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Steve Hickey and Hilary Roberts
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Sugar

“Pure, white and deadly” John Yudkin

In well-designed scientific studies, a “sugar pill” is a placebo or dummy, which is given to control subjects in a double-blind trial. Early scientists presumably assumed that sugar was safe and would have minimal effects on the body, making it ideal for comparison with the actions of a drug or treatment.

However, sugar has profound effects: as far as the body is concerned, it contains pure calories. Much of the energy intake of a modern diet is in the form of sugars and starches, which supply nutrient-free energy. High sugar consumption leads to weight gain, particularly when combined with low exercise levels.

Diets high in sugar and carbohydrates lead to poor health.⁷²⁵ A recent self-reported comparison of health and nutrition in 37,053 British people indicates the relationship of sugar to health.⁷²⁶ People who consumed large amounts of sugar-based snacks were about five times more likely to report poor health. Other dietary intakes associated with sugar intake, such as wheat, dairy, refined foods, tea and coffee, were also linked with reduced health. Additional foods related to feelings of illness were red meat and salt. Foods associated with good health had a relatively high ratio of effective antioxidants to carbohydrate; these include fresh fruit, vegetables, salad, seeds, nuts and oily fish.

Overeating is a major risk factor for cancer.⁷²⁵ Obese people are more likely to develop tumours, whereas people who eat less are more resistant. The problem of obesity is rising throughout the western world. The average American is now overweight,⁷²⁷ and two percent or more are severely obese.⁷²⁸ Lack of exercise and poor diet are leading causes of premature death.⁷²⁹ 14% of cancer deaths in men and 20% of those in women are related to obesity.⁷³⁰ This includes deaths from cancer of the breast, cervix, colon, oesophagus, gallbladder, kidney, liver, ovary, pancreas, prostate, stomach (male), and uterus.

The appeal of sugar is not limited to humans; it has a broad biological basis. Sugars provide easily assimilated energy for many organisms. As a result, the exchange of sugar is involved in many relationships between organisms.⁷³¹ For example, the sweet nectar of flowering plants is used to attract insects for pollination. Some plants,

such as a species of eucalyptus, excrete large amounts of sugars to attract ants, which provide protection against herbivores. Ants themselves farm caterpillars that excrete sugar. Keeping the caterpillars requires more energy than they excrete in the form of sugars, which indicates the importance of the sugar supply. In some cases, such as with the caterpillar *Maculinea arion*, the farmed animal will even eat ants.⁷³²

The relationship between cancer and its host also depends on the provision of sugars and other essential nutrients, to fuel abnormal growth. This chapter looks at the relationship between sugar and cancer.

We will consider prevention of cancer separately from treatment. This is because a diet to help people avoid cancer may differ from a diet that is optimal for a patient with advanced disease. As we consider the role of glucose, it will become apparent that it is intimately related to the actions of vitamin C in the cell. This interaction provides a means for preventing tumour growth and selectively killing cancer cells.

Restricting calories

Those who wish to avoid cancer or to live longer, if they already have the disease, are often advised to cut down on sugar.^{725,733,749}

Glucose deprivation generates oxidative stress within tumours, because of a possible defect in the metabolism of cancer cells.⁷³⁴ One anticancer diet is called Calorie Restriction with Optimal Nutrition (CRON). This consists of eating 70–80% of the calories required to maintain body weight, while getting an optimal level of vitamins and other nutrients. The core restriction is the total energy consumed.

Research has shown that a reduced calorie diet can extend average lifespan in many species, while also reducing the chronic disease load.⁷³⁵ In mice, energy restriction reduces the number of spontaneous tumours.⁷³⁶ Such deprivation inhibits the growth of mammary cancer, suppresses the growth of implanted tumours and increases survival rates.^{737,738} In Sweden, researchers found that women who had been treated for anorexia nervosa before they were 40 had a 53% lower incidence of breast cancer.⁷³⁹ Although this result is consistent with a large effect of calorie restriction, it is worth noting that these women's intake of specific vitamins and nutrients was also reduced.

Glucose metabolism

Sugar is the ultimate junk food; it is high in energy but contains no other nutrient value. Sugars and refined flour products make up a large portion of the western diet. Indeed, high carbohydrate diets seem to dominate official recommendations for healthy eating.

One approach to estimating the effect of these foods on the body is the glycaemic index (GI). The glycaemic index is a measure of the metabolic effects of dietary carbohydrates.^{740,741} The GI measures how a particular food increases blood sugar levels in the body. The higher the GI value, the greater are the body's glucose and insulin responses to the food in question. Since the body's response varies with the amount of food, this measure can be misleading. For this reason, a modification called the glycaemic load (GL) has been developed,⁷⁴⁰ to take into account the amount of food consumed.

For many people, this is a rather unworkable form of dietary advice. To illustrate the problem, we refer the reader to the publication of an international table of the glycaemic index and load of foods. In 2002, the table contained 750 entries.⁷⁴² Such a table is huge: even a simple meal could require a substantial amount of arithmetic, not to mention guesswork as to the effects of combining different foods. In the longer term, following such a diet may be impractical for the majority of people. However, the approach may be productive in helping to construct general dietary advice guidelines, or for studying the health impacts of manufactured food.

Several projects have investigated the association between glycaemic load and cancer. Controlled studies have shown that a high glycaemic load carries an increased risk of cancer in the higher gut,^{743,744} uterus,⁷⁴⁵ ovary,⁷⁴⁶ and colon.⁷⁴⁷ Studies of glycaemic index and glycaemic load provide valuable research tools, as high levels of glucose in the blood promote cancer.^{748,749,820} Indicators of diabetes, such as increased blood glucose, are associated with higher risk of colon cancer.⁷⁵⁰ Diabetes is associated with increased risk of cancer of the bowel,^{751,752,753,754,755,756} uterus,⁷⁵⁷ and pancreas.^{754,758} Furthermore, diabetes may be an independent predictor of mortality from cancer of the colon, pancreas, liver, bladder and female breast.⁷⁵⁴

Healthy people can benefit from learning the concept of glycaemic load and its relationship to different types of food. Prospective studies have shown an increased risk of cancer with high glycaemic load.^{759,760,761} Other studies suggest this risk is specific to sedentary, overweight subjects.^{762,763,764,765} As is often the case with such studies, others found no increase in risk, providing contradictory evidence.^{766,767,768,769}

Despite these conflicting findings, people wanting to maintain

good health would be wise to limit their intake of foods with a high glycaemic index. To a first approximation, the result of reducing the glycaemic load is similar to a low carbohydrate diet.

Treatments

There are many nutrient-based “treatments” for cancer. Several assume that making sure that the person is well nourished will allow them to fight the cancer. Although this may sound like common sense, there is no logical reason for thinking that such an approach will produce a favourable outcome. If true, the nutrients would need to benefit the person’s normal cells more than they would the cancer cells. Conversely, if the cancer’s growth was previously limited by the nutrient supply, then ensuring good nutrition could be catastrophic. For a nutrient-based approach to increase survival, differences between the host and cancer cells must be exploited.

We have mentioned that microevolution favours anaerobic cancer cells. As soon as anaerobic metabolism is mentioned, a biologist will think of sugar, the preferred energy source for such cells. Glucose is the main food supply for many cells in the body, including neurons in the brain. Most cells gain some of their energy from glucose, using glycolysis, which is the breakdown of glucose in a series of biochemical steps. Glycolysis is the early stage of glucose metabolism and is not dependent on oxygen. Cells typically have the ability to use glucose as a source of energy. However, the majority of healthy cells are more able than cancer cells to gain energy by breaking down fats and protein. Since cancer cells use glucose metabolism as their preferred method of generating energy, changing the balance of food consumed could be beneficial. A reduced calorie diet, based on fibre, proteins and fats, rather than glucose and starch, might starve the cancer. Healthy cells, on the other hand, would be better adapted to accommodate the modified fuel source.

A possible basis for an anticancer diet might be one that reduces carbohydrate intake. Indeed, at first glance, the low-carb diets popularised by the late Robert Atkins might have the unexpected benefit of generally lowering cancer incidence or, in some rare cases, providing a cure. Such a suggestion may aggravate many dieticians, nutritionists and vegetarians. Objections to the Atkins diet in this context are that it does not limit total energy intake and may not provide sufficient vitamins and

minerals. While not promoting the Atkins diet, we can risk the wrath of dietary experts and investigate the low-carb idea, to see if it might be productive.

Glucose deprivation could slow the growth of cancer cells, or even kill them. Glucose reduction leads to oxidative stress, which is preferentially toxic to cancer cells.^{770,771} Such oxidative stress is caused by the generation of hydrogen peroxide and superoxide in the cell body.^{772,773} The increased level of oxidants implies that glucose deprivation could be synergistic with the actions of substances such as vitamin C and others, which increase hydrogen peroxide in cancer cells.

Increased rates of cancer, which are associated with high blood glucose levels, are consistent with the microevolutionary model. Higher levels of glucose provide energy for anaerobic cells, facilitating cancer growth. Since glucose appears to be crucial for tumour cell microevolution, selection pressure should favour the development of mechanisms to increase its absorption. Cancer cells with the ability to pump glucose into their bodies are likely to have a selective advantage, leading them to dominate the colony. Supporting evidence includes the finding that glucose transporters are increased in cancer cells.^{774,775,776,777,778} Glucose molecules enter cancer cells preferentially, hence modified glucose molecules are used in taking medical images of cancers.^{779,780,781} Interestingly, patients may be asked to fast prior to such investigations, lowering their blood sugar, in order to increase the uptake of these glucose markers.⁷⁸² Cancer cells appear to have a scavenging mechanism to meet their glucose needs, which is consistent with the idea that a lowcarb diet would slow cancer growth by reducing glucose availability. The microevolutionary theory predicts that if cancer cells were able to stimulate high levels of glucose in the blood, they would have a selective advantage. Supporting this idea, glucose intolerance has been observed in cancer patients, as long ago as 1919. Such intolerance means the body becomes less efficient at converting carbohydrates into energy, resulting in raised blood glucose levels.

The relationship between cancer and glucose intolerance is unclear. One possibility is that people with pre-existing glucose intolerance may be more subject to cancer. High blood glucose levels are common in cancer patients, and a reduced efficiency of energy conversion contributes to weight loss (*cachexia*).^{783,784,785} Even a small tumour may disturb the body's glucose balance.⁷⁸⁶ Although the results are not entirely

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consistent,^{787,788,789,790} they imply that glucose metabolism is an important factor in understanding cancer.⁷⁹¹

These findings suggest that cancer cells are likely to use glucose at a high rate. Research confirms that increased glucose utilisation is a general

feature of cancer cells,^{792,793,794,795} as has been known since 1930.^{212,210} The ability to sustain high rates of glycolysis, even when oxygen is plentiful, is characteristic of cancer cells.^{796,797,798} Factors that alter cancer growth rates can act by modifying glucose metabolism and transport, indicating its importance to tumour progression.⁷⁹⁹ Glucose has even been suggested as a target for selective drug action.^{800,801} Drugs that inhibit the uptake of glucose or its use in cancer cells have been considered as potential treatments.

The microevolutionary model predicts that gene expression and biochemistry in cancer cells might be modified, to facilitate the use of glucose as the main energy source. This gene expression is a direct result of cell selection, acting in an environment where oxygen is scarce. Such adaptations support a greater role for enzymes involved in anaerobic metabolism. Gene expression and biochemical studies of cancer cells confirm this predication.^{802,803,804,805,806,807,808,809,810} There is little doubt that changes in glucose biochemistry are found in cancer cells. If glucose promotes cancer growth, then we might predict that cancer patients with low glucose levels would survive longer than would those with high levels. A recent paper backs up this suggestion,⁸¹¹ and further results, from patients with cancer of the pancreas, support this research.⁸¹²

Glucose and vitamin C

For many years, biophysicist John Ely has investigated the interactions between glucose and vitamin C. When blood sugar is low, vitamin C enters body cells more easily. The transporters that pump glucose into the cell also transport oxidised ascorbate. When a cancer cell boosts its glucose transport mechanisms, it increases its capacity to absorb vitamin C. Cancer patients are reported to have an enhanced tolerance for large oral supplements of vitamin C.⁸¹³ This may result from the cancer cells' capacity to absorb large amounts of the vitamin from the bloodstream.

In 1972, Ely warned Linus Pauling that it was essential to make sure patients had low sugar intakes, if they were benefit fully from megadose supplementation. This is because decreasing the availability of

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glucose helps increase the absorption of vitamin C into cells. Ely described this competition in his glucose-ascorbate antagonism theory. Later, he showed that the cross-linking of proteins by glucose, which occurs in diabetes, aging and cataracts, could be inhibited by vitamin C.^{814,815,816} He also related this cross-linking mechanism to increased risk of heart disease and cancer.⁸¹⁷ Ely proposed that sustained high plasma levels of vitamin C would greatly reduce cross-linking of proteins in the blood of diabetics. According to this idea, two grams of vitamin C every

two hours would maintain high levels of vitamin C in the blood,^a and prevent many of the complications of diabetes.

Ely made his point dramatically with the statement “aging is scurvy.” Aging is a process characterised by oxidation and crosslinking.⁸¹⁸ This claim implies that a person who does not have a constantly high level of vitamin C will age rapidly. Ely suggests that high blood glucose levels have other health effects, such as birth defects.^{819,820,821} He put forward an essential nutrient theory, which has similarities to Linus Pauling’s ideas of orthomolecular medicine. Ely proposed a role for the interaction of glucose and vitamin C in assisting cellular immunity. Glycolysis is the generation of energy from glucose, in the absence of oxygen. A related biochemical pathway, the pentose pathway or hexose monophosphate shunt, is used to power the synthesis of DNA and provide antioxidant electrons.⁷²⁵ The speed of this second pathway depends on the concentration of vitamin C in the cell.⁸²² Ely has pointed out that cell-mediated immunity requires this additional pathway. If vitamin C levels are low, as is common in the elderly, the shunt rate and cell-mediated immunity fall. Then, infectious diseases, cancer and mortality from all causes rise. Further evidence includes the finding that the behaviour of white blood cells is greatly inhibited in diabetics.^{823,824,825,826} People with compromised cell-mediated immunity, such as those suppressed following organ transplants, are at greater risk of developing some forms of cancer.

Raw fruit and vegetables

The effect of calorie and sugar intake on cancer is profound. Arthur Robinson, former President and Research Director of the Linus Pauling Institute, became interested in Edie Mae Hunsberger’s claim that she had cured herself of cancer by eating a diet of fresh fruit and vegetables.⁸²⁷ Robinson tested the claim on experimental mice suffering

^a We calculate the blood plasma level in a normal healthy young adult with this level of intake to be about 220 µM/L.

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from cancer, which he fed a diet of raw fruit and vegetables.⁸²⁸ The result was unexpected - a massive reduction in the number of lesions. Pauling described the result as a “bombshell”. It appeared that simply reducing calorie intake, by feeding raw fruit and vegetables, had extended the life of the cancer-ridden mice.

Mice given a diet of raw fruit and vegetables for two months showed a marked reduction in tumour growth. This reduction was not as great as that observed with massive doses of vitamin C, but was impressive nonetheless. The number of minor lesions was reduced from 54 in 60 animals (90%), to 11 in 40 animals (28%). Severe lesions were

also reduced, from 13 in 60 animals (22%), to just one in 40 animals (2.5%).^b The effect was largely independent of the type of vegetables provided. However, the benefit was lowered by the addition of seeds and nuts, which contain relatively large amounts of available energy. Adding apples and pears, which contain larger amounts of available sugar, also reduced the effect. Similarly, addition of protein inhibited the effects of raw fruit and vegetables. A combination of fruits and vegetables, taken with a human equivalent intake of 168 grams of vitamin C,^c gave results similar to those of vitamin C alone (four minor lesions and no severe lesions in 45 animals). The numbers of lesions in the mice are shown in the following table:

Robinson's mice after two months

Treatment Number of Mice

Minor

Lesions

Major

Lesions

Controls 60 54(90%) 13(22%)

Vitamin C 192g 45 7(16%) 0

Raw vegetables 40 11(18%) 1(2.5%)

**Raw vegetables +
165g vitamin C**

34 4(12%) 0

^b Percentages are average (arithmetic mean) lesions per mouse.

^c For simplicity, we are using Robinson's suggested conversion to human equivalent intake, ie 1g/Kg approximates to a one gram human dose. No-one is suggesting that this conversion is accurate, but it provides the reader with a basic heuristic.

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When the study was repeated, consistent results were obtained.

Robinson then carried out further experiments.⁸²⁸ Mice were irradiated to induce cancer, before being fed the experimental diet for four months.

Waiting four months could give a more accurate evaluation of the slower growth of some lesions. Four months is not a long period in human

terms, but laboratory mice have a total life expectancy of about three

years. The follow-up experiment confirmed the tumour growth reduction and anti-cancer effects of vitamin C and raw vegetables. In 56 control

mice, there were 218 minor lesions (3.9 per mouse) and 95 severe lesions (1.7 per mouse). 96 grams per day of vitamin C greatly reduced these

numbers, to 56 minor lesions (1.2 per mouse) and 13 severe lesions (0.29 per mouse) in 45 mice. Notably, a combined treatment of raw fruit and

vegetables, plus 25 grams of vitamin C, prevented the formation of

severe lesions almost completely, resulting in 24 minor (0.7 per mouse)

and 1 severe lesion (0.03 per mouse), in 34 mice. The following table

presents the results obtained:

Robinson's mice, after four months

Treatment Number of Mice

**Minor
Lesions**

**Major
Lesions**

Controls 56 218 95

Vitamin C 96g 45 56 13

Raw vegetables

+ 25g vit C

34 24 1

These effects can be explained if we assume that the availability of glucose inhibits the action of vitamin C. The results are consistent with anaerobic cancer cells requiring nutrients for growth.⁸²⁸ An appropriate analogy might be the different responses of infants and adults to reduction in available food. An adult would lose weight but could survive for long periods on limited food intake. However, an infant requires nutrients for growth. Even moderate food deprivation could reduce the growth rate in an infant. Prolonged deprivation might result in a permanent growth restriction, leading to an adult of short stature. Severe food deprivation would kill a child more quickly than an adult. In a

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similar way, if a malignant cancer is deprived of nutrients, particularly glucose, its growth rate may be reduced. Conversely, good nutrition may benefit a cancer more than it would the host.

The mice that died

The Linus Pauling Institute was set up by Arthur Robinson and Pauling himself, to study the effects of vitamin C and other nutrients. Pauling's popularisation of vitamin C and its effects generated great publicity. However, the medical establishment attacked Pauling, promoting the impression he was a quack.

Robinson carried out important experiments based on Pauling's ideas, during which he fed a meganutrient mix to mice with cancer. His results indicated that when he fed the mice a full supply of vitamins and minerals, their cancers got worse. This result was disturbing and not what might have been expected at the time. If true, the many popular books telling people what to eat when they have cancer might be doing more

harm than good. A simplistic interpretation of Robinson's result is that much current nutritional advice for cancer sufferers is wrong. Then came a further shock. To Robinson's surprise, he found that his mice died sooner if they were treated with nutritional doses of vitamin C. Vitamin C greatly affected the growth of the cancer in mice. As we have seen, later results showed that high intakes, equivalent to 50 grams or more in a human, inhibited cancer growth. However, Robinson's initial results indicated a human equivalent intake of 1.5 - 3 grams per day decreased survival times. The result was a heated and acrimonious dispute between Robinson and Pauling.^{827,830} Indeed, it ended their 16-year collaboration.

In Robinson's words,

"He [Pauling] was not willing to accept the experimentally proved fact that vitamin C in ordinary doses accelerated the growth rate of squamous cell carcinoma in these mice."

This was a particularly damaging event. Pauling believed that 75% of all cancers could be cured with vitamin C.¹⁵ However, results from his own institute appeared to invalidate his case. Pauling felt publication should be delayed until further results were obtained. Robinson, however, believed the experimental results were clear. We should add that the stakes were higher than simple damage to scientific reputations, as both were aware that the lives of millions could depend on the outcome. It was almost inevitable that the two should fall out over this question.

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As we shall now explain, **neither side was wrong**. The full set of results, later published by Robinson, is both interesting and informative. Robinson completed his series of experiments, using 1846 hairless mice.⁸³⁰ **Disturbingly, he found that diets apparently least suitable for healthy individuals greatly extended the lifespan of mice with cancer.** It appeared possible that dietary changes might extend the expected survival time of a cancer patient by a factor of 20. In human terms, this is the difference between having one year to live, compared to twenty years. The interpretation of these results requires some care. **Firstly**, mice were an unfortunate choice for the experiment, as they synthesise vitamin C in their bodies and normally have high blood levels. Humans are not in this fortunate position and have to obtain their vitamin C from the diet. **If mice are given supplemental vitamin C, their homeostatic control mechanisms might respond by producing less of their own.** For this reason, it is difficult to apply the results from mice given low, gram level vitamin C doses to humans. For example, unsupplemented mice might correspond to humans with an intake of several grams a day.

Pauling's objection to Robinson's results may have arisen from his knowledge of the differences between the physiology of humans and mice. He might have suggested that the biggest biochemical difference was that humans cannot manufacture vitamin C so, at these low intakes,

the results cannot easily be extrapolated to man. **Furthermore**, these mice were sick and their requirements are expected to be greater than those of healthy animals. Robinson was also correct in indicating that these results should not be concealed. Despite this, the experiments had not been completed and reporting the results would have been premature. **The full results support Pauling's assertion that vitamin C is an effective anticancer agent.**

Two months after irradiation, higher intakes of vitamin C were seen to have greatly suppressed the growth of cancers. The suppression effect was marked: no mice given high dose supplements (above 48 human-equivalent grams) had severe lesions. Furthermore, the number of minor lesions was reduced from about 54 lesions in 60 mice to seven lesions in 45 mice. Taken at face value, these results are astounding. They imply that dietary restriction or high dose vitamin C can be powerful methods for the treatment of cancer.

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Mice are not people

Robinson's results show that a raw fruit and vegetable diet with supplemental vitamin C will slow cancer growth – in mice. **The first objection a physician will state to this experiment is that mice are not people.** Some anticancer agents are effective in particular animals but not in humans. Robinson's result could provide false hope for victims and might make them avoid conventional therapy.

Robinson has said,

"if diet restriction were practiced by all cancer patients in the United States, the resulting life-extension might equal or surpass that resulting from the combined efforts of the entire current medical oncology effort"

We might normally dismiss this statement as having little value. However, the lifespan of his experimental mice was greatly extended. When Robinson suggests that some raw vegetable diets may help cancer patients, by reducing the supply of nutrients, he is making a measured statement and understands the biological differences between mice and humans.

In the first place, he embarked on this study because of reported effects in *people*. He was stimulated into studying raw vegetables by reports of their efficacy in human cancers, particularly in the book ***How I***

conquered cancer naturally, by Edie Mae Hunsberger.⁸²⁹ Healthy body cells may accommodate a nutrient shortage more effectively than the disturbed metabolism of cancer cells, whose growth depends on an adequate supply of specific nutrients.

Robinson gives the **example of a cancer patient with an inoperable throat cancer.**⁸³⁰ [Kehlkopfkrebs] The surgeon explained to the patient that he had run out of options and had only a short time to live. However, the patient sought the advice of a nutritionist, **Ann Wigmore**, and started a strict diet.

Notably, he followed Wigmore's early dietary advice, **eating nothing but raw vegetables.** Later variations on this diet involved a higher calorie intake.

After several months, the patient returned to his surgeon, who noticed three remarkable facts. **Firstly**, he was still alive and would normally have been dead by this time. **Secondly**, the patient looked like a concentration camp survivor, emaciated from malnutrition. **Finally, the cancer in his throat had completely gone.** The surgeon made sure the patient ate food containing essential nourishment and he regained his health, apparently free of cancer. John Ely has reviewed similar results¹⁹³

for glucose deprivation and vitamin C supplementation, both in mice and in humans.^{831,832}

Restricting carbohydrates in cancer patients may greatly increase the anticancer effects of vitamin C. In a study of 36 patients, carried out in 1957, only five of the patients, those with improved glucose tolerance, were reported to be tumour-free following treatment.^{725,833} In 1978 and 1979, two patients with Stage IV breast cancer decided to use ascorbate (10 grams per day or less) with restricted carbohydrate intake. Both patients were reported to be tumour free after six months. Moreover, both patients were still alive 13 years later, in 1992.⁸³¹ The patients reportedly suffered no side effects with the high ascorbate and low glucose therapy. Ely claims that this was the first description of such a rapid disappearance of a massive tumour burden, with any form of chemotherapy. His explanation is that the vitamin C caused the cancer cells to redifferentiate or revert to a more normal form. This redifferentiation is an experimentally established attribute of antioxidants.^{834,835}

John Ely and co-workers have demonstrated destruction of mouse tumours, by means of vitamin C and glucose restriction.⁸³⁶ Sugar reduction helps vitamin C to kill cancer cells, both directly and indirectly. Both ascorbate supplementation and nutritional deprivation can increase free radicals within cancer cells. Lack of glucose increases free radical damage within cancer cells. Moreover, nutrient deprivation acts at a basic physical level, preventing the supply of energy required for cancer

growth. The fundamental nature of this mechanism would be predicted to hinder adaptation by the cancer cells, as all cells require energy for growth.

A diet to eliminate cancer

Few people are willing to exist indefinitely on a diet with a severely restricted calorie intake. Despite the known risks to health, many people cannot resist the temptation to indulge their cravings for junk food, leading to an obesity epidemic in developed countries. Such people seem unable to overcome what amounts to an addiction to overeating. Even the most stoical patient would find it difficult to exist in a state of semi-starvation for their remaining lifespan. Many would consider such a cost greater than the benefit of life extension. For this reason, it is impractical to propose that cancer patients should embark on a lifelong strategy of dieting. Such a scheme would involve careful

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monitoring by qualified physicians and nutritionists, because of the potential for damage to the patient's health. However, the perceived cost/benefit analysis is a question for the patient themselves to answer.

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all the facts, **a patient might elect to eat normally and die more quickly, or to have an extended life but feel chronically hungry.** A proportion of people will choose the latter option.

Cancer cells, like any biological organism, find it difficult to multiply and grow without an adequate supply of energy or other factors essential to their survival. While different cancers will respond in diverse ways, it will be difficult for any cancer types to continue growing without sufficient food. In some cases, dietary restriction alone could cause the cancer cell population to become extinct.

Restriction of either calories or carbohydrates can slow the growth of established cancer. The extent to which the anticancer effect of restricting calories is explained by lack of available sugar is not clear from the current evidence. It could be that the benefits of calorie restriction can largely be obtained by a more acceptable, low carbohydrate diet. Lowering carbohydrate intake may provide the benefits of dietary restriction, in a more sustainable form.

Hoodia

Currently, the extent of dietary restriction needed to prevent cancer growth is not established. It may be possible to restrict the growth of many cancers with a moderate dietary restriction of carbohydrates and selected dietary nutrients. However, a severe, near-starvation diet,

consisting of raw vegetables alone, may be required for many slow growing cancers. Patients who decide it is worth starving themselves to gain the opportunity to live longer will need all the help they can get. Clearly, willpower is not sufficient, or we could all look like supermodels! However, **certain substances might make it easier to embark on a highly restricted diet, without feeling hungry all the time. One of these is Hoodia, an appetite suppressant used by the San Bushmen of the Kalahari Desert.** The Bushmen eat slices of the *Hoodia Gordonii* cactus on their hunting trips, allowing them to exist for long periods without food.⁸³⁷

Hoodia appears to act centrally in the brain to reduce appetite and maintain energy levels.⁸³⁸ Tom Mangold, a BBC reporter, has described its effects. Mangold visited the San Bushmen to experience the effects of Hoodia for himself.⁸³⁹ One evening, Mangold and his cameraman each ate a portion the size of half a banana, before driving back to Capetown.

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Both felt well on their four-hour journey and did not even think about food. The next day, they did not bother taking breakfast, as they were not hungry. Mangold ate lunch, but without pleasure. Their appetites returned gradually, after 24 hours.

In the 1960s, the South African Council for Scientific and Industrial Research (CSIR) isolated and patented an appetite suppressant molecule (P57) from Hoodia. In 1997, a British company called Phytopharm licensed P57. Following initial tests, Phytopharm sold the licence to Pfizer, one of the pharmaceutical giants. The shares of both companies rose, as investors looked forward to huge profits from this new aid to dieting. However, the companies were accused of exploiting the San Bushmen.

The chief executive of Phytopharm, Richard Dixey, claimed the San Bushmen “had disappeared” but, in fact, about 100,000 of them lived in a region spanning several countries, in southwest Africa. The San sued for a share of the profits, gaining 6% of the royalties from the CSIR license on commercial sales.

In a press release published in 2001, Phytopharm claim to have conducted a double-blind, randomised, placebo-controlled study of P57's effects.⁸⁴⁰ 19 overweight males received either P57 or a placebo, twice daily, for 15 days. Nine subjects in each group completed the study. The experimental group showed a significant reduction in their average daily calorie intake and a loss of body fat, compared to the controls. The enormous commercial possibilities of a safe appetite suppressant resulted in a large number of Hoodia-based products becoming available. However, many appear to contain little or no active ingredient. Use of Hoodia requires standardisation of its active

ingredients or its derived appetite suppressant substance, P57. Nevertheless, the existence of Hoodia indicates that it may be possible to undergo calorie restriction with less discomfort than expected. It appears possible that simple dietary changes could prevent many deaths from cancer. The mechanisms involved are based on oxidation and reduction, and are synergistic with the action of vitamin C. The use of natural supplements of L-ascorbic acid may be more efficient than "synthetic" vitamin C, as this form is more available to the cells. Ascorbate's effectiveness is greatly enhanced if combined with a reduction in sugar, and by using it with other nutrients, such as vitamin K.

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