# INTERNATIONAL HEALTH NEWS

*William R. Ware, PhD - Editor*

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It is well known among medical researchers that the failure to disclose conflicts of interest is common in spite of journal and institutional policies. One can find many articles in the medical literature where the authors went digging and found and described this phenomenon. The first thing some medical scientists do when examining a publication describing a clinical study is to search for the identity of the study sponsor and the conflicts of interest of the authors, i.e. in general those involved in the design, conduct and outcome analysis of the study. The motivation is simple. There have been too many reports where industry-sponsored and conducted clinical research produced results that differed from equivalent studies done entirely without industry involvement or potentially conflicted researchers. Also, it is well known that some drug companies will refuse for years to release important data for independent analysis and when the issue is forced, generally finally via shaming from the media, the reanalysis fails to duplicate the earlier published results and in fact may reveal negative results. Yet such studies are key to regulatory approval and subsequent sales.

Since most clinical studies of drugs and devices are financed by industry because almost no other source of the vast sums exists, it is hardly feasible to ignore all industry-sponsored studies. Indeed a serious problem. Conflicts of interest may also be involved in how clinical results are presented in papers. The opportunities for deceiving with spin are manifold and frequently successful because physicians who need the unvarnished truth may not have the time for detailed analysis of the clinical studies that provide the foundation for their practice, and the articles handed out by drug reps and at meeting booths are naturally selected to put the merits of the product in the very best light. Also, conflicts of interest can impact guidelines and strongly influence clinical practice.

With this background, the saga (scandal) being played out at the huge and world famous cancer hospital, Memorial Sloan Kettering (SKM) provides a very current example of this problem. Investigative reporting by the *New York Times* (NYT) started in September 2018 and is ongoing. Involved were strong financial relationships between SKM executives and senior researchers with pharmaceutical companies. Included were ties with start-up companies that were marketing products from research done at SKM, positions on boards of directors of companies that worked with SKM, and what are described as the

world's top breast cancer doctors found to have failed to disclose literally millions of dollars of income or its equivalent in publications related to their SKM research. The hospital's top medical officer and other leaders were found to have cultivated lucrative relationships with for-profit companies. The following headlines from the NYT tell most of the story:

- *Memorial Sloan Kettering curbs executives' ties to industry after conflict-of-interest scandals. The cancer center will now bar top officials from sitting on outside boards of for-profit companies and is conducting a wide-scale review of other policies. January 11, 2019.*
- *Top cancer doctor forced out over ties to drug makers, joins their ranks. AstraZeneca has hired Dr. Jose Baselga, former chief medical officer at Memorial Slone Kettering to lead its cancer research unit. January 7, 2019.*
- *When doctors serve on company boards. Some hospital executives and cancer researchers sit on boards of publicly-traded companies, raising question about whether their dual roles create a conflict of interest. December 21, 2018.*
- *Top cancer doctor resigns as editor of medical journal. Dr. Jose Baselga, former chief medical officer of Memorial Sloan Kettering Center, was asked to resign after he failed to disclose corporate ties in dozens of scientific articles. December 19, 2018.*

*Wishing you and your family good health,*

***William R. Ware, PhD, Editor***

**Highlights**



## A SUGGESTED PROTOCOL FOR CANCER PREVENTION

It is likely that practically every reader of this newsletter has friends or knows individuals who have been diagnosed with cancer. A wide variety of contributing factors are proposed involving genetics, cellular dysfunction, adverse environmental factors including carcinogenic chemicals, lifestyle including diet, nutritional deficiencies, etc. Combined, they only lead to a prevention program that does not appear targeted. Consider recent American Cancer Society guidelines for cancer prevention.

- Achieve and maintain a healthy weigh throughout life.
- Avoid excess weight gain at all ages.
- Get regular physical activity and limit high-calorie foods and drinks. Physical active means at least 20 minutes of moderate intensity per day.
- Eat a healthy diet with emphasis on plant foods. Details: limit processed meat and red meat, eat at least 2.5 cups of vegetables and fruits each day, and choose whole grains over refined grain products.
- Alcohol—one drink for women, 2 for men per day.

While each of these recommendations is supported by some evidence, the duration, likelihood of adherence and even meaningfulness of these efforts in this context are open to serious question.[1] Absolute benefits in long-term studies specifically with long-term, general cancer prevention as an endpoint appear unavailable or reveal only small absolute impact. Issues such as the importance of red meat and alcohol remain hotly debated. In fact, the above guidelines would just as well apply to simply staying healthy.

Then there is chemoprevention. The best-studied agents are tamoxifen, aspirin, and non-steroidal anti-inflammatories (NSAIDs). Tamoxifen is promoted on the basis of a relative risk reduction of 50% for ER-positive breast cancer. Impressive until one digs up the study and looks at the absolute risk reduction which is 1.8% over 7 years, i.e. about 98% did not benefit in terms of cancer prevention.[2] While accumulating evidence supports the use of aspirin in overall risk reduction for cancer incidence and mortality, high doses and use over a long period appear necessary. Aspirin has side effects which can be serious. Studies of where the focus is on colorectal cancer effectiveness have been inconsistent.[1] A randomized control study over about 18 years for aspirin use in breast cancer found only a small absolute risk reduction.[3] With NSAIDs there can also be genetic issues. For example, this class of drug has been found to produce large increases in 5-year survival for head and neck cancers, but only for those who carry the *PIK3CA* gene, and studies of primary NSAIDs chemoprevention with other cancer types such as colorectal and breast cancer have not controlled for this issue.[4] Thus over-the-counter and prescription drugs do not appear to qualify if one is seeking a preventive intervention for a given cancer or, more importantly, cancers in general that offer large clinically significant results.

It would appear that at present there is no really effective approach to primary prevention of specific cancers or more importantly cancer in general, and the current approaches, while producing small benefits, and are simply not good enough. Only not smoking stands out as quite effective.

Cancer cells are being generated in humans every day, driven by various internal mechanisms and external factors. To form a solid tumor those that escape being killed by natural defenses need to localize and grow to a mass which is estimated at about one billion cells, whereupon there is a clinical manifestation such as unusual bleeding, an odd lump, or one of a number of other indications. For some cancers, by the time there is this clinical evidence, cells from the tumor probably also have started spreading. This metastasis is what actually ends up being responsible for cancer mortality with

cancer established typically in the lung, liver, brain, or bone. It is this sequence of events that must be interrupted in a definitive manner to achieve prevention. Screening is coming under increasing criticism due to false positives, the detection of cancers that may never develop to present a significant risk, over-diagnosis and unnecessary but in many cases major or even aggressive treatment.

An obvious question is how did we evolve to naturally deal with what one might call adventitious cancer? The natural generation of cancer cells in fairly large numbers is not disputed and unless some natural mechanism exists for the body to destroy them, everyone would get cancer at a fairly young age. Starting in the 1990s, a theory was developed that there is a general protective mechanism that involves a certain class of organic chemicals in fruits and vegetables that takes advantage of unique enzyme present only in cancer cells which these chemicals target acting as substrates for the enzyme (CYP1B1) found expressed as a protein only in the cytoplasm of cancer cells.[5] They are metabolized and the metabolites either induce cell death (apoptosis) or cell division arrest which accomplishes the same end. These chemicals, called Salvestrols, are what chemists call polyphenols and while several dozen with this result of CYP1B1 action exist, scientists have identified those with the greatest potency to accomplish the above cancer cell death in cell culture studies and formulated an oral supplement. Just eating a variety of fruits and vegetables, while no doubt protective, does not in the 21st century offer the protection that existed prior to industrialized agriculture and in fact the only good sources of the selected fruits for Salvestrols come from organically grown heritage varieties, the latter retaining potency lost through hybridization to eliminate tart and bitter flavor, a characteristic of Salvestrols. This has been discussed repeatedly in IHN in the context of cancer therapy with emphasis on the fact that the above-described mechanism is not site specific since any cancer cell qualifies as a target.

As suggested by your editor in a recent journal article, there is considerable evidence that Salvestrols may also kill cancer stem cells thus interrupting a metastatic path.[6] The success of Salvestrols in cancer therapy has been demonstrated in a number of case histories where total remission was achieved.[7] Thus it is not surprising that the notion of using Salvestrols for primary prevention makes perfect sense if these polyphenols can deal successfully with the much larger number of cells in tumors, large enough to lead to the clinical diagnosis of cancer. They are fruit based, exquisitely targeted, independent of cancer type and totally non-toxic to normal cells. Sounds rather like the Holy Grail of cancer prevention and therapy. However, as explained above, to have a significant impact, the concentrated extract is necessary and the commercial product solves the problem of finding organic, heritage fruits.

Another non-site specific approach has recently received considerable attention. This is the ketogenic diet since cancer cells require large amounts of glucose for energy and are unable to use ketones which normal cells use when glucose is in short supply. This preventive action is closely related to the hypothesis that cancer is a metabolic disease centered in the dysfunction of the mitochondria, the cellular organelles involved in energy production.[8,9] The hypotheses is in competition with the deeply enshrined hypotheses that cancer is a genetic disease which seems to be losing supporters as

more is known about the immense and apparently quite hopeless complexity of cancer cell generics and epigenetics which hinders drug development,

It is suggested that readers consider the following protocol for comprehensive cancer primary prevention rather than just staying healthy in general. For such a protocol to be practical, it must focus on cancer, be simple, easy to implement and relatively inexpensive. The inclusion of diet is problematic since as the saying goes, it is easier to convince someone to change their religion than their diet. Nevertheless, the ketogenic diet is rapidly gaining popularity and individuals may find variations that are attractive and in addition, there are quite probably other health benefits.
This is a protocol presented for the reader's consideration.

- Three Salvestrol 2000 point capsules before breakfast, twice a week. Cost about $30 CDN ($25 US) per month. Include 500 mg of vitamin C with each dose since there appears to be significant synergistic effect. The unit *point* reflects the measured cancer cell cytotoxicity *in vitro*. Dosing was suggested in discussions with researchers**. From what has been described above, it should be clear that this is by far the most important component of the protocol.**
- Consider a ketogenic diet or a diet low in rapidly digested carbohydrates and especially sugar and refined and rapidly metabolized starches. Ideally, maximize organic food content and minimize prepared and ultra-prepared foods and watch out for added sugar.[10] The ketogenic diet is not well studied in this context and there are contradictory results.[11] The diet is described in many books and there are degrees of induced ketosis.
- High quality multivitamin with breakfast to provide Salvestrol enzyme cofactors.
- Get plenty of exercise.
- Maintain what is considered an optimum vitamin D level, with sun exposure or supplementation based on serum 25-hydroxy vitamin D measured in the fall and spring. Controversial as regards influence on cancer,[12] but the health benefits of maintaining a good or optimal vitamin D status justify its inclusion. Consensus appears to be that the good to optimal blood level of 25(OH)D is 50--80 ng/ml. Recent review: *Vitamin D Reduces Cancer Risk* by W. B. Grant (Google Orthomolecular News. Contains a list of news releases, February 6, 2019).
- Supplement with vitamin K2 (select supplement with MK-7 suggested). Considerable evidence of benefit in this context.[13] In addition, populations are commonly deficient. Note multivitamin preparations may not include vitamin K2 and also, vitamin K1 appears ineffective in this context.[14] Consensus on optimum dose in cancer prevention is about 50 micrograms/day of MK-7. Cheese and meat are the main food sources.
- Supplement with iodine, for example from kelp or seaweed extract or Lugol's solution (available from Amazon). Typical recommended dose by iodine experts is in the range of 1 to 1.5 mg mg/d. The average Japanese diet contains about 18 mg/d. The recommended daily allowance of 150 micrograms/day appears inadequate. Well-studied anti-cancer benefits (especially thyroid[15]) find that

iodine is commonly and seriously deficient. Also selenium has a synergistic effect. Possible risk reduction also found for breast cancer. Suggestion, read Dr. David Brownstein's book *Iodine: Why you need it, why you can't live without it.* In a recent blog, he mentions that many of his patients take 25 mg/day.

- Avoid aspartame because a case can be made for formaldehyde cell damage being in part carcinogenic.[16]

Additional dietary guidance is provided by Professor Gerry Potter, one of the scientists involved in the discovery and development of Salvestrols. It is called the red and green diet and provides guidance for selecting fruits and vegetables, ideally organic, to maximize natural Salvestrol intake. See the following link https://www.salvestrol.ca/doc/redgreendiet2.pdf

Critics would point out that that the above protocol has never been tested in long-term controlled studies. The simple answer is *of course not(!)* especially since the most important feature is a set of 4 encapsulated natural product extracts (polyphenols) which represent a targeted anti-cancer natural chemotherapy which cannot be patented. Therefore, we have to be satisfied at present with anecdotal evidence based on case histories and the informal reporting of physicians around the world who successfully use Salvestrols for cancer treatment and incidentally some also combine it with a ketogenic diet. Furthermore, the back extrapolation for treatment benefit to prevention is necessary since it is unlikely that there will be long-term prevention studies using this natural product, the principal reason being the required duration of such trials, the large required number of participants and the cost.

There seems little reason why this protocol cannot also be used to prevent recurrence after conventional treatment. For prostate cancer there is even a simple marker for recurrence, serum PSA. All too frequently, metastatic cancer is clinically manifest very late in its development and defies conventional treatment.

Finally, attention is called to a new (2nd) edition just published of the book *Salvestrols. Nature's Defense Against Cancer*, *Linking Diet and Cancer,* by Brian Schaefer (available at Amazon.com). It contains a significant amount of new information.

## MORE EVIDENCE OF THE DANGERS OF ARTIFICIALLY SWEETENED BEVERAGES

The latest is another study which again found a positive association between artificially sweetened beverages and cardiovascular acute events.[17] This study used data from the Women's Health Initiative and focused on postmenopausal US women. It was a follow-up study involved almost 82,000 women followed for a mean of about 10 years. Consumption was stratified according to never <1 drink/week, 1-4/week, 5-7/week and ≥2/day of artificially sweetened beverages. Enrollment was from 1993 to 1998. Outcomes were all stroke, ischemic stroke (clot), stroke due to bleeding (hemorrhagic), fatal and non-fatal coronary heart disease, and all-cause mortality. Only ≥ 2 drinks a day

consistently produced a positive association in all the endpoints except hemorrhagic stroke. Four models were used progressively adding confounding factors. Aside from the unadjusted model (not even age and race), all models produced statistical significant evidence of risk.

In the introduction the authors cite four studies for comparison. Three also yielded positive results. These were all follow-up studies based generally on one consumption assessment using a questionnaire. While in the study discussed here was no stratification by type of sweetener, the recruitment periods and follow-up suggest that the most common sweetener was aspartame and this was probably also true for the other studies the authors cited. Aspartame was approved for beverages in 1983 and the only serious competitor to appear later is sucralose which was approved in 1998 by the FDA.

If we focus on aspartame as the culprit in increasing both strokes and coronary heart disease events, fatal or non-fatal, as well as all-cause mortality, what might be the explanation or mechanism for the adverse effect? Readers of IHN probably know where this is leading since the dangers of aspartame have been discussed at length (June 2012 and November 2018 IHN issues). The aspartame molecule upon ingestion breaks up into two amino acids and methanol (aka methyl alcohol or wood alcohol). Once in the circulation, methanol poses no danger until it encounters an enzyme (alcohol dehydrogenase or ADH) that coverts it into formaldehyde. This occurs in the cytoplasm of cells that contain ADH. Formaldehyde is extremely reactive and generally does not get very far from the site of the reaction before reacting to produce in general a new molecule or modified polymer (e.g. DNA or RNA) that has deleterious effects in the context of not only heart and cardiovascular disease but in so-called diseases of civilization as well.[16] Actual methanol poisoning is very serious and frequently fatal if untreated.

The only antidote is ethanol, the alcohol in drinks. Ethanol competes with methanol for the services of the enzyme and if in high enough concentration essentially prevents the methanol to formaldehyde conversion and the methanol is naturally exerted. Thus the patient is kept quite drunk for several days. Chemists call this competitive inhibition. Ethanol is 16 times more effective than methanol in interacting with the AHD and thus fairly small levels of alcohol can allow most of the methanol to escape first pass oxidation in the liver and enter the circulation.

Now for the most curious aspect of this subject.[16,18,19] Humans are different than animals or non-human primates in how methanol is metabolized. Animals use the enzyme catalase to convert the alcohol to formaldehyde and then to formic acid. Human catalase is unable to metabolize methanol and the task is left to alcohol dehydrogenase (class 1). However, while the presence of catalase in humans and animals is widespread throughout the body, the ADH is not uniformly distributed but is present mainly in cells in the brain, blood vessels, skin, breast, kidney, bone, pancreas, lung and liver. If the encounter of a methanol molecule with AHD is in the liver and the result is formaldehyde, it may quickly encounter another enzyme, aldehyde dehydrogenase

present in large amounts in this organ and further metabolism will occur with no resultant toxicity. However, if this initial encounter occurs in a location where AHD is present but aldehyde dehydrogenase scarce or absent, and this is common, then the formaldehyde is free to react quickly with nearby molecules. These include basic proteins in the cytoplasm or nucleus and formaldehyde not only attaches but cross-links and inactivates them. Formaldehyde is much more toxic than folic acid, the next metabolite, which in fact is used as a food additive. However, the most important aspect of the problems formaldehyde create involves the localized nature of ADH.

The problem is that methanol is generally considered safe at concentrations commonly encountered due to smoking and eating canned fruit and bottled fruit juices. Methanol occurs in these foods because it is normally bound to pectin but the heating required for canning or juice pasteurization releases it. The supporting evidence for safety is overwhelmingly based on animal studies but animals have a high tolerance due to the rapid conversion of formaldehyde practically anywhere into non-toxic formic acid. Thus animals cannot be used to examine human toxicity but animal studies are very useful when industrial scientists wish to prove that methanol is not dangerous at low concentrations.

Getting back of the effect of formaldehyde on the vascular system which is one place where the enzyme ADH occurs, the reader should hear the story related by Woodrow Monte.[16] When he was early in his career as a nutritional scientist at Arizona State University, he was encouraged to assist is several autopsies to enhance his background and perspective. He spent a day assisting in three autopsies being done by the local medical examiner. They paid special attention to the conditions of the arteries, both in general and in the heart. The third autopsy was unforgettable. The subject was a hard-core alcoholic. In sharp contrast to the other two subjects, this individual had incredibly pristine arteries—no atherosclerosis at all, anywhere. The medical examiner noted Monte's surprise and commented that this was expected because the subject was an alcoholic. Pathologists, he was told, see this all the time and the phenomenon was independent of diet and smoking, the two principal sources of methanol if one does not consume aspartame. The perpetual drunk appears totally protected from atherosclerosis anywhere in the arteries by ethanol at high circulating concentrations. Alcoholics are taking daily the only methanol poisoning antidote and gaining protection mostly from the heavy load of methanol provided by smoking. The moral of the story is not the merits of continuous excessive ethanol consumption but the strong suggestion of a relationship between formaldehyde and atherosclerosis and thus heart disease, stroke, and other vascular problems.

Monte suggests that the before aspartame, a typical intake of methanol was about 8 mg/day.[16] Two 12 oz cans of a diet drink sweetened with aspartame would yield 50 mg, an 8 oz bottle of pasteurized fruit juice 18 mg. Also, 34 oz of diet soda is equivalent to the methanol inhaled by smoking a pack of cigarettes (83 mg).[16]

Studies of the dangers of artificially sweetened beverages generally do not consider aspartame special and the researchers appear to be unaware of the aspartame—

methanol—formaldehyde problem outlined above. Aspartame is unique among the currently popular sweeteners but it is considered safe based on animal studies which are not appropriate for examining its human toxicity. It is critical not to ignore the fact that the adverse effects are localized in the regions where the alcohol dehydrogenase is present, which localizes and focuses its toxicity. Furthermore, the results of formaldehyde reactions may be permanent and cumulative and thus long-term human studies with large cohorts concerning safety issues are needed but will probably never be carried out. Since medical science ignores the aspartame—methanol connection and the vast evidence base on which it exists (see Monte's 740 references for his book[16] available separately online). In the cited book, Monte makes the case for the role of aspartame in the so-called chronic diseases of civilization, all of which involve areas of the body where ADH is located.

More recently Walton and Monte make the case for the association between methanol and autism in a small study where the mothers of non-autistic children (550/711) consumed on average 9.5 mg of methanol per day whereas those with autistic children consumed 20.3 mg on average per day, mostly from aspartame and bottled or pasteurized fruit juice.[18]

## INTERMITTENT FASTING TO REVERSE TYPE 2 DIABETES

A recent study just reported found that intermittent fasting was able to reverse type 2 diabetes.[20] This study is actually made up of only three case histories and was published in the *British Medical Journal Case Reports.* Its importance includes the very long duration of the diabetes in the patients, ranging from 10 to 25 years. The male subjects all had insulin dependent diabetes, i.e. to control their blood sugar they needed insulin and patients 1 and 3 were on were on other diabetes medications as well. One of the investigators was Dr. Jason Fung who might be familiar to readers as the author of *The Obesity Code* discussed in IHN.

The intermittent fasting protocol involved a 24-hour fast three times a week for seven to eleven months. During the fast period, patients were allowed water, coffee, tea and bone broth and it was suggested they take a multivitamin supplement. On eating days, patients ate only lunch and dinner and were encouraged to consume a low sugar and low refined carbohydrate diet. The results presented in the table below were impressive.

| PATIENT | FAST DURATION MONTHS | FAST TYPE | INITIAL HbA1c (%) | FINAL HbA1c (%) | CHANGE WEIGHT Lbs. | DAYS TO COME OFF INSULIN | DIABETES YEARS |
|---|---|---|---|---|---|---|---|
| 1 | 7 | 3X/WEEK | 11 | 7.5 | 22 | 5 | 20 |
| 2 | 11 | 3X/WEEK | 7.2 | 6 | 23 | 18 | 25 |
| 3 | 11 | ALT DAYS | 6.8 | 6.2 | 18 | 13 | 10 |

All patients had a significant decrease in waist circumference. The initial HbA1c values reflect blood sugar control with anti-glycemic drugs and insulin replacement therapy.

Patients 2 and 3 had final HbA1c in the low prediabetes range, but patient 1 remained above 6.6% and was on one anti-glycemic drug (canagliflozin). However, he had a very high initial HbA1c and perhaps 7 months was not long enough if he suffered from severe beta cell dysfunction and poor glycemic control. Readers will recall that the New Castle Diet became much less effective in achieving reversal if the duration of the disease exceeded 6-8 years.

The patients described feeling excellent or terrific on fasting days suggesting that this protocol was not associated with unpleasantness or hardship. At the end of the study, two of the subjects were off all diabetic medications, whereas patient #1 was still on a drug in the same class as Jardiance discussed in the October IHN.

These results imply that the intermittent fasting protocol also restores beta cell function which is somewhat surprising since the New Castle Diet lost effectiveness when diabetes duration exceeded 6-8 years whereas these patients had long duration up to 25 years.[21] This raises questions that need exploring.

The authors of the report comment that they were unable to locate a single case study published in the las 30 years where therapeutic fasting was reported to as a cure for diabetes or eliminating the need for insulin. Note that the success achieved by Professor Roy Taylor involved about 700 calories per day for 2 months, not intermittent fasting.

As with other interventions, success is judged by more favorable markers rather than by observed decreases in diabetic-associated events such as cardiovascular, ocular etc. Over the years, many studies of drug interventions that lowered markers were found to yield very modest event rates. This highlights the issue of permanent damage which does not go away when the driving force is removed. This issue also remains in the context of the Newcastle Diet and will only be settled with a long-term follow-up of those with permanently reversed diabetes by looking for adverse acute events. The large number of subjects needed and the required 4-5 year trial would be exceedingly expensive.

In the book *The Obesity Code* Dr. Fung discusses intermediate fasting for diabetics on medication and points out that medical supervision is essential, that medication doses will need adjusting, that there is a danger of very low blood sugars which can be dangerous and even life threatening, and that constant blood sugar monitoring is essential. He also provides a bone broth recipe but Amazon sells powdered bone broth which can simplify making up this nutrient.

This study did not examine the question of the durability of the improved glucose metabolism and the three patients, i.e. the maintenance of the moderate or low-level prediabetic state. Taylor's group found that it was necessary to maintain the end-of-study weight in order to prevent relapse and this is probably true in the intermittent fasting study as well. In addition, some may find a maximum of 8 weeks of "semi starvation" but eating a selection of foods they like may be more attractive than fasting 3

times a week for 7 to 11 months with only bone broth plus common beverage. Both seem to work but the Newcastle diet has a much more extensive clinical base and in addition is supported by extensive research providing convincing physiological plausibility.[22]

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