

**WEBSITE REVIEW**  
**ALTERNATIVE MEDICINE**  
**Watching the Watchdogs at Quackwatch**

The founder and manager of [www.Quackwatch.com](http://www.Quackwatch.com), Stephen Barrett, M. D., gave a talk at our local skeptics group early this year in which he explained how helpful he has been to victims of quacks, including recovering their money. He recommended his website for general medical information. He has been a consultant for Consumers' Union. He has been Co-chairman of the Alternative Treatments Review Board for CSICOP since 7/80, and has written for their magazine *The Skeptical Inquirer*.

The website is available in 4 languages other than English, and is said to have had 2,300,000 visitors. At first glance it seems very complete and useful, with sections on Links to Other Web Sites, Consumer Strategy: Disease Management, Consumer Strategy: Tips for Provider Selection, Consumer Protection, Nonrecommended Sources of Health Advice, Questionable Products, Services, and Theories, Publications for Sale, About Quackwatch, and others. It does have a Search function. The Quackwatch Mission Statement on the website contains the following primary activities:

- Investigating questionable claims
- Distributing reliable publications
- Improving the quality of health information on the Internet
- Attacking misleading advertising on the Internet

A number of webpages (8) were selected arbitrarily because their topics were familiar to this reviewer, and these were examined minutely. The findings are used to make generalizations about the website.

The section titles below are from [www.Quackwatch.com](http://www.Quackwatch.com), as accessed on 31 Oct 01, each one followed by Comments based on the most reliable evidence I have found.

**“Tips for Lowering Your Dietary Fat Content**

Stephen Barrett, M.D. [No date given]

People whose blood cholesterol and LDL-cholesterol levels are undesirably high should consume a diet that is relatively low in total fat and saturated fat. To do

this systematically, it is necessary to become fully aware of what you are eating. This means getting into the habit of checking labels to determine the amount of cholesterol and the amount and type of fat. You should also pay attention to the "hidden" fats found in processed foods such as cookies, crackers, and snack cakes, and the kinds of fats and oils used in their own cooking.

The next step is to make substitutions. For example, leaner cuts of beef (select or choice rather than prime) should be used, and consumption of fish, poultry, fresh fruits and vegetables, beans, and other legumes should be increased. Foods high in complex carbohydrates-such as whole grains, beans, and vegetables-can be made the "main dish," with small amounts of red meats and cheeses becoming the "side dishes." Mixed dishes such as stews, casseroles, and pasta and rice meals can combine small amounts of meat with other foods, such as grains or vegetables.

Finally, evaluate your progress by having your blood cholesterol tested within a few months and then periodically as recommended by the professional who is guiding them. The goal should be a gradual but steady reduction in your total cholesterol and LDL-cholesterol levels. Low-fat eating has another potential benefit. Because obesity is often associated with a high-fat diet, some researchers suspect that low-fat eating offers promise as a weight-control measure. However, research in this area is in only its early stages. [Datillo AM. Dietary fat and its relationship to body weight. *Nutrition Today* 27(1):13-19, 1992]

If you have a serious cholesterol problem or want help in figuring out how to modify your diet, consult a registered dietitian.”

Later, under “Practical Tips”: “•Use only the egg whites or discard every other yolk in recipes requiring eggs...Or try commercial cholesterol-free egg substitutes.”

### **Comment**

The supposed relationship between eating cholesterol and fat and thereby developing some form of cardiovascular disease (called the diet-heart theory, DHT) has been disproven so many times that its refusal to die cannot be due to a true scientific dispute. There are some excellent descriptions of the chicanery that continues to promote the DHT to this day (McGee, 2001; Moore, 1989; Ravnskov, 2000; Smith, 1991). Based on the findings in a compendium of anti-cholesterol

trials, all of which were carried on long enough to obtain relative total death rates, the advice from Dr. Barrett is not supported. Specifically, in the American College of Physicians' 1996 Clinical Guideline, Part 2, 3 meta-analyses of cholesterol-lowering *diet* trials encompassing 24 trials found that there was no significant effect of diet on CHD and total mortality (Garber et al., 1996), a finding that was confirmed by three recent reviews (Ravnskov, 1998; Hooper et al., 2001; Taubes, 2001). Two recent studies found that the National Cholesterol Education Program (NCEP) guidelines for supposedly desirable (i. e., low) cholesterol levels (as preached by [www.Quackwatch.com](http://www.Quackwatch.com)) were of no value in predicting the existence of calcified plaques in coronary arteries as shown by electron beam tomography (Raggi et al., 2001; Hecht et al., 2001).

The recommendation for high-carbohydrate diets has no basis in fact. From 1955 to 1990, even as the percentage of calories declined from 41 to 35 % fat, the percentage of overweight Americans increased from 24 to 34 %. Many of the recommended starches, such as from wheat, rice and corn have high glycemic indices, higher, in fact than some those of some simple sugars (<http://www.mendosa.com/gilists.htm>); therefore, such foods are unhealthful for diabetics or pre-diabetics, and are major causes of obesity (Bernstein, 1997, p112,118,162; Gutierrez et al., 1998; Garg et al., 1994). The result of a small study on healthy adults 54-60 years old showed that the effect of a low-fat diet (the corollary of high-carbohydrate diet) on serum lipids was so deleterious that "...it seems appropriate to question the wisdom of recommending that all Americans should replace dietary saturated fat with...[carbohydrates]" (Abbasi et al., 2000). This confirmed a study on healthy postmenopausal women (Jeppesen et al., 1997). Two meta-analyses, one of 11 and one of 13 studies showed that lowering serum cholesterol through modified diets or drugs does not reduce morbidity or mortality from stroke in middle-aged men (Atkins et al., 1993; Hebert et al., 1995). It is possible that the health benefits achieved by a few people on low-fat diets was due to the decrease in the amount of toxic *trans* fats consumed (Oomen et al., 2001; de Roos et al., 2001); obviously these can be avoided in high-fat diets. Describing an actual experiment on himself, Uffe Ravnskov wrote:

"Numerous studies have shown that in people who eat a normal Western diet, the effect on blood cholesterol of eating 2 or 3 extra eggs per day over a long period of time can hardly be measured..."

"To find out how egg consumption influenced my own blood cholesterol, I once used myself as a human guinea pig without asking the ethics committee at my

university. Before and during the experiment I analyzed my [total serum] cholesterol. My usual egg consumption is one or two eggs per day, and my cholesterol value at the start of the experiment was 278 mg/dL, very close to a determination of [my] blood cholesterol made 10 years earlier.” On day 0, Dr. Ravnskov ate 1 egg; on day 1, 4 eggs; on day 2, 6 eggs; and on days 3-8, 8 eggs per day! “The data from my daring experiment showed that instead of going up, my cholesterol went down a little [to 246 mg/dL].” (Ravnskov, 2000, pp108-109).

“The low-cholesterol advocates have us believing that that eggs are evil. We know that egg yolks contain cholesterol...In fresh eggs the cholesterol is protected from the oxygen of the air by the eggshell and antioxidants in the yolk. Eating fresh eggs won't damage arteries because the cholesterol is pure. But when egg yolks are spray-dried in the process of making powdered eggs, oxysterol is formed. Spray-dried eggs are everywhere. They're used in many packaged foods such as cookies, crackers, and other commercially prepared baked goods because they are easier to handle than fresh eggs. What's worse, the ingredients label just lists them as eggs, so we can't even tell if they're in the foods we're eating.” (McCully, 2000).

An epidemiological study based on the food-frequency questionnaires used in the Health Professionals Follow-up Study on 38,000 men and the Nurses Health Study on 80,000 women found that consumption of up to 1 egg per day (the highest level investigated) did not increase the risk of CHD or stroke (Hu et al., 1999). Very few admitted to eating cholesterol-free egg substitutes, so this could not have been responsible.

### **“Low-Carbohydrate Diets**

Stephen Barrett, M. D. Posted 28 Apr 01

...Most low-carbohydrate diets do not attempt to limit the intake of proteins, fats, or total calories. (In other words, their fat content tends to be very high.) Promoters claim that unbalancing the diet will lead to increased metabolism of unwanted fat even if the calories are not restricted. This is not true, but calorie reduction is likely to occur because the diet's monotony tends to discourage

overeating.

“The most widely used low-carbohydrate diet is the one advocated by Robert C. Atkins, M. D., of New York City...[as in his books, such as his]...1992 update *Dr. Atkins’ New Diet Revolution*...The dieter is permitted to eat unlimited amounts of noncarbohydrate foods ‘when hungry’, but ketosis [formation of ketone bodies which may be detected in the urine with a test kit] tends to suppress appetite...

“...many individual experts have warned that unlimited intake of saturated fats under the Atkins food plan can increase the dieter’s risk of heart disease.”

Barrett then goes on to give examples of failures of the Atkins diet, undesirable side-effects causing dieters to drop the diet, and Atkins’ failure to keep records of his 60,000 patients.

### **Comment**

Barrett failed to make the connection that many of the people who have become obese or who react to carbohydrates in the diet with hypoglycemic depressions of energy and/or mood are genetically the ones more likely to have wide variations in blood glucose levels than normal people. In those with Type II diabetes no amount of injected insulin or oral anti-diabetic drugs will prevent the spiking of glucose and insulin concentration that does the damage to organs such as the kidneys; only restriction of high-glycemic index foods (also called rapid-acting carbohydrates because they are the ones that raise blood “sugar” rapidly) will prevent this damage (Bernstein, 1997, p43-47). A study of 65,173 nurses or former nurses found a strong association between diets high in starch, flour, and sweet foods and the development of Type II diabetes. Furthermore, consumption of minimally refined grain (such as bran without flour) lowered this risk. The combination of high glycemic foods and low intake of unrefined insoluble fiber was associated with a 2.5-fold higher incidence of diabetes. Eskimos and other hunting populations survive almost exclusively on protein and fat, and don’t develop cardiac or circulatory diseases (Bernstein, 1997, p45-47, 322-3). Other populations almost free from coronary artery disease include the Masai of Kenya, Africans of western Transvaal (Republic of South Africa), natives of Vilacamba, Ecuador, natives of Degestan, Russian Caucasus, elderly Ugandans, natives of Zaire, and semi-isolated residents of the Loetschental Valley of Switzerland (McGee, 1979, p87); as well as Indians in the Andes Mountains of Ecuador and the 8,000,000 residents of Hainan Island, China (McGee, 2001, p83, 89-90). What might all these disparate groups have in common? Diets low

in refined carbohydrate!

The [www.atkinscenter.com](http://www.atkinscenter.com) website homepage does make a distinction between high- and low glycemic index carbohydrates, while Barrett does not; nor does Barrett indicate any awareness that rapid-acting carbohydrates cause Type II diabetes (Zammit et al., 2001), which is a major risk-factor for early death . Barrett writes that better health will result from minimal consumption of animal fats, which are triglycerides and contain cholesterol. Quite the contrary, in the Scottish Heart Health Study on 11,629 subjects, the total death rates for men were not affected by HDL-cholesterol levels, total cholesterol or triglycerides; for women total cholesterol levels were not significant and the other two factors barely significant (Tunstall-Pedoe et al., 1997). In a Canadian study primarily designed to compare bypass surgery with stenting, the 5-year survival rates were distinctly *better* for patients with *hyperlipidemia* (Dzavik et al., 2001).

The primary source of body fat for most Americans is not dietary fat but carbohydrate, which is converted to blood sugar (glucose) and then, with aid of insulin, to fat by fat cells. Insulin is the primary fat-building enzyme (Bernstein, 1997, p112). With the aid of stable isotopes of carbon and hydrogen it was found that 50-80% of the lipids in human liver cells formed in 1 week were from synthesis by the cells, not absorbed from the nutrients provided; and that 80% of the carbon of the new lipid molecules came from glucose provided (Lee et al., 1995).

Daily consumption of animal fat, which typically consists of 25-50% saturated fat, in England and Wales fell about 5% absolute between 1930 and 1956; while in the same time frame death rates from coronary heart disease increased 6-fold; even *more* inverse relationships occurred in Italy, Portugal, Switzerland and France (Ravnskov, 2000, pp24, 31). Saturated fat is not even a slight cause of heart disease (Enig, 2000, pp77-78). Human milk fat is 45-50% saturated. The shorter-chain saturated fatty acids from human milk, coconut milk, and butter have worthwhile antimicrobial properties (Enig, 2000, pp87, 109-112).

The vast preponderance of honest data shows that the main premise from this page from Quackwatch is totally wrong. Low-carbohydrate diets, especially those low in refined and rapid-acting (high-glycemic index) carbohydrates, are particularly healthful, especially in people genetically disposed to Type II diabetes or hypoglycemia (Juntunen et al., 2002; Liu et al., 2002).

### **Chelation Therapy: Unproven Claims and Unsound Theories**

Saul Green, Ph. D. Revised 14 Sep 00

“Chelation therapy...is a series of intravenous infusions containing disodium EDTA [ethylenediaminetetraacetic acid] and various other substances. Proponents claim that EDTA chelation therapy is effective against atherosclerosis and many other serious health problems. Its use is widespread because patients have been led to believe that it is a valid alternative to established medical interventions such as coronary bypass surgery. However, there is no scientific evidence that this is so. It is also used to treat nonexistent ‘lead poisoning’, ‘mercury poisoning’, and other alleged toxic states...”

But later on p1: “After EDTA was found effective in chelating and removing toxic metals from the blood, some scientists postulated that hardened arteries could be softened if the calcium in their walls was removed.” Note the inconsistency! Green goes on to list 4 books he chose arbitrarily of the many that promote EDTA chelation. Then:

“The scientific jargon in these books may create the false impression that chelation therapy for atherosclerosis, and a host of other conditions, is scientifically sound. The authors allege that between 300,000 and 500,000 patients have safely benefited. However, their evidence consists of anecdotes, testimonials, and poorly designed experiments.”

Green then gives an early history of 3 apparent successes with chelation, and dismisses them all. He then describes how the procedure is to be carried out according to the main proponent organization in the USA, the American College for Advancement in Medicine (ACAM), the cost, “\$75-125 per treatment”, and the fact that most medical insurance will not cover this cost. He continues with a critique of a 1989 study that will be detailed below, 15 reports of trials that found no benefit, and 2 trials in the 1980s that supposedly found no benefit. A page that warns about the lack of safety of the treatment follows, then a refutation of 4 theories of how the treatment works, and finally a report of a study in Denmark in 1992 that Green considered to be of the highest quality. Green does not cite a single study he thinks shows any success from this treatment. His summary reinforces this conclusion: “The few well-designed studies that have addressed the efficacy of chelation for atherosclerotic diseases have been carried out by ‘establishment’ medical scientists. Without exception, these found no evidence that chelation worked.”

### **Specific Quotations and Comments**

“The sources used for this review included position papers of professional societies, technical textbooks, research and review articles, newspaper articles, patient testimonials [which Green considered inadmissible in his previous paragraph], medical records, legal depositions, transcripts of court testimony, privately published books, clinic brochures and personal correspondence.”

COMMENT: Green’s actual bibliography consisted of just 6 citations, all to papers in peer-reviewed journals, the most recent being from 1992. There were no reviews cited. Three of the citations lacked authors and titles, two of these lacked page numbers, and one of these had a misspelled journal title!

Green wrote of the success of the 1963 trial by Kitchell et al., his ref. 2, and Kitchell’s denial of success. Green wrote that the “improvement” (his quotes) was not significant because it was no better than than would be expected with proven methods, and there was no control group for comparison.

COMMENT: There was no description of the diagnosis of the patients in this trial, so one must assume that they suffered from atherosclerosis and one of its manifestations, such as intermittent claudication (blocked arteries). There were no “proven methods” at that time, no bypass surgery, no angioplasty. While the double-blind, placebo-controlled trial is the gold standard for treatment studies, it was not common before 1975. The patients were suffering from circulation conditions that would not be expected to improve without treatment, thus the patients served as their own controls. Even at present, since it is considered unethical not to treat a number of diseases, many trials do not utilize a placebo.

“In a retrospective study of 2,870 patients treated with NaMgEDTA [sic], Olszewer and Carter (1989) [sic] concluded that EDTA chelation therapy benefited patients with cardiac disease, peripheral vascular disease [this is blocked arteries] and cerebrovascular disease. These conclusions were not justified because the people who received the treatment were not compared to people who did not.”



COMMENT: No citation was given! The authors of this study (Olszewer, 1988) which utilized Na<sub>2</sub>MgEDTA, address the limitations of their study directly: “There was no placebo or control group and these studies are not double-blinded. It is a retrospective analysis of a large number of cases with standardized criteria for assessing improvement. Each patient served as his/her own control...the clinical response rates, especially for angina and intermittent claudication, are too high, i. e. from 77% to 91%, respectively, to be attributed to placebo-effect alone...”

Green then describes a study reported in 1990 by these same authors in which he ridiculed the double-blinding, and the actual results in exercise stress tests and blood pressure changes were not given by Green.

COMMENT: Since there was no citation, this sarcastic description cannot be evaluated with certainty; but there is another description of this trial in which 10 patients with intermittent claudication were divided into 2 groups. The treatment group received 10 infusions of EDTA, with the usual additives, while the control group received all of the additives without the EDTA; this was double-blinded. The treatment group more than doubled their walking distance compared with no significant change in the placebo group. The differences were so striking that it was considered immoral to continue, and both groups received EDTA from then on, resulting in an equal improvement in what had been the control group, and further improvement in what had been the treatment group (Cranton, 2001, p285-293).

Green wrote that a randomized, controlled, double-blind clinical trial of chelation therapy conducted by Curt Diehm at the University of Heidelberg Medical Clinic with Bencyclan, a blood-thinning agent, as the control (no placebo) gave similar results in both groups, and both groups were said to respond to the placebo effect.

COMMENT: The citation for this was given as follows: “Zeit. Deutsch Herzstiftung. Vol 10, July 1986.” An online search failed to locate this paper, thus this study could not be evaluated from the citation as given. Assuming that this was “The Heidelberg Study”, others have interpreted the raw results to indicate that the EDTA-treated group enjoyed 5 times the increase of walking distance of the Bencyclan group (Cranton, 2001, pp xxiii-xxiv, 339-340).

Green wrote that a randomized, controlled, double-blind clinical trial of chelation therapy

conducted by R. Hopf at the University of Frankfurt, with normal saline as the placebo, gave similar results in both groups, with the conclusion that EDTA was of no benefit.

COMMENT: The citation for this was given as follows: “Zeit. f. Kardiologie, 76, #2, 1987”. An online search of Zeitschrift für Kardiologie, Vol. 76, 1987, failed to produce this paper; thus, because of this possibly spurious citation, the work could not be evaluated.

Neither of the German studies was cited in any of the 3 major reviews in journals on EDTA chelation (Grier et al., 1993; Chappell et al., 1996; Ernst, 1997).

Green devotes a page to the supposed lack of safety of chelation, writing that there is no published scientific evidence that chelation will improve poor blood circulation, and that loss of zinc ion due to chelation is potentially serious. “People with coronary artery disease who need bypass surgery and choose chelation instead place themselves at great risk.” This was in the context of attacking an advertisement for chelation therapy, which was reproduced on the webpage.

COMMENT: While it accepted the use of EDTA chelation for treatment of lead poisoning, the US Food & Drug Administration (FDA) has been quite hostile to its use for anything else. An official request was sent from the FDA to state health and regulatory agencies across the US asking that any information relating to untoward results, poor results, or patient complaints about EDTA chelation therapy be forwarded to the FDA; no such reports were received (Cranton, 2001, p xv). Despite the loss of zinc to EDTA chelation, the authors of the study to which Green referred (above) recommended that zinc supplements be given, and that chelation be continued as both diagnosis and treatment for heavy metal poisoning (Allain et al., 1991). Bypass surgery has been shown in 3 major studies to have no significant effect on long-term survival rates, especially if function in the left main coronary artery had been adequate beforehand (McGee, 2001, pp24-28). A recent study from Canada is supportive in that bypass surgery improved 5-year survival rates only where disfunction in the left main coronary artery was treated (Dzavik et al., 2001). The immediate death rate from bypass surgery is about 6% (McGee, 2001, p28), while the immediate death rate from properly applied and administered EDTA chelation is nonexistent, and chelation has been called one of the safest therapies available (Cranton, 2001, p345-351).

The attack on the advertisement reproduced on this webpage of [www.Quackwatch.com](http://www.Quackwatch.com) certainly exemplified one of the activities in the Mission Statement, but the attack has been shown

in this review to be groundless.

Green devotes 1/4 of the EDTA webpage to rebutting 4 theories of how chelation is supposed to work biochemically. His attempts to debunk these theories are supposed to show that chelation cannot have any beneficial effect because none of the theories stand up scientifically. With each of his debunkings is added a hypothetical scenario intended to scare the user into avoiding chelation.

COMMENT: Green would appear to want to discourage the use of aspirin, morphine, codeine, general anesthetics, and antibiotics, since the biochemical explanation of these and other useful drugs were not understood when the drugs were adopted. For example, aspirin was used for more than 70 years before an explanation for its effects was found (Kauffman, 2000). The clinical benefits of these medical treatments were so obvious that double-blind studies would have been superfluous. The sad corollary is that modern prescription drugs taken for life to treat chronic diseases often have such weak effects, yet accompanied by such profound side-effects, that any overall benefits can be proven only by large-scale controlled trials, and even these are often inadequate (Cohen, 2001). All of Green's scare scenarios ignore the fact that chelation has been very safe as well as effective.

As a specific example of Green's use of supposed chemical knowledge, the following quotation is given from one of his explanations of why chelation cannot work: "Ionic iron has two electrons in its outermost or N shell and 14 electrons in its M shell. This configuration gives ionic iron the distinct characteristic of being able to accept three pairs of electrons from other ions... When iron is dissolved in water at a pH of 7.0 or more, its three pairs of electrons will be bound to three OH groups of the water."

COMMENT: In a transition metal such as iron the N shell is of lower energy than the M shell, so the N shell cannot be said to be outermost, especially since these two shells are combined to give hybrid orbitals. In elemental iron the total number of electrons in these outer shells, said to be the valence shells where electron transfer takes place in chemical reactions, is  $2 + 6 = 8$ , not 16. It follows that the unsolvated ions  $\text{Fe}^{2+}$  would have 6 and  $\text{Fe}^{3+}$  would have 5 electrons. Iron ions are not unique either in their ability to form a trihydroxide or to share 3 pairs of electrons. While solvated iron ion may share up to 6 pairs of electrons provided by a donor; it does not

provide a unique three pairs of its own (Holtzclaw et al., 1984).

As the clinching argument, Green saved the Danish Study for last:

“In 1992, a group of cardiovascular surgeons in Denmark published results of a double-blinded, randomized, placebo-controlled study of EDTA treatment for severe intermittent claudication [Guldager B et al., J Int Med 1992;231:261-267]. A total of 153 patients in two groups received 20 infusions of EDTA or a placebo for 5 to 9 weeks, in a clinical protocol duplicating the conditions used by Olszewer and Carter in 1990. The changes seen in pain-free and maximal walking distances were similar for the EDTA-treated and the placebo group, and there were no long-term therapeutic effects noted in 3-month and 6-month follow-ups. These investigators concluded that chelation was not effective against intermittent claudication.”

COMMENT: The Danish Study is the darling of the opponents of chelation, which includes 2 of the 3 reviews cited above (Grier et al., 1993; Ernst, 1997). This Danish study was flawed by the fact that those conducting it were cardiovascular surgeons whose livelihood was threatened by EDTA. They even went so far as to pre-announce their expectation of a negative effect from their study! Instead of using the best cocktail for the purpose, which includes magnesium, they omitted it, thus they were *not* duplicating the cocktail used by Olszewer and Carter in 1990. The Danish surgeons used iron as part of the oral supplements, which predictably chelated more strongly with the EDTA than either magnesium or calcium, guaranteeing a lesser effect. Also, 70% of the patients were smokers despite the fact that it has been shown that smoking will neutralize the effect of chelation. The Danish surgeons were informed by telephone and in writing that there were errors and omissions which would invalidate the trial. Finally they were investigated very grudgingly by the Committee on Investigation into Scientific Dishonesty of the Danish Medical Association, which found that the correct cocktail was not used, that a mineral (iron) was used that was contraindicated, that the double-blinding was broken, and that the surgeons claimed they had used the correct cocktail even when informed they had not (Douglass, 1995). A more recent review (Chappell, 1996) agrees with Douglass, and cites 4 more critical reviews of the Danish study published in peer-reviewed journals. On top of this, the treatment

group was pre-selected to be much sicker than the control group, with mean maximum walking distances of 119 meters before treatment for the EDTA group and 157 meters for the placebo group. When the raw data were examined the treatment group enjoyed an increase of 51% and the control group 24%, so this study was positive despite its denial by its own authors, who candidly admitted that they undertook the study to persuade the Danish government *not to pay* for chelation (Cranton, 2001, pp5-6)!

### **General Comment**

Discouraging sick people from undergoing an effective treatment is despicable, even more so when dangerous procedures with limited applicability such as bypass surgery are recommended instead. Because of the bias in mainstream medicine against chelation, most patients who accept it do so as a last resort after all conventional treatments have failed, although a majority would have been better off using chelation as a first treatment. It is estimated that more than a million patients have received more than 20 million infusions of EDTA with no ill effects when the procedure is correctly done, and that about 88% of patients improve (Cranton, 2001, pp4, 324). The motives of the anti-chelation cliques have been described in detail (Cranton, 2001, pp329-342; Carter, 1992).

### **“Glucosamine for Arthritis**

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Updated 18 May 2000

“In short-term controlled trials, glucosamine has been reported to be effective in relieving pain and increasing range of motion in patients with osteoarthritis. [One 4-week double-blind trial found that oral glucosamine sulfate (500 mg 3 x per day) was more effective than placebo in relieving symptoms. Two other double-blind trials, a 4-week and an 8-week, against ibuprofen showed equal effectiveness in relieving pain.] One Medical Letter consultant in an area where many patients are taking glucosamine has not detected any adverse events...

“Glucosamine appears to be safe and effective for treatment of osteoarthritis, but most published trials of the drug lasted only four to eight weeks and Medical Letter consultants find them unconvincing. As with other ‘dietary supplements’, the purity of the glucosamine products sold in pharmacies, health food stores, and supermarkets in the USA is unknown...

“Update... On March 15, 2000, the Journal of the American Medical Association published a meta-analysis [citation given] whose authors concluded: ‘As with many nutraceuticals that currently are widely touted as beneficial for common but difficult-to-treat disorders, the promotional enthusiasm often far surpasses the scientific evidence supporting clinical use.’”

COMMENT: This Quackwatch webpage, in its zeal to disparage the benefits of glucosamine, perhaps because it is perceived as an alternative to non-steroidal antiinflammatory drugs (NSAIDS) such as ibuprofen and aspirin, not to mention the newer COX-II inhibitors commonly used to treat arthritis, misses two important points. First, some NSAIDS, while relieving pain, worsen the progression of osteoarthritis (Reginster et al., 2001). Secondly, “NSAIDS can cause serious harm, even fatalities, from bleeding in the stomach or intestines. Bleeding can occur at any time and without warning, and older people are more likely to experience adverse effects from bleeding. Older adults are also more likely to have reduced liver and kidney function.” (Wolfe et al., 1999). Patients with newly diagnosed kidney failure who used aspirin or acetaminophen regularly were 2.5 times as likely to proceed to chronic kidney failure as those who did not. (Forel et al., 2001)

Since this webpage was written, it was shown in a double-blind placebo controlled trial lasting 3 years, patients on placebo suffered joint space loss and worsening symptoms, while those on 1500 mg glucosamine sulfate per day had minimum joint space narrowing and improvement in symptoms with no differences in safety or differential withdrawals from the trial (Reginster et al., 2001).

### **“Magnet Therapy**

Stephen Barrett, M. D. [Revised 26 Apr 01]

“During the past few years, magnetic devices have been claimed to relieve pain and to have therapeutic value against a large number of diseases and conditions. The way to evaluate such claims is to ask whether scientific studies have been published. Pulsed electromagnetic fields -- which induce measurable electric fields -- have been demonstrated effective for treating slow-healing [bone] fractures and have shown promise for a few other conditions. However few studies have been published on the effect on pain of small, static magnets marketed

to consumers.

“The main basis for the claims is a double-blind...study, conducted at Baylor College of Medicine in Houston, which compared the effects of magnets and sham magnets on knee pain. The study involved 50 adult patients with pain related to having been infected with polio virus when they were children. A static magnetic device or a placebo device was applied to the patient’s skin for 45 minutes. The patients were asked to rate how much pain they experienced when a ‘trigger point was touched’. The researchers reported that the 29 patients exposed to the magnetic device achieved lower pain scores than did the 21 who were exposed to the placebo device. Although this study is cited by nearly everyone selling magnets, it provides no legitimate basis for concluding that magnets offer any health-related benefit.

- Although the groups were said to be selected randomly, the ratio of women to men in the experimental group was twice that of the control group. If women happen to be more responsive to placebos than men, a surplus of women in the ‘treatment’ group would tend to improve that group’s score.

- The age of the placebo group was 4 years higher than that of the control group. If advanced age makes a person more difficult to treat, the ‘treatment’ group would again have a scoring advantage.

- The investigators did not measure the exact pressure exerted by the blunt object at the trigger point before and after the study...

“Two better-designed, longer lasting pain studies have been negative:

- Researchers at the New York College of Podiatric Medicine have reported negative results in a study of patients with heel pain. Over a 4-week period, 19 patients wore a molded insole containing a magnetic foil, while 15 patients wore the same type of insole with no magnetic foil. In both groups, 60% reported improvement, which suggests that the magnetic foil conveyed no benefit...[Caselli et al., 1997].

- ...researchers at the VA Medical Center in Prescott, Arizona conducted a randomized, double-blind, placebo-controlled, crossover study involving 20 patients with chronic back pain... Each patient was exposed to real and sham

bipolar permanent magnets during alternate weeks, for 6 hours per day, 3 days per week for a week, with a 1-week period between the treatment weeks. No difference in pain or mobility was found between the treatment and sham-treatment periods...” [Collacott et al., 2000].

Barrett then lists several legal cases where 4 companies were barred from advertising their magnets as having any medical benefit whatsoever. Finally, “There is no scientific basis to conclude that small, static magnets can relieve pain or influence the course of any disease...”

COMMENT: In the Baylor trial (Valbona et al., 1997) patients who received the magnets experienced an average pain score decrease of  $4.4 \pm 3.1$  ( $p < 0.0001$ ) on a 10-point scale vs. those on placebo, decrease  $1.1 \pm 1.6$ . The proportion of patients in the treatment group who reported a pain decrease greater than the average placebo effect was 76%, so the effect was not trivial. The proportion of women was higher (5:1 vs. 3:1) in the placebo group, as Barrett notes; but this does not necessarily invalidate the results; what if women happen to be *less* responsive to placebo than men? The ages of the treatment and control groups had SDs of  $\pm 10$  years. This means that 95% of the treatment group were 32-72 years old vs. 35-75 years old for the controls. Because of the very large overlap in ages, the difference between the means of ages does not mean much. And what if advanced age makes a person *more* responsive to treatment? In this double-blind study one would expect that the various pressures of the blunt object used to elicit pain would be randomly distributed. So this study remains indicative of probable benefit for the type of pain and the magnets used, and it should not be dismissed.

The NY College of Podiatric Medicine study showed that the type of magnets used had no effect on heel pain, not that every use of magnets for pain is useless. The foil magnets of 500 gauss at the surface were tested because they had previously been found to increase foot temperature by improving blood flow, to relieve muscular tension, and to alter the depolarization of C-fibers, so there was some therapeutic effect that prompted the trial.

The VA study showed that their bipolar magnets of 300 gauss did not help with back pain when the subjects were exposed for a total of 18 hours during the course of 1 week, not that all applications are worthless. How odd that Dr. Barrett did not mention that the ratio of women to men in this trial was 1:19.

In what the reader will now begin to realize is typical of [www.Quackwatch.com](http://www.Quackwatch.com), another



serious study with positive results was not cited. In a randomized, placebo-controlled study magnetic insoles significantly reduced diabetic neuropathy pain in the feet. One foot was used as the control, and there was a crossover. At the end of 4 months improvement was still pronounced in the diabetic patients. In the authors' own words: "The constant wearing of magnetic devices was able to dramatically suppress the neuropathic symptoms of burning pain and numbness and tingling in the diabetic cohort (90%) as compared to the non-diabetic cohort (33%). This response appears to be palliative but not curative since symptoms recur when... [the magnets are removed]. Nonetheless, it is intriguing that success was achieved in a condition felt to be 'disabling, intractable, and progressive'" (Weintraub, 1999). What could be the motivation of Stephen Barrett, M.D., who would discourage large numbers of advanced diabetics from obtaining such a simple form of relief? Or who cannot admit that some static magnets may relieve some kinds of pain?

### **Homeopathy: The Ultimate Fake**

Stephen Barrett, M.D. Updated 25 Aug 01

"Homeopathic 'remedies' enjoy a unique status in the health marketplace: They are the only category of quack products legally marketable as drugs.

"The 'Remedies' are Placebos — Homeopathic products are made from minerals, botanical substances, and several other sources. If the original substance is soluble, one part is diluted with either nine or ninety-nine parts of distilled water and/or alcohol and shaken vigorously (succussed); if insoluble, it is finely ground and pulverized in similar proportions with powdered lactose (milk sugar). One part of the diluted medicine is then further diluted, and the process is repeated until the desired concentration is reached. Dilutions of 1 to 10 are designated by the Roman numeral X (1X = 1/10, 3X = 1/1,000, 6X = 1/1,000,000). Similarly, dilutions of 1 to 100 are designated by the Roman numeral C (1C = 1/100, 3C = 1/1,000,000, and so on). Most remedies today range from 6X to 30X, but products of 30C or more are marketed...

"Actually, the laws of chemistry state that there is a limit to the dilution that can be made without losing the original substance altogether. This limit, which is related to Avogadro's number, corresponds to homeopathic potencies of 12C or 24X (1 part in  $10^{24}$ ). Hahneman himself [the originator of homeopathy] realized

that there was virtually no chance that even one molecule of original substance would remain after extreme dilutions. But he believed that the vigorous shaking or pulverizing with each step of dilution leaves behind a 'spirit-like' essence -- 'no longer perceptible to the senses' -- which cures by reviving the body's 'vital force'. This notion is unsubstantiated...

“Since many homeopathic remedies contain no detectable amount of active ingredient, it is impossible to test whether they contain what their label says. Unlike most potent drugs, they have not been proven effective against disease by double-blind clinical testing.”

Barrett goes on to cite and summarize several papers and reports of commissions in which a few of many trials had positive results. “Proponents trumpet the few ‘positive’ studies as proof that ‘homeopathy works’”. Barrett then shows how the FDA could produce extinction of homeopathy in the USA.

COMMENT: Your reviewer would be the first to admit that homeopathy should not have any beneficial effect beyond placebo for the obvious reasons. But a 1991 review from the University of Limburg in The Netherlands found that: “A survey of 293 general practitioners in The Netherlands showed that 45% of them think that homeopathic remedies are efficacious in treating upper respiratory tract infections or hay fever.” These reviewers “...could not believe the positive result...” found with pollen C30 in hay fever, so they did an exhaustive search for other reports on trials in homeopathy as skeptics. Assessment of the methodological quality of 107 controlled trials in 96 published reports was done using a list of predefined criteria, including proper randomization of patients and double-blinding. Of 105 trials with interpretable results, 81 trials indicated positive results, whereas 24 trials were negative. Of the 22 best quality trials with controls, 15 showed positive results and 7 showed no positive effect. In all the trials, using diagnoses from conventional medicine, certain conditions responded more than others, namely pollinosis, pain and mental problems. “...there is a legitimate case for further evaluation of homeopathy, but only by means of well-performed trials.” (Kleijnen, 1991).

Barrett did report that a later review of 184 trials found only 17 of reasonable quality, and that positive effects were found in some. His 2 citations were to non-peer-reviewed reports, of which one was from the National Council Against Health Fraud [NCAHF], which is associated

with and recommended by [www.Quackwatch.com](http://www.Quackwatch.com). But: “This Council, itself fraudulent, seems to have taken over the work of the Coordinating Conference on Health Information, the covert arm of the American Medical Association [AMA]... The NCAHF receives money from the AMA, the National Pharmaceutical Council, the food industry, and others.” (Carter, 1992, pp124-125). The NCAHF has also been accused of forcible interference with free speech on campus of Loma Linda University and the defamation of character of 2,500 physicians and others including Linus Pauling (Whitaker, 1997). It is believed that Dr. Whitaker’s letter and the actions of the California Health Freedom Movement team were responsible for the removal of the NCAHF from the University ([http://www.savedclark.org/by\\_whom2.htm](http://www.savedclark.org/by_whom2.htm) on 19 Jan 02).

As wrenching as the “logic” of homeopathic treatments appears to your rational reviewer, there is enough evidence of their effectiveness so as not to dismiss the technique altogether as Barrett has done.

### **“Dietary Supplements: Appropriate Use**

Stephen Barrett, M. D. Revised 11 May 01

“In general, supplements are useful for individuals who are unable or unwilling to consume an adequate diet... High (above-RDA) doses of vitamins should be regarded as drugs rather than supplements... Most high-dose recommendations by the health-food industry and its allies are not valid.” Here Barrett cites his own book in support (Barrett, 1994). He makes exceptions for very young children, pregnant teenagers, some vegetarians, and some sedentary seniors — anyone who does (or might) not consume “an adequate diet”.

A major exception is that: “Women should be sure that their intake of calcium is adequate to help prevent thinning of their bones (osteoporosis). The National Academy of Sciences advises Americans and Canadians at risk for osteoporosis to consume between 1000 and 1300 milligrams of calcium per day... This can be done with adequate intake of dairy products, but some women prefer calcium supplements. Women should discuss this matter with their physician or a registered dietitian.”

COMMENT: Studies exist which are quite contradictory to Dr. Barrett's position. In research supported by the Wallace Genetic Foundation and the American Cancer Society it was found that among 11,348 US adults of ages 25-74 years who were followed for 10 years, the 1,809 deaths were inversely related to the vitamin C intake. What makes this study especially useful is that males and females were considered separately, and those with a daily intake of 50+ mg from diet alone were compared with those with similar dietary intake plus regular supplements, which averaged 800 mg per day, far above the RDA of 60 mg per day. The relative risk (RR) of all-cause death with no supplements was set to 1.00. With supplements it dropped to 0.70 in males and 0.97 in females. Most of the gain was fewer deaths from cardiovascular diseases rather than cancer, with slightly fewer deaths from esophageal and stomach cancers in males (Enstrom et al., 1992). Others have interpreted this study to mean that men taking vitamin C supplements lived 6 years longer than those who did not, and women lived 1 year longer (Cranton, 2001, p23; McGee, 2001, 105).

In the Nurses Health Study of 8 years duration, women who took, on average, 200 IU of vitamin E as supplements for 2-8 years, had a RR of 0.59 for coronary diseases of several types and a RR of death of 0.87. Men who took 100-250 IU of vitamin E for 2-10 years had a RR for coronary diseases of several types of 0.63 (Kauffman, 2000).

In people with severe congestive heart failure (CHF), those on conventional therapy, including digitalis, diuretics and vasodilators had a 3-year survival rate of 25%. With added vitamin Q10 (also called coenzyme Q10 and ubiquinone) the 3-year survival rate was 75% (Folkers, 1986). The benefits of using this supplement in people with CHF was recently confirmed by a review from an unrelated research group (Mongthuon et al., 2001).

These 3 are sufficient examples to expose the quality of advice on this webpage of [www.Quackwatch.com](http://www.Quackwatch.com).

Contrarily, the admonition for women to take calcium is not exactly supported in the simplistic form in which it was given. According to Lynne McTaggart in *Medicine: What Works & What Doesn't* (1995), only about 13% of the cases of osteoporosis can be attributed to insufficient calcium intake. Lack of absorption of calcium may be caused by insufficient phosphorus, magnesium or vitamin D. Lieberman and Bruning in *The Real Vitamin & Mineral Book* (1990) wrote: "I advise you to take your calcium along with magnesium in the ratio of 2:1" [by mass, this is 1:1 in number of atoms]. Since the most persuasive evidence is often from peer-

reviewed medical journals, here are some examples: (1) A review of published data did not support calcium megadosing for the management of postmenopausal osteoporosis. When a dietary program emphasizing magnesium instead of calcium was tested on 19 postmenopausal women, a significant increase in bone density was observed within 1 year in 8 of the 15 who had bone density below the spine fracture threshold (Abraham et al., 1990). (2) In clinical studies, the efficacy of calcium, copper, manganese and zinc supplementation on bone mineral density of postmenopausal women was demonstrated (Saltman et al., 1993). (3) Estrogen's enhancement of magnesium utilization and uptake by soft tissues and bone may explain resistance of young women to heart disease and osteoporosis, as well as increased prevalence of these diseases when estrogen production ceases. With calcium supplementation in the face of commonly low magnesium intake, the risk of thrombosis increases (Seelig, 1993). (4) A total of 194 postmenopausal women of whom 70 were osteoporotic were studied with forearm bone densitometry. The dietary intake of calcium, phosphorus and magnesium were correlated with low bone density. Calcium and magnesium intakes were lower than the U. S. RDAs in this Ancona, Italy, based study. Supplements were recommended *before* menopause (Tranquilli et al., 1994). (5) No correlation was found between serum calcium levels and bone density in 14 subjects. In the 5 patients given magnesium supplements for 2 years a significant increase in bone density was observed (Rude et al., 1996).

### **“Stanislaw Burzynski and ‘Antineoplastons’**

By Saul Green, Ph.D.”

The following short webpage is reproduced whole for accuracy and tone from:  
<<http://www.Quackwatch.com/01QuackeryRelatedTopics/Cancer/burzynski1.html>>

The bold letters in brackets refer to specific Comments below.

“Unlike most "alternative medicine" practitioners, Stanislaw R. Burzynski has published profusely. The sheer volume of his publications impresses patients, but unless they understand what they are reading, they cannot judge its validity. To a scientist, Burzynski's literature contains clear evidence that his data do not support his claims. [A]

## Burzynski's Background and Credentials

Burzynski attended the Medical Academy in Lubin [sic],[**B**], Poland, where he received an M.D. degree in 1967 and an D.Msc. degree in 1968. He did not undergo specialty training in cancer or complete any other residency program. His bibliography does not mention clinical cancer research, urine, or antineoplastons during this period. [**C**]

In 1970, Burzynski came to the United States and worked in the department of anesthesiology at Baylor University, Houston, for three years, isolating peptides from rat brains. (Peptides are low-molecular-weight compounds composed of amino acids bonded in a certain way.) He got a license to practice medicine in 1973 and, with others, received a three-year grant to study the effect of urinary peptides on the growth of cancer cells in tissue culture. The grant was not renewed. [**D**]

In 1976, with no preclinical or clinical cancer research experience [**E**], Burzynski announced a theory for the cure of cancer based on his assumption that spontaneous regression occurs because natural anticancer peptides, which he named antineoplastons, "normalize" cancer cells. Since urine contains lots of peptides, he concluded that there he would find antineoplastons [**F**]. Less than one year later and based only on these assumptions, Burzynski used an extract from human urine ("antineoplaston A") to treat 21 cancer patients at a clinic he opened [**G**]. His shingle read, 'Stanislaw R. Burzynski, M.D., Ph.D.'

Burzynski's claim to a Ph.D. is questionable. When I investigated, I found:

- An official from the Ministry of Health in Warsaw informed me that when Burzynski was in school, medical schools did not give a Ph.D. [1]. [**H**]

- Faculty members from at the Medical Academy at Lubin [sic], [**B**] informed me that Burzynski received his D.Msc. in 1968 after completing a one-year laboratory project and passing an exam [2] and that he had done no independent research while in medical school [3]. [**I**]

•In 1973, when Burzynski applied for a federal grant to study "antineoplaston peptides from urine," he identified himself as "Stanislaw Burzynski, M.D, D.Msc." [4]

#### Analysis of Antineoplaston Biochemistry

Tracing the biochemistry involved in Burzynski's synthesis of antineoplastons shows that the substances are without value for cancer treatment. [J]

By 1985, Burzynski said he was using eight antineoplastons to treat cancer patients. The first five, which were fractions from human urine, he called A-1 through A-5. From A-2 he made A-10, which was insoluble 3-N-phenylacetyl amino piperidine 2,6-dione. He said A-10 was the anticancer peptide common to all his urine fractions. He then treated A-10 with alkali, which yielded a soluble product he named AS-2.5. Further treatment of AS-2.5 with alkali yielded a product he called AS-2.1. Burzynski is currently treating patients with what he calls "AS-2.1" and "A-10."

In reality, AS-2.1 is phenylacetic acid (PA), a potentially toxic substance [K] produced during normal metabolism. PA is detoxified in the liver to phenylacetyl glutamine [sic] (PAG), which is excreted in the urine. When urine is heated after adding acid, the PAG loses water and becomes 3-N-phenylacetyl amino piperidine 2,6-dione [sic] (PAPD), which is insoluble. Normally there is no PAPD in human urine.

What Burzynski calls "A-10" is really PAPD treated with alkali to make it soluble. But doing this does not create a soluble form of A-10. It simply reinserts water into the molecule and regenerates the PAG (Burzynski's AS-2.5). Further treatment of this with alkali breaks it down into a mixture of PA and PAG. Thus Burzynski's "AS-2.1" is nothing but a mixture of the naturally occurring substances PA and PAG.

Burzyski claims that A-10 acts by fitting into indentations in DNA. But PAG is too big a molecule to do this [L], and Burzynski himself has reported that PAG is ineffective against cancer [5,6].

PA may not be safe. In 1919, it was shown that PA can be toxic when ingested by normal individuals. It can also reach toxic levels in patients with phenylketonuria (PKU); and in a pregnant woman, it can cause the child in utero to suffer brain damage. [K]

Burzynski has never demonstrated that A-2.1 (PA) or "soluble A-10" (PA and PAG) are effective against cancer [M] or that tumor cells from patients treated with these antineoplastons have been "normalized." Tests of antineoplastons at the National Cancer Institute have never been positive. [N] The drug company Sigma-Tau Pharmaceuticals could not duplicate Burzynski's claims for AS-2.1 and A-10. The Japanese National Cancer Institute has reported that antineoplastons did not work in their studies. No Burzynski coauthors have endorsed his use of antineoplastons in cancer patients. [O]

These facts indicate to me that Burzynski's claims that his "antineoplastons" are effective against cancer are not credible. [P]

#### About the Author

Dr. Green is a biochemist who did cancer research at Memorial Sloan-Kettering Cancer Center for 23 years. He consults on scientific methodology and has a special interest in unproven methods. He can be reached at (212) 957-8029. This article is adapted from his presentation at the American Association for Clinical Chemistry Symposium in Atlanta in July, 1997.

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This article was posted on June 25, 2001.”

### Comments

[A] Green does not give any specific example to support this assertion. Julian Whitaker, M. D., for one, cites and accepts the evidence in at least one of Burzynski’s papers (Whitaker, 1999).

[B] Green means Lublin.

[C] These two sentences are merely attempts at invalidation, since lack of experience in a sub-specialty does not disqualify a person from *beginning* the sub-specialty.

[D] Since Green did not give a reason for the non-renewal of the grant, this fact must be regarded as another smear.

[E] According to Daniel Haley, who devoted a 48-page book chapter to antineoplastons, Burzynski had suspected, before he left Poland, that his peptides had anti-cancer activity after noticing that one of them was almost absent from the blood of a prostate cancer patient. In 1974 he co-authored an article which reported that the peptides caused up to 97% inhibition of DNA synthesis and cell division in cancer cells in tissue cultures (Haley, 2000, p346). Thus Green’s innuendo that antineoplastons (the anti-cancer peptides) sprang whole from nothing is probably false. Furthermore, Green’s implication that clinical or preclinical experience is needed is false; anyone can come up with a treatment worth investigating.

[F] This innuendo is false. Burzynski had first isolated antineoplastons from blood, and looked for them in urine in order to have a more convenient source of them (Haley, 2000, p347),

and by 1980, could synthesize them (Haley, 2000, p350).

[G] It is significant that Green did not reveal what happened to these 21 patients. It was reported that complete remission occurred in 4 cases, more than 50% remission in another 4 cases, and some improvement in another 4 cases. In 2 of the 5 patients who died, there was significant regression of the “neoplastic process”, and the deaths were not due to cancer or to any toxicity of the treatment (Burzynski, 1977).

[H] According to Daniel Haley, Burzynski earned both his M. D. and Ph. D. at age 24. Reacting to a paper in *JAMA* by this same Saul Green, Burzynski sent the *JAMA* a sworn statement from the President of the Medical Academy of Lublin confirming Burzynski’s Ph. D. in Biochemistry and M.D. with honors (Haley, 2000, pp345,362). Robert G. Houston, a medical writer, wrote in a letter to *JAMA* that “...Contrary to Green, I found Burzynski’s doctoral dissertation in biochemistry listed in the bibliography that Green claims omits it.” (Houston, 1993).

[I] An online search turned up 11 publications on research by Burzynski from 1964-1970, many in Polish.

[J] This statement smacks of one described above for EDTA chelation therapy, an intimation that understanding of Burzynski’s synthesis of antineoplastons would somehow prove that they could not be effective treatments for cancer. Surely one can see that effectiveness of substances for cancer is not related to a complete understanding of their biochemistry. Furthermore, the syntheses were by standard *in vitro* reactions, not biochemical (Burzynski et al., 1986).

[K] This innuendo fails to give any actual toxicity. According to the Merck Index, phenylacetic acid is used as an analgesic, antirheumatic and urinary antiseptic. Its sodium salt has been approved for human use by the FDA for treatment of hyperammonemia (Burzynski, 1993). Its LD<sub>50</sub> i. p. in mice is 2,710 mg/kg (Burzynski et al., 1986). By comparison, from the Merck Index, 9th ed., the common antineoplastic drug vincristine has an LD<sub>50</sub> i. v. in mice of 2mg/kg; this would extrapolate to just 100mg as the fatal dose for a human.

[L] The structures of Antineoplaston A-10 and the DNA-intercalating agent Doxorubicin, “the most important anticancer drug available” (Foye et al., 1995, p839) are shown in Fig. 1 drawn to the same scale and style. It should be obvious that A-10 (also called PAG) is not too big to fit into (intercalate) with DNA.

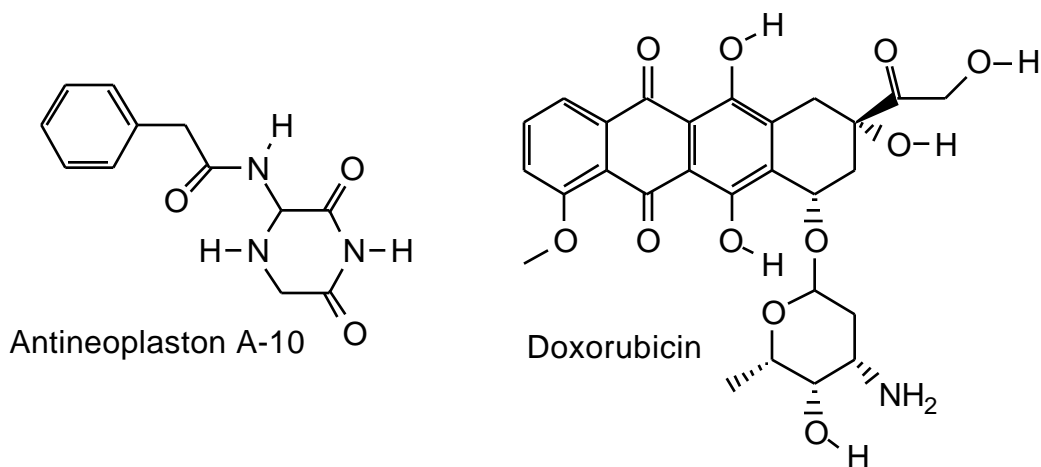


Fig. 1. Structural Formulas of Antineoplaston A-10 and Doxorubicin

[M] Quite the contrary, Burzynski has reported promising clinical results with AS2-1 in refractory cancer of the prostate (Burzynski et al., 1990) and with synthetic A10 (Liau et al., 1987), and with both in primary brain tumors (Burzynski et al., 1999).

[N] According to Robert G. Houston, the NCI saw the results of antineoplaston treatments in 7 cases and concluded that antitumor responses occurred (Houston, 1993). In a press-release the NCI claimed that these were the only patients of about 3,000 who had benefited (a lie), that Burzynski had sent the NCI incomplete patient information (also untrue), and when NCI-sponsored trials were done, the patients selected by the NCI were much more advanced and sicker than ones Burzynski had agreed to have treated. The NCI used lower doses than Burzynski advised, and withdrew the 2 improving patients to damage the evidence (Diamond et al., 1997, 674-685)!

[O] Since coauthorship implies endorsement, the presence of coauthors with Burzynski in at least 11 papers in peer-reviewed journals on the use of antineoplastons in cancer patients contradicts Green's assertion. These authors include Burzynski, B., Conde, A. B., Daugherty, J. P., Ellithorpe, R., Kaltenberg, O. P., Kubove, E., Liau, M. C., Mohabbat, M. P., Nacht, C. H., Peters, A., Saling, B., Stolzman, Z., Szopa, B., and Szopa, M.

[P] Ample evidence has been given to show that much of what Green wrote on this webpage is questionable. To make an informed decision on the effectiveness of antineoplastons, Julian Whitaker, M. D., over a 5-year period, visited the Burzynski Clinic 5 times, spoke with scores of patients, and evaluated their medical charts. He gave 4 examples of patients, 3 of them

children, who were offered no hope of long-term survival by conventional cancer practitioners, then successfully treated with antineoplastons, and calls this treatment “the most significant breakthrough in cancer research ever” (Whitaker, 1999). “Antineoplastons do not work all the time — nothing does. But there is enough data and case histories to demonstrate conclusively that these medicines do represent a breakthrough” (Haley, 2000, p386). “For Dr. Burzynski’s 3,000 patients, antineoplaston is a lifesaver; among ...alternative physicians, the treatment is gaining respect and credibility” (Diamond et al., 1997, pp684-685).

### **General Comment**

The discoverer of antineoplastons, Stanislaw R. Burzynski, earned his Ph. D. and M. D. degrees together in 1968 from the Medical Academy of Lublin, Poland, at age 24. His research for his doctorate in Biochemistry was on peptides, short chains of amino acids which are the building blocks of proteins. He noted that people with chronic kidney failure rarely develop cancer, and that these people have a superabundance of certain peptides in their blood. These peptides did turn out to have anti-tumor effects; Burzynski christened them antineoplastons.

Blood was an inconvenient source of antineoplastons, so Burzynski succeeded in finding them in the urine of healthy humans, and gradually identified about a dozen. These simple molecules, many related to phenylacetic acid, were found to stimulate the activity of “human suppressor genes”, that is, ones that turn off the activity of oncogenes. Thus the antineoplastons seem to act as a normal control mechanism for cell division, so they are not cytotoxic and indiscriminate as are the usual antineoplastic drugs used in cancer chemotherapy (Foye et al., 1995). This early work was published in peer-reviewed journals; many of the early papers were in Polish. Mixtures of these antineoplastons isolated from human urine produced up to 97% inhibition of DNA synthesis and mitosis in neoplastic cells in tissue culture (Burzynski, 1976).

Burzynski came to the USA around 1970 and worked at Baylor College of Medicine, Houston, TX. With others he obtained, in 1974, a Research Grant from the National Cancer Institute (NCI) of 3-year duration. Individual antineoplastons were identified and, by 1980, synthesized. By 1977 he had published on a study of the action of antineoplaston A on 21 patients considered end-stage and untreatable by conventional methods. It was reported that complete remission occurred in 4 cases, more than 50% remission in another 4 cases, and some improvement in another 4 cases. In 2 of the 5 patients who died, there was significant regression of the “neoplastic process”, and the deaths were not due to cancer or to any toxicity of the treatment

(Burzynski, 1977). Inexplicably by normal scientific standards, the grant was not renewed.

About this time Burzynski set up the Burzynski Research Institute in which cancer patients declared hopeless by mainstream practitioners were expected to pay for what was honestly called experimental treatment with antineoplastons. Success was considerable, especially for brain tumors in children (Burzynski, 1999). From the late 1970s to the present, every available government, state, and medical agency aligned with the cancer industry has done its best to shut down Dr. Burzynski's clinic. What made him a threat to the cancer industry from the beginning was the prospect that antineoplaston therapy represented a successful alternative to toxic and dangerous chemotherapy drugs, upon which a major part of the cancer industry's profits depend (Diamond, 1997). Like nutritional supplements, antineoplastons are natural products, and not patentable.

Attacks on Burzynski, both *ad hominem* and technical, have been launched from many directions. Burzynski attempted to complete New Drug Applications for the FDA, and to have trials carried out by the NCI, as well as doing his own with whatever approval from the FDA he could manage to obtain. On one pretext or another the US Food & Drug Administration (FDA) brought Burzynski to trial several times, and lost every case. The NCI is said to have faked clinical trials of antineoplastons in order to produce negative results (Haley, 2000; Diamond, 1997). After 20 years of such struggle, along with publication of many more papers in peer-reviewed journals, Burzynski was asked why he did not leave the USA; he replied that the science would prevail in the end. Ample evidence has been given here to show that much of what Green wrote on this webpage is questionable at best. Despite vicious attacks and distractions, such as raids by FDA operatives on Burzynski's clinic, and confiscation of many of his records, his research continues on what is probably an effective approach to the treatment of many types of cancers.

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All 8 pages from [www.Quackwatch.com](http://www.Quackwatch.com) that were examined closely for this review, which were chosen simply because their topics were familiar to this reviewer, were found to be contaminated with incomplete data, obsolete data, technical errors, unsupported opinions, and/or innuendo; no other pages were examined. Hostility to all alternatives was expected and observed from the website, but not repetition of groundless dogma from mainstream medicine, examples of which were exposed. As a close friend and colleague reminded me, the operators of this site and I

may have the same motivation — to expose fraud. It remains a mystery how they and I have interpreted the same body of medical science and reached such divergent conclusions. While Dr. Barrett may (or may not) have helped many victims of quacks to recover funds and seek more effective treatment, and while some of the information on pages of the website not examined in this review may be accurate and useful, this review has shown that it is very probable that many of the 2,300,000 visitors to the website have been misled by the trappings of scientific objectivity. At least 3 of the activities in the Mission Statement:

- Distributing reliable publications
- Improving the quality of health information on the Internet
- Attacking misleading advertising on the Internet

...have been shown to be flawed as actually executed, at least on the 8 webpages that were examined. Medical practitioners such as Robert Atkins, Elmer Cranton and Stanislaw Burzynski, whom I demonstrated are not quacks, were attacked with the energy one would hope to be focused on real quacks. The use of this website is not recommended. It could be deleterious to your health.

Acknowledgment. Expert online searches and editorial aid were provided by Leslie Ann Bowman. Additional aid from other faculty at The University of the Sciences in Philadelphia was obtained from Gina Kaiser, Robert C. Woodley and Sylvia Tarzanin. Ted Pollard made valuable contributions.

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Disclaimer: Any recommendations herein are based on studies published in peer-reviewed scientific journals. I am not an M. D. and cannot engage in the practice of medicine. My degrees are: B. S. in Chemistry from the Philadelphia College of Pharmacy and Science, and a Ph. D. in Organic Chemistry from the Massachusetts Institute of Technology. My experience includes about 10 years of exploratory drug development at the former, now called the University of the Sciences in Philadelphia, and 4 years at the Massachusetts College of Pharmacy, where the major effort was on synthesis of potential anticancer drugs under contract with the National Cancer Institute (NCI), and writing the chapter on Cancer Chemotherapy in the 2nd and 3rd eds. of W. O. Foye, Ed., *Principles of Medicinal Chemistry*; this also appeared as Kauffman, J. M., & Foye, W. O. (1979), The Nature and Treatment of Cancer, *The Apothecary* 91, May/June, 7; and Kauffman, J. M., & Foye, W. O. (1979), Antineoplastic Drugs, *The Apothecary* 91, July/August, 7. Later I served as consultant to the Franklin Research Center in Philadelphia, PA, partially in connection with their contract with the NCI to develop anticancer drugs.

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