a recent study that looked at all-cause mortality among a large cohort of US Seventh-day Adventist men and women.[7] Four types of vegetarians were represented: vegan (eggs, fish and dairy less than one time per month) lacto-ovo (eggs and dairy OK), pesco (fish, eggs and dairy OK) and semi (added non-fish meats but no more than once a week). The majority were lacto-ovo. In this follow-up study vegetarians were compared with an equal number of non-vegetarians who otherwise shared the same general lifestyle. Follow-up was a mean of about 6 years.

The authors present a table giving the number of participants in each vegetarian group as well as the number of both vegetarians and non-vegetarians and in addition, the number of deaths observed for each group. For other than the pesco vegetarians, the mortality rates were slightly larger for vegetarians than non-vegetarians (vegan, 3.55%, lacto-ovo 3.85%, pesco 2.99%, and semi 3.65% and non-vegetarian 3.24%). When all vegetarians are compared with non-vegetarians, the mortality rate differences were 3.65% vs. 3.24%, a difference of 0.41% of ***increased*** absolute risk for vegetarians. In other words, no differences of a significant magnitude were found. The authors then performed a multivariate analysis taking into account a large number of potential confounders and obtained a 12% ***relative*** risk reduction. One can make a rough estimate of the confounder adjusted absolute all-cause mortality risk reduction comparing all vegetarians with non-vegetarians. The result is 0.4% favoring vegetarians, i.e. for 99.6%; there was no benefit with the vegetarian diet. Also, when the crude risk reduction favors the non-vegetarian diet, one is given cause for concern regarding the statistical analysis that resulted in the opposite, but clinically insignificant result.

It was concluded that vegetarian diets are associated with lower all-cause mortality. The authors comment: “These favorable associations should be considered carefully by those offering dietary guidance.” It would appear that the results fail to support this enthusiasm.

A similar study with risk of colorectal cancer as an endpoint found negligible absolute benefits and again the crude data showed no benefit except for pesco vegetarians.[8]

The above results are consistent with a study conducted in the UK which involved over 60,000 individuals. The study involved a pooled analysis of mortality data from three prospective studies. No significant differences were found in all-cause mortality between vegetarian and non-vegetarian diet groups. There was also no difference for coronary heart disease mortality nor was there a significant difference in malignant cancer mortality.[9]

BOTTOM LINE
Vegetarian diets appear to confer little if any benefit as compared to non-vegetarian diets in terms of mortality or cancer risk. Studies are consistent and some would consider the evidence of no benefit compelling. In addition, it needs to be emphasized that vegetarians must pay close attention to potential nutritional deficiencies inherent in this dietary approach. This is especially true for pure plant-based diets.

DOES ALCOHOL INCREASE THE RISK OF DIABETES?

Studies have in fact shown that moderate alcohol consumption lowers the risk of type 2 diabetes but there is little information on how this depends on individual characteristics and lifestyle. A recent meta-analysis based on over 700,000 individuals has provided a clearer picture of this issue.[10] The investigators defined light consumption as 0-12 g/day, moderate as 12-24 g/day and heavy ≥ 24 g/day. For perspective, a 12 oz glass of 5% beer, a 5 oz glass of table wine, a 3 oz glass of fortified wine such as port or sherry, and a 1.5 oz glass of spirits all have about 13-14 g of alcohol. Table wine varies in alcohol content from about 12% to 15% and a 5 oz glass thus varies in round numbers from 13 to 17 g. A 750 mL bottle of 15% wine contains about 90 g of alcohol. The alcohol is ethanol or ethyl alcohol, an important point since there are a number of other alcohols in common use for non-beverage purposes and they are toxic.

This study examined the risk of diabetes over an intake range of 0 to 60 g/day. It was found that for both men and women, this range carried a reduced risk compared to abstinence. Graphs were what are termed U-shaped and looked like inverted and slightly distorted parabolas. For men, 20 g/day was most protective, but even at 50 g/d, the risk was less than abstainers. For women the maximum benefit was seen at 30 g/day, an amount considered too high in general for women. Differences in benefit when moderate consumption was compared with abstinence were observed for age, body mass index, smoking status, physical activity and family history of type 2 diabetes, but the differences within each category, for example current vs. never smoking, never

reached statistical significance except for age. The risk of heavy consumption needs to be qualified since the definition used of ≥ 24 g is deceptive given that even 50 g/day for men was still protective (statistically significant). This study does not provide information that allows direct calculation of absolute benefit. However, a rough estimate yields 1% to 3%. For anyone with no risk associated with moderate alcohol consumption, this small benefit seems reassuring for those that enjoy moderate drinking and are at no risk of addiction.

As has been discussed in IHN, the issue of the safe level of consumption of alcohol by women is complex and related to prenatal effects on children, breast cancer and heart disease. Current opinion ranges for abstinence to one drink per day.

U-shaped risk curves showing protection for a diverse and unrelated set of chronic diseases at moderate alcohol consumption are remarkably common and appear to have no accepted explanation. It has been suggested that one explanation may be that beverage alcohol (ethanol) prevents the metabolism of methyl alcohol (methanol) to formaldehyde, thus preventing the highly deleterious effects of formaldehyde on organs and the vascular system. Formaldehyde is highly toxic even in small amounts and is also recognized as a carcinogen. However, the enzyme is localized and thus the damage from formaldehyde is localized. The U shaped curve is postulated to occur because the same enzyme is involved in the metabolism of both ethanol and methanol, but is much more active toward ethanol and thus this alcohol ties up the enzyme and prevents it metabolizing much methanol which allows time for it to be excreted. The metabolism of ethanol does not yield formaldehyde.[11] Exposure to methanol can arise from smoking, consuming canned and bottled fruits and vegetables and consuming the artificial sweetener aspartame which is 11% methyl alcohol by weight and a major source due to the heavy consumption of diet drinks. It is even available in supermarkets in one or two pound packages. Formaldehyde toxicity arises from cellular and DNA damage. This subject is explored in detail in a book by Woodrow Monte.[12] Monte, a Professor Emeritus of Nutrition at Arizona State University, attributes the dramatic increase over a number of decades of the chronic diseases of civilization to methanol toxicity. See IHN June 2012 and December 2012 for a discussion of this hypothesis. It is interesting that formaldehyde outgassing from laminated flooring makes the evening news while the common sources mentioned above which are capable of producing much higher exposure are ignored.

SALT RESTRICTION. HOW COMPELLING IS THE EVIDENCE?

Over the past few years there has been a movement to demonize dietary salt with the resultant appearance of public health initiatives to publicize the danger and encourage the limitation of intake to an officially approved limit, typically about 2 g/day. Proponents argue that high sodium intake increases blood pressure and thus the risk of cardiovascular events or disease and assert that salt reduction policies will produce major public health benefits. The World Health Organization is on board with a recommendation of < 2 g/day. It is quite likely that this notion is well on the way to

becoming an established dogma similar to the now discredited recommendation regarding the limitation of saturated fat intake to prevent heart disease and other disorders.

The enthusiastic acceptance of this notion by public health and medical organizations gives the impression that the matter is settled. One is reminded of the stance of the climate change scientists who like to say that science has spoken and the deniers are to be ignored. A recent article in the *International Journal of Epidemiology* attempts to generate a perspective through which the salt hypothesis can be viewed.[13] They examined the literature published between 1978 and 2014 and included primary studies, systematic reviews, guidelines, comments, letters and narrative reviews. Each was classified as either supportive or refuted the link between reduced sodium intake and lower rates of heart disease, stroke, and death. More than half of the 269 reports were published since 2011. They found that 54% were supportive of the hypothesis, 33% not supportive and 13 inconclusive. They found strong evidence for the presence of polarization with two distinct bodies of scholarship, each driven by a few prolific authors who tended to cite other researchers who shared their points of view. Markedly absent was intelligent debate and in fact the body of literature could be characterized by strong citation bias and the bias associated with a tenaciously held belief. The selection of studies to include in systematic reviews was found to be biased. The choice of which primary evidence studies to cite directly influenced the conclusions of systematic reviews.

The proponents appear to have prevailed, and the anti-salt movement is gaining strength by the day. It has the potential to equal the anti-fat movement as a marketing tool.

The authors cite an interesting commentary which appeared in 2010 in the journal *Science* which reminds readers that one of the hallmarks of a scientist is critical, rational skepticism and discusses the decline in the emphasis on this activity as part of contemporary educational practice.[14] Medicine seems to be an excellent example where once a hypothesis is elevated to dogma, opposition is not merely discouraged but there is an organized and well-orchestrated approach to suppressing debate. Critical articles are frequently publishable only in minor journals and critical letters to editors frequently rejected. The *British Medical Journal* is an outstanding counter example where debate and contrary views are welcome and encouraged, judging by what they accept for publication.

BOTTOM LINE

Limit salt if you want, but realize that not only is the matter not settled, but the deniers are numerous and the need for debate and resolution of conflicting views is necessary before one should accept salt limitation as if science has spoken. In fact, the data from the deniers includes J or U shaped risk curves suggesting that there is a level of restriction below which risk increases and also that there is level of intake that is beneficial. It should also be noted that ordinary supermarket table salt is enriched with iodine. Low salt intake will reduce this involuntary supplementation and low iodine is a

serious health problem (see Dr. David Brownstein's book on iodine as well as his book on *Salt Your Way To Health).*

MYSTERIOUS 20TH CENTURY EPIDEMIC OF HEART DISEASE

From 1900 to 2010 there has been an amazing fluctuation in the per capita deaths from coronary heart disease (CHD) well documented in the US and UK. The rate was low or very low in the early 1900s but then rose strongly to peak in the period around 1970. This was followed by a decline which in the UK was even sharper than the rise.[15, 16] In the US, the same general pattern was seen but with a slower decline. In both countries the maximum was at around 500 deaths per 100,000. In the UK, the rate in 2010 was about 25 per 100,000 and in the US about 175 and is lower now. The sharp rise and fall has been described as an epidemic and it now appears to be petering out. This time line is not common to chronic diseases but rather, a characteristic of an epidemic of an infectious disease.[17]

There does not appear to be a satisfactory explanation for this time-line of CHD mortality seen from 1900 to 2010, but there are a number of possibilities. Consider first the rise to a maximum around 1970. To get CHD mortality, one first needs atherosclerosis and ultimately plaque rupture or heart failure or sudden "electrical" failure. With heart attacks the development of atherosclerosis a dominant mortality factor. What risk factors were on the rise during this period from 1900 up to about 1960-1970? A list might include:

- Industrialization of food and a sharp increase in the use of processed food like white flour and sugar, and the vastly increased intake of trans-fats and omega-6 fatty acids.
- Increase in exposure to toxins in air, water and food.
- Psychological stress from two world wars with a severe and long lasting financial depression in between. This was followed by the Korean War. The widespread psychological stress would quite likely have had an impact on the development of atherosclerosis and subsequent arterial blockage and plaque rupture.
- In the period 1900 to 1950 the managing of acute coronary problems was rather primitive compared to what developed after about 1980.
- Tobacco smoking increased from almost nil to a high prevalence peaking about 1960 at close to 50% of the adult population.

Then a remarkable reversal took place with a rapid and steady decline in the CHD mortality rate. This decline was overcoming factors present that were working to increase the rate including the continued and accelerated industrialization of food, increasing levels of toxins, and increasing incidence of type 2 diabetes, obesity, and the metabolic syndrome. Exercise was declining. In a presentation the AHA Scientific sessions in November 2015, Xanthakis showed results on the twenty-year trends in scores that reflect how healthy Americans were. There has been a steady decline in

cardiovascular health during the reversal of the mortality trend and this ultimately translates into cardiovascular related mortality, again working against the observed decline. Actions that might partly account for the decline in the rate of CHD mortality include the dramatic decrease in the prevalence of smoking. The US Surgeon General estimated that between 1964 and 2012, the prevalence of smoking declined from 42% to 18% and smoking is a strong risk factor. In fact, the graph of smoking prevalence looks almost exactly like the graph of the CHD mortality rates in the US. Interventions for acute coronary events improved dramatically during this period of decline, as did the treatment of heart failure. Sudden coronary mortality also declined with better responses both in hospital and outside. Emergency responders were equipped with defibrillators and they were made available in public places. The Blood pressure was better controlled but the absolute benefit turns out to be small. Population cholesterol levels change by such a small amount that it unlikely that this was a significant factor, especially when the decrease with brought about by medication which had and still have a very small, even negligible impact on mortality.

While one can point to factors that might account for the dramatic drop in mortality rate, it is difficult to present a quantitative argument given the number of factors in play and the lack of adequate data and thus opinions differ and the mystery appears to remain unresolved.[18, 19] One hypothesis attributes the epidemic to infections. In a letter in the *Quarterly Journal of Medicine*, Harvey suggests that the time course of the epidemic in heart disease is almost identical to that of duodenal ulcers, and that the decline phase for that epidemic cannot be explained by medical intervention or the treatment of *H. pylori* infections.[17] Many diseases caused by bacteria show the same temporal features, with epidemics peaking and then eventually disappearing without targeted therapy. In fact, many for which vaccination is used as preventative today had already dramatically peaked and then declined to very low incidence before the appropriate vaccine was even developed.

The potential role of infection in coronary atherosclerosis was suggested in 2012 by Ravnskov *et al* in the *American Journal of Medical Sciences*.[20] They proposed an interaction between microbes and LDL cholesterol and the resultant complexes become enlarged by action of antibodies against oxidized LD that has reacted with homocysteine. These large complex molecules were capable of blocking the artery's own microcirculation system (vasa vasorum) and ultimately leading to local oxygen deficiency (hypoxia) and both arterial plaque and vulnerable plaque. They comment that over 100 years ago the famous physician William Osler suggested that vulnerable coronary plaque seen at autopsy looked like micro abscesses.

BOTTOM LINE

The solution to this mystery would bring considerable insight into the subject of heart disease. Unfortunately, it is not a high profile mystery and there are papers which simply dismiss it by giving vastly more weight to the beneficial changes since 1970 while ignoring the strong driving forces in the opposite direction.

REFERENCES

(1) Ravnskov U, Dinicolantonio JJ, Harcombe Z, Kummerow FA, Okuyama H, Worm N. The questionable benefits of exchanging saturated fat with polyunsaturated fat. *Mayo Clin Proc* 2014 April;89(4):451-3.

(2) Ramsden CE, Hibbeln JR, Majchrzak SF, Davis JM. n-6 fatty acid-specific and mixed polyunsaturate dietary interventions have different effects on CHD risk: a meta-analysis of randomised controlled trials. *Br J Nutr* 2010 December;104(11):1586-600.

(3) Patterson E, Wall R, Fitzgerald GF, Ross RP, Stanton C. Health implications of high dietary omega-6 polyunsaturated Fatty acids. *J Nutr Metab* 2012;2012:539426.

(4) Maingrette F, Renier G. Linoleic acid increases lectin-like oxidized LDL receptor-1 (LOX-1) expression in human aortic endothelial cells. *Diabetes* 2005 May;54(5):1506-13.

(5) Hibbeln JR, Nieminen LR, Lands WE. Increasing homicide rates and linoleic acid consumption among five Western countries, 1961-2000. *Lipids* 2004 December;39(12):1207-13.

(6) Tjonneland A, Overvad K, Bergmann MM et al. Linoleic acid, a dietary n-6 polyunsaturated fatty acid, and the aetiology of ulcerative colitis: a nested case-control study within a European prospective cohort study. *Gut* 2009 December;58(12):1606-11.

(7) Orlich MJ, Singh PN, Sabate J et al. Vegetarian dietary patterns and mortality in Adventist Health Study 2. *JAMA Intern Med* 2013 July 8;173(13):1230-8.

(8) Orlich MJ, Singh PN, Sabate J et al. Vegetarian dietary patterns and the risk of colorectal cancers. *JAMA Intern Med* 2015 May;175(5):767-76.

(9) Appleby PN, Crowe FL, Bradbury KE, Travis RC, Key TJ. Mortality in vegetarians and comparable nonvegetarians in the United Kingdom. *Am J Clin Nutr* 2016 January;103(1):218-30.

(10) Li XH, Yu FF, Zhou YH, He J. Association between alcohol consumption and the risk of incident type 2 diabetes: a systematic review and dose-response meta-analysis. *Am J Clin Nutr* 2016 February 3.

(11) Monte WC. Methanol: a chemical Trojan horse as the root of the inscrutable U. *Med Hypotheses* 2010 March;74(3):493-6.

(12) Monte WC. *While Scence Sleeps, a sweetener Kills*. Author published. Available from Amazon.com as paperback or e-book; 2012.

(13) Trinquart L, Johns DM, Galea S. Why do we think we know what we know? A metaknowledge analysis of the salt controversy. *Int J Epidemiol* 2016 February 17.

(14) Osborne J. Arguing to learn in science: the role of collaborative, critical discourse. *Science* 2010 April 23;328(5977):463-6.

(15) Grimes DS. An epidemic of coronary heart disease. *QJM* 2012 June;105(6):509-18.

(16) Ford ES, Roger VL, Dunlay SM, Go AS, Rosamond WD. Challenges of ascertaining national trends in the incidence of coronary heart disease in the United States. *J Am Heart Assoc* 2014 December;3(6):e001097.

(17) Harvey R. Another 20th century epidemic. *QJM* 2012 October;105(10):1034-5.

(18) Cooper R, Cutler J, Desvigne-Nickens P et al. Trends and disparities in coronary heart disease, stroke, and other cardiovascular diseases in the United States: findings of the national conference on cardiovascular disease prevention. *Circulation* 2000 December 19;102(25):3137-47.

(19) Ford ES, Ajani UA, Croft JB et al. Explaining the decrease in U.S. deaths from coronary disease, 1980-2000. *N Engl J Med* 2007 June 7;356(23):2388-98.

(20) Ravnskov U, McCully KS. Infections may be causal in the pathogenesis of atherosclerosis. *Am J Med Sci* 2012 November;344(5):391-4.

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