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William R. Ware, PhD - Editor

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The US is the acknowledged leader in research in medicine in the world. In 2014, almost 200,000 journal articles in the area of medicine attributable to the US were published with China next at 63,000 and Canada at about 30,000. World Bank figures indicate that the US led the world in money spent (health care costs) per GDP at 17% in 2013, with the Netherlands next at about 13%. To gain some perspective, consider the following statistics. Mortality at age < 5 per 1000 live births continues to hold the record for developed countries in North America and Europe. In 2013 it was 6.9 with Canada second at 5.2 and the UK third at 4.6. The US also holds the record for a similar comparison of the probability of lifetime maternal mortality for > age 15 at 1/1800 births with the next contender being Canada at 1/5200. Scandinavian countries come in at 1/12,000 to 1/15,000, a huge difference. For the 2010 prevalence of diabetes among seven major areas of the world, North America holds the record with the prevalence (standardized to age distributions) at 10.2% of the population. When the US is compared with the major countries of Europe, the age-adjusted prevalence of heart disease in 2004 was also on top with 26.4% for males and 19.7% for females (percentages of the respective populations). The population percentage considered obese was also top of the list by a wide margin compared to major European countries and Canada. In a comparison of life expectancy at birth, for 2013, the US was 28th among a list of 35 of the world's developed countries and on this list above only the Czech Republic, Poland, Estonia, Mexico, the Slovak Republic Hungary and Turkey.

While one can argue that there are extenuating circumstances, that these comparisons depend on the comparative group in question and that these numbers may not be as bad as they look, nevertheless it appears to be a rather dismal report card if one is concerned about major non-contagious diseases or the success of the system in serving the needs of mothers and their newborn children or providing a system and culture that yields a level of life expectancy near the top rather than the bottom of lists of developed countries. Perhaps it is time to give more emphasis to preventive medicine, given that the US is no doubt a leader in acute care which is still in most medical schools the major emphasis of the final years of training.

In this issue, we lead off with a discussion of the downside of statins. This is followed by a closely related subject, coenzyme Q-10 and heart failure. A recent study involving treating heart failure with coenzyme Q-10 is an example of the sort of research that needs to be vastly expanded. If evidence-based medicine continues to reign supreme, then randomized controlled clinical trials of non-patentable therapies are required and only government or private funds can be looked to for the quite considerable sums needed to conduct such trials. Even vitamin D is a candidate, given the publicity in the medical news services when someone finds a benefit but

points out that the results should be ignored pending such trials. While the industry may be happy with the snail's pace of such trials for non-patentable therapies, the general public should be unhappy. There is little doubt that there are significant therapies that achieve results that are better than patent medicines but are withering away due to lack of support for studies to satisfy the critics of case control or cohort studies, not to mention case histories, the latter historically a principal path to progress in medicine.

In this issue we expand on the topic of what to eat or not to eat discussed in the September issue of IHN as a review and perspective. An attempt is made in this issue to justify the Do Not Eat or Drink list. In addition soy products are given a separate lengthy discussion. The issue of what to eat goes beyond the matter of preventing chronic diseases or in general non-communicable disease and also involves aging driven by basic causes. However, it is the chronic diseases which must be delayed for successful aging with a high quality of life.

Wishing you and your family good health,

William R. Ware, PhD, Editor

Highlights

DOWNSIDE OF STATINS AND OTHER CONCERNS

Anyone who reads the cardiovascular medical literature has seen statins described as miracle or lifesaving drugs. Such positions, either stated directly or implied, seem almost obligatory for the authors to meet the current standards of correct behavior and avoid the risk of potential negative peer pressure.

Readers of IHN have repeatedly been told that for primary prevention, the number who do not benefit is around 99% and for secondary prevention is typically 95-98%. These numbers are mostly based on studies before industrial affiliation was a required disclosure or clinical trial registration mandated. When studies published after this became a requirement in the EU and US, what has been seen is that for all clinical lipid lowering trials, no significant beneficial effects have been reported for the prevention of coronary heart disease, i.e. statins are ineffective in preventing coronary heart disease related events.[1] This is in sharp contrast to the views of the majority of scientists who claim the contrary even though their views are based on the above dismal percentages who do not benefit. Thus the number who do not benefit may be even closer to 100% than indicated above. This problem of either very modest (the fashionable term for slight) or negligible benefit, mostly dependent on primary vs. secondary prevention, shifts the major issue to risks of what is frequently lifelong statin therapy. Okuyama *et al* have just published a comprehensive review and perspective in *Expert Reviews of Clinical Pharmacology* of two most important aspects of this issue, while touching on others.[1] One of the coauthors is

Peter Langsjoen whose research on heart failure is discussed in this issue of IHN. They take the position based on the published literature and their clinical experience, that statins stimulate (not a misprint) atherosclerosis and heart failure. One is a prerequisite for coronary heart disease, the other viewed by some as currently at epidemic levels. They base their case on the following points:

- Statins are mitochondria toxic. The mitochondria, tiny intracellular organelles which are numerous especially in heart muscle cells, are where the energy generating biochemical processes are localized and this biochemistry depends upon coenzyme Q-10 (Q10). The mechanism has been well understood for decades. Statins inhibit Q10 synthesis and thereby, among other things, interfere with energy production required for the proper pumping function of the heart which has particularly high energy requirements. The authors simply call statins general cell toxins. In its reduced form (ubiquinol) Q10 is also a clinically relevant antioxidant, especially in the mitochondria, where it protects DNA from damage. Mitochondrial DNA is particularly vulnerable to oxidative damage as compared to so-called nuclear DNA as in ordinary cells. Thus statins are implicated in heart failure and other critical damage as well.
- Statins decrease levels of enzymes including those containing selenium that protect against oxidative stress, including superoxide dismutase, catalase and glutathione peroxidase, and decreases have been shown clinically to be inversely associated with CHD events and positively with event-free survival in patients with CHD in a follow-up study of about 5 years.
- The authors speculate that the reduction by statins of proteins containing selenium (selenoproteins), which are involved in several steps of glucose metabolism, may be partly responsible for the increase in diabetes associated with statins. This subject was discussed recently in IHN (July-August 2015).
- Statins inhibit vitamin K_2 synthesis which is necessary since the main source of vitamin K is K_1 which must be converted first to K_3 and then K_2 and statins inhibit this last conversion. A deficiency in K_2 is known to accelerate coronary artery calcification, the hallmark of coronary atherosclerosis.
- Statin adverse effects on skeletal muscles are the most commonly reported side effect and a common reason for stopping therapy. Statins decrease the concentration of mitochondria, energy production and mitochondrial levels in muscles. That is, they are toxic to muscles.
- Statins are specifically implicated in the impairment of heart muscle function (cardiomyopathy) when not explainable by other underlying pathophysiology. The authors cite clinical experience where 130 cases were identified during 4 years in one solo practice. They point out that cardiomyopathy induced by statins appears to be permanent which may limit the benefits of stopping the drug and taking Q10 partly because DNA damage is involved. They suggest that statins may be a major contributing factor to the heart failure epidemic responsible for over a million hospitalizations every year. This is not the first time this has been suggested. The connection was made long ago.

The expert commentary the authors offer is that cardiologists disagree with the position that there is no clinical evidence for *the lower the better* as regards LDL, a notion that has been in vogue for quite some time. By adopting the conclusions of studies concerning efficacy reported only after 2004 and by scientists reporting research essentially free of conflicts of interest, they

take this position that will no doubt be heavily criticized, while the very serious risk of side effects will be dismissed as minor compared to the benefits, an issue made more urgent by the growing contrary evidence of a significant link between statin treatment and diabetes, which is unacceptable, especially in primary prevention and in all women, given what still appear to be negligible benefit. This difference in opinion in part reflects the conventional acceptance of meta-analyses that include what the authors view as potentially corrupted studies.

It is hard to miss the irony of the this new position presented by Okuyama *et al* , since in involves rejecting a therapy prescribed to prevent adverse CHD and CVD events while suggesting that in fact it appears to increases the risk of developing coronary plaque, heart failure and diabetes, the latter also increasing the risk of cardiovascular disease. They quote Robertson Davies, “The eyes see only what the mind is prepared to comprehend.”

THE BOTTOM LINE

The risks of statins appear to exceed the benefits in primary prevention. For secondary prevention, it is a matter of the weight given to small benefits which accrue to only a small percentage against the growing evidence of severe side effects.

COENZYME Q-10 AS A THERAPY FOR HEART FAILURE

If the current guidelines for treatment of HF are followed, drugs are used to lower blood pressure, improve blood flow and decrease the work load on the heart (ACE inhibitors and Angiotensin II receptor blockers). Also used are drugs that slow the heart rate and drugs, such as diuretics, which help prevent and eliminate fluid buildup. Potassium-sparing anti-hypertensive drugs are also used. By and large, this approach does not address underlying causes but makes it easier for the patient to cope. Nevertheless, HF is progressive, has eventually a strong impact on the quality of life, and finally many patients die from HF or a stroke or a heart attack. HF is also a common aftermath of a heart attack.

While seemingly counterintuitive, statin therapy for heart failure has been popular. A recent meta-analysis by Preiss *et al* examined the randomized controlled trials from 1994 to 2014. When treated and controls subjects were compared, statins reduced the numbers of patients experiencing non-fatal HF hospitalization by 0.23% and 0.18% for a composite of the above endpoint and HF death, respectively. The numbers needed to treat from these absolute risk reductions are 430 and 560 to prevent one event which most clinicians would judge as no effect, but the approved term is modest effect. Many patients with HF are on statins simply because they have been on them for years and were left on them after a heart attack and even when HF developed.

The biological plausibility of a connection between coenzyme Q-10 (Q10) and heart failure seems clear from the paper by Okuyama *et al* discussed above and the obvious question is, why not treat with Q10, a readily available over the counter supplement, some preparations now with greatly enhanced bioavailability? Until recently the answer has always been, it does not work, but the evidence was based on studies that either used Q10 of low bioavailability or at what turn out to be too low doses. A paper by Mortensen *et al* recently published in the *Journal of the American College of Cardiology—Heart Failure* finally appears to have been a properly designed and executed randomized and controlled trial (RCT) taking into account the dose issue and actually measuring baseline and on-treatment Q10 blood levels.[2] As will be discussed

below, it was found that long-term treatment with Q10 of patients with chronic heart failure very significantly improved symptoms and reduced major adverse cardiovascular events.

In the study of Mortensen *et al* the treated and control groups had baseline Q10 levels of about 1 microgram/mL which over the early weeks of the trial increased in the treated group to 3 micrograms/mL. It is interesting to compare statins and Q10. Mortensen *et al* found that for death from heart attack, heart failure or sudden cardiac death, the absolute risk reduction over 2 years was 5.8% (NNT = 17), for hospitalization for worsening HF or acute HF, 7.3% (NNT = 14). From survival plots, the absolute reduction in major cardiovascular events over 2 years was 18% (NNT = 6) and all-cause mortality 10% (NNT = 10). Such absolute risk reduction numbers and numbers needed to treat to prevent one event from randomized clinical trials are usually regarded as a sensational success. After all, for primary prevention of a cardiovascular event, statins have NNT of typically 70 to 100 or higher and they are regarded as miracle drugs.

Important perspective concerning Q10 and HF can be gained by considering the views of Dr. Peter H. Langsjoen, a cardiologist who has been involved in Q10 research since 1985 and has published extensively in this area. He was recently interviewed and the transcript is available on the internet.[3] He points out that early on, it was believed that if HF patients typically had Q10 levels around 0.5 microgram/mL and normal individuals had levels of around 1microgram/mL, then when one was trying to treat HF, the use of supplementation to bring the value up to about 1microgram/mL was indicated. When this was tried, not much improvement was seen. Further research revealed that there was a significant blood level threshold at about 2.5 microgram/ml above which HF patients appeared to have some benefit and severe HF patients were helped by supplementation once the level achieved was greater than 3.5 microgram/mL. This was pointed out in a 2008 paper in *Biofactors.*[4] Even in an earlier paper[5] in the same journal the threshold of > 3.5 microgram/mL blood level was discussed and justified. The results of early research have been clearly ignored in most studies of Q10 and HF and this has an unfortunate impact on the design of a number of trials with highly influential negative results.

It is also interesting to compare the RTC of Mortensen with a study that appeared in 2008 which took into account the necessity of high levels of Q10. This study should not have been ignored when most of the subsequent studies were designed and previous studies evaluated. As might be expected, the authors of the report were Langsjoen and Langsjoen [4]. They report on seven *consecutive* patients who had worsening HF (NYHA Class IV-the highest) who were on maximal medical therapy and taking large doses of the ubiquinone form of Q10 which was not, from the point of view concerning the importance of the >3.5 microgram/mL threshold, adequately elevating blood levels in the context of severe HF, simply because of bioavailability. Some patients were taking 900 mg/day of ubiquinone and were still well below the threshold. This could have been written up and presented as a negative study demonstrating that without a doubt Q10 supplementation did not work at all for severe HF. Not so. Patients were switched from an average dose of ubiquinone of 450 mg/day to the ubiquinol form with a similar average dose yielding a change in average blood Q10 from 1.6 to 6.5 µg/mL. The table below provides the detailed results of changes in the fraction (EF) and NYHA class change indicating the decline in the severity of HF after ubiquinol was used rather than ubiquinone. Ubiquinol is the reduced form and the most prevalent form in the humans. Here are the results.

Case #	Blood Q10 (µg/mL)	EF, % **	NYHF Class *	Treatment Duration (months)
1	2.0 -> 9.3	15 -> 60	IV -> I	20
2	0.9 -> 2.6	35 -> 50	IV -> III	3
3	1.5 -> 8.9	10 -> 10	IV -> III	12
4	1.7 -> 5.1	35 -> 60	IV -> I	10
5	1.5 -> 5.6	30 -> 55	IV -> II	9
6	2.0 -> 5.7	10 -> 20	IV -> III	10
7	1.6 -> 6.5	22 -> 39	IV -> II	10

** Class I HF is asymptomatic. Class II involves mild shortness of breath and/or angina with little limitation of ordinary activity. Class III involves significant limitation of activity due to symptoms, even to the point of problems walking short distances. Class IV is sufficiently severe as to apply mostly to bed ridden patients.*
*** Ejection fraction*

The HF class changes listed are impressive and obviously of huge significance to most of the patients involved. A normal ejection fraction is 55-70%. Note the high baseline Q10 levels, which are at the upper extreme of the modern laboratory reference range, and yet the individuals had Class IV HF which was then strongly impacted by changing the supplement to achieve greater bioavailability and thus achieving much higher Q10 levels. Note also the individual variations. This table in fact nicely states the case for treating HF patients to a high target even if their Q10 levels are already high by traditional standards. Four out of 7 patients regressed to NYHA I or II. These results should have suggested the urgent need for a much larger study. Finally it has been done and reported.

The story of Q10 and HF is a classic example of the fundamental problem faced by alternative and integrative medicine. Here we have a coenzyme that we make in our bodies and which is also available in bioidentical form over the counter as either ubiquinone or ubiquinol. Preparations are now available which use ubiquinol and have high bioavailability. Yet it has taken from 2008 until 2014 for a properly designed RCT to be conducted and published in a peer review journal. It confirms benefits already reported in a case history report, the latter unfortunately being regarded by evidence based medicine as about a low as one can go on the scale of credibility. The conventional wisdom is that Q10 supplementation was a waste of money because it does not work, an opinion based on flawed or irrelevant studies. It is encouraging to see an editorial comment on the Mortensen *et al* study in the same journal titled *Time to Energize Coenzyme Q10 for Patients With Heart Failure.*[6]

THE BOTTOM LINE

The discussion of earlier work given above illustrates how hard it is for case studies to have any impact on future research on a more sophisticated level. This does a great disservice to patients with disorders which strongly impact their quality of life and ignores the strong if not overwhelming impediment that exists for clinical research on non-patentable but strongly biologically active compounds. Individuals with heart failure should consult with their physician about the advisability of trying Q10, especially since now there is a randomized controlled trial. Also, if Q10 is tried, the patient should insist on baseline and follow-up blood tests to determine the blood Q10 level for comparison with the results of Langsjoen.

EVIDENCE FOR THE DO NOT EAT OR DRINK LIST IN THE SEPTEMBER REVIEW AND PERSPECTIVE

Before the industrialization of the raising and processing of food became the norm, many of the issues we face were simply not present. Animals grazed, chickens ran around outside and neither were fed processed food or given drugs. The addition of chemicals other than perhaps salt and sugar was not involved in processed food and in fact home canning was common. Animals ate natural uncontaminated food. Over the centuries, the hunter-gatherers learned to identify toxic plants by trial and error and avoid them, and this knowledge was passed from generation to generation. Eating food that did not make one sick was the norm. As discussed in the last IHN, populations living isolated from "civilization" even in the early 20th century did not suffer from the chronic diseases that are now the major causes of morbidity and mortality. Today, it is almost impossible to make the connection between food and other sources of toxins and disease since chronic diseases are rampant and the explanations of fundamental causes can be very weak or even unknown. Those made sick by eating toxic food don't stand out since almost everyone over 65 has one or more chronic diseases.[7] In guidelines, only lip service is paid to the role of toxins if the subject is mentioned at all and doing something really significant and comprehensive about this potentially critical aspect of the cause of chronic disease is off the radar, partly for political reasons.

Although there has been no negative feedback, readers may have reacted unfavorably to the contents or length of the "Do-Not Eat or Drink" list in the review of diet and health included in in the September IHN issue. It would be natural to decide that this is rather extreme. The common assumption is that governments and regulatory bodies keep the public safe from hazardous levels of toxins in food and water. That may have been the original justification for governmental involvement in these issues, but to believe that they are effective requires a big leap of faith. The demonstration that this is a fantasy involves research on the presence of toxins in food and water, not a popular area of research. Convincing research that safe levels are neither realistic nor even evidence-based, especially when the definitive human studies are next to impossible and animal studies not dependable, frequently industry supported, and the results not reliably transferable to humans. There are two big issues, agricultural practices and industrial food processing of agricultural products into the hundreds of offerings in the supermarket.

Largely consistent with Do Not Eat or Drink list is the Dutch Undesirable Nutrient and Food list which generates a score for use in dietary studies.[8] It includes 11 food groups high in sodium and/or added sugar, processed vegetables, fruit juice and sugar sweetened beverages, processed meat, refined grains, hard margarines, ready meals and soups, spreads and snacks. It also includes items based, it appears; only on the notion that fat is bad, which was ignored in the list in the IHN review and perspective. The Dutch list as does the one under discussion, focuses on items that are undesirable from the metabolic point of view. The second major concern in the Do Not Eat or Drink list involves toxins that historically were absent or present only in trace amounts in food and have been introduced by farming practices and industrial food processing. Modern agriculture is obviously the source of most of the food offered to us by food vendors, mostly supermarkets, and originates from the food processors who produce boxes, bags and cans of heavily manipulated agricultural produce. What is going on in the food processing industry directly relates not only to components that are metabolically undesirable, but also to this toxic burden which comes from both the produce they use and the chemicals they add.

The sources of contamination of soils, crops and the marketed end products involve the following farming production practices: (a) Biosolids, sewage sludge and animal manure utilization, all applied to the farm land; (b) Agricultural chemicals including chemical fertilizer and pesticides and herbicides; (c) Contaminants in irrigation water.

Biosolids are aerobically and/or anaerobic digested sewage sludge, a by-product of municipal waste treatment plants. Animal manure may be treated or untreated. We are talking about 10 million tons of dry biosolids per year, about half of which are land applied. Food contaminants linked to farming practices are unnaturally high levels of nitrogen and phosphorous compounds associated with cardiovascular and carcinogenic risks, and as well heavy metals such as nickel, cadmium, lead and arsenic, pesticides and bacterial pathogens. The list of adverse effects is long and involves allergic, carcinogenic, neurological, and cardiovascular impairments as well as oxidative stress, liver disease, nutritional interference, kidney disease, and spontaneous abortion and infant mortality. This is only a partial list since the adverse effects of many pesticides and herbicides are not well studied but generally acknowledged to be potentially serious. For full documentation, see the large number of citations provided by Udeigwe *et al.*[9]

Agricultural products turn up in the produce departments of supermarkets and other markets. Some go into the feed of animals used for food, providing a feedback circuit. What is most important is that many become the raw materials for the processed food industry which then takes this contaminated food and adds additional synthetic chemicals to enhance flavor, texture, appearance and shelf-life. Packaging and can liners (coatings) can contribute additional toxic chemicals.

For non-processed meats such as beef and pork purchased by the cut or ground and for chicken, whole, or pieces, there is a problem with contamination by antibiotics and growth hormones and other drugs given to animals raised for human food. In addition, such meat will have residues of toxins from the food the animals are fed, some of which may be cumulative and reach significant levels and survive cooking.

The end result is a level of toxic chemical content the extent of which the human body has never historically encountered. Traces of heavy metals have always been in the food supply, but the practices of modern agriculture have increased the levels substantially as well as the levels of pesticide and herbicide residues.[9] The only simple way around this is to maximize the organic component of the diet in the hope of reducing the intake of toxic chemical, both inorganic such as toxic minerals and synthetic chemicals.

The official guidance available that can be passed on to consumers is highly variable depending on the organization issuing the guidelines. Most are now out of date because of the shifting view of the dangers of saturated fat and cholesterol. However, what is missing is giving any importance to the toxic content of food or the absence of satisfactory evidence regarding safety associated with long-term steady consumption. When discussed, the argument is that there is no evidence, but this is a taboo subject. How can one study the relative merits of various foods and diet without correcting for the content of toxins? Furthermore, the content is probably increasing rapidly as agricultural practices change, driven by Big Agriculture and Big Chemical. The authors of these guidelines know that the absence of evidence of risk does not mean the absence of risk.

Given the problems of avoidance just associated with the Do Not Eat or Drink list, one defensive action is to attempt to detoxify. This is discussed in earlier issues of IHN and includes saunas. See the archive index.

THE BOTTOM LINE

The Do Not Eat or Drink list can be justified on the basis of some evidence and common-sense caution. It is unrealistic to expect strong evidence since this is not a research area where it is easy to get funding and even positive results do not yield much enhancement of the investigators reputation, and may even result in alerting strong and powerful financial interests which triggers defensive action, a smear campaign and difficulty in publishing the results through industry control of journal editorial boards. Avoidance generally means a significant change in where one obtains food and frequently presents an unsurmountable challenge—the origin of the view that we are all doomed in the long run, a notion which neglects the enhanced survival of those and their progeny that succeed in avoiding the dangers presented by the modern world. Finally, the issue of what to eat goes beyond the matter of preventing chronic diseases or in general non-communicable diseases and also involves aging driven by basic causes, a highly complex area which is frequently impacted by expanding appreciation of the importance of stem cells, the microbiome, and the epigenic genome as deeply involved. The problem of understanding aging to the point of having meaningful interventions appears to be in its infancy. But it is the chronic diseases, although not independent of the driving some causes of aging, that must be delayed for successful aging with a high quality of life. Evidence that many fail in this endeavor is compelling. Some consider life extension a bad idea, given what they perceive as the expected quality of life. A good place to start is with increased attention to what one does or does not eat and drink while acknowledging the complexity of the issues associated with what to eat.[10]

WHY IS SOY ON THE WHAT NOT TO EAT OR DRINK LIST?

In the review and perspective in last IHN, soy was included in the list of foods to avoid. Why? Soy has been cultivated in Asian cultures for thousands of years, but the original purpose was not for human consumption but for nitrogen replacement in soil. It was not considered digestible until it was discovered that bacterial fermentation alleviated the problem. The fermented products such as natto, miso, tempeh and soy sauce ultimately became staples in the Asian diet, in particular in Japan. Traditional tofu was fermented. In the West, human consumption of soy became popular after aggressive processing methods were developed to render it suitable for human food without fermentation and over the last several decades it has become a part of the regular Western diet, mostly from processed foods. The harvested beans are de-hulled and extruded or otherwise treated to generate a high-fat soy meal. Fat is removed with a solvent extraction and the residue subjected to an acidified ethyl alcohol-water extraction to produce protein concentrates which are then used for both animal and human food such as so called soy protein isolates and texturized vegetable protein. Fragile proteins are denatured in this process, and toxic lysinoanaline and highly carcinogenic nitrosamines produced. The metal vats generally used for the acid treatment result in contamination from aluminum, a highly undesirable element to eat. There is also contamination from manganese which is beneficial at very low levels but highly toxic at high levels of ingestion. Thus one of the reasons soy products are condemned is this toxic load and why they are on the “do not eat” list. It is also the poster child for a modern food only eaten recently except in fermented form.

Another reason is that almost all soy today is genetically modified to withstand the pesticide Roundup which is then used extensively to eliminate weeds from the soy fields. The combination of being a GMO and contaminated by everything in the Roundup mix is nevertheless said by the industry and some governments to still produce a completely safe

product, but critics, as most readers know, strongly disagree based mostly on the GMO content rather than the pesticide and herbicide residues. Other problems with processed soy are thought to be associated with digestive distress, immune-system dysfunction, cognitive decline, cancer and heart disease, and include[11, 12]

- Soy products contain high levels of phytic acid which reduces the assimilation of dietary calcium, magnesium, coper, iron and zinc.
- Soy inhibits the production of the digestive enzyme trypsin and interferes with digestion.
- Soy products contain plant estrogens (phytoestrogens) which disrupt endocrine function and have the potential to promote breast cancer in adult women. These phytoestrogens can also cause hypothyroidism and may be involved in thyroid cancer.
- Soy increases the need for vitamin B12 and vitamin D.
- Soy consumption has been linked to kidney stones.
- Soy products contain hemagglutinin which is associated with cell clumping.
- Soy phytoestrogens impact reproductive health.[13]

These health issues do not appear to apply to fermented soy products common in Asia and around the world where Asian food is appreciated. However, modern tofu used in Chinese cooking and widely available in Chinese grocery stores is not fermented. It is also noteworthy that pure vegans use processed soy products frequently as a source of protein.

The issue of health risks such as described above and various benefits aggressively promoted by the industry makes it difficult to examine the issues critically because the scientific research literature consists of a mix of industry and non-industry studies, and a significant number of conflicting results. Any useful review will need to separate these two types of study as they apply both to risks and benefits, a huge task considering the magnitude of the earlier literature where disclosure was not required. A recent review article concluded that additional studies are necessary to elucidate the issues raised above and that the long-term health effects of consuming highly processed modern soy foods is unknown.[11] This is the final reason for avoiding soy foods.

However, such avoidance is difficult since soy-containing infant formulas are commonly used when babies are lactose intolerant, and soy protein and soy isolates are used extensively in prepared foods, although recent laws in some countries make it mandatory that this be declared on the contents label. Maternal consumption during gestation is of significant concern. Also, two recent studies suggest an association between soy infant formula and autistic behavior and seizures in autistic children.[14, 15]

Finally, a counter argument involves the hypothesis that soy isoflavones exert both estrogenic and anti-estrogenic effects, some of which may be beneficial, and this is an area of active research. Hormonal and other potential benefits of soy may eventually complicate considerably the risk/benefit problem with soy, but the distinction between fermented and unfermented soy producers is a potential confounder.[11]

THE BOTTOM LINE

Limit or avoid soy products. Eat only the traditional fermented soy sources. Humans got along very nicely before soy recently became a “Western” food. However, avoiding soy products is a challenge since it is hidden in numerous processed foods. Use the internet to obtain lists of label terms that conceal soy. The lists are long. However, recognize that the case against soy is far from closed.

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