More of the Mounting Evidence that Dextrose High Glycemic Sugars Causes and or Aggravates Cancer and Many More Diseases

UCSF Scientists Declare WAR on Sugar in Food

Like alcohol and tobacco, sugar is a toxic, addictive substance that should be highly regulated with taxes, laws on where and to whom it can be advertised, and even age-restricted sales, says a team of UCSF scientists. (University of California San Francisco)

In a paper published in Nature on Wednesday, they argue that increased global consumption of sugar is primarily responsible for a whole range of chronic diseases that are reaching epidemic levels around the world. The healthcare expense of sugar caused diseases is massive.

Sugar is so heavily entrenched in the food culture in the United States and other countries that getting people to kick the habit will require much more than simple education and awareness.
Childhood Cancer rises alongside sugar use
Dextose Processed Sugar weakens the cancer immune defense

SKY NEWS
HEADLINES
1,500 People die from Cancer each Day

In the 1960's one person in twenty got Cancer.
In the 1970's one person in fifteen got Cancer.
In the 1980's one person in twelve got Cancer.
In the 1990's one person in nine got Cancer.
In the 2000's one person in four got Cancer.
In 2010 one person in three gets Cancer.
Some say we are winning the battle against Cancer. They are Misled.
Metabolism’s Unexpected Role in Cancer A geneticist at the Salk Institute discusses his incredible discoveries. Mitzi Perdue Metabolism’s Unexpected Role in Cancer The discoveries made in Reuben Shaw’s lab could influence how we treat diabetes, Alzheimer’s, and even aging itself.

[© sheelamohanachandran - Fotolia.com] The relationship between metabolism, cancer, and genetics was for decades obscured in part by chance, but in the last decade, the relationship has been rediscovered, also at least in part by chance. Reuben Shaw, Ph.D., a geneticist and researcher at the Salk Institute, is at the center of this story, and interestingly, the discoveries made in his lab have not only resulted in new targets for cancer therapy, but longer term, they’re also likely to influence how we treat diabetes, Alzheimer’s, and even aging itself.

Lost Information To begin with the chance part of the story, what we now know to be true—that metabolism influences cancer—was well known at least 90 years ago. Back then, Otto Heinrich Warburg, a German physiologist, observed that tumor cells utilize glycolysis more than their normal counterpart cells despite being in normal oxygen conditions (the “Warburg Effect”). In 1931, Warburg won a Nobel Prize for his work on mitochondria. Subsequently he formulated the Warburg Hypothesis, that one of the causes of cancer is defective mitochondria.

In the 1980s, however, the discovery of “oncogenes” that directly caused cancer led researchers to believe that the Warburg Hypothesis for cancer causation was simply wrong. As the data on cancer-causing genes became both more comprehensive and more productive, cancer research switched to decoding genes, and a generation of researchers began ignoring metabolism as a factor.

Every single person who has cancer has a pH that is too acidic.

Dr. Otto Warburg won the Nobel Prize in 1931 for proving that cancer can’t survive in an alkaline, oxygen rich environment but thrives in an acidic, low oxygen environment.
Chance Intervenes Things changed, however, when Dr. Shaw, who was trained as a cancer researcher at MIT and Harvard Medical School, was accepted at the Molecular and Cell Biology Laboratory at the Salk Institute. As Dr. Shaw puts it, “Salk is the only place that has a strong and deep history of cancer and diabetes research that also has the laboratories for both housed in one building. This means that some of the top people in the country get to interact fluidly, including not only sharing knowledge but also their tools and equipment.”

From Dr. Shaw’s point of view, the location of both the cancer and diabetes researchers in the same building meant that he was benefiting on a daily basis from the unique tools and discoveries of both the cancer and diabetes researchers at Salk and the cross-fertilization of these two fields. He was therefore able to pursue his investigations of the connections between the two diseases in ways that might not have happened if he were in a silo-type building where all his colleagues were researching cancer alone or diabetes alone.

The Cancer-Diabetes Connection Before coming to Salk, he was already interested in a possible connection between the two diseases. As a postdoctoral fellow at the Harvard Medical School, he made the unexpected discovery in 2003 that LKB1, a gene causing 30% of lung cancers and 25% of cervical cancers was directly activating the enzyme AMPK, known to modulate diabetes and metabolism.

At this point, Dr. Shaw asked himself two seminal questions: “What did a diabetes gene have to do with cancer? And did the cancer gene have anything to do with diabetes?”

The answer turned out to be revelatory. AMPK is an ancient metabolic checkpoint that senses energy deprivation in the cells. Early in evolution, cells needed a sensor regulating their need for energy, and AMPK is found in organisms from simple yeasts to man and everything in between. AMPK responds to caloric restriction, exercise, hypoxia, low glucose, and metabolic hormones such as ghrelin or adiponectin.

In 2005, Dr. Shaw and his lab showed that metformin operates through LKB1 and AMPK to lower blood glucose. Since it is well-tolerated, it is the frontline treatment for type 2 diabetes with more than 120 million people taking it every day. However, as Dr. Shaw had postulated, at this time it was also becoming known that metformin reduces the risk of cancer in diabetic patients.

In 2008, now at Salk, Dr. Shaw and his lab discovered that AMPK directly shuts off a major oncogene called TOR, but it only does so when nutrients are low. This oncogene is the causal biochemical event in a number of human cancers, including kidney cancer, tuberous sclerosis, and LAM.

“LKB1 and AMPK act as a fuel gauge in our cells,” he explained in a recent interview, “and when energy is low, they instruct the cells to slow their metabolism. When tumor cells lack LKB1 or other parts of its pathway, they have, in effect, lost the sensor to know if their fuel levels are low.”
Interfering with Cancer’s Sweet Tooth Knowing that cells lacking LKB1 had lost their fuel gauges, Dr. Shaw wondered if this could be an entry point for disrupting tumor growth. Dr. Shaw already knew that factors such as exercise and calorie restriction could stimulate AMPK’s signaling ability, but were there, he wondered, drugs that could accomplish the same thing? Interestingly, the answer is yes.

The drugs metformin and phenformin both inhibit mitochondria; however, phenformin is nearly 50 times as potent as metformin. Dr. Shaw and his postdoctoral fellows tested both metformin and phenformin as chemotherapeutic agents in mice genetically engineered to mutate different cancer genes in adult lung cells, which results in the mice developing advanced-stage lung tumors. Only in mice lacking the LKB1 cancer gene did Dr. Shaw and his team observe that, after three weeks of treatment with phenformin, there was a major reduction in tumor burden in the mice.

Cancer’s Achilles’ Heel Knowledge of this leads to a profound impact on therapies for cancer because, as Dr. Shaw now knew, it was possible to interfere pharmacologically with this pathway. Disruptions of the “fuel sensing” mechanism means that with cancer cells, they could cause nutrient and oxygen deprivation. This had the medically important effect of signaling AMPK to arrest cell growth. The cancer cells would be influenced to cease proliferating.

But that’s not the end. The other side of the coin of being able to induce a faulty fuel-sensing mechanism is that the cancer cells may act as if they have all the energy and nutrients they need, even when they don’t. These results in the continuation of cell growth, and in the absence of fuel, the cells continue dividing until they run out of all energy stores and die.

Possible Clinical Trials “These studies,” he said, “are the tip of the iceberg. We are in the midst of decoding new links between metabolism and cancer that are going to result in new druggable targets. They are likely to be important in treating many different cancers, and they may also be effective for other diseases such as type II diabetes. In the future we may find that aberrations in these same pathways and the metabolic disturbances that result may underpin neurodegenerative diseases and other broad disease categories as well.”

A lot is at stake. The 90-year-old Warburg Hypothesis, re-evaluated by Dr. Shaw and his colleagues, could have an outsize impact on modern medicine. Let the clinical trials begin!

Mitzi Perdue, GEN’s corresponding editor, holds degrees from Harvard and George Washington University. She has authored more than 1,600 newspaper and magazine articles on science R&D and clinical medical applications, as well as on food, agriculture, and the environment. Perdue has a strong understanding of complex scientific and mathematical concepts. For 22 years, she was a syndicated columnist for the Scripps Howard News Service and before that, California’s Capitol News. Perdue is also
the author of the newsletter from the professional association, Academy of Women’s Health. She has produced and hosted more than 400 interview shows, often in conjunction with scientists at the University of California at Davis. She is a former Commissioner for the U.S. National Commission on Libraries and Information Science and a former Trustee for the National Health Museum.

KEYWORDS: Cancer, Diabetes, Metabolic Diseases, Metabolomics, Salk Institute

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<th>High GI</th>
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<tr>
<td>All-bran cereal (8.42)</td>
<td>Beets (5.64)</td>
<td>Popcorn (8.72)</td>
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<td>Watermelon (4.72)</td>
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<td>Carrots (3.47)</td>
<td>Pineapple (7.59)</td>
<td>Whole wheat flour bread (9.71)</td>
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<tr>
<td>Peanuts (1.14)</td>
<td>Sucrose, i.e. table sugar (7.68)</td>
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<tr>
<td>Strawberries (1.40)</td>
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<tr>
<td>Sweet Corn (9.54)</td>
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</tbody>
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Med GL: Apple juice (11.40)
Bananas (12.52)
Fettucine (18.40)
Orange juice (12.50)
Sourdough wheat bread (15.54)
Life Cereal (16.68)
New potatoes (12.57)
Wild rice (18.57)
Cheerios (15.74)
Shredded wheat (15.75)

High GL: Linguine (23.52)
Macaroni (23.47)
Spaghetti (23.47)
Couscous (23.65)
White rice (23.64)
Baked Russet potatoes (26.85)
Cornflakes (21.81)

Source: Revised International Table of Glycemic Index (GI) and Glycemic Load (GL), *The American Journal of Clinical Nutrition*, July 2002
It is well known that obesity is a leading cause of diabetes, a disease where the body fails to control blood sugar levels. High blood sugar levels are characteristic in obesity and diabetes. What is less well known is that diabetes and obesity are also linked to an increase in cancer risk. That is, the diabetic population has up to double chances to suffer pancreatic or colon cancer among others, according to well sustained epidemiological studies. With obesity in British and Spanish children reaching 16%, the highest in Europe, this epidemic has major health implications. How obesity or diabetes increase cancer risk has been a major health issue. Scientists led by Dr. Custodia Garcia-Jimenez at the University Rey Juan Carlos in Madrid have uncovered a key mechanism that links obesity and diabetes with cancer: high sugar levels, which increase activity of a gene widely implicated in cancer progression. Dr Garcia Jimenez's laboratory was studying how cells in the intestine respond to sugars and signal to the pancreas to release insulin, the key hormone that controls blood sugar levels. Sugars in the intestine trigger cells to release a hormone called GIP that enhances insulin release by the pancreas. In a study published in Molecular Cell, Dr Garcia Jimenez's team showed that the ability of the intestinal cells to secrete GIP is controlled by a protein called β-catenin, and that the activity of β-catenin is strictly dependent on sugar levels. Increased activity of β-catenin is known to be a major factor in the development of many cancers and can make normal cells immortal, a key step in early stages of cancer progression. The study demonstrates that high (but not normal) sugar levels induce nuclear accumulation of β-catenin and leads to cell proliferation. The changes induced on β-catenin, the molecules involved and the diversity of cancer cells susceptible to these changes are identified. Figure 1 shows the connection of High Glycemic abuse insulin resistance to many diseases.
NEW MRI RESEARCH REVEALS CANCER CELLS THRIVE ON PROCESSED SUGAR

rawforbeauty.com
source.naturalnews.com
Glycemic load, glycemic index, and carbohydrate intake in relation to pancreatic cancer risk in a large US cohort

Alpa V. Patel · Marjorie L. McCullough · Alexandre L. Pavlack · Eric J. Jacobs · Michael J. Thun · Eugenia E. Calle

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Abstract

Background Consumption of diets with high glycemic load has been hypothesized to increase pancreatic cancer risk by raising postprandial glucose levels and insulin secretion.

Methods The authors analyzed data from the American Cancer Society Cancer Prevention Study II (CPS-II) Nutrition Cohort to examine the association between pancreatic cancer and glycemic load, glycemic index (GI), and intake of carbohydrates. Diet was assessed among 124,907 men and women who were cancer-free and non-diabetic at baseline in 1992 using a validated 86-item food frequency questionnaire (FFQ). During 9 years of follow-up, 401 incident pancreatic cancer cases were identified. Cox proportional hazards modeling was used to compute hazard rate ratios (RR) adjusted for potential confounding factors.

Results We found no association between glycemic load, GI, or carbohydrate intake and risk of pancreatic cancer in this population. The hazard rate ratio (RR) was 1.01 (95% CI 0.75–1.37, trend P = 0.80) for glycemic load, 0.92 (95% CI 0.68–1.24) for GI, and 1.10 (95% CI 0.80–1.51) for carbohydrate intake among men and women in the highest quintile compared to the lowest quintile of each measure. We also found no significant association between these measures and pancreatic cancer risk among individuals who show a greater susceptibility towards insulin insensitivity, such as those who are overweight or more sedentary.

Conclusion Overall, our data do not support the hypothesis that glycemic load or index, or carbohydrate intake are associated with a substantial increase in pancreatic cancer risk; however, a weak positive association cannot be ruled out.

Keywords Glycemic load · Glycemic index · Carbohydrates · Pancreatic cancer · Prospective cohort

Abbreviations

RR rate ratio
CI confidence interval
CPS-II Cancer Prevention Study II
ICD International Classification of Diseases
NDI National Death Index
GI glycemic index
GL glycemic load
CHO carbohydrates
BMI body mass index
MET metabolic equivalent
SE standard error

Introduction

Pancreatic cancer is the fourth leading fatal cancer among US men and women [1]. Over 31,000 new cases and an almost equal number of deaths due to pancreatic cancer are projected for 2006 [1]. Cigarette smoking and diabetes mellitus are the only risk factors
<table>
<thead>
<tr>
<th>Glycemic Index</th>
<th>Value</th>
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<tr>
<td>High</td>
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<tr>
<td>Moderate</td>
<td>56–69</td>
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<tr>
<td>Low</td>
<td>&lt;55</td>
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Extra high levels of dextrose in the blood will diminish white blood cells and thus weaken immunity, irritate nerves, weaken cell membranes, lessen healing repair, and over-all decrease health.
One of the most important things to remember about cancer is it is NOT a chemotherapy disease, it is NOT a radiation disease and it is not a Vitamin C disease. Cancer is actually a metabolic dysfunction tied to genetic mutations, and the first step in fighting it is on the metabolic level. Let’s learn how oxygen plays a role in the development and treatment of cancer.

Every cancer has a trigger: infections, chemical toxins or heavy metal toxins are a few of the main ones. Early changes are seen through metabolic shifts that ultimately cause mutation, continually pushing genetic changes, growth and spread throughout the life of the cancer. Let’s take a look at how changes in oxygen metabolism are some of the first metabolic signs of difficult cancers.

Cancer is Fueled by Sugar and Destroyed by Oxygen

Oxygen’s Important Role In Cell Metabolism and Cancer Growth

Cancer is a very difficult to understand disease and there are many misconceptions associated with it. But one of the main keys of understanding, treating and ultimately winning the raging war against cancer is none other than oxygen. Eighth on the periodic table, oxygen is responsible for the breathing of cells and are essential role in providing energy.[1] However, cancerous, mutated cells thrive in anaerobic, or oxygen-lacking environments.
When growing, cancer cells show a change where they have lower levels of oxygen. This may stem from dysfunctions in the cell’s mitochondria (known as cellular “factories” that play a major role in cell respiration). If these issues go unchecked, it leads to further complications and malfunctions in apoptosis (programmed cell death). You may remember from biology class, mitochondria have two main functions: energy creation and policing uncontrolled division of cells.

Nobel Prize winner Dr. Otto Warburg famously hypothesized “...the prime cause of cancer is the replacement of the respiration of oxygen in normal body cells by a fermentation of sugar,” meaning, cancer is caused by a lack of oxygen. Today’s modern cancer cell biology has shown he was on the right track as mitochondrial health and shifting to a more oxygen-rich environment may protect healthy cells and further neuter cancer cells.

Furthermore, malignant, rapidly growing tumor cells typically have glycolytic rates up to 200 times higher than those of their normal tissues of origin. This means cancer has a much higher need for sugar than normal cells; this has been proven by the abnormally high level of insulin receptors found on all cancer cells. Because cancer cells favor the lack of oxygen, they shift to glycolytic pathways; put simply they use glucose as their source of energy. Cancer cells average about 16 times more insulin receptor sites than normal cells.

It’s important to realize that the genetics of a cancer in its early stages and its late stages are completely different. This is what makes late-stage cancer so complex and difficult to treat – you’re essentially trying to overcome these numerous advanced metabolic changes. Let’s look at some of the gene pathways that make this issue even more compounded.

**Angiogenesis and Genes That Fuel Cancer Growth**

**Angiogenesis** is a normal, healthy cell process through which new blood vessels form from pre-existing vessels. However, it’s also the fuse which sets off unchecked growth, turning benign tumors into malignant steamrollers. It’s also what transitions the metabolism of the cancer, making it that much harder to kill.

**Hypoxia** is when a portion of the body doesn’t have adequate oxygen supply. **Hypoxia-inducible factor 1-alpha**, (HIF-1-alpha,) is a protein that is encoded by the HIF1A gene, playing an essential role in cellular and systemic responses to hypoxia. Cancer cells use this protein to grow their blood supply and spread.
According to a study by the Liver Cancer Institute at Zhongshan Hospital and Shanghai Medical School in Shanghai, “HIF-1alpha in HCC [hepatocellular carcinoma, the most common form of liver cancer] plays an important role in predicting patient outcome. It may influence HCC biological behaviors and affect the tumor inflammation, angiogenesis and act in concert with the oncogene MYC [a gene found in many cancers]. Attaching importance to HIF-1alpha in HCC may improve the prognostic and therapeutic technique.” [2]

Epidermal Growth Factor Receptor (EGFR) is normally used to tell cells to grow. It is found in all cancer cells. However, EGFR over-expression has been linked to numerous cancers, such as lung, prostate, colon, breast, anal and others.

This receptor is also associated with increased chemotherapy resistance, leading to tumors that are untreatable. Additionally, EGFR is linked to insulin, making it the metabolic gasoline that fuels changes and growth in the cell.

This also links back to HIF-1 alpha. According to a study by the Department of Pathology at the VU University Medical Centre in Amsterdam, “In invasive breast cancer, HIF-1alpha is associated with angiogenesis, and expression of growth factors [including] the receptor EGFR. Thus, agents targeting HIF-1 may combine different pathways of inhibiting breast cancer growth, including angiogenesis and growth factors.” [3]

Discovery of Tumor M2-PK Proves Cancer Cells Shift From Oxygen to Glucose as Source of Energy

M2-PK (also known as PKM2) is an enzyme that is important in tumor metabolism, discovered in 2010 by Harvard Medical School. Tumor M2-PK helps cancer cells shift to greater glycolytic pathways. It is only found in cancer cells and not in normal healthy cells, making M2-PK an excellent marker for monitoring excelled growth or tracking improvement in treatment, depending if levels are high or low.

Reactive Oxygen Species and Chemotherapy

Chemotherapy and radiation therapy both rely on Reactive Oxygen Species (ROS) to work, augmenting ROS stress. ROS are essential toxic substances like hydrogen peroxide and others that can cause damage to cells in high concentrations. ROS are natural byproducts of the metabolism of oxygen, however, more resistant cancers actually produce their own antioxidants to fight these toxic substances.
Earlier stage cancers do not appear to have the same defense mechanisms that are found in more resistant later stage cancers. This explains why chemotherapy and radiation therapy may not work in late-stage cancers. The answer may involve actually increasing ROS levels so therapy can kill cancer cells once again – this is the therapeutic aim of oxidative medicine, giving high doses of antioxidants and creating ROS instead of destroying it. Therefore, the dosing and delivery change the entire mechanism of action of integrative treatments.

In this form of ROS, oxygen is what actually allows chemotherapy and radiation to work. Several types of DNA damage are caused by ROS-related oxidation. That is the goal of effective cancer treatment, to not only kill cancer cells but their genetics as well. In many cases, when oxidative therapy is combined with correctly-tested chemotherapy you can improve overall treatment for patients.

Everyone’s metabolism is different and therefore, every cancer patient’s tumor’s metabolism is different. By using the oxygen metabolism and other signaling pathways like EGFR and M2-PK, doctors can find the specific metabolism and make the strongest push in their favor. This is one major focus of our group, as we see cancer for what it is: a metabolic dysfunction pushing for constant genetic mutations, which aids its spread.
The 10 Most Cancer Causing Foods

The truth is that many common food items have been scientifically shown to increase cancer risk, and some of them substantially.

Here are 10 of the most unhealthy, cancer-causing foods that you should never eat again:

1) **Genetically-modified organisms (GMOs)**

   It goes without saying that GMOs have no legitimate place in any cancer-free diet, especially now that both GMOs and the chemicals used to grow them have been shown to cause rapid tumor growth. But GMOs are everywhere, including in most food derivatives made from conventional corn, soybeans, and canola. However, you can avoid them by sticking with certified organic, certified non-GMO verified, and locally-grown foods that are produced naturally without biotechnology.

2) **Processed meats**

   Most processed meat products, including lunch meats, bacon, sausage, and hot dogs, contain chemical preservatives that make them appear fresh and appealing, but that can also cause cancer. Both sodium nitrite and sodium nitrate have been linked to significantly increasing the risk of colon and other forms of cancer, so be sure to choose only uncured meat products made without nitrates, and preferably from grass-fed sources.

3) **Microwave popcorn**

   They might be convenient, but those bags of microwave popcorn are lined with chemicals that are linked to causing not only infertility but also liver, testicular, and pancreatic cancers. The U.S. Environmental Protection Agency (EPA) recognizes the perfluorooctanoic acid (PFOA) in microwave popcorn bag linings as “likely” carcinogenic, and several independent studies have linked the chemical to causing tumors. Similarly, the diacetyl chemical used in the popcorn itself is linked to causing both lung damage and cancer.

4) **Soda pop**
Like processed meats, soda pop has been shown to cause cancer as well. Loaded with sugar, food chemicals, and colorings, soda pop acidifies the body and literally feeds cancer cells. Common soda pop chemicals like caramel color and its derivative 4-methylimidazole (4-MI) have also specifically been linked to causing cancer.

5) ‘Diet’ foods, beverages

Even worse than conventional sugar-sweetened soda pop, though, is “diet” soda pop and various other diet beverages and foods. A recent scientific review issued by the European Food Safety Authority (EFSA) of more than 20 separate research studies found that aspartame, one of the most common artificial sweeteners, causes a range of illnesses including birth defects and cancer. Sucralose (Splenda), saccharin and various other artificial sweeteners have also been linked to causing cancer.

6) Refined ‘white’ flours

Refined flour is a common ingredient in processed foods, but its excess carbohydrate content is a serious cause for concern. A study published in the journal Cancer Epidemiology, Mile Markers, and Prevention found that regular consumption of refined carbohydrates was linked to a 220 percent increase in breast cancer among women. High-glycemic foods in general have also been shown to rapidly raise blood sugar levels in the body, which directly feeds cancer cell growth and spread.

7) Refined sugars

The same goes for refined sugars, which tend to rapidly spike insulin levels and feed the growth of cancer cells. Fructose-rich sweeteners like high-fructose corn syrup (HFCS) are particularly offensive, as cancer cells have been shown to quickly and easily metabolize them in order to proliferate. And since cookies, cakes, pies, sodas, juices, sauces, cereals, and many other popular, mostly processed, food items are loaded with HFCS and other refined sugars, this helps explain why cancer rates are on the rise these days.

8) Conventional apples, grapes, and other ‘dirty’ fruits

Many people think they are eating healthy when they buy apples, grapes, or strawberries from the store. But unless these fruits are organic or verified to be pesticide-free, they could be a major cancer risk. The Environmental Working Group (EWG) found that up to 98 percent of all conventional produce, and particularly the type found on its “dirty” fruits list, is contaminated with cancer-causing pesticides.

9) Farmed salmon

Farmed salmon is another high-risk cancer food, according to Dr. David Carpenter, Director of the Institute for Health and the Environment at the University of Albany. According to his assessment, farmed salmon not only lacks vitamin D, but it is often contaminated with carcinogenic chemicals, PCBs (polychlorinated biphenyls), flame retardants, pesticides, and antibiotics.

10) Hydrogenated oils
They are commonly used to preserve processed foods and keep them shelf-stable. But hydrogenated oils alter the structure and flexibility of cell membranes throughout the body, which can lead to a host of debilitating diseases such as cancer. Some manufacturers are phasing out the use of hydrogenated oils and replacing them with palm oil and other safer alternatives, but trans fats are still widely used in processed foods.
# Vitamin D and Cancer

<table>
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<th>Vitamin D Effects</th>
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<tr>
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<tr>
<td>Loss of sensitivity for growth inhibiting signals</td>
<td>Enhances cell differentiation</td>
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<td>Unlimited growth potential</td>
<td>Activates apoptosis</td>
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<td>Insensitivity for active cell death (= Apoptosis)</td>
<td>Inhibits angiogenesis in tumours</td>
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<td>Continuous neo-angiogenesis</td>
<td>Decreases metastatic potential</td>
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<tr>
<td>Tissue-invasion and growth in other organs</td>
<td>Activates the immunsystem</td>
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So we must deal with:

1. radiation
2. fatty acid deficiency
3. smoke
4. toxins
5. viruses
6. stress
7. sugars
8. anti-biotics
9. toxic foods (bologna)
10. mental disturbances
11. general all around health
Specific Sugar Molecule Causes Growth of Cancer Cells

Sep. 16, 2013 — The process of glycosylation, where sugar molecules are attached to proteins, has long been of interest to scientists, particularly because certain sugar molecules are present in very high numbers in cancer cells. It now turns out that these sugar molecules are not only present but actually aid the growth of the malignant cells. In the long term this discovery is an important step towards a cure that can stop the growth of cancer cells.

In co-operation with a research group from Singapore, scientists at University of Copenhagen have shown that immature sugar molecules in the form of truncated O-glycans aid growth properties of cancer cells. Previously, scientists have not been able to decode the significance of these truncated O-glycans, and therefore, the results, which were recently published in the journal PNAS, represent an important contribution to understanding the growth of cancer cells as well as the work towards developing a cure that can limit or stop the growth.

Catharina Steentoft, PhD student at Copenhagen Center for Glycomics and one of the scientists behind the results, stresses that this is basic science and there is still a long way from the results to actually developing a treatment or using them for diagnostic purposes. The results are still a cause for optimism, though.

"This is part of how we will proceed in the battle against cancer. When you know a certain process is important for the development of cancer you can start to consider ways to affect this process in a way that stops the cancer cell from taking advantage of it," explains Catharina Steentoft.

Sugar molecules affect proteins
Sugar molecules play an important role in almost all of the processes taking place in the body. One of the ways in which sugar molecules affect us is through glycosylation, a process where sugar molecules are attached to proteins. The proteins are basically the building bricks of the body, whilst sugar molecules affect the proteins, and therefore play a significant role in the human organism. A flaw in a chain of sugar molecules can lead to protein malfunctioning and disease.

As early as 1982, scientists around the world realized the importance of sugar molecules for cancer. The American doctor and scientist Georg F. Springer discovered that a certain type of sugar molecules, the truncated O-glycans, were particularly prominent in cancer cells. The discovery of Catharina Steentoft and colleagues builds on the foundation of this knowledge.

Pinpointing ways to proceed
For 30 years, scientists all over the world have worked on using the truncated O-glycans as biomarkers for diagnostics and outcome-prediction, but now the group of researchers from Singapore and Copenhagen has finally pinpointed the significance of these sugar molecules -- that they actually cause the cancer cells to grow and the cancer to spread more aggressively.

“We have now taken the first step towards understanding how cancer cells can change their glycosylation and produce these truncated O-glycans. It is a rather big step forward since it gives us an entirely new understanding of something we have worked many years to grasp. It guides our entire field of research towards new ways to proceed in the battle against cancer,” Catharina Steentoft says.
Sugar molecules on the surface of cells change their characteristics during development of cancer. Normal cells, as shown on the left, typically have long chains of sugar molecules (illustrated by circles filled with different colours and with the protein, that the chains are attached to, illustrated as a black line) that end in our blood type antigen like ABO. Cancer cells, on the contrary, often have truncated, immature chains of sugar molecules, as shown on the right. (Credit: University of Copenhagen)
High-Glycemic Foods Linked to Colon Cancer

Insulin resistance linked to diabetes may promote colon tumor growth

By Sid Kirchheimer
WebMD Weight Loss Clinic
Reviewed By Brunilda Nazario, MD

Feb. 3, 2004 -- A diet rich in foods that trigger a quick and drastic jump in blood sugar levels can do more than just boost risk of type 2 diabetes and contribute to obesity. A new study indicates they may also lead to colon cancer.

- Magnesium-rich foods lower type 2 diabetes risk
- Get the real facts about nutrition
- Eat well -- even with diabetes

Researchers at Harvard and UCLA find that the future risk of colorectal cancers is nearly three times higher in women who eat the most high glycemic-load foods compared with those who eat lesser amounts. These foods include breads, pastas, pancakes, and other carbohydrates made from refined "white" grains, as well as other processed or sugary foods such as cakes, cookies, and other snacks.

What Is Glycemic Index?
A food's glycemic index is a number that tells how much and how quickly blood sugar increases after eating a food that has carbs.
"We find a very straightforward and clear association between high-glycemic foods and the risk of colorectal cancers," researcher Simin Liu, MD, ScD, tells WebMD. "It's because these foods seem to trigger a greater tendency toward insulin resistance."

Insulin resistance, already linked to type 2 diabetes, is believed to create an environment in the colon that is conducive to tumor growth, says Liu, director of nutrition research at Harvard's Brigham and Women's Hospital and associate professor of medicine and epidemiology at Harvard Medical School.

His study, published in the Feb. 4 issue of the *Journal of the National Cancer Institute*, is now the fourth in recent years to find a link between high-glycemic foods and colon cancers, and comes as people with diabetes -- and those at risks of developing diabetes -- are increasingly urged to pay closer attention to the glycemic index of foods they eat.

With insulin resistance, cells' ability to respond to the action of insulin is hampered and blood sugars are not decreased to a normal range. To compensate, the pancreas secretes more insulin to help maintain a normal blood glucose level. Since high-glycemic foods are quickly digested, they provide a sudden rush of blood sugars that are not easily metabolized, prompting obesity as well as diabetes. Conversely, foods with a low-glycemic load -- typically those rich in fiber and slower to be digested -- raise blood sugar more gradually.

"This finding doesn't surprise me at all," says Marji McCullough, ScD, RD, nutritional epidemiologist and a senior researcher for the American Cancer Society. "The idea that insulin and its associated hormones elevate cancer risk is very plausible, and this mechanism is being actively studied in cancer research."

Already, she says, there is evidence that a diet rich in high-glycemic load foods can boost risk of pancreatic cancer, and there is some research -- which hasn't been verified -- that indicates these foods may also boost breast cancer risk.

As a general rule, low-fat, high-fiber foods -- fruits, vegetables, and whole grains and legumes -- often prescribed to manage weight and help prevent diabetes and other health conditions, have a low-glycemic index. Conversely, starchy and processed foods such as potatoes, breads, and cereals usually have a high-glycemic index.

**Problem Hard to Pinpoint**

"It's hard to pinpoint the real impact of a high-glycemic load diet, because it varies on a number of things, such as how much the food is processed, how big is the meal, and what other foods are in the meal," she tells WebMD. "If you have large portions, for instance, that also raises blood sugars very quickly."
These other factors may explain why the largest and longest study to date -- tracking nearly 50,000 Canadian women for 16 years -- found no link between intake of high-glycemic foods and future risk of colon cancers. That study was published last June.

"Because there have been very few studies of this issue to date, it is too early to make a final decision regarding an association with glycemic load to colon cancer," says Paul Terry, PhD, of the National Institute of Environmental Health Sciences in North Carolina, who led that study. "But the results of this study are interesting."

In Liu's study, some 40,000 American women filled out detailed questionnaires about their eating habits and their rates of colorectal cancers were tracked for nearly eight years. Overall, women consuming the most high-glycemic foods were three times more likely to develop colon cancers, but some women observed had a sixfold risk.

Although there are exceptions -- potatoes, All-Bran cereal, and bananas, for instance, have a high-glycemic load. But most "whole" foods rich in fiber have a low-glycemic load and since they take longer to digest, are less likely to trigger insulin resistance.

"If one was to make recommendations, I'd say you should replace refined grains with whole grains, replace potatoes with other vegetables, and eat more nuts, which have a low-glycemic load," Liu tells WebMD. "It's hard to pinpoint whether it's the fiber per se, or the fact that foods that contain high fiber also have phytochemcials that also help in preventing colon cancer."

SOURCES: Higgenbotham, S, *Journal of the National Cancer Institute*, Feb. 4, 2004; vol 96: pp 229-233. Terry, P. *Journal of the National Cancer Institute*, June 18, 2003; vol 95: pp 914-916. Simin Liu, MD, ScD, director, nutrition research, Brigham and Women's Hospital; assistant professor, epidemiology; assistant professor, medicine, Harvard Medical School, Boston. Marji McCullough, ScD, RD, nutritional epidemiologist; senior researcher, American Cancer Society, Atlanta. Paul Terry, PhD, research fellow, epidemiology branch, National Institute of Environmental Health Sciences, Research Triangle Park, N.C.
According to researchers at the University of California, San Francisco, sugar poses a health risk—contribute to around 35 million deaths globally each year. So high is sugar's toxicity that it should now be considered a potentially toxic substance like alcohol and tobacco. Its link with the onset of diabetes is such that punitive regulations, such as a tax on all foods and drinks that contain "added" sugar, are now warranted, the researchers concluded. They also recommend banning sales in or near schools, as well as placing age limits on the sale of such products.

Sugar's harmful effects do not stop at diabetes, metabolic syndrome, hyper- and hypoglycemia, GERD and heart disease. Sugar and cancer are locked in a death grip, yet oncologists often fail to do what's necessary to stop their patients from feeding their cancers with sweets.
Whereas many within the mainstream medical community insist on promoting the belief that the link between certain types of food with an increased risk of cancer is "weak" or only "nominally significant." They believe that research "linking foodstuffs to cancer reveals no valid medical patterns." We also find such superficial attitudes promoted in the medical press—all of which lack any kind of medical depth.

An increasing number of medical scientists and many alternative practitioners know that the most logical, effective, safe, necessary and inexpensive way to treat cancer is to cut off the supply of food to tumors and cancer cells, starving them with a lack of glucose. The therapeutic strategy for selective starvation of tumors by dietary modification (ketogenic diet) is one of the principle forms of therapy that is necessary for cancer patients to win their war on cancer.

Researchers at Huntsman Cancer Institute in Utah were one of the first to discover that sugar "feeds" tumors. The research published in the journal Proceedings of the National Academy of Sciences said, "It's been known since 1923 that tumor cells use a lot more glucose than normal cells. Our research helps show how this process takes place, and how it might be stopped to control tumor growth," says Don Ayer, Ph.D., a professor in the Department of Oncological Sciences at the University of Utah.

Dr. Thomas Graeber, a professor of molecular and medical pharmacology, has investigated how the metabolism of glucose affects the biochemical signals present in cancer cells. In research published June 26, 2012 in the journal Molecular Systems Biology, Graeber and his colleagues demonstrate that glucose starvation—that is, depriving cancer cells of glucose—activates a metabolic and signaling amplification loop that leads to cancer cell death as a result of the toxic accumulation of reactive oxygen species (ROS).[1]

Refined sugars are strongly linked to cancer, not only as a cause of it but also as something that feeds the cancer cells once a person has the disease—Nothing could be more important to consider in the attempt to improve the outcome of cancer treatments. The kinds of sugar so prevalent in today's standard American diet lead to cancer directly by causing inflammation throughout the body but in some places more than others depending on the individual and their constitution. Listen to this video and hear how simple this all really is. Once cancer cells are established in the body, they
depend on steady glucose availability in the blood for their energy; they are not able to metabolize significant amounts of fatty acids or ketone bodies,[2]. so they need sugar.

**Suppress/ Delay/ Slow/ Kill Cancer**

Carbohydrates of one of the three macronutrients—the other two being fats and protein. There are simple carbohydrates and complex carbohydrates. Simple carbohydrates include sugars found naturally in foods such as fruits and fruit juices, sodas, some vegetables, white bread, white rice, pasta, milk and milk products, most snack foods, sweets, etc. But let us not forget the simple sugars added to foods during processing and refining that we may have no awareness of. It's the simple sugars that get most of the credit for causing the insulin response and glycation-associated inflammation that can lead to cancer.

Thus by reducing the amount of simple carbohydrates in the diet, the emergence of cancer can be suppressed or delayed, or the proliferation of already existing tumor cells can be slowed down, stopped and reversed by depriving the cancer cells of the food they need for survival.

Drs. Rainer Klement and Ulrike Kammerer conducted a comprehensive review of the literature involving dietary carbohydrates and their direct and indirect effect on cancer cells, which was published in October 2011 in the journal *Nutrition and Metabolism*, concluding that cancers are so sensitive to the sugar supply that cutting that supply will suppress cancer.[3] "**Increased glucose flux and metabolism promotes several hallmarks of cancer such as excessive proliferation, anti-apoptotic signaling, cell cycle progression and angiogenesis.**"

Also, eating white sugar (or white anything) causes **magnesium mineral deficiencies** because the magnesium has been removed in the processing, making sugar a ripe target as a major cause of cancer because deficiencies in magnesium are not only pro-inflammatory but also pro-cancer.

**More Ways to Cause Cancer with Sugar**

**High fructose corn syrup** (HFCS) causes cancer in a unique way because much of it is contaminated with mercury due to the complex way it is made. High fructose corn
syrup causes selenium deficiencies because the mercury in it binds with selenium, driving selenium levels downward. Selenium is crucial for glutathione production and its deficiency in soils tracks mathematically with cancer rates. Selenium and mercury are also eternal lovers having a strong affinity to bond with each other.

Already touched on briefly, excess sugar spikes insulin levels and insulin's eventual depletion. High insulin and insulin-like growth factor (IGF-1) are needed for the control of blood sugar levels that result from chronic ingestion of high-carbohydrate meals (like the typical American diet, that is full of grains and sugars). Increased insulin levels are pro-inflammatory and pro-cancer and can directly promote tumor cell proliferation via the insulin/IGF-1 signaling pathway.

**Dr. Christine Horner** has a lot to say to women about insulin and breast cancer:

> When it comes to breast cancer, insulin is no friend. One of the biggest reasons is due to the fact that both normal breast cells and cancer cells have insulin receptors on them. **When insulin attaches to its receptor, it has the same effect as when estrogen attaches to its receptor: it causes cells to start dividing.** The higher your insulin levels are, the faster your breast cells will divide; the faster they divide, the higher your risk of breast cancer is and the faster any existing cancer cells will grow.

There's also another detriment that high insulin levels can inflict. It makes more estrogen available to attach to the estrogen receptors in breast tissue. Insulin regulates how much of the estrogen in your blood is available to attach to estrogen receptors in your breast tissue. When estrogen travels in the blood, it either travels alone seeking an estrogen receptor, or it travels with a partner, a protein binder, that prevents it from attaching to an estrogen receptor. Insulin regulates the number of protein binders in the blood. So, the higher your insulin levels are, the fewer the number of protein binders there will be and therefore the more free estrogen that will be available to attach to estrogen receptors.

In other words, when your insulin levels are up, free-estrogen levels are up, and both of them speed up cell division. That's why high insulin levels increase your risk of breast cancer so much. Eating sugar increases your risk of breast cancer in another way. **It delivers a major blow to your immune system with the force of a prizefighter.**
Dr. Horner talks about a study conducted by Harvard Medical School (2004) that found that women who, as teenagers, ate high-glycemic foods that increased their blood glucose levels had a higher incidence of breast cancer later in life. “So, encouraging your teenage daughter to cut back on sugar will help her to lower her risk of breast cancer for the rest of her life,” she said.
Sugar, Inflammation, Angiogenesis & Cancer

Sugars and the inflammation and acidic environments they create are important constituents of the local environment of tumors. In most types of cancer inflammatory conditions are present before malignancy changes occur. "Smoldering inflammation in tumor microenvironments has many tumor-promoting effects. Inflammation aids in the proliferation and survival of malignant cells, promotes angiogenesis and metastasis, subverts adaptive immune responses, and alters responses to hormones and chemotherapeutic agents."[4]

Dr. Mark Sircus, Ac., OMD, DM (P) (acupuncturist, doctor of oriental and pastoral medicine) is a prolific writer and author of some astounding medical and health-related books. His books are heavily referenced, and for many years Dr. Sircus has been researching into the human condition and into the causes of disease; he has distilled many of the divergent medical systems into a new form of medicine that he has coined Natural Allopathic Medicine.
ON A BRISK SPRING Tuesday in 1976, a pair of executives from the Sugar Association stepped up to the podium of a Chicago ballroom to accept the Oscar of the public relations world, the Silver Anvil award for excellence in "the forging of public opinion." The trade group had recently pulled off one of the greatest turnarounds in PR history. For nearly a decade, the sugar industry had been buffeted by crisis after crisis as the media and the public soured on sugar and scientists began to view it as a likely cause of obesity, diabetes, and heart disease. Industry ads claiming that eating sugar helped you lose weight had been called out by the Federal Trade Commission, and the Food and Drug Administration had launched a review of whether sugar was even safe to eat. Consumption had declined 12 percent in just two years, and producers could see where that trend might lead. As John "JW" Tatem Jr. and Jack O'Connell Jr., the Sugar Association's president and director of public relations, posed that day with their trophies, their smiles only hinted at the coup they'd just pulled off.

Their winning campaign, crafted with the help of the prestigious public relations firm Carl Byoir & Associates, had been prompted by a poll showing that consumers had come to see
sugar as fattening, and that most doctors suspected it might exacerbate, if not cause, heart disease and diabetes. With an initial annual budget of nearly $800,000 ($3.4 million today) collected from the makers of Dixie Crystals, Domino, C&H, Great Western, and other sugar brands, the association recruited a stable of medical and nutritional professionals to allay the public's fears, brought snack and beverage companies into the fold, and bankrolled scientific papers that contributed to a "highly supportive" FDA ruling, which, the Silver Anvil application boasted, made it "unlikely that sugar will be subject to legislative restriction in coming years."

The story of sugar, as Tatem told it, was one of a harmless product under attack by "opportunists dedicated to exploiting the consuming public." Over the subsequent decades, it would be transformed from what the New York Times in 1977 had deemed "a villain in disguise" into a nutrient so seemingly innocuous that even the American Heart Association and the American Diabetes Association approved it as part of a healthy diet. Research on the suspected links between sugar and chronic disease largely ground to a halt by the late 1980s, and scientists came to view such pursuits as a career dead end. So effective were the Sugar Association's efforts that, to this day, no consensus exists about sugar's potential dangers. The industry's PR campaign corresponded roughly with a significant rise in Americans' consumption of "caloric sweeteners," including table sugar (sucrose) and high-fructose corn syrup (HFCS). This increase was accompanied, in turn, by a surge in the chronic diseases increasingly linked to sugar. Since 1970, obesity rates in the United States have more than doubled, while the incidence of diabetes has more than tripled. (The chart below uses sugar "availability" numbers rather than the USDA's speculative new consumption figures.)
Precisely how did the sugar industry engineer its turnaround? The answer is found in more than 1,500 pages of internal memos, letters, and company board reports we discovered buried in the archives of now-defunct sugar companies as well as in the recently released papers of deceased researchers and consultants who played key roles in the industry's strategy. They show how Big Sugar used Big Tobacco-style tactics to ensure that government agencies would dismiss troubling health claims against their products. Compared to the tobacco companies, which knew for a fact that their wares were deadly and spent billions of dollars trying to cover up that reality, the sugar industry had a relatively easy task. With the jury still out on sugar's health effects, producers simply needed to make sure that the uncertainty lingered. But the goal was the same: to safeguard sales by creating a body of evidence companies could deploy to counter any unfavorable research.
Glycemic index: overview of implications in health and disease

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Abstract

The glycemic index concept is an extension of the fiber hypothesis, suggesting that fiber consumption reduces the rate of nutrient influx from the gut. The glycemic index has particular relevance to those chronic Western diseases associated with central obesity and insulin resistance. Early studies showed that starchy carbohydrate foods have very different effects on postprandial blood glucose and insulin responses in healthy and diabetic subjects, depending on the rate of digestion. A range of factors associated with food consumption was later shown to alter the rate of glucose absorption and subsequent glycemia and insulinemia. At this stage, systematic documentation of the differences that exist among carbohydrate foods was considered essential. The resulting glycemic index classification of foods provided a numeric physiologic classification of relevant carbohydrate foods in the prevention and treatment of diseases such as diabetes. Since then, low-glycemic-index diets have been shown to lower urinary C-peptide excretion in healthy subjects, improve glycemic control in diabetic subjects, and reduce serum lipids in hyperlipidemic subjects. Furthermore, consumption of low-glycemicindex diets has been associated with higher HDL-cholesterol concentrations and, in large cohort studies, with decreased risk of developing diabetes and cardiovascular disease. Case-control studies have also shown positive associations between dietary glycemic index and the risk of colon and breast cancers. Despite inconsistencies in the data, sufficient, positive findings have emerged to suggest that the dietary glycemic index is of potential importance in the treatment and prevention of chronic diseases.
INTRODUCTION

The concept of a glycemic index was developed to provide a numeric classification of carbohydrate foods on the assumption that such data would be useful in situations in which glucose tolerance is impaired. In many ways, the glycemic index concept was an extension of the dietary fiber hypothesis of Burkitt and Trowell (1), who suggested that foods that are more slowly absorbed may have metabolic benefits in relation to diabetes and to the reduction of coronary heart disease (CHD) risk. At the same time the dietary fiber hypothesis was formed, the concept of a cluster of diseases related to central adiposity and intraabdominal fat mass with attendant insulin resistance was being developed (2–5). The similarity of many of the issues that were raised after the formulation of both concepts further defined possible preventive and therapeutic roles for the glycemic index classification of foods. The necessary research in this area was greatly facilitated by the compilation of comprehensive glycemic index food tables (6).

GLYCEMIC INDEX AND GLYCEMIC LOAD

The glycemic index is the indexing of the glycemic response of a fixed amount of available carbohydrate from a test food to the same amount of available carbohydrate from a standard food consumed by the same subject (initially, the standard “food” was glucose, but more recently it has been white bread; 7, 8). The blood glucose area after consumption of the test food was expressed as a percentage of the standard. The glycemic load, which assesses the total glycemic effect of the diet and has proved very useful in epidemiologic studies, is the product of the dietary glycemic index and total dietary carbohydrate (9–11). In general, the insulin responses, when measured, related well to glycemic responses (12, 13). It also appeared that the rate of digestion of the food was an important determinant of glycemic response (14, 15). Thus, the rate of liberation of the carbohydrate products of digestion in vitro over 3–5 h reflected the blood glucose area in vivo (14). Intrinsic and extrinsic factors that alter the rate of gastrointestinal motility, digestion and absorption, and the nature of the starch, cooking method, particle size, and the presence of fiber, fat, and proteins were all found to result in differences in the glycemic index (16, 17). The starchy staples of traditional cultures were often foods that had lower glycemic indexes, such as pasta, whole-grain pumpernickel breads, cracked wheat or barley, rice, dried peas, beans, and lentils (18, 19). It appears that the traditional use of low-glycemic-index carbohydrate foods in the diet was particularly prevalent among cultures that are now experiencing high rates of diabetes, eg, the Pima Indians and the Australian Aborigines, and where the change to high-glycemic-index foods has been a more recent phenomenon (20–22). Obviously, many other factors, such as obesity and reduced physical activity, must play major roles in increasing diabetes risk. Nevertheless, over time the desire for sweet foods, which resulted from rapid carbohydrate breakdown of starch in the mouth, may have resulted in the selection of rapidly digested (and hence high-glycemic-index foods) as cultures became more affluent (18). Thus, foods with high glycemic indexes are proposed further as a dietary factor that favors the development of chronic disease.

CONCERN OVER UTILITY OF THE GLYCEMIC INDEX CLASSIFICATION

It is said that the glycemic index concept lacks clinical utility because differences in glycemic indexes between foods are lost once these foods are consumed in a mixed meal (23). Part of the reason for this is that when a mixed meal consists of several carbohydrate sources, the effect of the lower glycemic index component is diluted in proportion to the amount of carbohydrate from other foods. Appropriate calculation of the mixed-meal glycemic index is therefore required (8). Small amounts of fat added to the meal have also been considered to greatly alter the glycemic response. However, in studies in which 8–24 g fat was fed in mixed meals containing 38–104 g carbohydrate, the added fat had little effect on predicted glycemic response (24). Furthermore, although large deviations in the dietary macronutrient profile will occur over time, these differences by definition will also average out over time. Only in those subjects in whom there are substantial differentiations in daily macronutrient intake are changes in the dietary glycemic index likely to be obscured, and in such individuals any meaningful attempt at dietary modification is also likely to be difficult.

It is said that the glycemic index concept adds further needless complications and restrictions to the dietary management of diseases and that such factors cannot be justified by the modest gains that might accrue (25). An alternative view might be that the glycemic index is simply a tool for alerting the potential consumer to new starchy foods they may not otherwise have considered eating. Over time, the introduction of new foods will expand the range of food choices available, providing foods to be selected not only for their glycemic index, but also for their range of health advantages. A certain amount of dietary understanding is certainly required, eg, carrots with a high glycemic index are not taboo. It is realized that there are other considerations relevant to the consumption of carrots,
and that the glycemic index is not significant in low-energy foods in which the ratio of other desirable factors (eg, minerals, vitamins, and fiber) to available carbohydrate is high.

MECHANISMS OF ACTION

The hypothesized metabolic effects relate to the rate at which glucose is absorbed from the small intestine. A reduced rate of glucose absorption after the consumption of low-glycemic-index carbohydrate foods will reduce the postprandial rise in gut hormones (eg, incretins) and insulin. The prolonged absorption of carbohydrate seen over time will maintain suppression of the free fatty acids (FFA) and the counterregulatory responses, while at the same time achieving lower blood glucose concentrations (Figure 1). Over time, with the reduction in FFA concentrations and the rise in the respiratory quotient with tissue insulinization, glucose is withdrawn from the circulation at a faster rate. Consequently, blood glucose concentrations return toward baseline despite continued glucose absorption from the small intestine. The rise in peak postprandial blood glucose is the reforereduced together with the incremental blood glucose are above baseline. Studies in healthy men have shown this effect after a glucose solution was sipped at an even rate over 180m in a supposed to being consumed a sabolusatzerotime. A marked economy in insulin secretion with sipping the glucose solution was also seen, together with improved glucose clearance for intravenous glucose at 4 h. This was coincident with the lower serum FFA concentrations compared with those after the bolus intravenous-glucose-tolerance test. In part, this improvement, which was also seen after consumption of low-glycemic-index meals, may be the result of sustained tissue insulinization, suppression of FFA release, and the absence of a counterregulatory endocrine response. Other studies that used low-glycemic-index meals showed an improved second meal carbohydrate tolerance that was reminiscent of the Staub-Traugott effect (ie, in which the first meal improves the glucose tolerance of the second meal) and related the improved postprandial glycemia of the second standard meal to lower FFA concentrations.

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FIGURE 1.

Hypothetical effect of feeding diets with a low (A) or high (B) glycemic index on gastrointestinal glucose absorption and postprandial blood glucose.
FIGURE 2.

Mean (±SE) blood glucose, serum free fatty acids (FFA), serum insulin, serum C-peptide, and plasma gastric inhibitory polypeptide (GIP) concentrations after consuming a glucose solution (50 g in 700 mL H₂O) as a bolus over 5 min at time 0 (▪) or after sipping the same solution over 0–3.5 h at an even rate (□).

In addition, increased food frequency, as a model for mimicking the slow digestion of low-glycemic-index foods, has been shown to reduce glycemic and insulimemic responses over the course of a day in diabetic subjects (30, 31). In the longer term, increased food frequency has been associated with altered adipose tissue enzyme concentrations (32) and reduced fasting blood lipid concentrations, even though the same foods were eaten in the same amount in any given 24-h period (33–38). For reasons that are not clear, not all studies have shown these effects (39). However, spreading the nutrient load does not appear to be advantageous in terms of increased thermogenic effects that would favor weight reduction (40).

EFFECTS IN HEALTH AND DISEASE

In healthy young men, low-glycemic-index diets have minimal effects in the short term (Table 1♭;41, 42. In one euglycemic hyperinsulinemic clamp study, glucose disposal was impaired after 3 wk of a low-glycemic-index diet at high, but not low, insulin infusion rates (42). However, in another study of healthy men, 24-h urinary C-peptide output was found to be reduced with low-glycemic-index diets (41). In addition, LDL-cholesterol concentrations were reduced with the low-glycemic-index diet as was the serum C-peptide response to a standard breakfast after 2 wk. Nevertheless, there were higher blood glucose concentrations at 45 and 60 min that were associated with the lower C-peptide response. This apparent impairment in glucose tolerance may have been related to gut adaptive responses with less incretin secretion because the intravenous glucose tolerance test was similar in both treatments (41). On the other hand, middle-aged, insulin-resistant women, many of whom had already suffered a myocardial infarction, showed improved insulin sensitivity after an insulin tolerance test (43). In studies of persons with type 1 and 2 diabetes, most studies (10 of 14) (Table 14;44–55 showed improvement in glycated proteins, and in one study, plasminogen activator inhibitor 1 concentrations were also reduced (54). These effects occurred despite large variations in the glycemic index difference between the test and control treatments, the short duration of many studies, and the limited numbers of subjects in others. However, in an assessment of the effects of monounsaturated fat compared with high-carbohydrate diets and low- compared with high-glycemic-index diets in patients with diabetes, the effects of both interventions on glycated proteins were comparable (Figure 3♭;59). After consuming a low-glycemic-index diet for 1 mo, patients with hyperlipidemia showed reduced LDL-cholesterol and triacylglycerol concentrations (in those with higher triacylglycerol concentrations), despite no significant difference in body weight (58). These data are not definitive but suggest a potential therapeutic utility of the glycemic index concept.

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Controlled studies of the effects of low-glycemic-index (GI) diets on carbohydrate and lipid metabolism in healthy, diabetic, and hyperlipidemic subjects.

Table 1

Mean (±SD) difference from the control diet in glycated proteins (hemoglobin A1c or serum fructosamine) in diabetic subjects consuming either low-glycemic-index (GI) or high-monounsaturated–fatty acid (MUFA) diets. The vertical line indicates no effect. Adapted from reference 59.

Epidemiologic Insights

Two studies (one that used the third National Health and Nutrition Examination Survey database and the other a British study) showed a negative relation between glycemic index and HDL cholesterol, suggesting that low-glycemic-index diets may preserve HDL cholesterol and thus have a potentially positive effect in reducing CHD risk (Table 2). In relation to CHD, the Nurses' Health Study showed a negative relation between fatal and nonfatal myocardial infarctions and glycemic index, as well as glycemic load (11). Of particular interest was the observation that there was no association of dietary glycemic index with CHD in persons with a body mass index (in kg/m²) <23, suggesting that the effect of dietary glycemic index may be increasingly important in those with a greater degree of insulin resistance (Table 2). On the other hand, no significant association of glycemic index or glycemic load and CHD was seen in older men in the Zutphen study (67). The relatively small number of subjects in this study (<1500) and their age at the start of the study (65–84 y) may be part of the explanation: large numbers of the original cohort had already died or were excluded because of diabetes or CHD (Table 2). The population left was therefore preselected and may have been less vulnerable to environmental factors.

Table 2

Cross-sectional and cohort studies of the relation of glycemic index (GI) to the risk of cardiovascular disease, diabetes, and cancer and its association with HDL and glycated hemoglobin (Hb A1c). In relation to diabetes outcome, both the Nurses’ Health Study (9) and the Health Professionals Studies (10) showed an inverse relation between glycemic index and the risk of developing diabetes by using a validated food-frequency questionnaire. In the case of the Health Professionals Study, both the association and the trend became significant only after adjustment for fiber intake (10). The Iowa Women's Health Study, although it also showed a negative
association between cereal fiber intake and the risk of diabetes, showed no significant association between glycemic index or load and diabetes incidence (69). This discrepancy may relate to the frequency of application of the food-frequency questionnaire during the study, the glycemic index database used, and the age range of the subjects studied. Older cohorts selected as free of disease at the outset of a study may already have excluded a significant proportion of vulnerable subjects. In this respect, the Iowa Women’s Health Study subjects were generally older than the subjects in the Nurses’ Health Study (Table 2). The glycemic index may have relevance to cancer prevention. In addition, insulin resistance and insulin-like growth factors have been implicated in the so-called diet-related cancers: colon, breast, and prostate (73, 74). Preliminary data support this association for colon cancer (75). A case-control study showed a direct association between dietary glycemic index and colon cancer risk. A sedentary lifestyle in conjunction with a high-glycemic-index diet increased risk relative to a sedentary lifestyle with a low dietary glycemic index or relative to an active lifestyle with a high dietary glycemic index (76). An Italian case-control study reported that the dietary glycemic index was related to colorectal cancer risk, i.e., the higher the glycemic index, the greater the risk of colorectal cancer (71). The same relation of glycemic index and disease was also shown for breast cancer (72). Prostate and ovarian cancers, among other forms of cancer, may be influenced by the dietary glycemic index. In these cases, insulin resistance and insulin-like growth factors have also been implicated (72, 77). Therefore, the greater part of the epidemiologic literature provides additional support for a role of dietary glycemic index in disease.

NEWER ASPECTS OF GLYCEMIC INDEX RESEARCH

There is considerable interest in the relations between insulin resistance, the generation of reactive oxygen species, tissue damage, and the liberation of proinflammatory cytokines and acute phase proteins, the latter appearing to be powerful markers of chronic diseases, notably CHD (78). The dietary glycemic index may play a role in this sequence of events.

Studies have shown that the postprandial rise in glucose is consistent with depression of serum antioxidants, including lycopene and vitamin E (79, 80). Presumably, the higher the glycemia, the greater the postprandial depression of serum antioxidants (80). Finally, supplementing subjects’ diets with the antioxidant vitamin E has been shown to improve glycemic control (81). Studies such as these suggest a possible beneficial role for low-glycemic-index diets by reducing oxidative damage.

It has been suggested that obesity is related to glycemic index or glycemic load (28, 82, 83). Studies on altering glycemic index and load have indicated that the lower the glycemic index and load of the first meal, the less food is consumed in the subsequent meal (28). Longer-term studies are required to define the relevance of these interesting findings.

Finally, more studies are required to assess the relation of glycemic index to chronic diseases, including cancer, CHD, and diabetes. In addition, large-scale intervention studies are underway and more are required to define the therapeutic utility of the glycemic index concept.

CONCLUSION

The dietary glycemic index concept suggests a possible role for the rate of carbohydrate digestion in the prevention and treatment of chronic disease, including those diseases that have been highlighted in the dietary fiber hypothesis and are now associated with insulin resistance. This concept is no longer novel; pharmacologic approaches to slowing carbohydrate absorption, notably the use of α-glycoside hydrolase inhibitors, are now accepted in the management of diabetes.

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Footnotes

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and cut off our funding?!
and upset our profit
the hell you have...
Broccoli Cures Cancer

- George Carlin

Anti-Cancer Superfruits

Blueberries
Goji Berries
Acai Berries
Pomegranate
Grapes
Avocado
Soursop
Strawberries
Mangosteen
Noni
Apple
Kiwi
Sugar industry's secret documents echo tobacco tactics

Sugar Association's intent to use science to defeat critics uncovered by dentist


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When Cristin Couzens went on the hunt for evidence that Big Sugar had manipulated public opinion, she had no idea what she was doing. She was a dentist, not an investigative reporter. But she couldn't let go of the nagging suspicion that something was amiss.

Her obsession started in an unlikely place, at a dental conference in Seattle in 2007 about diabetes and gum disease. When one speaker listed foods to avoid, there was no mention of sugar. "I thought this was very strange," Couzens said. And when a second speaker suggested sugary drinks were a healthy choice, she chased him down at the end of the conference to make sure she'd heard him correctly. "How could you possibly recommend sweet tea as a healthy drink?" she asked the speaker, who paused just long enough to say, "There is no evidence that links sugar to chronic disease," before he bolted out the door.

Cristin Couzens publicized secret documents in a magazine article titled Big Sugar's Sweet Little Lies. (Eric Weber)

"I was so shocked by that statement," she said, "I felt obligated to do a little bit of research, thinking perhaps the sugar industry had somehow had an influence over the lack of advice to limit sugar intake to prevent and control diabetes. That's what set me off."

She quit her job, exhausted her savings and spent 15 months scouring library archives. Then one day she found what she was looking for, in a cardboard box at the Colorado State University archives.

"The first folder that I opened jumped right out at me," she said. "It was on the Sugar Association letterhead which is the trade association in Washington for cane and beet sugar producers. And the word "confidential" was right under the letterhead. So the first document I saw was a confidential Sugar Association memo talking about their PR strategies in the 70s."

What Couzens found was something food industry critics have been seeking for years — documents suggesting that the sugar industry used Big Tobacco tactics to deflect growing concern over the health effects of sugar.
"So I had lists of their board reports, their financial statements, I had names of their scientific consultants, I had a list of research projects they funded, and I had these memos where they were describing how their PR men should handle conflict of interest questions from the press," she said.

The documents survived in the Colorado University Library Archives only because they helped explain a photograph of three men and a trophy. When the Great West Sugar Company went out of business in the 1980s, someone put the files in a box so that librarians would know who the men were and why they were being honored. So who were they?

“That was a picture of sugar industry executives being awarded the Silver Anvil, which is like the Oscars of the PR world,” Couzens said. In the 1976 photo, the president of the Sugar Association and its director of public relations smile as they pose with their prize for their successful campaign “forging public opinion,” in the face of mounting consumer and government concern over the health risks of sugar.

“It's a little bit shocking to me that an industry would be rewarded for manipulating scientific evidence,” Couzens said. “At the time the award was given in 1976, there was a controversy. Many people thought sugar was harmful, the sugar industry wanted to turn public opinion toward thinking sugar was safe so they forged public opinion on how the public viewed the effects of sugar,” she said.

As Couzens sorted through the documents, the full extent of that campaign to forge public opinion emerged. The documents describe industry lobby efforts to sponsor scientific research, silence media reports critical of sugar, and block dietary guidelines to limit sugar consumption.

The Sugar Association's president reported to the Board of Director's meeting in October, 1976 that, "in confronting our critics we try never to lose sight of the fact that no confirmed scientific evidence links sugar to the death-dealing diseases. This crucial point is the life blood of the association."

Part 1: **Food cravings engineered by industry**

And the Sugar Association was clear about its intention to use science to defeat sugar’s critics. To support “our desire to maintain research as a main prop of the industry's defence,” the member companies were told that $230,000 was being reserved to fund scientific research, according to a report from the Sugar Association's annual meeting of the board of directors, in Chicago in 1977.

At the next meeting, a year later in Washington, the Sugar Association updated the board of directors on the research program, listing 17 different scientists at some of the world's most prestigious universities, including M.I.T., Harvard, and Yale, receiving sugar industry money.

Some of the money was supplied by “contributing research members,” heavy sugar users including "Coca-Cola, Hershey, General Foods, General Mills and Nabisco” who contributed the funds specifically for the lobby group's scientific effort. The document reports that those companies each “contributed $10,000 to our general research fund.”
Targeting media coverage

The documents also reveal efforts to manipulate media coverage. The Sugar Association had been stung by a New York Times headline: "The Bitter Truth About Sugar," written by a prominent nutritionist in June 1976. "It's bad for the health, bad for the teeth, and we eat more of it than we think", the article declared.

The Sugar Association president warned the board of directors that this "shoddy piece" was being followed up by "naive writers working for other papers around the country." But the association had saved the day. Thanks to an inside tip, they were able to keep Reader's Digest from running a condensed version of the same article.

"Thanks to friendly sources who alerted us, a progressive approach to research by the Digest and persistence on our part, we were able to persuade them to cancel the story," the document reports. "We did it in two stages. We failed in our initial conversations, but succeeded when we took our case to the editor-in-chief. Our telegraph to him is included in your folders and might be helpful should you be confronted by criticism."

The documents Couzens found in that cardboard box also reveal that the Sugar Association was busy trying to block dietary guidelines that would recommend limits on sugar consumption. At the time, the US Senate Select Committee on Nutrition and Human Needs, headed by Senator George McGovern, had released "Dietary Goals for the United States," which recommended that Americans should reduce their sugar intake by 40 per cent.

The Sugar Association had been warned by a committee insider that "the final conclusions would hang sugar," the association's president reported to the board in 1977. And now that the committee's report had been released the results "certainly bear this prediction out," he added.

But the lobby group had a plan. "The McGovern Report has to be 'neutralized'" that document reports, assuring the members that the Sugar Association would fight back, because "the consequences of losing this battle and permitting dietary goals to become a basic reference are too grave to be taken lightly."

Sugar industry committed to sucrose consumption

When Couzens approached the sugar industry for comment, she was told the documents were ancient history. "They gave a comment like, that was in the past and does not reflect the mission of the sugar association currently," she said. But then she found one more document, an internal Sugar Association e-newsletter from August, 2003 that announced "the Sugar Association is committed to the protection and promotion of sucrose consumption. Any disparagement of sugar will be met with forceful, strategic public comments and the supporting science."

'It's very, very difficult to find those kinds of ...[documents]

, so it was a real treasure.' — Marion Nestle

Couzens said that document showed "the sugar industry is still very active in nominating scientists to serve on the dietary guidelines advisory committee, and it is still publishing research through connections with the World Sugar Research Organization, based in London. These scientific reviews that are published by the sugar industry are still
considered in the evidence review for the dietary guidelines, so they’re still serving to balance out the evidence,” she said.

Armed with all of these documents, Couzens’ next challenge was to make them public somehow. She sought out author Gary Taubes, an American science writer who has reported extensively on the health effects of sugar. He listened to her story and offered to help her get the documents published in the independent news magazine Mother Jones, under the provocative title “Big Sugar’s Sweet Little Lies.” What happened when this bombshell was finally revealed, after months of research and sacrifice? Nothing, Couzens said. The story failed to be picked up by the mainstream media.

Couzens blames it on Hurricane Sandy. The media was distracted. But, she said, the story “has gotten particular attention from food policy folks here in the U.S. and also from researchers who had been studying the tobacco industry who see the parallels,” she said.

“I thought it was just wonderful and I got in touch with her right away,” said Marion Nestle, author of Food Politics, a professor at New York University and vocal food industry critic. “Those kinds of things are very, very hard to come by. One of the things that brought down the cigarette companies was finding enormous documentation of the efforts of cigarette companies to cover up the fact that they knew that cigarettes were unhealthful. It’s very, very difficult to find those kinds of things, so it was a real treasure.”

When CBC News asked the Canadian Sugar Institute for comment on the revelations that the sugar lobby group influenced science and public opinion, it didn’t answer the question. Instead we received this reply: “The Canadian Sugar Institute (CSI) is the national, non-profit association representing Canadian sugar manufacturers on nutrition and international trade affairs. I am providing you with a summary of the scientific consensus on some common misconceptions about sugars and health.”

Chiara DiAngelo, the Canadian Sugar Institute’s coordinator of nutrition communications, also offered the names of several university professors to talk to for more information about industry-sponsored research.
The Prof. Nelson Cancer as Recovery Exercise

1. Stop feeding the cancer with high glycemic foods, stop dextrose, use fructose.
2. Plug up the holes in the cell membranes with good fatty acids, not bad fatty acids; eat fresh and raw vegetables and vegetable juice, no cooked oil, suplement fatty acids.
3. Detoxify the body of toxins, get the body’s natural detoxifiers to all work well.
4. Stop adding carcinogenic toxins, smoke, radiation, fluorine, SInthetic chemicals, etc.
5. Take natural more safe chemotherapy not SInthetic.
6. Increase water, nutrition, good air... Food is the best medicine.
7. Use fasting as nature’s surgery
8. Exercise 20 mins a day, 5 day a week, work to a sweat.
9. Mirth, merriment and mental meditation; laughter is your best medicine; the best sign of mental stability is to be able to laugh at oneself.
10. CARE. Find joy and fun, release the negative, selfish, self pity, anger, greed, arrogance, and delusion. Face your false beliefs and grow in mind, body, spirit, environment, and social networks, find spiritual friends and talk out your troubles with laughter, respect, and care.
Here is the complete story on what to do

http://www.downloads.imune.net/medicalbooks/3D%20views%20on%20natural%20cancer%20therapies.pdf