INTERNATIONAL HEALTH NEWS

Your Gateway to Better Health!

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Welcome to the beginning of our 11th year of publication on the World Wide Web. As usual, this issue is packed with information aimed at keeping you healthy. We begin another series by Prof. William Ware. This time he discusses "The Diet Zoo" and yes, it certainly is a "zoo" out there when it comes to choosing what constitutes a healthy diet. The low fat, high carbohydrate diet advocated by the Establishment for many years is, fortunately, on its way out after no doubt having contributed to millions of cases of obesity and type 2 diabetes. The question now is, "What is a healthy diet?" Dr. Ware addresses this question in three parts – Part I deals with basic nutrition science, Part II explores information from scientific studies relating to the effects of fat, carbohydrate and protein consumption, and Part III discusses the pros and cons of current Establishment diet recommendations as well as such popular diets as the Atkins and South Beach diets. A must read!

Also in this issue our New Zealand correspondent, Dr. Maurice McKeown, discusses the wound healing properties of Manuka honey. And if this isn't enough, we bring you our usual Abstracts and Newsbriefs to keep you up to date on health news from around the world. Enjoy!

Wishing you and yours a Happy Holiday Season and good health in the coming year,

Hans Larsen, Editor

December/January Highlights

Fish oils benefit the heart

SOUTHAMPTON, UNITED KINGDOM. It is well established that populations with a high consumption of oily fish have a lower incidence of heart disease and several studies have confirmed that fish oils (eicosapentaenoic acid [EPA] and docosahexaenoic acid [DHA]) are the protective components. There is also impressive evidence that they help prevent atherosclerosis, lower blood pressure, reduce triglyceride levels, and are highly protective against both fatal and non-fatal heart attacks. Fish oils also have antiarrhythmic effects and help prevent blood clotting.

Recent research concludes that perhaps the most important effect of fish oils, when it comes to preventing cardiovascular disease, is their ability to stabilize atherosclerotic plaque by reducing the infiltration of inflammatory and immune cells (lymphocytes and macrophages) into the plaque. Heart attacks are now believed to involve the rupture of an atherosclerotic plaque. These plaques come in two main varieties, those with a thin, unstable fibrous cap and those with a thick, stable fibrous cap. A recent study showed that supplementation with 1.4 grams/day of fish oil significantly reduced macrophage infiltration and resulted in a substantial shift towards a preponderance of stable, thick-capped plaques. At least two studies have shown that the beneficial

effects of fish oils on heart health become clear after about 2 months.

Calder, Philip C. New evidence in support of the cardiovascular benefit of long-chain n-3 fatty acids. **Italian Heart Journal**, *Vol. 4, July 2003, pp. 427-29*

All milk is not the same

BRISBANE, AUSTRALIA. Cow's milk contains about 34 grams of protein per liter. The protein has two components, whey and casein; there are several types of casein in milk, but the main ones are A1 and A2 beta casein. The two types break down differently when digested with A1 casein producing a bioactive peptide, which is very similar to the digestion product of gluten. Research has uncovered a strong association between the consumption of beta casein A1 and the incidence of childhood (type I) diabetes. More recently, a strong correlation has also been found between the incidence of heart disease and the consumption of beta casein A1. The consumption of A1 in Ireland, for example, is 3.5 times higher than in France and so is the incidence of heart disease. Just recently scientists at the University of Queensland demonstrated (in animal experiments) that beta casein A1 promotes atherosclerosis and high cholesterol levels while beta casein A2 does not.

The ratio of A1 to A2 casein in cow's milk varies from herd to herd. Beta casein from Red Danish cows typically contains 70% A1 whereas beta casein from Guernsey herds contains 70% of the A2 version. A2 Corporation, a New Zealand dairy company, has found that it is possible, by selective breeding, to increase A2 content to 100% and is now marketing A2 milk in NZ. A2 milk is essentially free of the detrimental A1 fraction.

Tailford, Kristy A., et al. A casein variant in cow's milk is atherogenic. **Atherosclerosis**, *Vol. 170, September 2003, pp. 13-19*

www.a2corporation.com

Holter recordings may help identify sleep apnea

ST. LOUIS, MISSOURI. Sleep-disordered breathing (sleep apnea syndrome) is fairly common, especially among patients with coronary heart disease or hypertension (high blood pressure). Sleep apnea is, unfortunately, not simple to diagnose and it is estimated that 80-90% of all cases remain undiagnosed. This is clearly a problem as sleep apnea has been linked to an increased risk of heart attack, stroke, angina, ventricular arrhythmias, hypertension, and decreased heart rate variability. The interrupted sleep pattern characterizing sleep apnea can result in an increased risk of accidents, depression, anxiety, memory loss, and diminished sexual function.

Diagnosis involves as overnight stay in a sleep-testing laboratory where the patient is hooked up to a polysomnograph during sleep. The polysomnograph continuously records such variables as brain waves, eye movements, muscle activity, heart rate, and blood oxygen level. Some equipment even produces a snoring index.

Researchers at the Washington University School of Medicine now propose that a relatively simple Holter monitor recording done overnight may be useful as a screening tool for sleep apnea. Their study involved 57 patients 33 of which had been diagnosed with sleep apnea through polysomnography. By examining the patients' Holter monitor recordings the researchers were able to identify all patients who DID NOT have sleep apnea (100% sensitivity). They also found that all patients, whose change in heart rate during an apnea episode were equal to or exceeded 20 beats/minute for at least 20% of the total recording time, had significant obstructive sleep apnea. Overall, they were able to identify correctly 33% of all patients with a polysomnographic diagnosis of sleep apnea.

The researchers believe that nighttime Holter monitor recordings may be useful in screening for sleep apnea and other sleep-related disorders such as restless leg syndrome. They do point out that test results may be misleading for patients with major autonomic dysfunction as well as for those who take beta-blockers or have frequent arrhythmias.

Stein, Phyllis K., et al. A simple method to identify sleep apnea using Holter recordings. **Journal of Cardiovascular Electrophysiology**, *Vol. 14, May 2003, pp. 467-73*

More on DVT and air travel

CANBERRA, AUSTRALIA. Australian researchers have just released a study relating hospital admissions for deep vein thrombosis (DVT) to air travel. They found that 46 out of 4.8 million Australian airline passengers arriving in Western Australia during 1981-1999 had been admitted with DVT within 2 weeks of arrival. Out of 4.6 million non-Australians arriving over the same period 200 were admitted. Thus total hospital admissions for DVT was 26.5 per million passengers. The corresponding mortality from pulmonary embolism (a possible end result of DVT) was 1 per 2 million passengers or 2% of passengers diagnosed with DVT. The DVT rate was fairly low until about age 40 years, after which it increased quite dramatically to reach 140 per million at age 75 years and older. The main predisposing factors for DVT are blood clotting disorders, cancer, heart failure, obesity, leg fracture, recent surgery, infection, and pregnancy. The risk is also increased by conditions common during air travel such as sitting still for long periods of time, dehydration, smoking, and alcohol consumption.

Kelman, CW, et al. Deep vein thrombosis and air travel: record linkage study. **British Medical Journal**, *Vol. 327, November 8, 2003, pp. 1072-76*

Editor's comment: International air travel now accounts for about 1.56 billion person trips each year. Applying the Australian findings to this number would result in a total worldwide incidence (based on hospital admissions) of DVT related to air travel of some 41,000 passengers per year and a total mortality of about 780 passengers. Of course, it is entirely possible that some passengers may have developed DVT beyond the 2-week survey period or, in the case of foreign travellers, may have left Australia before DVT was diagnosed.

Other researchers have provided very different estimates of DVT risk related to air travel. Scurr et al at the Royal Free and University College in London, UK estimate that as many as 10% of all air travellers develop symptomless DVT in the calf[1]. American researchers observed that 17% of patients with DVT admitted to hospital developed their symptoms during or after a flight[2]. Considering that the overall annual incidence of DVT (including air travel) is about 48 per 100,000 people, the total number of air travellers suffering DVT would be about 75,000 per year[3]. The LONFLIT study carried out recently found an overall DVT risk of 1.5% among 744 long distance air travellers[4]. All passengers affected by DVT in the LONFLIT study had either cardiovascular disease or used pharmaceutical drugs. Assuming that even as little as 5% of all airline flights are long distance, the LONFLIT data would indicate that over 1 million passengers are at risk for DVT every year. Airhealth.org, an organization dedicated to research into the risk of DVT related air travel, estimates that there are 100,000 deaths in the US alone every year from DVT associated with air travel[5].

Professor Gianni Belcaro of G d'Annunzio University in Italy, who has conducted a number of travel-related DVT studies, led a team of experts who examined 568 passengers flying between the UK and Italy from May to September this year. The passengers, aged between 25 and 65 years, were screened by ultrasound for blood clots both before and after their flight. The researchers discovered clots in 4.3% of the high-risk subjects after the flight, with two passengers going on to develop pulmonary embolisms possibly related to their trip. Those with an increased risk of DVT are women on the birth control pill and HRT, people who have recently had surgery, and pregnant women. Alcohol also increases the risk. Prof. Belcaro said, "The results show passengers are at risk of developing blood clots even on short flights. In fact, our research suggests most blood clots develop in the first 2 to 3 hours of a journey and grow larger and more dangerous with time". Two of the 568 passengers involved in Dr. Belcaro's study went on to develop thromo embolism so extrapolating his numbers to the 1.56 billion annual passengers would mean that 15 million travellers would develop a clot and over 600,000 would develop DVT.

It is clear that estimates of DVT vary substantially and that there is as yet no consensus as to the magnitude of the problem. Nor does there appear to be a clear understanding of how many clots detected by ultrasound actually end up producing clinical DVT or pulmonary embolism. Until authorities get a better handle on the situation and can provide a realistic risk estimate it would seem prudent to follow the preventive measures outlined in the November 2003 issue of *IHN*.

References

[1] Scurr, JH, et al. Frequency and prevention of symptomless deep-vein thrombosis in long-haul flights: a randomised trial. The Lancet, Vol. 357, May 12, 2001, pp. 1485-89

[2] Eklof, B, et al. Venous thromboembolism in association with prolonged air travel. Dermatol Surg, Vol. 22, July 1996, pp. 637-41
[3] Silverstein, MD, et al. Trends in the incidence of deep vein thrombosis and pulmonary embolism: a 25-year population-based study. Archives of Internal Medicine, Vol. 158, March 23, 1998, pp. 585-93
[4] Belcaro, G, et al. Venous thromboembolism from air travel: the LONFLIT study. Angiology, Vol. 52, June 2001, pp. 369-74
[5] Reynolds, M. Deep-vein thrombosis in long-haul flights. The Lancet, Vol. 358, September 8, 2001, p. 838 (letter to the editor)

Paradoxical pharmacology makes waves

The medical community and the pharmaceutical industry may be facing major changes in the way drugs are prescribed. Recent research has shown that some drugs that were previously thought to be highly detrimental in the treatment of a disease now turn out to be highly effective. A good case in point is congestive heart failure (CHF) and beta-blockers. Patients with CHF have reduced pumping power so need more adrenaline or adrenaline-mimicking drugs to get the heart to work harder. Beta-blockers block adrenaline receptors so conventional pharmacology dictated that beta-blockers should never be used in CHF. About 30 years ago a Swedish cardiologist discovered that giving CHF patients low doses of beta-blockers over the long-term actually strengthened their heart and decreased mortality by 65%. Thus paradoxical pharmacology was born. In this approach, drugs that actually make the patient worse in the short-term are given and result in a very significant long-term improvement. The improvement is believed to be due to the body adapting to a drug and producing a beneficial change in the number or status of the receptors for the particular drug. The approach has been used with spectacular success in CHF and preliminary indications are that it may also be useful in asthma therapy – again using small doses of beta-blockers. Other prominent examples of paradoxical pharmacology are in the treatment of hyperactive children where Ritalin (an amphetamine) is given to calm them down and acne where retinoic acid (a skin irritant) has been found quite effective. Richard Bond, a pharmacologist at the University of Texas, believes that paradoxical pharmacology may prove to be an effective approach to the treatment of 5-10% of all diseases.
Martindale, Diane. What doesn't kill you. **New Scientist**, *October 25, 2003, pp. 38-41*

Microwaving destroys vital nutrients

MURCIA, SPAIN. A team of Spanish researchers reports that microwaving broccoli virtually eliminates its vital antioxidant nutrients. The researchers cooked broccoli in 4 different ways – steaming, pressure cooking, boiling, and microwaving. They found that steaming preserved about 90% of the bioflavonoid antioxidant content, pressure cooking preserved about 45% (the rest being lost in the water), boiling preserved about 35%, and microwaving only about 2%. In other words, 98% of perhaps the most important components of broccoli as far as health protection and cancer prevention are concerned were lost in microwaving. The same group of researchers has also found that broccoli heads lose 50-80% of their vital nutrient content between harvest and actual sale at the retail level.

Meanwhile a team of Finnish researchers has discovered that blanching vegetables prior to freezing them results in a loss of about 30% of their antioxidant content. Freezing in itself also causes a small loss of antioxidants.

Microwave cooking zaps nutrients. **New Scientist**, *October 25, 2003, p. 14*
Vallejo, F, et al. Health-promoting compounds in broccoli as influenced by refrigerated transport and retail sale period. **J Agric Food Chem**, *Vol. 51, May 7, 2003, pp. 3029-34*

Editor's comment: Raw food contains more nutrients than cooked food, but unfortunately, in many cases cooking is required in order to make these nutrients absorbable in the body. The Spanish research clearly shows that steaming is the optimum way of cooking vegetables. It also clearly shows that microwaving destroys pretty well all the really important nutrients in broccoli. If microwaving destroys nutrients in broccoli, is there any reason to believe that it would not also destroy vital nutrients in other foods? Considering that the microwave oven was introduced for general domestic use about 30 years ago it really is quite astounding that it is only now being realized that microwaving has a devastating effect on vital food components.

Finally, it is clear that the vital nutrient content in broccoli is dramatically reduced through transportation, storage, and cooking. The optimum way of obtaining all the benefits of broccoli is by sprouting organic broccoli seeds. Sprouted broccoli seeds have about 40 times more of cancer preventive nutrients than does cooked broccoli and animal experiments have shown that these sprouted seeds are highly protective against prostate cancer and perhaps other cancers as well.

Safety and efficacy of vitamins

TORONTO, CANADA. The most recent issue of the *Journal of Orthomolecular Medicine* is devoted entirely to a discussion of the benefits and safety of vitamins. Among the highlights of the dozen articles on the subject:

- Vitamin A is highly effective in preventing a flu from taking hold. The recommended dosage is 100,000 IUs at onset and 100,000 IUs in the evening. Patients usually feel great the next morning. If more in-depth immune support is required 100,000 IUs can be taken twice a day for no longer than 1 week – after this liver toxicity can become a problem.
- Niacin flush is usually not a problem in dosages of 100 mg or less. It can be minimized by gradually increasing the dosage, taking it right after a meal, and accompanying it with vitamin C. Talking aspirin for 2 days before beginning niacin therapy will also reduce flushing intensity. Once the first flush has been experienced aspirin is no longer required.
- Although niacin is not toxic to the liver it can result in elevated liver function tests. This effect can be avoided by taking 1.2 g of lecithin twice daily along with the niacin.
- Niacin may increase homocysteine levels, but this effect is easily counteracted by supplementation with folic acid, vitamin B6 and vitamin B12.
- Vitamin B6 deficiency is common and associated with elevated homocysteine levels, premenstrual syndrome (PMS), kidney stones, and asthma. Supplementation with 40-100 mg/day of vitamin B6 (pyridoxine) helps alleviate the above conditions.
- Total body sun exposure provides the equivalent of 10,000 IUs (250 micrograms) of vitamin D. Thus the safe limit for daily vitamin D intake is probably around 10,000 IU/day. Dr. Reinhold Veith, a noted vitamin D researcher, says that many people are deficient in vitamin D, particularly those living in northern latitudes. He believes the current recommended daily intake of 400 IU/day is too low by a factor of 10. Vitamin D deficiency has been associated with osteoporosis, rickets, multiple sclerosis, hypertension, congestive heart failure, several types of cancer, and scleroderma.
- Vitamin E helps normalize blood pressure, strengthens and regulates the heart beat, helps relieve angina, prolongs prothrombin clotting time, and decreases platelet adhesion. Normal dosage is 400-800 IU/day, but up to 3,000 IU/day may safely be used to treat specific conditions. A dose of 2,000 IU/day was used safely in elderly patients to slow down the progression of Alzheimer's disease.

The overall conclusion of the reports is that vitamins have an important role in both the prevention and treatment of many disorders, that they are generally safe, and that not a single fatality has ever been associated with vitamin supplementation.

The safety and efficacy of vitamins. **Journal of Orthomolecular Medicine**, *Special Issue, Vol. 18, 3rd & 4th Quarters, 2003*

Vitamin C and longevity

LONDON, UNITED KINGDOM. Researchers at the London School of Hygiene and Tropical Medicine report that older people with high blood levels of vitamin C live longer than people with low levels. Their study involved 1214 people between the ages of 75 and 84 years. All participants had a blood sample drawn for analysis of vitamin C (ascorbate), vitamin E, vitamin A (retinol), and beta-carotene levels. They also completed detailed food

frequency questionnaires during a personal interview.

The researchers found that ascorbate concentrations decreased markedly with age and that participants with the highest blood levels of ascorbate (greater than 66 micromol/L) had about half the risk of dying during the 4-year follow-up period as did participants with the lowest blood levels (less than 17 micromol/L). Blood levels of the other antioxidants measured did not correlate with mortality.

Ascorbate levels correlated well with fruit and vegetable intake of at least 5 servings per day. Only 17% of the participants took vitamin C supplements and doing so did not affect the correlation between blood levels of ascorbate and mortality. Men, smokers, sedentary people, and people of a lower socioeconomic status were more likely to be vitamin C deficient. The researchers conclude that an effort should be made to increase ascorbate levels in older people preferably through an increased intake of fresh fruit and vegetables.
Fletcher, Astrid E., et al. Antioxidant vitamins and mortality in older persons. **American Journal of Clinical Nutrition**, *Vol. 78, November 2003, pp. 999-1010*

Editor's comment: Other research has shown that supplementing with 400 mg/day of vitamin C yields an equilibrium ascorbate level of about 70 micromol/L in healthy young volunteers. It is likely that older people may require more, so 300-500 mg 3 times a day is probably a good aim. Vitamin C should always be taken in divided doses throughout the day as it is fairly rapidly excreted. It is also prudent to supplement with a combination of vitamin C and the bioflavonoids usually found with it in nature rather than with just pure ascorbic acid.

NEWSBRIEFS

Homocysteine levels linked to stroke. Researchers at the Sheba Medical Center in Israel have confirmed that a high blood level of homocysteine (a sulfur-containing amino acid derived from methionine) increases the risk dramatically of suffering an ischemic stroke. Their study involved over 3,000 men and women with congestive heart failure. They conclude that a high homocysteine level increases the risk of stroke nearly 5-fold even after adjusting for traditional risk factors such as hypertension, high cholesterol/triglyceride levels, diabetes, and smoking. They also point out that high homocysteine levels are a risk factor for atherosclerosis, cognitive decline and Alzheimer's disease, and can worsen the symptoms of Parkinson's disease. NOTE: Elevated homocysteine levels can be safely and effectively lowered by supplementation with folic acid (400-800 micrograms/day), vitamin B6 (50-100 mg/day) and vitamin B12 (1 mg/day taken sublingually).
Italian Heart Journal, Vol. 4, September 2003, pp. 577-79

Cholesterol-lowering drug should be taken at night. Simvastatin (Zocor) is a widely prescribed cholesterol-lowering drug. It is not clear whether the timing of taking the drug is important. British researchers now report that simvastatin is significantly more effective in lowering both total cholesterol and LDL cholesterol (the "bad" one) if taken in the evening. A similar effect was not observed with another statin drug, atorvastatin (Lipitor) probably because it remains in the body longer.
British Medical Journal, Vol. 327, October 4, 2003, p. 788

Life span and fatty acids. Australian researchers have just released the results of a fascinating study which links longevity with the fatty acid composition of cell membranes. They discovered animals that have a preponderance of saturated fatty acids in their membranes have a slower metabolism and live far longer than animals that have lots of polyunsaturated fats in their membranes. The key fatty acid would seem to be the highly polyunsaturated docosahexaenoic acid (DHA), a major component of fish oils. It turns out that cell membranes rich in DHA are a lot more fluid than membranes rich in saturated fatty acids. This results in a faster metabolism and quicker reaction times all around, especially in the brain and eyes. Unfortunately, DHA is also an easy target for free radicals that leak out of mitochondria as they produce energy. The end result is that fluid, unsaturated membranes deteriorate and age faster than more viscous, saturated ones. Free radical attacks can also damage proteins and DNA. On the other hand, a shortage of DHA in cell membranes can lead to serious problems like high blood pressure, heart disease, diabetes, and depression.

So you guessed it – you can't win! About the only proven way of increasing longevity is by restricting calorie intake which apparently removes some DHA from membranes.
New Scientist, November 1, 2003, pp. 42-45

Antidote to antibiotics. When an antibiotic is taken orally most of it enters the blood stream after absorption in the small intestine. Some of it, however, is not absorbed and is carried on into the large intestine (bowel, colon) where it cannot be absorbed. Antibiotics can linger in the colon for up to 2 weeks and during this time they not only kill beneficial bacteria in the bowel, thus allowing overgrowth with candida, but they also allow antibiotic- resistant bacteria to develop in the gut. A Finnish company, Ipsat Therapies of Helsinki, has come up with an elegant solution to this problem. They have developed an enzyme that destroys most common antibiotics. They enclose the enzyme in an enterically coated capsule so that it is only released when it reaches the colon. Tests on volunteers have shown that the enzyme capsule does not affect blood levels of antibiotic at all, but does completely inactivate any antibiotic that has made it unabsorbed to the colon.
New Scientist, October 25, 2003, p. 14

GM crops impact seriously on wildlife. Most genetically modified (GM) seeds are synthesized so that the resulting plants will be highly resistant to powerful weed killers. Not surprisingly, concerned citizens have asked the question, "What impact do these weed killers have on wildlife?" The answer is now in. During an impressive 4-year investigation sponsored by the British government scientists made 4,000 visits to some 283 GM crop fields and collected and analyzed 1 million plants, 1.5 million insects and small animals, and 750,000 seeds. Their conclusion was that two out of the three GM crops investigated (oilseed rape and sugar beet) had a serious negative impact on both the variety and number of wildlife in the fields. The scientists point out that the problem is not so much that a crop has been genetically modified as the fact that it leads to a massive increase in the application of wildlife-destroying weed killers. Experts conclude that the results of the investigation are so negative that it is unlikely that the British government will ever open up the country to GM crops.
New Scientist, October 25, 2003, p. 3 and 21

Salivary cortisol test is accurate. Researchers at the University of Milan report that the salivary cortisol test provides an accurate measurement of the body's cortisol level. They compared the results from the salivary tests with those from a serum cortisol test and a urinary free cortisol test taken at midnight from 41 patients with Cushing's syndrome (excessively high cortisol levels) and 27 controls. They found that all 3 tests diagnosed Cushing's syndrome with a 95% accuracy. They recommend that the salivary test be used as the first-line test when diagnosing patients suspected of having Cushing's syndrome.
Journal of Clinical Endocrinology & Metabolism, Vol. 88, September 2003, pp. 4153-57

Smallpox vaccine lasts a lifetime. In the aftermath of 9/11 the Centers for Disease Control and Prevention claimed that the effect of immunization against smallpox only lasts for about 3-5 years. This led to a massive effort to revaccinate large numbers of military personnel and healthcare workers in the US. The Israel Defense Forces Medical Corps more recently issued a statement indicating that vaccination retained its effect for at least 30 years. German researchers now conclude that smallpox vaccination is likely to be protective for life. They studied the vaccination history of people who contracted smallpox during epidemics in Liverpool, UK in 1902-1903 and 1950-1971. They found that 77% of study participants were still protected against fatal smallpox 70 years after their vaccination. The protective effect against severe (including fatal) smallpox declined by 1.41% per year, so that 50 years after vaccination about 50% of the protective effect had been lost.
American Journal of Epidemiology, Vol. 158, October 15, 2003, pp. 717-23

Electric blankets linked to breast cancer. A study involving 304 African-American women diagnosed with breast cancer and 305 matched controls concludes that the use of an electric blanket or mattress pad is associated with a 50% increase in the risk of breast cancer. The risk increases the more often the heating device is used and with the number of years it is used. Women who used it more than one of the 4 seasons had a 4-fold increase in risk and those who used it for 10 years or more had a 6-fold risk increase. Risk was substantially reduced it the heating device was only used to warm the bed and was then turned off. Overall risk was higher in premenopausal women than in post menopausal women.
American Journal of Epidemiology, Vol. 158, October 15, 2003, pp. 798-806

Therapeutic Value of Honey as a Skin Healer

by Maurice McKeown, BDS, PhD

Mainstream and alternative medicine frequently confront one another in many areas of health care. Sadly such confrontations rarely result in any clear-cut victory for either side. That could, however, be set to change.

Researchers in New Zealand and Australia are currently building a strong case that some of their local honeys can promote wound healing which is vastly superior to that provided by mainstream medication; indeed even superior to traditional honeys. Healing of persistent leg ulcer is for example, a major problem. The treatment of damage to skin is after all, a field in which the results are clearly visible. There is a substantial body of medical literature, which has investigated the potential beneficial effects of honey in recent years. Professor Peter Molan at the University of Waikato in New Zealand has reviewed 73 published articles on the subject. Professor Molan has been a key figure in the research on Manuka honey in New Zealand. (*Primary Intentions 1998 Vol 6 (4) 148-158*)

One such study involved the investigation of superficial burn injuries published in the *British Journal of Surgery* in 1991. One hundred and four patients were included. Fifty-two were treated by conventional medical methods and the remainder with honey. Of the wounds treated with honey 87% healed within 15 days as against 10% in the conventional group. (*Subrahmanyam 1991 Topical application of honey in treatment in burns. Vol 78: 497-498*)

Honey has provided a therapeutic ointment from time immemorial. It is only recently however that we have been able to explore the biochemical mechanisms which are involved. Any ointment applied to damaged skin whether it be burned or cut, has a legion of tasks to perform. It has, above all, to maintain the integrity of the skin involved, expel noxious invaders and promote the healing process. It seems that some honeys can supply such benefits and may in addition be able to defeat even dreaded antibiotic-resistant bacteria.

New Zealand folklore has fostered the belief that some types of honey have substantial curative powers. This led to the investigation of honey produced from bees that fed upon our native tea trees here in New Zealand. The trees were so named because early explorers, desperate for a decent cup of tea, were forced to use their leaves to produce a drinkable beverage. (I have to say that having tasted the beverage, I would definitely prefer water.)

It should be pointed out that the early discoverer/explorer Captain Cook did not precisely differentiate all the plants that he discovered around the world. Thus in both Australia and New Zealand we have plants called 'Tea' trees. Many readers will be familiar with tea tree oil. The first oil to be marketed originated in Australia. It is derived from the plant – Melaluca, while New Zealand tea tree oil - a much more recent commercial product, comes from a different plant – Leptospermum. The oils produced from each are dramatically different in their chemistry. To add further confusion to the matter Leptospermum also grows in a small part of Australia. It is Leptospermum that we are concerned with here, as bees in New Zealand and a few of their Australian cousins produce their honey by feasting upon Leptospermum. In Australia the honey is called Jelly Bush honey and in New Zealand it is called Manuka honey. It appears that of the many varieties of Leptospermum in Australia only one produces the therapeutic honey considered here.

This Leptospermum derived honey is now believed to have healing properties far beyond the traditional healing power of other honeys. Research in New Zealand has led to the marketing of a special honey, which is claimed to have specific curative powers. It carries an identification label specifying the strength of the antibacterial component; similar to the protective gradation applied to sun protection formulas. The reason is that the curative factor is present in uncertain quantities in any specific batch of honey produced by bees feeding upon Manuka bushes. The reasons are unclear, either the bees or the bushes are inconsistent. The product claims to provide a guarantee of anti-bacterial potency.

Interestingly the precise component present in this honey is currently unknown and the designation on the product label reflects that. The term UMF factor 15+ for example means that the Unknown Manuka Factor present in the honey is designated as level 15. Batches of honey are tested to determine anti-

bacterial potency. Manuka honey is available in a range of different potencies. The most potent is sold in a tube. The key anti-bacterial (and possibly antiviral) components in the honey which are believed to be extremely potent, have eluded researchers to date but recent research at the University of New South Wales in Australia has thrown important light on both the anti-bacterial mechanisms involved in honeys generally and on Manuka-type honeys in particular.

How does ordinary honey help wound healing?
Honey helps to keep a wound moist**.** Research in the early 1960s confirmed that wounds kept moist, heal quicker. Unfortunately a moist environment also promotes bacterial growth; thus a successful dressing material must also be anti-bacterial.

Standard honeys are mildly anti-bacterial. One reason is that they are high in sugars, which bind to water molecules denying bacteria the moisture which they need to grow. The main anti-bacterial action in honey is the result of the production of hydrogen peroxide by the action of the enzyme glucose oxidase, an enzyme which bees add to honey. Unfortunately the valuable anti-bacterial effect of hydrogen peroxide is limited by the fact that the body has an enzyme which breaks it down rapidly.

Jelly Bush and Manuka honeys
Shona Blair, a PhD student at the University of Sydney, has been researching the virtues of such honeys for some years. In a radio interview on ABC (the Australian Broadcaster) on 31 October this year, she disclosed the findings of her latest work.

She has determined that honey kills bacteria by turning off genes that "allow the bacteria to reproduce and … the honey seemed to be activating huge numbers of the bacteria's defence genes (over 100)." She believes that the honey was in effect overwhelming the bacteria by attacking it on many different levels at once.

Shona looked at many genes and concluded that "about 70% of the genes looked at were up-regulated, which meant that they were turned on more by the honey, and about 40% were down-regulated so that they were in effect made less effective by the honey."

It has also been found that the UMF factor is not broken down in the same way as hydrogen peroxide and is thus more persistently antibacterial. In addition UMF factor is claimed to be able to penetrate skin easily, thus being capable of reaching deep-seated infection. Ms Blair's research has also uncovered a variety of other likely mechanisms, which are involved.

She has found that honey stimulates white blood cells to produce cytokines, particularly interlukin-1, interlukin-6 and tumor necrosis factor. She believes that applications of honey speed up healing and reduce scarring.

Her research has included laboratory studies in which differing concentrations of honey were applied to E Coli bacteria in a culture medium. Preliminary results of her work, published in a news release by the University of Sydney in Australia, provide evidence that these special honeys may be vitally important in the fight against antibiotic resistant organisms.

She commented "I found honey diluted to one percent inhibits the growth of drug resistant staph aureus for approximately three hours, a two percent honey solution inhibits growth for five hours, three percent for 10 hours and there was no detectable growth in four percent solution over a 24-hour period."

It appears that bees and nature in general, still have a competitive edge over the medical miracles of our latest medical science.

It is quite simple to use such treatment. Honey should be applied to a wound and covered by a band aid for practical reasons. It should be replaced once a day, or more often in cases of serious or persistent injury.

The New Zealand version of the honey discussed above is marketed internationally by Comvita New Zealand and on the Internet at www.comvita.com. The standard product of uncertain potency does not have a label indicating the UMF value of the product. The special version has the designation - for example - Active UMF 15+.

Note: Some honeys are artificially heated to promote fluidity. It is best to buy honeys at a reputable health food store.

Honey is not patented – yet! It is unlikely, one hopes, that the drug industry will leap on the commercial bandwagon. In the meantime a tube of Manuka honey might be a valuable addition to your medicine cabinet.

The Diet Zoo: Does Science Provide Guidance? – Part I

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"The foolish faith in authority is the worst enemy of truth." Albert Einstein, 1901

INTRODUCTION. Dieting has a long history, initially associated with creating a desired image, e.g. the slender beauty, or dealing with a medical problem for which the conventional wisdom of the time used dietary modification as a therapy. At the turn of the century, North Americans ate lots of meat and fat, some put thick layers of lard on their bread, and many ate fresh produce only when in-season, and yet cardiovascular problems and adult onset diabetes were relatively rare, and obesity was not the subject of constant comment in the press nor regarded as an epidemic of great public health concern. The French Paradox is approximately the modern-day equivalent. A big change occurred some 30-40 years ago when both the US government and the medical and nutritional authorities took the position that all fat was unhealthy and responsible for the growing incidence of heart disease and cancer. While this view was strongly opposed by a minority of scientists, it became the true dogma by which many chose to live. The oversimplified notion that all fat is bad was deeply embedded, both among layman, the medical profession and the nutritional scientists, and a low-fat diet was accepted as the answer to many health problems. Many health care professionals and much of the general public still hold this view. The net result was the replacement of fat by carbohydrate, in many cases rapidly digested carbohydrate, which constituted a significant change in the diet of North Americans. This subject was reviewed in *International Health News* (IHN) newsletters, November 2002 through January, 2003 [1]. It is also discussed in two widely quoted articles by Gary Taubes [2,3] The Science article is currently available free at http://nasw.org/mem-maint/awards/The%20soft%20science.pdf. The New York Times Magazine article can be purchased by non-subscribers for a nominal fee from their website www.nytimes.com.

It was not until the early seventies that a contrary voice was loud enough to be heard by the general public, the voice of the late Robert Atkins, M.D., a cardiologist, who took a position diametrically opposed to The Establishment (the American Heart Association, the American Diabetes Association, the USDA, the American Medical Association, etc.) and promoted a diet high in fat and protein and low in carbohydrates with emphasis on slowly digested carbohydrates. Initially, his primary concern was weight loss. This diet flew in the face of the conventional wisdom and was vigorously and vehemently condemned by the government, the medical establishment, and nutrition experts. Atkins had to appear before a congressional committee to defend himself, as though he was some sort of public enemy. Nevertheless, his first book became a best seller, and subsequent editions have followed the same course, making books on the Atkins diet the most widely read diet guides in the history of man.

In the past decade a number of other similar low-carbohydrate diets have also become very popular. Most have been and are still labeled by their critics with the apparently intentionally derogatory term "fad diets," and this collection of diets defines one boundary of the great diet controversy that has arisen and now rages. A visit to a large book store like Boarders or Chapters or Barnes and Noble will reveal the present state of the popular diet book market, with hardcovers and paperbacks on every imaginable diet topic and philosophy, displayed typically in 10 to 20 linear feet of 6 to 8 foot high book shelves. This is the popular print version of the Diet Zoo.

The word "diet" has many connotations and a varied usage, but this review will be concerned with the meaning related mainly to changes in eating habits, either self-motivated or suggested by someone, generally a health care professional. Also, there is the very general question, *what should I eat*? Whatever the motivation, it is important to have clearly in mind the answer to the question*, why am I dieting or why am I following a particular diet philosophy?* While the following list is not complete, it covers the most common reasons.

- Overweight or obese, or avoiding these conditions.
- Bad lipid profile, e.g. high LDL cholesterol, low HDL cholesterol, high triglycerides.
- Presence of the characteristics of the metabolic syndrome or insulin resistance.
- Hypertension, and/or diabetes.

- Primary prevention of coronary heart disease (CHD), cardiovascular disease (CVD) and diabetes.
- Secondary prevention of CHD or CVD related adverse events, i.e. after a heart attack or stroke, or the existence of stable angina, transient ischemic attacks, etc.
- The desire to become more beautiful and attractive or more closely resemble some idol. Includes the extreme and pathological disorders anorexia and bulimia.
- Body building and training for sports.
- Maintenance of good health, i.e. concern that the present diet may carry risks even in the absence of any evidence from symptoms or a physical exam. For example, deciding to adopt a so-called heart-healthy diet, and thus being confronted with the question of what in fact is a heart-healthy diet.

It would seem that diets undertaken in response to a health crisis must be judged by different criteria than those embarked upon for less pressing reasons. The main theme of most diet books and magazine articles on diet would suggest that weight loss is the primary motivation, although it appears likely that many individuals embarking on such an endeavor may be totally unaware of other aspects of their health that frequently accompany being overweight or obese and also respond to dietary intervention. In fact, the implication of the above list is that specific diets can be used as a therapeutic approach to health problems that transcend or accompany being overweight or obese. The flip side is that diet may be implicated in the same health problems. This is an important and fascinating area. *For example, cardiovascular disease and diabetes are major health problems in the developed world which are now thought to be susceptible to dietary and lifestyle intervention, with the potential for sensational reductions in risk* [4,5,6,7,8].

This review will attempt to probe the basic science involved in the issues that accompany the Diet Zoo and will attempt to evaluate the evidence, which allows a judgment call on questions of benefits and risks of various dietary approaches. This review will also examine selected popular fad-diet schemes as well as current recommendations from what will continue to be called The Establishment.

METABOLISM AND WEIGHT LOSS. To metabolize essentially means "to change" and it involves chemical processes that transform the nutrients (chemical substances) derived from food to chemical substances that can be used in the multitude of chemical transformations that occur in the body. Food enters the system via the mouth, and the digestion processes occur there, in the stomach and in the intestines. It is important to realize that this digestive system is *outside* the body, being continuous and "open at both ends," and that substances in the food must pass through the walls of this system to get into the body proper. Many chemical transformations are required for digestion and absorption of food, and undigested material, including fiber, is simply eliminated, never having really been "inside" the body. Proteins, carbohydrates and fats are each processed differently in the digestive system to yield amino acids, simple sugars, and emulsified fat subunits, respectively, which pass into the body and constitute the raw materials for metabolism.

Weight loss is a very common goal of dieting. Every day we take in a certain amount of liquid and solid material which includes considerable water, both in food and consumed separately. In addition, there are gases absorbed, mainly by the lungs. This then is the mass intake. Mass is lost through breathing, sweating, urinating and defecating. The "complete burning" of food fuel or body fat and muscle to produce energy yields water, carbon dioxide, and if necessary, certain nitrogen compounds. Carbon dioxide and some water are lost through the lungs, some water leaves via sweat, the remainder of the water and various waste chemicals are lost in urine and stool. It is important to understand that when chemical reactions occur during metabolism, *mass is conserved*, and that mass loss must occur via the routes just described. We are not like nuclear reactors where mass is converted directly into energy in a closed system. Also, many chemical reactions involved in metabolism do not involve "complete burning" but simply result in the generation of other chemicals that are then used by the body for a wide variety of purposes. Thus to loose weight one must have a mass loss greater than the mass gain that comes from eating, breathing and drinking. The calorie counting view is oversimplified and ignores both the complexities of metabolism, the ability of the body to metabolically adjust to reduced calorie intake, and the fact that mass loss (weight loss) is only accomplished by breathing, sweating and by liquid and solid waste elimination. Many of the processes associated with metabolism simply generate, from the point of view of mass, equivalent masses of other chemical species, some of which must be eliminated to yield a weight loss. In fact, when these chemical

reactions have either a net heat energy gain or deficit, the energy comes almost entirely from differences in the chemical bond energies of reactants as compared to products of these reactions. Nevertheless, it is possible to incorporate these principles and generalize to the extent of having a rough, empirical number for the caloric intake required to maintain weight, based on body mass and activity or direct observation. When caloric intake falls below this level, then the deficit in energy is made up by utilizing non-food sources such as body fat and muscle, and some of the end products of the chemistry are eliminated to give one a weight loss.

The caloric intake required to maintain weight varies with body mass and activity at work and recreation. Typical values range from 2000 to 3000 calories (actually kcal) per day. The consensus opinion seems to be that eating less than 1200 calories per day is not a good idea, and that starvation diets can have serious health consequences including the loss of muscle mass, deleterious deficiencies in essential nutritional fats and potentially serious hormonal imbalances [9].

METABOLIC SYNDROME X AND INSULIN RESISTANCE. Because dietary intervention can impact both the metabolic syndrome and insulin resistance, it is important to develop some background in these and related problems. This subject has recently been discussed in an IHN research report by Hans Larsen with the title *Insulin Resistance and Diabetes.* The metabolic syndrome was recently given a new, high profile by virtue of its inclusion in the latest position paper of National Cholesterol Education Program Adult Treatment Panel (ATP-III) as a risk factor for diabetes, CHD and CVD. The ATP-III [10] definition of the presence of the metabolic syndrome is the presence of *three or more* of the following:

- Waist Circumference greater than 102 cm (M), 88 cm (F)—i.e. excess abdominal fat or an "apple shape."
- Triglycerides greater than 150 mg/dL (1.7 mmol/L)
- HDL cholesterol less than 40 mg/ dL (1.03 mmol/L)
- Hypertension. Blood Pressure equal or greater than 130 systolic, 80 diastolic mmHg
- Fasting glucose equal to or greater than 110 mg/dL (6.1 mmol/L)

Several studies indicate that this definition produces an incidence rate of about 24% in the U.S. population [11]. A similar definition by the World Health Organization produces 25% [12]. This average value is deceiving. A recent review [11] finds a strong variation of incidence with both age, body mass and ethnic background. For example, in white males, the prevalence goes from about 15% at age 30 to 40% at age 70, and with white men who are not overweight, prevalence ranges from about 7 to 10%, but if they are obese, the prevalence can reach 75%. It will be noted that the ATP-III definition includes commonly measured or observed parameters obtained during routine physical examinations. It is also important to recognize that there are individuals who are obese but metabolically healthy, i.e. they have none of the attributes of the metabolic syndrome aside from obesity. It may be important to identify this group to avoid inappropriate treatment, to lessen the psychological impact of obesity in these individuals, and to avoid confounding in research studies [13]. However, as discussed below, the health risks associated with obesity may go beyond those related to the metabolic syndrome.

The metabolic syndrome, as defined above, is the latest incarnation of numerous descriptions of a collection of clinical attributes that, according to many studies, lead to greatly increased risks of heart disease and diabetes. Dr. Gerald Reaven [14] of Stanford University is acknowledged as the ground breaker in this fascinating area. The syndrome has also been called Syndrome X, the Metabolic Syndrome X, the Dismetabolic syndrome, and the Deadly Quartet, to name the principal variations on this theme. The importance lies in its association with a large increase in the risk of CHD, CVD, and diabetes. The presence of four or five of the above attributes further increases the risk. It is of interest that LDL cholesterol levels are not included in the ATP-III definition. Part of the reason is that the ATP-III committee is making the metabolic syndrome an independent risk factor over and above elevated LDL. It is interesting in this context that there are those who consider that an elevated triglyceride level coupled with a low HDL cholesterol level may confer at least as great a risk of CHD as an elevated LDL level alone [15,16]. The bottom line is simply that the metabolic syndrome is shockingly prevalent, appears to confer serious risk of CHD and diabetes, and should be taken very seriously by both individuals and their physicians. The relevance to this review is that the metabolic syndrome can be avoided or reversed in many individuals by diet and lifestyle [14].

When the fasting blood glucose level is between 110 and 125 mg/dL (6.1-7.0 mmol/L), the condition is called impaired fasting glucose (IFG). Closely related to IFG is impaired glucose tolerance (IGT). Glucose tolerance is determined by measuring the serum glucose level after giving a fasting patient a drink containing 75 g of glucose, the so-called glucose challenge. At two hours, a blood glucose level of 140-199 mg/dL (7.8-11.0 mmol/L) is regarded as evidence of IGT. In all prevalence studies up to 2002, only half or less of the people with IFG have IGT, and an even lower proportion (20-30%) with IGT have IFG. It is argued that IGT and IFT represent different metabolic disturbances [17], but this is still not clear. The glucose tolerance test, also called the oral glucose tolerance test (OGTT), is not popular, presumably because it requires the patient to wait or revisit the office or clinical lab. Those in favor of the test feel it provides valuable additional information over the fasting glucose measurement [18], whereas those opposed [19] feel that combining the fasting glucose measurement with other clinical observations [20] allows the physician to adequately assess the risks of heart disease and diabetes without "inconveniencing" the patient! However, there is also evidence the OGTT is a better predictor of deaths from all causes as well as CVD as compared to fasting glucose values [21]. The reluctance to do an OGTT is curious considering the serious nature of the problems being investigated. IFG and/or IGT, even as isolated conditions, should be considered warning signals, taken very seriously, and hopefully the recognition would stimulate lifestyle and diet modifications.

Individuals who have IFG or IGT may or may not have what is called insulin resistance, a malfunctioning of the insulin-glucose system wherein abnormally large amounts of insulin are required to maintain blood glucose under control both fasting and after eating. Reaven [14] considers insulin resistance to be at the root of the problems associated with the metabolic syndrome, and *to be almost always present when patients present with high triglycerides, low HDL cholesterol, and abdominal obesity*. Either the fasting or post glucose challenge insulin level can provide some indication of the presence of insulin resistance. Reaven et al [22] recently reported that an abnormally high fasting or two-hour post glucose challenge serum insulin levels are the best simple clinical indicators of insulin resistance. However, Reaven holds the position that no test easily used in normal clinical practice at present is really satisfactory for demonstrating the presence of insulin resistance, mainly due to the lack of standardization of serum insulin assays [23].

In view of the close connection between the characteristic risk factors of diabetes and cardiovascular disease, as formalized in the concept of the metabolic syndrome, the hypothesis that diabetes and cardiovascular disease arise from a "common soil" has become attractive, i.e. adult onset diabetes should perhaps be viewed basically as a *vascular disease.* This point was recently made by Harvard's Frank Hu and Mier Stampfer in an editorial [24] concerning a study by Hunt et al [25] that linked the development of type 2 diabetes and the development and progression of atherosclerosis (as measured by elevated carotid artery intima-media thickness levels). This may well be a turning point in the matter of thinking about and preventing type 2 diabetes, since it emphasizes the need to reduce the risk of atherosclerosis and to treat the risk factors such as hyperlipidemia (elevated cholesterol and triglycerides) and hypertension aggressively, hopefully first with diet and lifestyle changes. A recently reported study on elevated CVD risk prior to the clinical diagnosis of type 2 diabetes also supports the "common soil" hypotheses [26]. In this follow-up study, women who went on to develop type 2 diabetes had a relative risk of 3.75 (3.17 after adjusting for BMI, smoking and other CVD risk factors) for a heart attack and 2.30 for stroke in the period *prior* to the diabetes diagnosis, as compared to women who did not go on to develop diabetes.

WHY DO PEOPLE BECOME OVERWEIGHT AND OBESE? The answer depends on to whom one listens. Being overweight, incidentally, is generally defined as having a Body Mass Index (BMI) of between 25 and 30, whereas obesity involves a BMI of greater than 30. Thus the healthy range is generally considered to be below 25, although very low numbers, especially if they are decreasing, should prompt an investigation of pathological causes. A low BMI can also be related to smoking which dulls the appetite in some individuals. BMI is calculated by multiplying 703 (unit conversion factor) times weight in pounds and dividing by the square of the height in inches (kg/m^2). Today it has largely replaced older "ideal weight" tables both in clinical practice and research.

The conventional explanation for being overweight or obese is that simply not enough exercise or physical and metabolic activity occurs to compensate for the calories consumed, i.e. more calories are consumed than are required to maintain

normal weight for a given level of physical activity. For any given level of physical activity, if more calories are consumed than needed, then the excess is stored as fat. Consumption of high levels of rapidly digested carbohydrates can produce high transient levels of insulin, and these high levels of insulin are thought to facilitate the storage of fat from carbohydrates converted into fat. This also fits in well with the theory [27] that the increase in overweight and obesity in the past 20-30 years is due in part to the replacement of dietary fat by sugar and starchy foods.

However, there can be little doubt that the question posed at the beginning of this section is far from simple. Consider the so-called French Paradox. The French do not have the overweight or obesity problem nearly to the extent seen in North Americans. Their incidence of heart disease is dramatically lower (about one-third) than ours. They eat baguettes, greasy croissants, liver pate, butter, cheese, lots of olive oil, etc., i.e. lots of fat. Low-fat or fat-free foods are almost unknown on French grocery store shelves. It is estimated that they get 35-45% of their calories from fat. Their obesity rate is about 8% and constant compared to 30% and growing in North America. One company in the US that specializes in oversized coffins to accommodate the obese reports a 20% annual growth rate in sales (National Post, September 28)! Needless to say, this paradox has attracted considerable attention over a number of years. The French Paradox obviously goes against the thesis that fat makes one fat, which was discussed at in a review in IHN [1] with particular reference to the views and research of Dr. Walter Willett, who regards this notion as a fallacy [28,29]. Foreyt and Poston have recently presented a "symposium consensus " view on this subject [30] which emphasized the role of lifestyle, excessive caloric consumption from all sources, and decreased physical activity as causes of obesity.

A very interesting view of the French Paradox is presented by Dr. Will Clower in the new book *The Fat Fallacy* [31]. Clower is a neurophysiologist on the faculty of the University of Pittsburgh. He spent two years as a research fellow in Lyon, France and availed himself of the opportunity to study in an informal way the French Paradox. The French have radically different eating habits as compared to North Americans. They eat smaller portions of a greater variety of food. Their meals tend to be, whenever possible, special occasions and extend, whenever time permits, over a much greater period of time than do ours. They take small bites, and rarely stuff their mouths' as do many North Americans. They prepare, eat and enjoy food with almost religious fervor. The idea of heaping plates and a quick, gobble-it-down-and-run meal are foreign to their eating philosophy. The French also have salad after the main meal. When there is vinegar in the dressing, this tends to slow down digestion, as does the cheese that generally follows. Desserts, if offered, are generally small and may well consist of fruit. Their meat consumption is lower than ours and their use of olive oil much greater. Between-meal snacking is uncommon, as is the heavy consumption of the standard fare of what we find in the local doughnut shop or they find in their *boulangerie-pâtisserie*, and their pastries incidentally tend to be very pricey. The consumption of what Cower calls "faux-foods", e.g. highly processed foods from the super market freezer, canned goods shelves or in the form of ready-to-eat dinners, is much lower. The drinking of wine with meals is almost universal, and starts at a fairly early age. Food is generally prepared with great care and much attention to flavor, taste and presentation. With many cooks, it comes close to being an art form. While there are fast food restaurants in France, they do not represent a significant aspect in French eating habits.

An interesting and popular theory regarding the tendency to become obese or overweight, the "thrifty genotype" hypotheses, holds that many individuals are very efficient at metabolic pathways that lead to the storage of fat during times of feasting, since this would have had a big survival advantage tens of thousands of years ago when humans had to endure long periods of food shortages. We have essentially the same genetic makeup. Today the situation is one of feast rather than famine, every day, at least in North America, and this feast now has large portion sizes, low-fat foods, unknown until very recently, loaded with refined carbs, a fast-food industry making a big play on "super sizing," and all-you-can-eat buffets that are the rage.

Finally there are familial and genetic predispositions to obesity, as well as endocrine causes. The latter include Cushing's syndrome, and some hypothalamic tumors, and as well, hypothyroidism can cause weight gain by reducing the metabolic rate. One would hope that physicians dealing with patients presenting with obesity problems would examine these possibilities.

Thus stability of body weight or weight gain appears to involve a complex set of factors, such as the

amount of daily exercise, the amount of food eaten and how it is eaten, the nature and content of meals and snacks, the amount of food that is required to produce satisfaction, the rate of return of hunger and the urge to eat again, and restraint regarding the portions offered or available. Willett's recent book *Eat, Drink and be Healthy* (Chapter 3) contains an interesting and relevant review of this subject [27].

BEING OVERWEIGHT OR OBESE CARRIES SERIOUS RISKS. The number of Americans that are overweight or obese has rapidly increased in the last two decades from 47% to about 60% of the population. Below are summarized some of the unfavorable health aspects associated with obesity or being overweight.

- The risk of death from all causes increases throughout the range of moderate to severe overweight for both men and women in all age groups. The risk associated with a high BMI is greater for white as compared to black individuals [32]. Many attempts to correlate mortality with BMI show a U shaped curve, but the interpretation of the increased risk at *low* BMI is subject to much debate, is related to the inclusion of smokers, and is difficult to correct for long term effects of diseases that cause wasting and are ultimately fatal [33]. This also causes a problem when attempts are made to establish guidelines for a "healthy weight" [33].
- Increased body weight is associated with increased death rates from all cancers combined and for cancers at specific sites, in particular esophagus, colon and rectum, liver, gallbladder, pancreas, kidney, non-Hodgkin's lymphoma and multiple myeloma. Significant trends of risk and higher BMI were seen for death from stomach and prostate cancer in men and breast, uterus, cervix and ovary cancer in women [34]. Thus avoidance of weight gain appears to be an important factor in cancer prevention [35].
- Being elderly, female and overweight carries an enhanced risk of Alzheimer's disease, according to a study published in 2003 [36].
- An interesting study just published indicates that abdominal obesity is an independent and potent risk factor for ischemic stroke in all race-ethnic groups, and is a stronger risk factor than BMI. Also a greater effect was observed among younger persons [37].
- It has been theorized that insulin resistance and obesity are involved in a vicious circle, where once obesity occurs, basal lipolysis in the enlarged fat mass causes increased free fatty acid levels which tend to increase insulin resistance and hyperinsulinemia, and the hyperinsulinemia ultimately leads to fat storage and more obesity, and thus the cycle continues [38]. Unless steps are taken early to halt the weight increase and abdominal fat deposition, the patient may be "swept away in the morbidity and mortality of the metabolic syndrome" [38].
- There seems little question that obesity is related to an elevated risk of hypertension, elevated plasma insulin and insulin resistance, adult-onset diabetes, and unfavorable blood lipid profiles, with the end result of increased risk of CVD as well as non-CVD complications associated with diabetes [39]. There is also growing evidence that emerging risk factors—insulin resistance, a proinflammatory state, and a prothrombotic state are common in obese persons and are independent risk factors for the development of atherosclerosis [40].

This is by no means a complete listing of the suspected or documented adverse aspects of being overweight or obese. For example, the adverse effects on joint health have been omitted. Also, space does not permit a detailed discussion of the influence of gender, age, or ethnic origin on the risks of obesity or being overweight, but there are definitely substantial differences observed. When risk factors are examined as a function of BMI, the relationship is frequently an increasing linear relationship, or one that curves upward, with increased risk having a threshold in what is considered the upper limit of the normal range, i.e. about 23-25 kg/m^2 [33,34]. Thus adjusting diet to halt weight gain at its onset is easily justified. While with the obese, weight reduction to achieve a BMI in the normal range is in many cases unrealistic, studies consistently indicate substantial declines in risk for many health problems with only a 10 or 15% decrease in BMI, even though the patient is still overweight or obese [33].

The implications are clear. The percentage of the population in North America that is overweight or obese is already very high and is growing at an alarming rate. Thus far the recommendations of The Establishment, followed by large numbers of individuals for several decades, have not resulted in a change in the bleak picture. Americans were told to decrease their fat consumption, which they did (as a percentage of total calories), but they only collectively got fatter [2,27]! This is clearly a crisis situation, and as we will see below, The

Establishment is slowly changing its position. But unless dramatic changes occur in actual eating habits among the general public, some of whom have become highly skeptical or cynical regarding flip-flops in Establishment dietary recommendations, the implications as regards the coming impact on the heath care systems, the health care insurance industry, and the taxpayer are almost beyond comprehension. The situation is especially critical because of the impact of the obesity epidemic includes major areas of illness, i.e. cancer, CVD, and diabetes. The name of the game, so to speak, is to avoid or delay the onset of CVD, diabetes and other health problems by not being overweight or obese. Clearly, the related metabolic syndrome is something to avoid like the plague. There is considerable evidence suggesting that diet can play an essential role in avoiding the metabolic syndrome or at least significantly reducing the magnitude of the problems that characterize it, and insulin resistance appears to be reversible if caught early enough [14,9]. Interested readers may also wish to consult the book by Challem et al [41] for more information on reversing insulin resistance.

While this review will not discuss such lifestyle aspects as exercise and stress reduction, these must be very seriously considered along with diet in context of achieving or maintaining health. The role of exercise in both weight control and weight reduction cannot be overemphasized. Recent studies suggest that between 50% and 70% reductions in the incidence of heart disease and diabetes can be achieved by *diet and lifestyle* alone [4,5,6,7,8]. By normal standards of risk reduction, these numbers are sensational.

DIET AND CANCER IN GENERAL. Clearly an important question when judging diets. It is a difficult area since prospective cohort and case-control studies frequently disagree. This was discussed in the IHN review [1] in connection with cancer and fat. In terms of positive risk factors, large prospective studies have not supported the role of dietary fat. Instead, according to a recent review by Willett [42], positive energy balance, reflected in early age for the onset of menstruation and weight gain as an adult, appear to be the important determinants of the risk of both colon and breast cancer. Likewise, lack of physical activity has been shown to be associated with positive risk for these diseases. Thus diet enters indirectly when it contributes to being overweight or obese. In terms of decreasing risk, the role of fruits and vegetables appears to be overstated [42], but Willett emphasizes the potential role of folic acid in connection with both breast and colon cancer. The effects of folic acid consumption appear strongest among persons who regularly consume alcohol, which itself is associated with a risk of these cancers. A recent study by Terry et al [43] found from a large prospective study that individuals who consumed *very low* amounts of fruit and vegetables had an enhanced risk of colorectal cancer. The connection between dairy products and prostate cancer appears to be absent, but there is a modest risk associated with a large intake of calcium >2000 mg/day vs. <700 mg/day [44]. Willett's review ends with the following conclusions which are also similar to those given in his book [27]:

- Weight control and regular physical activity should be high priorities for the prevention of cancer.
- Decisions about dietary fat should be made primarily on it relationship with CVD, not cancer. The 2002 review by Moyad [45] takes essentially the same position.
- Consuming an abundance of fruits and vegetables and eating grains in a minimally processed, high fiber form is desirable, but the benefits appear greater for CVD than for cancer.
- Adequate folic acid intake appears important in reducing cancer risk, particularly if alcohol is consumed regularly. Taking a multiple vitamin is the most reliable approach.
- Aside from the above, one must wait for the results of 30 large prospective studies that are underway in order to form additional significant conclusions regarding diet and cancer.

Specific reference will be made in Part II to recent studies that also bear on this subject in the context of either fat and red meat or carbohydrate.

Part II of this review will explore some of the information from scientific studies that relates to fat, carbohydrate and protein consumption, with emphasis on questions of the impact of choices regarding the nature, quantity and quality of these macronutrients on health. Part III will discuss both the so-called fad-diets and the current Establishment diet recommendations, using the information from Part II as a guide.

References

[1] Ware, W. R., *Dietary fat and heart disease. Is there a connection?.* International Health News, Hans Larsen, Editor, 2002.

[2] Taubes, G., "Nutrition. The soft science of dietary fat," *Science*, vol. 291, no. 5513, pp. 2536-2545, Mar.2001.

[3] Taubes, G. What If It Has Been a Big Fat Lie? New York Times Magazine [July 7, 2002]..

[4] Hu, F. B. and Willett, W. C., "Optimal diets for prevention of coronary heart disease," *JAMA*, vol. 288, no. 20, pp. 2569-2578, Nov.2002.

[5] Knowler, W. C., Barrett-Connor, E., Fowler, S. E., Hamman, R. F., Lachin, J. M., Walker, E. A., and Nathan, D. M., "Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin," *The New England Journal of Medicine*, vol. 346, no. 6, pp. 393-403, Feb.2002.

[6] Tuomilehto, J., Lindstrom, J., Eriksson, J. G., Valle, T. T., Hamalainen, H., Ilanne-Parikka, P., Keinanen-Kiukaanniemi, S., Laakso, M., Louheranta, A., Rastas, M., Salminen, V., and Uusitupa, M., "Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance," *The New England Journal of Medicine*, vol. 344, no. 18, pp. 1343-1350, May2001.

[7] Hu, F. B., Manson, J. E., Stampfer, M. J., Colditz, G., Liu, S., Solomon, C. G., and Willett, W. C., "Diet, lifestyle, and the risk of type 2 diabetes mellitus in women," *The New England Journal of Medicine*, vol. 345, no. 11, pp. 790-797, Sept.2001.

[8] Stampfer, M. J., Hu, F. B., Manson, J. E., Rimm, E. B., and Willett, W. C., "Primary prevention of coronary heart disease in women through diet and lifestyle," *The New England Journal of Medicine*, vol. 343, no. 1, pp. 16-22, July2000.

[9] Schwarzbein, D. D. N., *The Schwarzbein Principle* Deerfield Beach. FL: Health Communications, Inc., 1999.

[10] "Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III)," *JAMA*, vol. 285, no. 19, pp. 2486-2497, May2001.

[11] Park, Y. W., Zhu, S., Palaniappan, L., Heshka, S., Carnethon, M. R., and Heymsfield, S. B., "The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994," *Arch.Intern.Med*, vol. 163, no. 4, pp. 427-436, Feb.2003.

[12] Ford, E. S. and Giles, W. H., "A comparison of the prevalence of the metabolic syndrome using two proposed definitions," *Diabetes Care*, vol. 26, no. 3, pp. 575-581, Mar.2003.

[13] Sims, E. A., "Are there persons who are obese, but metabolically healthy?," *Metabolism*, vol. 50, no. 12, pp. 1499-1504, Dec.2001.

[14] Reaven, G., *Syndrome X* New York: Simon & Schuster, 2000.

[15] Jeppesen, J., Hein, H. O., Suadicani, P., and Gyntelberg, F., "Relation of high TG-low HDL cholesterol and LDL cholesterol to the incidence of ischemic heart disease. An 8-year follow-up in the Copenhagen Male Study," *Arterioscler.Thromb.Vasc.Biol.*, vol. 17, no. 6, pp. 1114-1120, June1997.

[16] Ballantyne, C. M., Olsson, A. G., Cook, T. J., Mercuri, M. F., Pedersen, T. R., and Kjekshus, J., "Influence of low high-density lipoprotein cholesterol and elevated triglyceride on coronary heart disease events and response to simvastatin therapy in 4S," *Circulation*, vol. 104, no. 25, pp. 3046-3051, Dec.2001.

[17] Carnevale Schianca, G. P., Rossi, A., Sainaghi, P. P., Maduli, E., and Bartoli, E., "The Significance of Impaired Fasting Glucose Versus Impaired Glucose Tolerance: Importance of insulin secretion and resistance," *Diabetes Care*, vol. 26, no. 5, pp. 1333-1337, May2003.

[18] Tuomilehto, J., "Point: a glucose tolerance test is important for clinical practice," *Diabetes Care*, vol. 25, no. 10, pp. 1880-1882, Oct.2002.

[19] Davidson, M. B., "Counterpoint: the oral glucose tolerance test is superfluous," *Diabetes Care*, vol. 25, no. 10, pp. 1883-1885, Oct.2002.

[20] Stern, M. P., Fatehi, P., Williams, K., and Haffner, S. M., "Predicting future cardiovascular disease: do we need the oral glucose tolerance test?," *Diabetes Care*, vol. 25, no. 10, pp. 1851-1856, Oct.2002.

[21] "Glucose tolerance and cardiovascular mortality: comparison of fasting and 2-hour diagnostic criteria," *Arch.Intern.Med*, vol. 161, no. 3, pp. 397-405, Feb.2001.

[22] Tuan, C. Y., Abbasi, F., Lamendola, C., McLaughlin, T., and Reaven, G., "Usefulness of plasma glucose and insulin concentrations in identifying patients with insulin resistance," *Am J Cardiol.*, vol. 92, no. 5, pp. 606-610, Sept.2003.

[23] McLaughlin, T. L. and Reaven, G. M., "Beyond type 2 diabetes: the need for a clinically useful way to identify insulin resistance," *Am J Med*, vol. 114, no. 6, pp. 501-502, Apr.2003.

[24] Hu, F. B. and Stampfer, M. J., "Is type 2 diabetes mellitus a vascular condition?," *Arterioscler.Thromb.Vasc.Biol.*, vol. 23, no. 10, pp. 1715-1716, Oct.2003.

[25] Hunt, K. J., Williams, K., Rivera, D., O'Leary, D. H., Haffner, S. M., Stern, M. P., and Gonzalez, V. C., "Elevated Carotid Artery Intima-Media Thickness Levels in Individuals Who Subsequently Develop Type 2 Diabetes," *Arterioscler.Thromb.Vasc.Biol.*, vol. 23, no. 10, pp. 1845-1850, Oct.2003.

[26] Hu, F. B., Stampfer, M. J., Haffner, S. M., Solomon, C. G., Willett, W. C., and Manson, J. E., "Elevated risk of cardiovascular disease prior to clinical diagnosis of type 2 diabetes," *Diabetes Care*, vol. 25, no. 7, pp. 1129-1134, July2002.
[27] Willett, W. C., *Eat, Drink and Be Healthy. The Harvard Medical School Guide to Healthy Eating* New York: Fireside, 2001.
[28] Willett, W. C., "Is dietary fat a major determinant of body fat?," *American Journal of Clinical Nutrition*, vol. 67, no. 3 Suppl, pp. 556S-562S, Mar.1998.
[29] Willett, W. C. and Leibel, R. L., "Dietary fat is not a major determinant of body fat," *Am J Med*, vol. 113 Suppl 9B pp. 47S-59S, Dec.2002.
[30] Foreyt, J. P. and Poston, W. S., "Consensus view on the role of dietary fat and obesity," *Am J Med*, vol. 113 Suppl 9B pp. 60S-62S, Dec.2002.
[31] Clower, W., *The Fat Fallacy* New York: Three Rivers Press, 2003.
[32] Calle, E. E., Thun, M. J., Petrelli, J. M., Rodriguez, C., and Heath, C. W., Jr., "Body-mass index and mortality in a prospective cohort of U.S. adults," *The New England Journal of Medicine*, vol. 341, no. 15, pp. 1097-1105, Oct.1999.
[33] Willett, W. C., Dietz, W. H., and Colditz, G. A., "Guidelines for healthy weight," *The New England Journal of Medicine*, vol. 341, no. 6, pp. 427-434, Aug.1999.
[34] Calle, E. E., Rodriguez, C., Walker-Thurmond, K., and Thun, M. J., "Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults," *The New England Journal of Medicine*, vol. 348, no. 17, pp. 1625-1638, Apr.2003.
[35] Bianchini, F., Kaaks, R., and Vainio, H., "Overweight, obesity, and cancer risk," *Lancet Oncol.*, vol. 3, no. 9, pp. 565-574, Sept.2002.
[36] Gustafson, D., Rothenberg, E., Blennow, K., Steen, B., and Skoog, I., "An 18-year follow-up of overweight and risk of Alzheimer disease," *Arch.Intern.Med*, vol. 163, no. 13, pp. 1524-1528, July2003.
[37] Suk, S. H., Sacco, R. L., Boden-Albala, B., Cheun, J. F., Pittman, J. G., Elkind, M. S., and Paik, M. C., "Abdominal obesity and risk of ischemic stroke: the Northern Manhattan Stroke Study," *Stroke*, vol. 34, no. 7, pp. 1586-1592, July2003.
[38] Girod, J. P. and Brotman, D. J., "The metabolic syndrome as a vicious cycle: does obesity beget obesity?," *Med Hypotheses*, vol. 60, no. 4, pp. 584-589, Apr.2003.
[39] Kopelman, P. G., "Obesity as a medical problem," *Nature*, vol. 404, no. 6778, pp. 635-643, Apr.2000.
[40] Grundy, S. M., "Obesity, metabolic syndrome, and coronary atherosclerosis," *Circulation*, vol. 105, no. 23, pp. 2696-2698, June2002.
[41] Challem, J., Berkson, B., and Smith, M. D., *Syndrome X* New York: John Wilen & Sons, 2000.
[42] Willett, W. C., "Diet and Cancer: One View at the Start of the Millennium," *Cancer Epidemiology Biomarkers Prevention*, vol. 10, no. 1, pp. 3-8, Jan.2001.
[43] Terry, P., Giovannucci, E., Michels, K. B., Bergkvist, L., Hansen, H., Holmberg, L., and Wolk, A., "Fruit, vegetables, dietary fiber, and risk of colorectal cancer," *J Natl.Cancer Inst.*, vol. 93, no. 7, pp. 525-533, Apr.2001.
[44] Rodriguez, C., McCullough, M. L., Mondul, A. M., Jacobs, E. J., Fakhrabadi-Shokoohi, D., Giovannucci, E. L., Thun, M. J., and Calle, E. E., "Calcium, Dairy Products, and Risk of Prostate Cancer in a Prospective Cohort of United States Men," *Cancer Epidemiology Biomarkers Prevention*, vol. 12, no. 7, pp. 597-603, July2003.
[45] Moyad, M. A., "Dietary fat reduction to reduce prostate cancer risk: controlled enthusiasm, learning a lesson from breast or other cancers, and the big picture," *Urology*, vol. 59, no. 1, pp. 51-62, Apr.2002.
[46] Kerstetter, J. E., O'Brien, K. O., and Insogna, K. L., "Low protein intake: the impact on calcium and bone homeostasis in humans," *J Nutr*, vol. 133, no. 3, pp. 855S-861S, Mar.2003.
[47] Layman, D. K., Boileau, R. A., Erickson, D. J., Painter, J. E., Shiue, H., Sather, C., and Christou, D. D., "A reduced ratio of dietary carbohydrate to protein improves body composition and blood lipid profiles during weight loss in adult women," *J Nutr*, vol. 133, no. 2, pp. 411-417, Feb.2003.
[48] Roughead, Z. K., Johnson, L. K., Lykken, G. I., and Hunt, J. R., "Controlled high meat diets do not affect calcium retention or indices of bone status in healthy postmenopausal women," *J Nutr*, vol. 133, no. 4, pp. 1020-1026, Apr.2003.
[49] Layman, D. K., Shiue, H., Sather, C., Erickson, D. J., and Baum, J., "Increased dietary protein modifies glucose and insulin homeostasis in adult women during weight loss," *J Nutr*, vol. 133, no. 2, pp. 405-410, Feb.2003.
[50] Hu, F. B., Stampfer, M. J., Manson, J. E., Rimm, E., Colditz, G. A., Speizer, F. E., Hennekens, C. H., and Willett, W. C., "Dietary protein and risk of ischemic heart disease in women," *American Journal of Clinical Nutrition*, vol. 70, no. 2, pp. 221-227, Aug.1999.
[51] Farnsworth, E., Luscombe, N. D., Noakes, M., Wittert, G., Argyiou, E., and Clifton, P. M., "Effect of a high-protein, energy-restricted diet on body composition, glycemic control, and lipid concentrations in overweight and obese hyperinsulinemic men and women," *American Journal of Clinical Nutrition*, vol. 78, no. 1, pp. 31-39, July2003.

[52] Dawson-Hughes, B. and Harris, S. S., "Calcium intake influences the association of protein intake with rates of bone loss in elderly men and women," *American Journal of Clinical Nutrition*, vol. 75, no. 4, pp. 773-779, Apr.2002.
[53] Dawson-Hughes, B., "Interaction of dietary calcium and protein in bone health in humans," *J Nutr*, vol. 133, no. 3, pp. 852S-854S, Mar.2003.
[54] Rapuri, P. B., Gallagher, J. C., and Haynatzka, V., "Protein intake: effects on bone mineral density and the rate of bone loss in elderly women," *American Journal of Clinical Nutrition*, vol. 77, no. 6, pp. 1517-1525, June2003.
[55] Kerstetter, J. E., Svastisalee, C. M., Caseria, D. M., Mitnick, M. E., and Insogna, K. L., "A threshold for low-protein-diet-induced elevations in parathyroid hormone," *American Journal of Clinical Nutrition*, vol. 72, no. 1, pp. 168-173, July2000.
[56] Appel, L. J., "The effects of protein intake on blood pressure and cardiovascular disease," *Curr.Opin.Lipidol.*, vol. 14, no. 1, pp. 55-59, Feb.2003.
[57] Knight, E. L., Stampfer, M. J., Hankinson, S. E., Spiegelman, D., and Curhan, G. C., "The impact of protein intake on renal function decline in women with normal renal function or mild renal insufficiency," *Ann Intern.Med*, vol. 138, no. 6, pp. 460-467, Mar.2003.
[58] Cordain, L., *The Paleo Diet* New York: JohnWiley & Sons, 2002.
[59] Cordain, L., Miller, J. B., Eaton, S. B., Mann, N., Holt, S. H., and Speth, J. D., "Plant-animal subsistence ratios and macronutrient energy estimations in worldwide hunter-gatherer diets," *American Journal of Clinical Nutrition*, vol. 71, no. 3, pp. 682-692, Mar.2000.
[60] Fung, T. T., Willett, W. C., Stampfer, M. J., Manson, J. E., and Hu, F. B., "Dietary patterns and the risk of coronary heart disease in women," *Arch.Intern.Med*, vol. 161, no. 15, pp. 1857-1862, Aug.2001.
[61] Steward, H. L., Bethea, M. C., Andrews, S. S., and Balart, L. A., *The New Sugarbusters!* New Your: Ballantine Books, 2003.
[62] Brand-Miller, J., Wolever, T. M. S., Foster-Powell, K., and Colagiuri, S., *The New Glucose Revolution* 2003.
[63] Reaven, G. M., "Insulin resistance/compensatory hyperinsulinemia, essential hypertension, and cardiovascular disease," *J Clin Endocrinol.Metab*, vol. 88, no. 6, pp. 2399-2403, June2003.
[64] Liu, S., Willett, W. C., Stampfer, M. J., Hu, F. B., Franz, M., Sampson, L., Hennekens, C. H., and Manson, J. E., "A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women," *American Journal of Clinical Nutrition*, vol. 71, no. 6, pp. 1455-1461, June2000.
[65] Liu, S. and Willett, W. C., "Dietary glycemic load and atherothrombotic risk," *Curr.Atheroscler.Rep.*, vol. 4, no. 6, pp. 454-461, Nov.2002.
[66] Brand-Miller, J. C., "Glycemic load and chronic disease," *Nutr Rev.*, vol. 61, no. 5 Pt 2, pp. S49-S55, May2003.
[67] Franceschi, S., Dal Maso, L., Augustin, L., Negri, E., Parpinel, M., Boyle, P., Jenkins, D. J., and La Vecchia, C., "Dietary glycemic load and colorectal cancer risk," *Ann Oncol.*, vol. 12, no. 2, pp. 173-178, Feb.2001.
[68] Augustin, L. S., Dal Maso, L., La Vecchia, C., Parpinel, M., Negri, E., Vaccarella, S., Kendall, C. W., Jenkins, D. J., and Francesch, S., "Dietary glycemic index and glycemic load, and breast cancer risk: a case-control study," *Ann Oncol.*, vol. 12, no. 11, pp. 1533-1538, Nov.2001.
[69] Augustin, L. S., Gallus, S., Bosetti, C., Levi, F., Negri, E., Franceschi, S., Dal Maso, L., Jenkins, D. J., Kendall, C. W., and La Vecchia, C., "Glycemic index and glycemic load in endometrial cancer," *Int.J Cancer*, vol. 105, no. 3, pp. 404-407, June2003.
[70] Jonas, C. R., McCullough, M. L., Teras, L. R., Walker-Thurmond, K. A., Thun, M. J., and Calle, E. E., "Dietary glycemic index, glycemic load, and risk of incident breast cancer in postmenopausal women," *Cancer Epidemiol.Biomarkers Prev.*, vol. 12, no. 6, pp. 573-577, June2003.
[71] Terry, P. D., Jain, M., Miller, A. B., Howe, G. R., and Rohan, T. E., "Glycemic load, carbohydrate intake, and risk of colorectal cancer in women: a prospective cohort study," *J Natl.Cancer Inst.*, vol. 95, no. 12, pp. 914-916, June2003.
[72] Michaud, D. S., Liu, S., Giovannucci, E., Willett, W. C., Colditz, G. A., and Fuchs, C. S., "Dietary sugar, glycemic load, and pancreatic cancer risk in a prospective study," *J Natl.Cancer Inst.*, vol. 94, no. 17, pp. 1293-1300, Sept.2002.
[73] Abbasi, F., McLaughlin, T., Lamendola, C., Kim, H. S., Tanaka, A., Wang, T., Nakajima, K., and Reaven, G. M., "High carbohydrate diets, triglyceride-rich lipoproteins, and coronary heart disease risk," *Am J Cardiol.*, vol. 85, no. 1, pp. 45-48, Jan.2000.
[74] Marlett, J. A., McBurney, M. I., and Slavin, J. L., "Position of the American Dietetic Association: health implications of dietary fiber," *J Am Diet.Assoc.*, vol. 102, no. 7, pp. 993-1000, July2002.
[75] Fuchs, C. S., Giovannucci, E. L., Colditz, G. A., Hunter, D. J., Stampfer, M. J., Rosner, B., Speizer, F. E., and Willett, W. C., "Dietary Fiber and the Risk of Colorectal Cancer and Adenoma in Women," *The New England Journal of Medicine*, vol. 340, no. 3, pp. 169-176, Jan.1999.
[76] Montonen, J., Knekt, P., Jarvinen, R., Aromaa, A., and Reunanen, A., "Whole-grain and fiber intake and the incidence of type 2 diabetes," *American Journal of Clinical Nutrition*, vol. 77, no. 3, pp. 622-629, Mar.2003.
[77] Bazzano, L. A., He, J., Ogden, L. G., Loria, C. M., and Whelton, P. K., "Dietary Fiber Intake and Reduced Risk of Coronary Heart Disease in US Men and Women: The National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study," *Archives of Internal Medicine*, vol. 163, no. 16, pp. 1897-1904, Sept.2003.

[78] Liu, S., Buring, J. E., Sesso, H. D., Rimm, E. B., Willett, W. C., and Manson, J. E., "A prospective study of dietary fiber intake and risk of cardiovascular disease among women," *J Am Coll Cardiol.*, vol. 39, no. 1, pp. 49-56, Jan.2002.
[79] Rosamond, W. D., "Dietary fiber and prevention of cardiovascular disease," *J Am Coll Cardiol.*, vol. 39, no. 1, pp. 57-59, Jan.2002.
[80] Hu, F. B., Manson, J. E., and Willett, W. C., "Types of dietary fat and risk of coronary heart disease: a critical review," *J Am Coll Nutr*, vol. 20, no. 1, pp. 5-19, Feb.2001.
[81] Austin, M. A., Hokanson, J. E., and Edwards, K. L., "Hypertriglyceridemia as a cardiovascular risk factor," *Am J Cardiol.*, vol. 81, no. 4A, pp. 7B-12B, Feb.1998.
[82] Assmann, G., Schulte, H., and von Eckardstein, A., "Hypertriglyceridemia and elevated lipoprotein(a) are risk factors for major coronary events in middle-aged men," *Am J Cardiol.*, vol. 77, no. 14, pp. 1179-1184, June1996.
[83] Fruchart, J. C. and Duriez, P., "HDL and triglyceride as therapeutic targets," *Curr.Opin.Lipidol.*, vol. 13, no. 6, pp. 605-616, Dec.2002.
[84] Rosenson, R. S., Shott, S., and Tangney, C. C., "Hypertriglyceridemia is associated with an elevated blood viscosity Rosenson: triglycerides and blood viscosity," *Atherosclerosis*, vol. 161, no. 2, pp. 433-439, Apr.2002.
[85] Stavenow, L. and Kjellstrom, T., "Influence of serum triglyceride levels on the risk for myocardial infarction in 12,510 middle aged males: interaction with serum cholesterol," *Atherosclerosis*, vol. 147, no. 2, pp. 243-247, Dec.1999.
[86] Hellerstein, M. K., "Carbohydrate-induced hypertriglyceridemia: modifying factors and implications for cardiovascular risk," *Curr.Opin.Lipidol.*, vol. 13, no. 1, pp. 33-40, Feb.2002.
[87] de Lorgeril, M., Renaud, S., Mamelle, N., Salen, P., Martin, J. L., Monjaud, I., Guidollet, J., Touboul, P., and Delaye, J., "Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease," *Lancet*, vol. 343, no. 8911, pp. 1454-1459, June1994.
[88] He, K., Merchant, A., Rimm, E. B., Rosner, B. A., Stampfer, M. J., Willett, W. C., and Ascherio, A., "Dietary fat intake and risk of stroke in male US healthcare professionals: 14 year prospective cohort study," *BMJ*, vol. 327, no. 7418, pp. 777-782, Oct.2003.
[89] Hung, T., Sievenpiper, J. L., Marchie, A., Kendall, C. W., and Jenkins, D. J., "Fat versus carbohydrate in insulin resistance, obesity, diabetes and cardiovascular disease," *Curr.Opin.Clin Nutr Metab Care*, vol. 6, no. 2, pp. 165-176, Mar.2003.
[90] Sacks, F. M. and Katan, M., "Randomized clinical trials on the effects of dietary fat and carbohydrate on plasma lipoproteins and cardiovascular disease," *Am J Med*, vol. 113 Suppl 9B pp. 13S-24S, Dec.2002.
[91] Ascherio, A., "Epidemiologic studies on dietary fats and coronary heart disease," *Am J Med*, vol. 113 Suppl 9B pp. 9S-12S, Dec.2002.
[92] Bernstein, R. K., *Dr. Bernstein's Diabetes Solution* New York: Little, Brown and Company, 2003.
[93] Kushi, L. and Giovannucci, E., "Dietary fat and cancer," *Am J Med*, vol. 113 Suppl 9B pp. 63S-70S, Dec.2002.
[94] Smith-Warner, S. A., Spiegelman, D., Adami, H. O., Beeson, W. L., van den Brandt, P. A., Folsom, A. R., Fraser, G. E., Freudenheim, J. L., Goldbohm, R. A., Graham, S., Kushi, L. H., Miller, A. B., Rohan, T. E., Speizer, F. E., Toniolo, P., Willett, W. C., Wolk, A., Zeleniuch-Jacquotte, A., and Hunter, D. J., "Types of dietary fat and breast cancer: a pooled analysis of cohort studies," *Int.J Cancer*, vol. 92, no. 5, pp. 767-774, June2001.
[95] Cho, E., Spiegelman, D., Hunter, D. J., Chen, W. Y., Stampfer, M. J., Colditz, G. A., and Willett, W. C., "Premenopausal fat intake and risk of breast cancer," *J Natl.Cancer Inst.*, vol. 95, no. 14, pp. 1079-1085, July2003.
[96] Hill, M., "Meat, cancer and dietary advice to the public," *Eur.J Clin Nutr*, vol. 56 Suppl 1 pp. S36-S41, Mar.2002.
[97] Norat, T. and Riboli, E., "Dairy products and colorectal cancer. A review of possible mechanisms and epidemiological evidence," *Eur.J Clin Nutr*, vol. 57, no. 1, pp. 1-17, Jan.2003.
[98] Salmeron, J., Hu, F. B., Manson, J. E., Stampfer, M. J., Colditz, G. A., Rimm, E. B., and Willett, W. C., "Dietary fat intake and risk of type 2 diabetes in women," *American Journal of Clinical Nutrition*, vol. 73, no. 6, pp. 1019-1026, June2001.
[99] van Dam, R. M., Willett, W. C., Rimm, E. B., Stampfer, M. J., and Hu, F. B., "Dietary fat and meat intake in relation to risk of type 2 diabetes in men," *Diabetes Care*, vol. 25, no. 3, pp. 417-424, Mar.2002.
[100] Schulze, M. B., Manson, J. E., Willett, W. C., and Hu, F. B. Processed meat intake and incidence of Type 2 diabetes in younger and middle-aged women. Diabetologia [October, 2003].
[101] Hu, F. B., "Dietary pattern analysis: a new direction in nutritional epidemiology," *Curr.Opin.Lipidol.*, vol. 13, no. 1, pp. 3-9, Feb.2002.
[102] Kris-Etherton, P. M., Etherton, T. D., Carlson, J., and Gardner, C., "Recent discoveries in inclusive food-based approaches and dietary patterns for reduction in risk for cardiovascular disease," *Curr.Opin.Lipidol.*, vol. 13, no. 4, pp. 397-407, Aug.2002.
[103] Hu, F. B., Rimm, E. B., Stampfer, M. J., Ascherio, A., Spiegelman, D., and Willett, W. C., "Prospective study of major dietary patterns and risk of coronary heart disease in men," *American Journal of Clinical Nutrition*, vol. 72, no. 4, pp. 912-921, Oct.2000.

[104] Terry, P., Suzuki, R., Hu, F. B., and Wolk, A., "A Prospective Study of Major Dietary Patterns and the Risk of Breast Cancer," *Cancer Epidemiology Biomarkers Prevention*, vol. 10, no. 12, pp. 1281-1285, Dec.2001.
[105] Missmer, S. A., Smith-Warner, S. A., Spiegelman, D., Yaun, S. S., Adami, H. O., Beeson, W. L., van den Brandt, P. A., Fraser, G. E., Freudenheim, J. L., Goldbohm, R. A., Graham, S., Kushi, L. H., Miller, A. B., Potter, J. D., Rohan, T. E., Speizer, F. E., Toniolo, P., Willett, W. C., Wolk, A., Zeleniuch-Jacquotte, A., and Hunter, D. J., "Meat and dairy food consumption and breast cancer: a pooled analysis of cohort studies," *Int.J Epidemiol*, vol. 31, no. 1, pp. 78-85, Feb.2002.
[106] Fung, T., Hu, F. B., Fuchs, C., Giovannucci, E., Hunter, D. J., Stampfer, M. J., Colditz, G. A., and Willett, W. C., "Major Dietary Patterns and the Risk of Colorectal Cancer in Women," *Archives of Internal Medicine*, vol. 163, no. 3, pp. 309-314, Feb.2003.
[107] van Dam, R. M., Rimm, E. B., Willett, W. C., Stampfer, M. J., and Hu, F. B., "Dietary patterns and risk for type 2 diabetes mellitus in U.S. men," *Ann Intern.Med*, vol. 136, no. 3, pp. 201-209, Feb.2002.
[108] Fung, T. T., Rimm, E. B., Spiegelman, D., Rifai, N., Tofler, G. H., Willett, W. C., and Hu, F. B., "Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk," *American Journal of Clinical Nutrition*, vol. 73, no. 1, pp. 61-67, Jan.2001.
[109] Allan, C. B. and Lutz, W., *Life Without Bread* Los Angeles: Keats Publishing, 2000.
[110] Atkins, R. C., *Atkins for Life* New York: St. Martin's Press, 2003.
[111] Atkins, R. C., *Dr. Atkins' New Diet Revolution* New York: Harper Collins-Quill, 2002.
[112] Volek, J. S. and Westman, E. C., "Very-low-carbohydrate weight-loss diets revisited," *Cleve.Clin J Med*, vol. 69, no. 11, pp. 849, 853, 856-849, 853, 858, Nov.2002.
[113] Brehm, B. J., Seeley, R. J., Daniels, S. R., and D'Alessio, D. A., "A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women," *J Clin Endocrinol.Metab*, vol. 88, no. 4, pp. 1617-1623, Apr.2003.
[114] Foster, G. D., Wyatt, H. R., Hill, J. O., McGuckin, B. G., Brill, C., Mohammed, B. S., Szapary, P. O., Rader, D. J., Edman, J. S., and Klein, S., "A randomized trial of a low-carbohydrate diet for obesity," *The New England Journal of Medicine*, vol. 348, no. 21, pp. 2082-2090, May2003.
[115] Samaha, F. F., Iqbal, N., Seshadri, P., Chicano, K. L., Daily, D. A., McGrory, J., Williams, T., Williams, M., Gracely, E. J., and Stern, L., "A low-carbohydrate as compared with a low-fat diet in severe obesity," *The New England Journal of Medicine*, vol. 348, no. 21, pp. 2074-2081, May2003.
[116] Westman, E. C., Yancy, W. S., Edman, J. S., Tomlin, K. F., and Perkins, C. E., "Effect of 6-month adherence to a very low carbohydrate diet program," *Am J Med*, vol. 113, no. 1, pp. 30-36, July2002.
[117] Sharman, M. J., Kraemer, W. J., Love, D. M., Avery, N. G., Gomez, A. L., Scheett, T. P., and Volek, J. S., "A ketogenic diet favorably affects serum biomarkers for cardiovascular disease in normal-weight men," *J Nutr*, vol. 132, no. 7, pp. 1879-1885, July2002.
[118] Agatston, A., *The South Beach Diet* Rodale, 2003.
[119] Eades, M. R. and Eades, M. D., *The Protein Power Lifeplan* New York: Warner Books, 2003.
[120] Eades, M. R. and Eades, M. D., *The 30-Day Low-Carb Diet Solution* New York: John Wiley & Sons, 2003.
[121] Sinatra, S. T., Sinarta, J., and Lieberman, R. J., *Heart Sense for Women* New York: Penguin, 2000.
[122] Pearson, T. A., Blair, S. N., Daniels, S. R., Eckel, R. H., Fair, J. M., Fortmann, S. P., Franklin, B. A., Goldstein, L. B., Greenland, P., Grundy, S. M., Hong, Y., Miller, N. H., Lauer, R. M., Ockene, I. S., Sacco, R. L., Sallis, J. F., Jr., Smith, S. C., Jr., Stone, N. J., and Taubert, K. A., "AHA Guidelines for Primary Prevention of Cardiovascular Disease and Stroke: 2002 Update: Consensus Panel Guide to Comprehensive Risk Reduction for Adult Patients Without Coronary or Other Atherosclerotic Vascular Diseases. American Heart Association Science Advisory and Coordinating Committee," *Circulation*, vol. 106, no. 3, pp. 388-391, July2002.
[123] "Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III)," *JAMA*, vol. 285, no. 19, pp. 2486-2497, May2001.
[124] Franz, M. J., Bantle, J. P., Beebe, C. A., Brunzell, J. D., Chiasson, J. L., Garg, A., Holzmeister, L. A., Hoogwerf, B., Mayer-Davis, E., Mooradian, A. D., Purnell, J. Q., and Wheeler, M., "Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications," *Diabetes Care*, vol. 26 Suppl 1 pp. S51-S61, Jan.2003.
[125] Gifford, K. D., "Dietary fats, eating guides, and public policy: history, critique, and recommendations," *Am J Med*, vol. 113 Suppl 9B pp. 89S-106S, Dec.2002.

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Fish oil rich in omega 3 fatty acids aids in weight loss, promotes healthy skin and hair and helps to manage heart diseases. Moreover, Fish oil gives relief from depression.Â Many of the health benefits of fish oil can be attributed to the presence of omega-3 essential fatty acids like docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), according to the USDA National Nutrient Database. Other useful essential fatty acids in fish oil include alpha-linolenic acid or ALA and gamma-linolenic acid or GLA. Here is a brief on omega-3 fatty acids: There are three types of omega-3 fatty acids, namely alpha-linolenic acid (ALA), docosahexaenoic acid (DHA), and eicosapentaenoic acid (EPA). All three are important for the body. A study found that fish oil benefits the heart by counteracting the effects of mental stress in several measurements of cardiovascular health, such as heart rate and muscle sympathetic nerve activity (MSNA). In conducting the study, a team of researchers at Michigan Technological University worke.Â The olive oil served as a control as it does not provide the same cardiovascular beneficial effects as fish oil. After eight weeks of the oil interventions, the researchers conducted the same tests again on the participants. The results of the study revealed that the test results remained the same in the two groups when they were at rest. However, the team saw changes in the tests when the two groups underwent mental stress. Modern beliefs in the heart health benefits of fish oil stem from observational studies of Greenland Inuit in the 1970s, who found that their diets were extremely high in oily fish and they suffered fairly low rates of heart disease. In the years since, the American Heart Association has routinely recommended that people eat fish at least twice a week to, among other things, slow the rate of plaque growth in the arteries, lower their blood pressure a bit, and generally stay heart healthy.Â Fish oil, Kopecky said, â€œis really the standard of our treatment now for the majority of people with high triglycerides,â€ alongside increasing physical activity and lowering carb intake. Taking omega-3 fish oil supplements is often touted as a simple way to protect your heart - but experts say the evidence that it does any good is flimsy at best. Cochrane researchers looked at trials in over 100,000 people and found little proof that it prevented heart disease. They say the chance of getting any meaningful benefit from taking omega-3 is one in 1,000. Eating oily fish, however, can still be recommended as part of a healthy diet. The review mainly looked at supplements rather than omega-3 from eating fish. Experts still believe the latter is good for the heart as well as general