

# Cancer Patients' use of Sweeteners: A 7-Year, Controlled Study

Colleen Huber\*

Naturopathic Cancer Society, Tempe, Arizona, USA

\*Corresponding author: Colleen Huber, Naturopathic Cancer Society, Tempe, Arizona, USA, Tel: (480) 839-2800; E-mail: [ch@naturopathyworks.com](mailto:ch@naturopathyworks.com)

Received date: 15 Mar 2016; Accepted date: 26 Apr 2016; Published date: 30 Apr 2016.

Citation: Huber C (2016) Cancer Patients' use of Sweeteners: A 7-Year, Controlled Study. Int J Cancer Res Mol Mech 2(2): doi <http://dx.doi.org/10.16966/2381-3318.127>

Copyright: © 2016 Huber C. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

## Abstract

**Background:** Correlation between blood glucose or glycemic load and neoplastic growth has been established for a number of types of cancer. Previous studies involved mice and/or fewer than 20 human subjects and/or were retrospective. This is a 7-year interventional study of 317 consecutive human cancer patients at one clinic, who were treated naturopathically, with cancer-disrupting nutrients and herbs, plus the abstention from sweetened foods as the dietary intervention.

**Methods:** Mortality vs survival was recorded of sweetened food eaters among cancer patients at one clinic over a seven-year period. Since 2006, this clinic has recorded data on consumption of sugar and other sweeteners in cancer patients, and has consistently recommended, but never mandated, avoidance of sweetened foods, except with extracts of the plant *Stevia rebaudiana*, which does not contain saccharides or sugar alcohol. In this controlled interventional study, the diets and outcomes are reported for all 317 patients with a diagnosis of cancer who were treated at the clinic, and who stayed at least two weeks in treatment. All results are reported in this paper.

**Results:** Achievement of remission was quite different for the following two categories: all patients: 151/317=48% and those who ate sweetened foods: 9/29=31%. The difference between these two groups was much stronger for the cohort of patients who continued treatments until either remission or death. Comparing all patients who were steadfast in the recommended treatments with the sweetened food eaters who were steadfast in all but dietary recommendations, 151/183=83% of all completely steadfast patients achieved remission, but only 9/25=36% of the steadfast sweetened food eaters achieved remission. Remission was defined as no visibly active tumor on MRI imaging of the same area that had previously active tumor growth. Of all patients who were steadfast in the treatments (including the sweetened food eaters), 32/183=17% died while still under the care of the clinic, but considering only the sweetened food eaters who otherwise consistently pursued the recommended treatments, 16/25=64% died.

**Conclusions:** In this first ever, long-term interventional study of glycemic restriction in hundreds of cancer patients, we found that sweetened foods (other than stevia-sweetened foods) were highly correlated with patient mortality across all types and all stages of cancer. Stevia is therefore recommended as the only sweetener to be used by cancer patients.

**Keywords:** Blood glucose; Cancer; Stevia

## Background

Maintaining low blood glucose in cancer patients has been found to be essential for optimal outcomes. A majority of studies tracking blood glucose and tumor growth finds a direct relationship between them. For tumor tracking, PET/CT fusion imaging is most informative, finding blood glucose uptake in tumors to be disproportionate and considerably stronger than in normal tissue, illustrating the glucose-dependent metabolism of neoplastic cells. In fact, the contrast between uptakes of glucose in a malignant tumor compared to normal tissue is so clear that the edges of a tumor may be seen on a PET image simply from the delineation of where glucose uptake is high next to where it is relatively low.

A correlation between blood sugar or glycemic load and cancer growth has been established for breast cancer [1-5], colorectal cancer [6-9], endometrial cancer [10,11], gastric cancer [12,13], liver and biliary tract cancers [14,15], ovarian cancer [16,17], pancreatic cancer [18-21], and prostate cancer [22,23].

## Methods

Mortality vs survival was recorded of sweetened food eaters among outpatients with a cancer diagnosis at one clinic. Since 2006, this clinic has recorded data on consumption of sugar and other sweeteners in

cancer patients, and has consistently recommended, but never mandated, avoidance of sweetened foods, except with extracts of the plant *Stevia rebaudiana*, which does not contain saccharides or sugar alcohol. This clinic has no inpatient facilities and no food service. All patients selected and purchased all of their own food, all of which originated from and was almost entirely consumed outside of the clinic. Data from all 317 consecutive patients with a diagnosis of cancer from outside of the clinic are reported in this interventional study, excluding only those cancer patients who chose to forgo further treatment after less than two weeks in treatment.

Natural methods such as intravenous nutrients with cancer-disrupting effect were the only ones offered, choosing among both oral and intravenous, herbal and nutritional interventions, choosing those that patients found tolerable and that we observed to work synergistically, adjusting for individual tolerances and requirements, in accordance with the naturopathic principle of "Treat the whole person".

The breadth of this evidence makes the prudent physician reluctant to condone cancer patients' use of sweeteners. Therefore, all cancer patients in the clinic were asked to avoid sweeteners, such as sugar, honey, maple syrup, corn syrup, high fructose corn syrup, alcohol, alcohol sugars and plant nectars, as well as fruit juices, because of the high sugar content and

fast availability of glucose to the circulation from these foods. Stevia was the only sweetener recommended for use due to its lack of saccharide content. Other refined carbohydrates, specifically flour products were discouraged. Whole natural foods: eggs, dairy and other animal proteins, vegetables, fruits and some whole grains, were encouraged as the entire diet, with seasonal selections and the widest available variety among them. Among the patients arriving to the clinic, there are vegans, vegetarians and omnivores and some avoid grains entirely. The patients were not encouraged to adopt one or another of these diets, because the focus on elimination of sweeteners was the single priority from which the patients were not to be distracted. This seemed to minimize the patients' forgetting and eating sweetened foods. The patients were not discouraged from eating any other kinds of foods, only sweetened foods. Patients were encouraged in gentle ways to find alternatives to sweeteners that would be satisfactory for them. Through ongoing reminders, the choice of whether to indulge in or to skip dessert would be a choice in which their doctor's reminder would be hard to ignore.

Oncologists have generally not been in agreement with this recommended diet. Chemotherapy treatment rooms are places where candy dishes are not out of sight, or are actively carted around to a captive audience of patients receiving chemotherapy infusions. The oncologists' exhortation to the patients to maintain weight regardless of nutritional quality of food, has led to consumption of some of the worst quality food by cancer patients. Other physicians less specifically credentialed to treat cancer patients are unlikely to challenge the oncologists in matters related to cancer patients' diet.

By 2006, when the collection of data for this study began, a glycemic correlation with cancer growth had already been long-established in the medical literature, thereby motivating the departure from the standard oncology dietary recommendations. Therefore, the patients at this clinic were advised to avoid sweeteners in their diet.

### **Sugar and its effect on the body**

"Sugar" in popular parlance generally refers to the sucrose that is refined from sugar cane. Sucrose is a disaccharide of glucose and fructose, composed equally of both. High-fructose corn syrup (HFCS) differs in that it has a higher proportion of fructose to glucose. Fructose bypasses the regulating step of phosphofructokinase (PFK) in the liver, delivering it faster to the circulation without that obstacle. Sucrose offers nothing that is taken in or utilized nutritionally by the body; it is devoid of fat, protein and complex carbohydrates. There are no nutrients in refined sugar: no amino acids, no lipids, no fiber, no vitamins, minerals, flavonoids or other antioxidants. Yet the "empty calories" alone do not account for all the damage inflicted on human health.

Sugar consumption has been estimated to be 40 pounds per person per year in the U.S. in 1986 [24]. By the early 2000s, Americans consumed 90 pounds per person per year; at the same time diabetes had affected 14 million Americans, and one-third of Americans were categorized as obese [25]. We know that when sugar rises quickly in the circulation the liver is strained by the task of converting excess sugar to fat, principally palmitate, which is a saturated fatty acid. Triglycerides are produced and put into the circulation, and insulin resistance results as discussed below.

Sucrase and isomaltase glycoside hydrolases break down polysaccharides in the duodenum, and disaccharides such as sucrose are further broken down in the jejunum. Ingestion of pure sucrose or sucrose-laden liquids especially, raises blood glucose quickly. Sweets accompanied by protein, fats, or fiber slow the entry of sugar to the bloodstream, with a lower amplitude wave of glycemia and insulinemia than from sweets alone in a refined carbohydrate food, such as a donut, for example. Slowly or not, the pancreas releases insulin. A rush of sudden sugar in the circulation results in a lot of insulin secreted. A repetitive pattern of this for years

causes the pancreas to become depleted and unable to maintain effective delivery of glucose to cells. Complicated by cells' insulin resistance, blood glucose rises to levels that are difficult to control, beyond the threshold for a Type II diabetes diagnosis. Studies of sugar bingeing in animals initiated this process in only a few weeks [26]. We know that chronic insulinemia also results in atherosclerosis and hypertension, and unfavorable total cholesterol to HDL ratios.

Cancer afflicts a remarkably similar population. The WHO International Agency for Research on Cancer found a higher prevalence of cancer in populations where there is obesity, diabetes and metabolic syndrome [27]. The likely mechanism is that sugar consumption provokes insulin secretion, and that insulin and its closely related hormone, insulin-like growth factor, stimulate tumor growth. IGF-1 promotes entry of sugar into a cell. However, elevated IGF-1 can result from high protein diets. IGF-1 binds to insulin receptors, and both utilize a tyrosine kinase receptor. IGF-1 acts as a growth factor in breast cancer [28], lung cancer [29] and prostate cancer [30].

On imaging, cancer cells demonstrate greater glucose uptake than normal cells, and the above-cited studies demonstrate glucose's contribution to tumor growth. This intake of glucose requires insulin as a delivery mechanism in both normal cells and cancer cells. Therefore, insulin is also implicated in tumor growth. Although receptors on normal cells are down-regulated beyond a certain level of glucose intake, cancer's appetite for glucose appears insatiable, and this glucose appears correlated with cancer's rapid and uncontrolled growth. Mutations have been found that increase insulin's delivery of glucose to cells. Craig Thompson MD, President of Memorial Sloan Kettering Cancer Center in New York, has concluded, regarding insulin and IGF's influence on cancer cells, that insulin is what drives malignant tumors to take up increasing amounts of glucose and to metabolize it, and that it is this process that allows many pre-cancerous cells to undergo the mutations that make them malignant [25].

How is glucose so important to a cancer cell's growth? We know that sugar and other refined carbohydrates are quickly metabolized fuels, providing subjective relief of hypoglycemic symptoms in patients even within seconds. Cancer cells grow more quickly than normal cells; thus the expedience of using sugar as a fuel is easily understood. However, unlike normal cells, cancer can also thrive in hypoxic environments. Cancer cells then preferentially undergo anaerobic glycolysis, known as the Warburg Effect, with pyruvate going to lactate, (as NADH is converted to NAD+) rather than going to CO<sub>2</sub> and Acetyl CoA, entering the citric acid cycle. Otto Warburg [31] discovered this difference between normal and malignant cells in 1924. Initially, it seemed that cancer cells could only undergo anaerobic glycolysis, but it is now known that aerobic glycolysis may also proceed in a cancer cell. Cancer cells convert glucose to lactic acid in such high production; it has been proposed to be cancer's chief function, as a way to eliminate toxic levels of glucose quickly to provide a usable fuel for the brain and heart [32]. Without oxygen the electron transport chain is not able to produce the high level of ATP that it does for cells undergoing aerobic metabolism. Even in the presence of adequate oxygen levels, cancer cells appear to default to fermentation rather than oxidative phosphorylation for ATP production, although the electron transport chain and oxidative phosphorylation form far higher amounts of ATP than does fermentation. For the large amount of sugar metabolized in fermentation, little ATP is formed. It is possible those only high levels of glucose enable rapid tumor growth, due to the inefficiency of anaerobic glycolysis, and that this is the most plausible reason for cancer's high dependence on glucose.

This begs the question: can we inhibit or stop cancer growth by withholding glucose? It has been observed that a ketogenic diet, a diet that severely restricts carbohydrates, has been followed by improved results in patients with various cancers. The classic ketogenic diet, limits total

carbohydrates to 20 to 40 grams per day, while the total fat consumed is four times the total of protein plus carbohydrates, by weight. Simple carbohydrates, sweeteners, fruits, grains, and starchy vegetables, are avoided in the classic ketogenic diet. A later development adds medium-chain triglycerides, such as coconut oil, and a wider selection of proteins and carbohydrates than in the classic ketogenic diet. Given the lack of carbohydrates in this diet, oxidation of fats to produce energy is then the primary metabolic mechanism of the ketogenic diet. This oxidation of fats to fatty acids and ketone bodies in the liver provides usable fuel for the brain in a low glycemic environment. Animal studies of glioma in a ketogenic environment showed cancer cells behaving more like normal cells [33]. Macroscopically, a ketogenic diet was found either to decrease size of tumors or to retard growth of tumors in glioblastoma [34], prostate cancer [35], gastric cancer [36], and lung cancer [37]. Advanced metastatic disease in a variety of cancers with a ketogenic diet demonstrated improved measures of quality of life [38].

Perhaps it is premature to conclude that sugar causes cancer, or even that eliminating sugar or dropping blood glucose as low as tolerable would serve to eliminate cancer. However, a growing body of evidence suggests that dietary sugars and rising blood glucose are high risk for cancer patients, and that oncologists and other health care practitioners would do well to be alert to and to caution against high glycemic foods in the diet of their cancer patients.

## Results

Of 317 patients at one naturopathic clinic, 29 patients told us that they had not complied with our one dietary recommendation; that is, at some time during the months of their treatment at the clinic, they had consumed sweetened foods at least one time. Clinic doctors and staff are careful to avoid any appearance of scolding or disapproval when patients acknowledge that they have eaten sweetened foods. When a patient informs us of having eaten foods with sweeteners, we adopt a co-responsible (or perhaps co-dependent) approach, in that we need to be more effective at helping the patient identify foods and eating habits that substitute for sweetened foods in a way that is satisfactory and sustainable long-term. In physician-patient consults we look for more and more individually suitable and feasible alternatives to sweetened foods. For example, one person may be more drawn to an all-carbohydrate breakfast with little protein or fat, while another is more drawn to ice cream. Another patient may have a coffee habit in which coffee seems incomplete or unsatisfactory without sweeteners. For others, it is chocolate that satisfies. This approach is not

always successful, but it seems to encourage honest exploration of current and past food choices and planned future food choices. Because we are not always successful in convincing all patients to adopt our recommended diet, those who disregarded our dietary recommendation are noted below, unless they were able to eliminate sweeteners at the beginning of treatment and were able to maintain that diet through our most recent contact with them prior to the recording of the data below.

As of July 1, 2013 we stopped collecting new data for this paper. 20 patients up until that time had died while still exclusively in our care, although they followed all of our protocols as well as our dietary recommendation. These included all consecutive patients without restriction as to type of cancer and stage of cancer, with a preponderance of patients in the more advanced stages (due to the unfortunate delay of naturopathic cancer care to long past an optimal time). 12 more patients had died while still in our care, having returned to eating sweetened foods. 16 of our cancer patients had come out of remission. 5 of those then went back in remission. 4 of those 16 had discontinued avoiding sweetened foods.

Tables 1-3 show comparable information for three groups of patients: Table 1 summarizes all 317 consecutive cancer patients who presented to our clinic for cancer treatment, and who stayed in our treatments for at least two weeks. Table 2 shows the same information for those who chose to eat sweetened foods. Table 3 shows the same information for those who chose to avoid sweetened foods. The remission rate is different for all patients: 151/317=48% and those who ate sweetened foods: 9/29=31% and those who avoided sweetened foods: 142/288=49%. However, the difference in these three groups is even more pronounced if we consider those patients who stayed with our treatments until either remission or death, as in tables 4-6.

Comparing all patients who were steadfast in the treatments (Table 4) with the sweetened food eaters, who were steadfast in all but dietary recommendations (Table 5), we see that 151/183=83% went into remission, but only 9/25=36% of the sweetened food eaters went into remission. 90% of the steadfast patients who avoided sweeteners went into remission.

Of all patients who were steadfast in the treatments (including our sweetened food eaters), 32/183=17% died, but considering only the sweetened food eaters who were otherwise steadfast in the treatments, 16/25=64% died. Of the steadfast patients who avoided sweeteners, 16/158=10% died.

| Outcome  | Number of patients | Number in each group also receiving chemotherapy | Number in each group also receiving surgery |
|--|--------------------|--|---|
| Remission or assumed remission   | 151                | 7  | 47  |
| Died while still only in our care, following all of our protocols and diet                   | 20                 | 0  | 1   |
| Iatrogenic death in hospitals or by MDs  | 20                 | 14   | 7   |
| Of those who left before finishing treatment, number who died after leaving (except for DDD) | 46                 | 1  | 10  |
| Death after dietary dispute  | 12                 | 1  | 2   |
| Still being treated, not yet in remission  | 18                 | 3  | 10  |
| No current information but never known to be in remission                                    | 33                 | 3  | 9   |
| Waiting to know status, or conflicting information   | 17                 | 0  | 2   |
| <b>Total</b>   | <b>317</b>         | <b>29</b>  | <b>88</b>                                   |

**Table 1:** Outcomes of 317 cancer patients after naturopathic treatment  
'DDD: death after dietary dispute.

| Outcomes of sweetened food eaters   | Number of patients | Number in each group also receiving chemotherapy | Number in each group also receiving surgery |
|---|--------------------|--|---|
| Remission or assumed remission  | 9                  | 0  | 5   |
| Died while still only in our care, following all of our protocols   | 0                  | 0  | 0   |
| Iatrogenic death in hospitals or by MDs   | 1                  | 0  | 0   |
| Of those who left before finishing treatment, number who died after leaving (except for DDD) <sup>*</sup> | 0                  | 0  | 0   |
| Death after dietary dispute   | 12                 | 1  | 2   |
| Still being treated, not yet in remission   | 0                  | 0  | 0   |
| No current information but never known to be in remission   | 7                  | 2  | 0   |
| Waiting to know status, or conflicting information  | 0                  | 0  | 0   |
| <b>Total</b>  | <b>29</b>          | <b>3</b>   | <b>7</b>                                    |

**Table 2:** Outcomes of 29 cancer patients after naturopathic treatment, and with whom there was a dietary dispute regarding sweetener consumption  
<sup>\*</sup>DDD: death after dietary dispute.

| Outcomes of sweetener avoiders  | Number of patients | Number in each group also receiving chemotherapy | Number in each group also receiving surgery |
|---|--------------------|--|---|
| Remission or assumed remission  | 142                | 7  | 42  |
| Died while still only in our care, following all of our protocols and diet                                | 20                 | 0  | 1   |
| Iatrogenic death in hospitals or by MDs   | 19                 | 14   | 7   |
| Of those who left before finishing treatment, number who died after leaving (except for DDD) <sup>*</sup> | 46                 | 1  | 10  |
| Death after dietary dispute   | 0                  | 0  | 0   |
| Still being treated, not yet in remission   | 18                 | 3  | 10  |
| No current information but never known to be in remission   | 26                 | 1  | 9   |
| Waiting to know status, or conflicting information  | 17                 | 0  | 2   |
| <b>Total</b>  | <b>288</b>         | <b>26</b>  | <b>81</b>                                   |

**Table 3:** Outcomes of 288 cancer patients treated naturopathically, all of whom were able to avoid consumption of sweeteners  
<sup>\*</sup>DDD: death after dietary dispute.

| Stage                                 | Total patients treated until remission or death | Remission  | Died                                  | Remission/Total=Success rate |
|---------------------------------------|---|------------|---------------------------------------|------------------------------|
| I                                     | 65  | 64         | 1                                     | 98%                          |
| II                                    | 30  | 29         | 1                                     | 97%                          |
| III                                   | 17  | 14         | 3                                     | 82%                          |
| Early IV                              | 49  | 37         | 12                                    | 76%                          |
| Late IV                               | 22  | 7          | 15                                    | 32%                          |
| <b>Total</b>                          | <b>183</b>                                      | <b>151</b> | <b>32<sup>a</sup></b>                 | <b>83%</b>                   |
| <b>Stage I through early Stage IV</b> | <b>161</b>                                      | <b>144</b> | <b>17 (including DDD)<sup>*</sup></b> | <b>89%</b>                   |

**Table 4:** Steadfast patients, by stage of cancer-all patients  
<sup>a</sup>This number includes those who did not follow our dietary recommendations.  
<sup>\*</sup>DDD: death after dietary dispute.

| Stage                                 | Total patients treated until remission or death | Remission | Died      | Remission/Total=Success rate |
|---------------------------------------|---|-----------|-----------|------------------------------|
| I                                     | 5   | 4         | 1         | 80%                          |
| II                                    | 4   | 3         | 1         | 75%                          |
| III                                   | 3   | 0         | 3         | 0%                           |
| Early IV                              | 10  | 2         | 8         | 20%                          |
| Late IV                               | 3   | 0         | 3         | 0%                           |
| <b>Total</b>                          | <b>25</b>                                       | <b>9</b>  | <b>16</b> | <b>36%</b>                   |
| <b>Stage I through early Stage IV</b> | <b>22</b>                                       | <b>9</b>  | <b>13</b> | <b>41%</b>                   |

**Table 5:** Steadfast patients, by stage of cancer- sweet eaters



| Stage                                 | Total patients treated until remission or death | Remission  | Died      | Remission/Total=Success rate |
|---------------------------------------|---|------------|-----------|------------------------------|
| I                                     | 60  | 60         | 0         | 100%                         |
| II                                    | 26  | 26         | 0         | 100%                         |
| III                                   | 14  | 14         | 0         | 100%                         |
| Early IV                              | 39  | 35         | 4         | 90%                          |
| Late IV                               | 19  | 7          | 12        | 37%                          |
| <b>Total</b>                          | <b>158</b>                                      | <b>142</b> | <b>16</b> | <b>90%</b>                   |
| <b>Stage I through early Stage IV</b> | <b>139</b>                                      | <b>135</b> | <b>4</b>  | <b>97%</b>                   |

**Table 6:** Steadfast patients, by stage of cancer-sweetener avoiders

## Conclusions

In this first ever, long-term, interventional study of glycemic restriction in hundreds of cancer patients, we found that consuming sweetened foods (other than stevia-sweetened foods) made a significant difference in patient outcome across both all stages and all types of cancer among patients presenting to our clinic. Mortality was significantly increased by consumption of sweetened foods. We therefore recommend that the diet of cancer patients not contain sweeteners other than stevia.

## References

- Tavani A, Giordano L, Gallus S, Talamini R, Franceschi S, et al. (2006) Consumption of sweet foods and breast cancer risk in Italy. *Ann Oncol* 17: 341-345.
- Larsson SC, Bergkvist L, Wolk A (2009) Glycemic load, glycemic index and breast cancer risk in a prospective cohort of Swedish women. *Int J Cancer* 125: 153-157.
- Wu AH, Yu MC, Tseng CC, Stanczyk FZ, Pike MC (2009) Dietary patterns and breast cancer risk in Asian American women. *Am J Clin Nutr* 89: 1145-1154.
- Bradshaw PT, Sagiv SK, Kabat GC, Satia JA, Britton JA, et al. (2009) Consumption of sweet foods and breast cancer risk: a case-control study of women on Long Island, New York. *Cancer Causes Control* 20: 1509-1515.
- Jiang Y, Pan Y, Rhea PR, Tan L, Gagea M, et al. (2016) A Sucrose-Enriched Diet Promotes Tumorigenesis in Mammary Gland in Part through the 12-Lipoxygenase Pathway. *Cancer Res* 76: 24-29.
- Wang B, Bobe G, LaPres JJ, Bourquin LD (2009) High sucrose diets promote intestinal epithelial cell proliferation and tumorigenesis in APC(Min) mice by increasing insulin and IGF-I levels. *Nutr Cancer* 61: 81-93.
- Wang B, Bobe G, LaPres JJ, Bourquin LD (2009) Dietary carbohydrate source alters gene expression profile of intestinal epithelium in mice. *Nutr Cancer* 61: 146-155.
- Nayak SP, Sasi MP, Sreejayan MP, Mandal S (2009) A case-control study of roles of diet in colorectal carcinoma in a South Indian Population. *Asian Pac J Cancer Prev* 10: 565-568.
- Williams CD, Satia JA, Adair LS, Stevens J, Galanko J, et al. (2009) Dietary patterns, food groups, and rectal cancer risk in Whites and African-Americans. *Cancer Epidemiol Biomarkers Prev* 18: 1552-1561.
- King MG, Chandran U, Olson SH, Demissie K, Lu SE, et al. (2013) Consumption of sugary foods and drinks and risk of endometrial cancer. *Cancer Causes Control* 24: 1427-1436.
- Mulholland HG, Murray LJ, Cardwell CR, Cantwell MM (2008) Dietary glycaemic index, glycaemic load and endometrial and ovarian cancer risk: a systematic review and meta-analysis. *Br J Cancer* 99: 434-441.
- Ikeda F, Doi Y, Yonemoto K, Ninomiya T, Kubo M, et al. (2009) Hyperglycemia increases risk of gastric cancer posed by *Helicobacter pylori* infection: a population-based cohort study. *Gastroenterology* 136: 1234-1241.
- Bertuccio P, Praud D, Chatenoud L, Lucenteforte E, Bosetti C, et al. (2009) Dietary glycemic load and gastric cancer risk in Italy. *Br J Cancer* 100: 558-561.
- Fedirko V, Lukanova A, Bamia C, Trichopolou A, Trepo E, et al. (2013) Glycemic index, glycemic load, dietary carbohydrate, and dietary fiber intake and risk of liver and biliary tract cancers in Western Europeans. *Ann Oncol* 24: 543-553.
- Moerman CJ, Bueno de Mesquita HB, Smeets FW, Runia S (1995) Consumption of foods and micronutrients and the risk of cancer of the biliary tract. *Prev Med* 24: 591-602.
- Augustin LS, Polesel J, Bosetti C, Kendall CW, La Vecchia C, et al. (2003) Dietary glycemic index, glycemic load and ovarian cancer risk: a case-control study in Italy. *Ann Oncol* 14: 78-84.
- Silvera SA, Jain M, Howe GR, Miller AB, Rohan TE (2007) Glycaemic index, glycaemic load and ovarian cancer risk: a prospective cohort study. *Public Health Nutr* 10: 1076-1081.
- Chan JM, Wang F, Holly EA (2009) Sweets, sweetened beverages, and risk of pancreatic cancer in a large population-based case-control study. *Cancer Causes Control* 20: 835-846.
- Rossi M, Lipworth L, Polesel J, Negri E, Bosetti C, et al. (2010) Dietary glycemic index and glycemic load and risk of pancreatic cancer: a case-control study. *Ann Epidemiol* 20: 460-465.
- Mueller NT, Odegaard A, Anderson K, Yuan JM, Gross M, et al. (2010) Soft drink and juice consumption and risk of pancreatic cancer: the Singapore Chinese Health Study. *Cancer Epidemiol Biomarkers Prev* 19: 447-455.
- Larsson SC, Bergkvist L, Wolk A (2006) Consumption of sugar and sugar-sweetened foods and the risk of pancreatic cancer in a prospective study. *Am J Clin Nutr* 84: 1171-1176.
- Freedland SJ, Aronson WJ (2009) Dietary intervention strategies to modulate prostate cancer risk and prognosis. *Curr Opin Urol* 19: 263-267.
- Drake I, Sonestedt E, Gullberg B, Ahlgren G, Bjartell A, et al. (2012) Dietary intakes of carbohydrates in relation to prostate cancer risk: a prospective study in the Malmö Diet and Cancer cohort. *Am J Clin Nutr* 96: 1409-1418.
- Glinsmann WH, Irausquin H, Park YK (1986) Evaluation of health aspects of sugars contained in carbohydrate sweeteners. Report of Sugars Task Force, 1986. *J Nutr* 116: S1-S216.
- Taubes G (2011) Is sugar toxic? *New York Times*, New York, USA.
- Pagliassotti MJ, Prach PA, Koppenhafer TA, Pan DA (1996) Changes in insulin action, triglycerides, and lipid composition during sucrose feeding in rats. *Am J Physiol* 271: R1319-R1326.
- Jaggers JR, Sui X, Hooker SP, LaMonte MJ, Matthews CE, et al. (2009) Metabolic syndrome and risk of cancer mortality in men. *Eur J Cancer* 45: 1831-1838.

28. Foekens JA, Portengen H, Janssen M, Klijn JG (1989) Insulin-like growth factor-1 receptors and insulin-like growth factor-1-like activity in human primary breast cancer. *Cancer* 63: 2139-2147.
29. Kaiser U, Schardt C, Brandscheidt D, Wollmer E, Havemann K (1993) Expression of insulin-like growth factor receptors I and II in normal human lung and in lung cancer. *J Cancer Res Clin Oncol* 119: 665-668.
30. Iwamura M, Sluss PM, Casamento JB, Cockett AT (1993) Insulin-like growth factor I: action and receptor characterization in human prostate cancer cell lines. *Prostate* 22: 243-252.
31. Warburg O, Posener K, Negelein E (1924) About the metabolism of the tumors. *Biochemical Journal* 152: 319-344.
32. Seneff A (2013) *Cancer to the rescue? The Weston A. Price Foundation*, Washington, DC 20016, USA
33. Scheck AC, Abdelwahab MG, Fenton KE, Stafford P (2012) The ketogenic diet for the treatment of glioma: insights from genetic profiling. *Epilepsy Res* 100: 327-337.
34. Tisdale MJ, Brennan RA (1988) A comparison of long-chain triglycerides and medium-chain triglycerides on weight loss and tumour size in a cachexia model. *Br J Cancer* 58: 580-583.
35. Freedland SJ, Mavropoulos J, Wang A, Darshan M, Demark-Wahnefried W, et al. (2008) Carbohydrate restriction, prostate cancer growth, and the insulin-like growth factor axis. *Prostate* 68: 11-19.
36. Otto C, Kaemmerer U, Illert B, Muehling B, Pfetzer N, et al. (2008) Growth of human gastric cancer cells in nude mice is delayed by a ketogenic diet supplemented with omega-3 fatty acids and medium-chain triglycerides. *BMC Cancer* 8: 122.
37. Allen BG, Bhatia SK, Buatti JM, Brandt KE, Lindholm KE (2013) Ketogenic diets enhance oxidative stress and radio-chemo-therapy responses in lung cancer xenografts. *Clin Cancer Res* 19: 3905-3913.
38. Schmidt M, Pfetzer N, Schwab M, Strauss I, Kämmerer U (2011) Effects of a ketogenic diet on the quality of life in 16 patients with advanced cancer: A pilot trial. *Nutr Metab (Lond)* 8: 54.