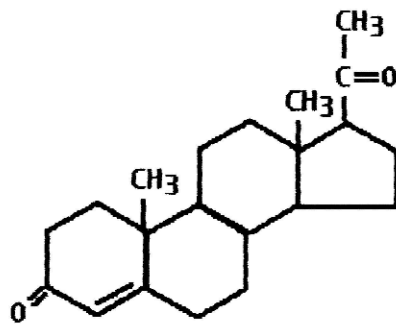


THE JOURNAL OF
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Progesterone

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PROGESTERONE

A bioidentical hormone cannot be patented, so synthetic progestagens are intentionally different from the bioidentical hormone in order to be patentable. As a consequence, such progestagens will have a somewhat different hormonal activity and are more likely to have negative side effects. Bioidentical progesterone, although apparently safer than synthetic progestagens, has not yet been well studied in long-term use. It should be considered a “naturally occurring drug” and no drug is completely safe. Women with a history of blood clots in the legs (thrombophlebitis) or liver disease should not use either bioidentical progesterone or any of the progestagens without close medical supervision.

Use of bioidentical progesterone in pregnancy also requires careful physician surveillance since added progesterone can aggravate the common complaints of pregnancy such as bloating, breast tenderness, weight gain, headache, and moodiness. Although natural progesterone tends to have fewer side effects than synthetic progestagens, either may cause worsening of some medical conditions such as asthma. During pregnancy, progesterone inhibits lactation, whether produced by the body or supplemented. The fall in progesterone levels following delivery is one of the triggers for milk production.

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CONTENTS

EDITORIAL

- Where has all the omega-3 gone? 79
James Heffley, Ph.D., CCN, DANLA

REVIEW ARTICLE

- Fish oil supplementation in people with diabetes: A review of the research 81
Gretchen K. Vannice, MS, RD and Jill Kelly, Ph.D.

COMMENTARY

- Alzheimer's Dementia, Vitamins B12 and B6, Lithium, Gingko biloba,
Dental Mercury, Genetic Risk, and Drinking Water Fluoridation 84
Joseph G. Hattersley

- LITERATURE BRIEFS** 99

- BOOK REVIEWS** 104

- Natural Alternatives to Vioxx, Celebrex & Other Anti-Inflammatory Prescription Drugs:
What to Use to Help Relieve Arthritis Pain and Inflammation
Carol Simontacchi

- We're Killing Our Kids: How to End the Epidemic of Overweight and
Sedentary Children
Todd Hollander

- BOOKS RECEIVED FOR REVIEW** 105

WHERE HAS ALL THE OMEGA-3 GONE?

There is a mounting body of evidence showing that the dietary intake of omega-3 fatty acid (OM3) in the typical American diet has declined over the decades. Evidence is also accumulating that reversing this trend, increasing the dietary intake of OM3, can decrease the risk of complications in numerous degenerative diseases, such as coronary heart disease, hypertension, diabetes, cancer, rheumatoid arthritis, inflammatory bowel disease, asthma, and depression. In addition, the role of OM3s in the regulation of gene expression adds to its importance during pregnancy and breastfeeding. If some malevolent force had decided to inflict the maximum damage to the health of Americans, it probably could not have chosen a more effective avenue of attack than to reduce our intake of OM3.

So how did it happen? It appears that the malevolent force was simply ignorance.

The first fatty acid to be labeled “essential” was linoleic acid, an omega-6 fatty acid. Once its essentiality was confirmed, for years it was generally assumed to be the only essential fatty acid, and as long as the American diet contained linoleic acid it was deemed adequate. It was not until 2002 that the Food and Nutrition Board of the Institute of Medicine established adequate dietary intake levels for OM3s and even then it was primarily for alpha-linolenic acid, although it was conceded that infants under 1 year of age also needed eicosapentaenoic acid and docosahexaenoic acid.

By then, for several reasons, the dietary intake of OM3 had been dropping for decades. The most abundant food sources of OM3 are nuts and seeds, which are typically high in total fat. A wave of fat-phobia has made nuts and seeds “politically incorrect” in some circles, even though research completely exonerates them.

Animal sources of OM3 also tend to be high in both total and saturated fat, and high saturated fat intake is generally discouraged. Animals do not synthesize OM3s, so all the OM3 supplied in animal flesh is derived from their diet. In the past Americans, perhaps by necessity, consumed more wild game that is naturally higher in OM3. Today the only “wild” food still available to most Americans is fish, which is still an acceptable source of OM3, though contaminated to some degree with mercury and pesticide residue.

Even though dark green vegetables can supply only a small amount of OM3 compared to nuts and seeds, this source of OM3 can add up, especially if concentrated by juicing. Dark green vegetables like seaweed, broccoli, spinach, kale, cabbage, Brussels sprouts, and parsley are a reasonable source of OM3 if consumed regularly. Unfortunately these vegetables are becoming rare in the American diet and few avail themselves of the benefits of juicing.

It seems less and less likely that we will see a big turnaround in the food choices Americans habitually make, so the solution to the problem of how to restore OM3 intake in the American diet is likely to be twofold: food fortification and food supplements. Increasing the OM3 in eggs by simply feeding hens a diet high in OM3 has been commercially successful. Raising domestic animals on a high OM3 diet and “finishing out” on flaxseed, a superb source of OM3, rather than grain will improve the OM3 level in their meat. Milk and other dairy products with enhanced levels of OM3 are already available.

Fish oil supplements have been available since our grandparents’ time but gained a reputation for producing an unpleasant fishy “burp.” The recent availability of “prescription grade” molec-

ularly distilled fish oil products largely eliminates this excuse for not taking fish oil supplements.

If ignorance led to our present situation in which OM3 may be the most serious deficiency in our American diet, we now have the information to reverse this long-standing dietary insuffi-

ciency and bring about vastly improved health in our country. We are extraordinarily unwise if we let this opportunity pass by.

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FISH OIL SUPPLEMENTATION IN PEOPLE WITH DIABETES: A REVIEW OF THE RESEARCH

Gretchen K. Vannice, MS, RD
Jill Kelly, Ph.D.

The *World Health Organization* and the *International Diabetes Federation* have recently reported that 3.2 million deaths a year (1) can be attributed to diabetes, a complex condition of related metabolic disorders, including dyslipidemia, as well as a high risk of cardiovascular complications. Indeed cardiovascular disease has been confirmed as the major cause of death in people with both type 1 and type 2 diabetes (2). Recently, the role of supplementation of long-chain omega-3 (ω -3) fatty acids from fish oil in the management and treatment of both insulin-dependent diabetes mellitus (type 1 DM) and non-insulin-dependent diabetes mellitus (type 2 DM) has been the subject of numerous clinical trials.

First, research shows that fish oil supplementation significantly lowers triglyceride levels. A 1998 meta-analysis of 26 trials of fish oil supplementation in people with diabetes (both type 1 and type 2) that analyzed their serum lipids and glucose tolerance showed a decrease of almost 30% in triglyceride (TG) levels (3). The authors concluded that 3 g/day (the dose most commonly used) of ω -3 fatty acids was a safe and effective dosage for lowering TG levels in people with type 2 DM. A second meta-analysis (4) in 2000 of 823 diabetic subjects (mostly type 2 men between 55–65 years of age), which included some of the same trials, found comparable results: fish oil supplementation significantly lowered triglycerides (weighted mean: -0.56mmol/l), especially in those with hypertriglyceridemia (weighted mean: -0.73 mmol/l). The authors of the 2000 review con-

cluded that fish oil supplementation is a helpful and reasonable therapeutic strategy in patients with high TG levels; in addition, they reported that in those with normal TG levels, no clinically significant effects on glycemic control were found (4).

While the triglyceride-lowering effect of fish oil supplementation is consistently found in clinical trials, the impact of these ω -3 fatty acids on LDL-cholesterol (LDL) is less clear but still promising. For example, in the 1998 review cited above, LDL levels were raised only slightly, but significantly, in type 2 DM subjects (mean change 0.20 mmol/l) (3). The findings in the 2000 review also showed an increase in LDL levels (average of 0.21mmol/l) (4). Contradicting both these findings, a related randomized, double-blind, placebo-controlled study of 51 men and women with type 2 DM taking either 4g EPA or DHA (the principal fatty acids of fish oil) or olive oil found that no significant changes in total cholesterol, LDL, or HDL occurred (5). Similarly, in an 8-week placebo-controlled trial of randomized groups of 42 subjects with type 2 DM who were administered 4g/d of fish oil or corn oil, the results showed lowered TG and raised HDL_{2b} levels but no significant effects on LDL or HDL (6). However, in a review of 36 crossover and 29 parallel studies comparing the essential fatty acids (EFAs) in flaxseed oil and fish oil, total cholesterol was not materially affected by ω -3 fatty acids but LDL tended to rise 5–10% and HDL by 1–3%, while serum TG concentrations decreased by 25–30% (7). (The authors also noted that very large amounts of

flaxseed oil were needed to reduce TG levels to the levels that fish oil did).

Fish oil also has a favorable impact on blood pressure. In a clinical trial of 19 obese, hypertensive, and dyslipidemic patients with diabetes (OHD+DM) or without (OHD-DM), a 13-day protocol of programmed fasting and fish-oil feeding (8) lowered BP in both groups, from 159/81 to 146/73 mmHg in the OHD-DM group, and 158/83 to 142/76 mm Hg in the OHD+DM group. Serum TG levels were also reduced, from 159 to 108 mg/dl in the OHD-DM group, and from 209 to 153 mg/dl in the OHD+DM group. In addition, HDL increased significantly in the OHD+DM group. A favorable reduction in hemostasis parameters (platelet aggregation) was seen among the non-diabetic patients only (8). Similarly, a meta-analysis of 36 trials of fish oil supplementation (median dose 3.7 g/d) and blood pressure in adult men and women showed that fish oil has a small hypotensive effect on BP, especially in those who are older and have hypertension. The mean duration of these trials was 11.7 weeks. The mean reductions in blood pressure were significant, with systolic being reduced by 2.1 mmHg and diastolic reduced by 1.6 mmHg (9). It has been shown that even relatively small BP reductions may reduce stroke and coronary event risk (10).

Lastly, fish oil seems to have a beneficial effect on endothelial function, either by decreasing endothelial activation or by improving endothelium-dependent vasodilation in patients who are at high risk for cardiovascular disease, including those with diabetes (11). How ω -3 fatty acids are atheroprotective is unclear; however, they appear to influence lipid concentrations, as we have seen, as well as the size and oxidizability of lipids, platelet aggregation (12), and arrhythmia (13).

All of these lipidemia factors may well explain why a prospective study of 5,103 female nurses with type 2 DM but free of cardiovascular disease at baseline showed that those diabetic

women with higher consumption of fish and fish oil supplementation had a lower rate of coronary heart disease and total mortality (14).

There has been concern that fish oil supplementation may raise blood glucose levels. However, evidence suggests that there may be no reason for concern, particularly in a risk-benefit analysis. No significant effects on blood glucose levels were seen in the 2000 meta-analysis, involving 18 trials and >800 type 2 DM subjects (4). And while the 1998 study showed small but borderline significant ($p = 0.06$) increases in fasting blood glucose levels in the type 2 DM subjects (and a lowering of fasting blood glucose levels in the type 1 DM subjects), the authors concluded that 3 g/day of ω -3 fatty acids were safe and effective for lowering TG levels in patients with diabetes (3). Furthermore, in a randomized double-blind crossover of 12 type 2 diabetic men who took 6g daily of fish oil or sunflower oil for 2 months, results showed fasting plasma insulin, glucose, and HbA_{1c} levels were similar in the two groups, and there was no increase in basal hepatic glucose production (15). In another study of 16 type 2 DM patients with hypertriglyceridemia who were randomly assigned to either fish oil (2.7g/d for 2 months, then 1.7g/d for 4 more months) or olive oil, there was a long-term significant reduction in plasma TG and VLDL-TG, a significant enrichment in the erythrocyte phospholipid content of long-chain ω -3 fatty acids, and no deterioration of blood glucose control (16). No changes in the variables were observed in the placebo (olive oil) group. The authors did note that this amount of ω -3 fatty acids was unable to improve insulin resistance in the subjects (16). And the *Italian Fish Oil Multicenter Study* (17), a randomized double-blind placebo-controlled study evaluated the possible worsening of glycemic control after fish oil supplementation in hypertriglyceridemic patients with and without glucose intolerance or diabetes. A total of 935 adult men and women were given 3 g/day (1 g, 3x/d) of ω -3 fatty acids for 2 months or placebo, then 2 g/day

(1 g, 2x/d) for 4 months or placebo. As expected, there was a significant decrease in triglycerides (21.53% at 6 months with a tendency toward a progressive reduction over time); in addition, there was no alteration in the major glycemic indexes, including fasting glucose levels. What's more, in the subgroup with diabetes, no alterations in HbA1c or serum insulin were noted. And finally, the subgroup with impaired glucose tolerance underwent oral-glucose-tolerance tests before and after the study period; no alterations were seen following the supplementation (17).

In summary, in people with diabetes, multiple human clinical trials have shown that fish oil supplementation significantly reduces triglyceride levels, improves HDL levels, reduces blood pressure by a small yet significant amount, and does not adversely affect blood glucose levels. Furthermore, fish oil supplementation, especially from purified sources, has been noted to be safe and effective by several investigators. The diverse and beneficial effect of fish oil supplementation on the many aspects of diabetes makes supplementation a promising therapeutic.

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ALZHEIMER'S DEMENTIA, VITAMINS B12 AND B6, LITHIUM, GINGKO BILOBA, DENTAL MERCURY, GENETIC RISK, AND DRINKING WATER FLUORIDATION

Introduction. An estimated 2.3 million Americans now have AD, characterized by confusion, difficulty concentrating, loss of memory, marked change of personality which can lead to outbursts of violence, hallucinations, wandering away, and early death. Prevalence doubles every five years after the age of 60, increasing from 1 percent among 60- to 64-years-old up to 40% of those aged 85 years and older. Nursing home care costs about \$47,000 per patient annually and rises steadily, a huge burden on the health-care system. The disease is also terrible for the patients' caregivers. In what experts are calling "a looming public health disaster," statistics suggest there will be between 5 and 7 million patients with Alzheimer's dementia (AD) over the next 10 years. (1)

It is interesting to consider what proportion of AD may result from undernutrition. (2) Some nutritional approaches might greatly lighten the threatened public health disaster. In this article I inform you about three safe, inexpensive, at-home nutritional methods for prevention and virtual elimination of AD. These methods do not simply lower the risk, but may completely prevent the condition or even turn it off after it starts.

The first uses vitamin B12, the second uses low-dose lithium orotate, the third uses long-term megadose vitamin B6; I present others too. The approach using B12 has the additional virtue of greatly lowering the risk of a range of mental and non-mental diseases, and also reducing violent behavior. After that I examine two very serious AD risk factors: dental mercury and fluoridated water. Finally I discuss what

needs to be done to get these simple, successful, harmless techniques into wide use.

Vitamin B12. John V. Dommissie, MD, who practices medicine in Tucson, Arizona, has confirmed (3, 4, 5) that AD appears to result from too-low serum vitamin B12, and repletion of the vitamin succeeds despite other risk factors. Full repletion of B12, i.e. restoring serum levels to or near to those found at birth, can reverse 75 percent of B12 deficiency dementias, when discovered early enough. (6) The neurological and cerebral manifestations of B12 deficiency require dosages larger, (7) and extending over a longer time, (8) than to reverse hematological effects. (9) And there is no reason to run the risk of not catching deficiency in time, or go to any expense and inconvenience. Bluntly: try the therapy and see if it helps the patient.

"The only question now," writes Dommissie, "is what proportion of cases of mood-disorder is caused by B12-deficiency and what percentage is idiopathic." Almost all of Dommissie's uni- and bipolar patients have had B12 levels in the lowest one third of the so called normal (to prevent pernicious anemia) range, levels which he now regards as deficient (for adequate affective, cognitive and other mental functions). When their levels have been raised to the highest one third of that 'normal' range, every one of those patients has done and felt better. In some cases they came out of their depression or mood-swing disorder, this was the only new or different treatment they received. When such patients' affective disorder has got worse, their B12 level had again dropped.

Mammals including humans are born with

serum levels about 2,000 pg/ml (picograms, i.e. trillionths of a gram per milliliter) of B12, which decline throughout human life owing to common practices in Western societies. (10) Below 550 to 600 pg/ml, deficiencies start to appear in the cerebrospinal fluid. (11, 12) Most cases of AD are actually missed B12 deficiency cases because of the too-low normal range for B12. (3) U.S. clinical laboratories regard 200 pg/ml as the lower range of normal. That low limit was set with hematologic (related to blood) criteria. But neuropsychiatric criteria, which are much higher, have now become more critical.

It is essential to measure serum B12, but tests for methylmalonic acid and homocysteine (13) are not necessary. Simply supplementing B12 in the dark may miss the mark badly. And to really know the patient's whole picture, the ION Panel (NEEDS 1-800-634-1380) is well worth its cost of about \$600. (14)

The normal range for serum B12, states Dr. Domisse, should be 600 to 2,000 pg/ml. Japan's "normal" range is 500–1,300 pg/ml; (15) and he says this may explain why Japan has such a low rate of AD, (16) compared to the United States. (17) By some estimates, as many as 80 percent of elderly American patients may share hidden B12 insufficiency. (18, 19, 20) B12 deficiency is common with folate deficiency in dementia, (21, 22, 23) and worsens over time as the deficiency increases. (24) The impact was seen first on neuropsychiatric measures, and augmentation of B12 and folate materially improved scores on cognitive performance tests. (25, 26)

For at-home use, it is not feasible to pump up serum levels of vitamin B12 using diet and vitamin supplements. Oral supplementation, writes world-renowned Jonathan V. Wright, MD, "usually just doesn't raise blood levels high enough to produce a therapeutic effect in individuals who experience B12 deficiency." (27) However, oral supplementation of 2 mg cyanocobalamin daily was more effective than one milligram IM monthly. After 4 months of

therapy in a test, the serum B12 had risen in the oral therapy group from 95 to 1,005 pg/ml and in the IM group from 95 only to 325 pg/ml. (28) There's nothing to prevent weekly or semiweekly IM, though, when it is done at home.

The most direct method is intramuscular injection, which requires a doctor's prescription; another feasible approach uses inexpensive sublingual B12 at 2,500–5,000 mcg (2.5 to 5 mg), which anyone can buy at a health food store. Intramuscular injection is about as difficult technically as pushing a pin into a ripe orange, (29) and IM can be very economical if a patient can self-administer or a companion can administer.

B12 deficiency can also cause:

- Depression (30, 31, 32, 33, 34, 35)
- Bipolar-1 disorder (manic depressive) and more commonly bipolar-2 (cyclothymic personality) (36, 37, 38)
- Psychotic forms of depression have been particularly associated with B12 deficiency (39) This has also been linked to paranoid psychoses (40)
- Chronic fatigue syndrome (41, 42, 43)
- Weakened immunity leading to susceptibility to cancer and recurrent infections
- Asthma
- Disrupted sleep/wake rhythms
- Environmental illness
- Low stress tolerance
- Osteoporosis
- AIDS
- Premature aging (44)
- Multiple sclerosis (45, 46)
- As well as increasing risk of cardiovascular disease, cancer (47) and much more, by hindering remethylation of the toxic sulfur amino acid homocysteine back into the nontoxic essential amino acid methionine. (48)
- IM B12 has yielded seeming miracle cures in a range of desperate illnesses. (49, 50)

And in numerous cases, when B12 was depleted, with or without other changes in life, violent behavior disappeared. (51, 52, 53, 54, 55)
WHEW!

And so, would restoring ample serum B12 levels prevent many or most of those adverse conditions? Evidence shown below, writes Dr. Dommissie, suggests the answer is “Yes, at least in the case of depression.”

Causes of B12 insufficiency. Several features of modern life accelerate the decline of B12 in serum throughout life.

Widely used microwave ovens resulted in 30 to 40 percent degradation of milk’s vitamin B12 in six minutes; with conventional heating, 25 minutes of boiling would be needed to depress B12 that much. (56) Also the heat of microwaving is certain to destroy all the enzymes in ingested food, which may be required to enable absorption and utilization of the food. Thus by microwaving much of their food, both at home and in meals “out,” tens of millions of Americans are making themselves increasingly vulnerable to AD.

B12 stores tend to be insufficient among millions who have for decades eaten vitamin- and mineral-depleted processed Western diets—which are also big sources of disease-creating free radicals. (40) Too low levels of omega-3 essential fatty acids in Western diets also must contribute to insufficient B12 levels. (57) Omega-3 supplementation may yield its benefits largely through augmenting vitamin B12. Too-low levels of acetyl-carnitine and folic acid appear to worsen risk of the condition. (58, 59)

Most commonly, B12 insufficiency results directly from *Hypochlorhydria*, (60, 61, 62) insufficient hydrochloric acid (HCl) in the stomach. The acid should be concentrated enough to dissolve a nail within an hour. (63) Hypochlorhydria is likely caused by zinc/vitamin B6 deficiency (40) and shortage of ionized calcium, (64, 65) which are typical of older people; or even achlorhydria, no HCl at all. Lack of enough pepsin or HCl in the stomach to gener-

ate the bond between B12 and its carrier protein typically shows with atrophic gastritis (66, 67). Lack of either is a risk factor for gastric cancer. (68)

B12 deficiency can also result from inadequate stomach secretion of the tiny open-ended protein capsules known as intrinsic factor, or from presence in the gut of bacterial overgrowth; (69) from ingestion of cobalamid, a B12 antagonist, (70), or failure of absorption for other reasons. (71)

Antacids, both prescribed and over-the-counter (OTC), are chronically overused by tens of millions of elderly. When all the acid is mopped up daily by antacids, “the B-vitamins never even get to first base.” (72) For example, B12 absorption is dramatically reduced when the drug Prilosec (omeprazole), which has recently gone over the counter, is used. (73, 74) “A significant percentage of patients taking omeprazole are also being treated for or are at high risk of heart disease, and therefore are almost always instructed to eat a diet low in red meat (or devoid of it completely) and of animal products, which of course are the best source of vitamin B-12.” (75) Also, omeprazole reduces gastric ascorbic acid (vitamin C) levels. (76)

This is another egregious example of iatrogenic disease created by tunnel-visioned one-organ specialists using a “Band-Aid” approach to treatment of a symptom or test reading, oblivious to the possibly disastrous long-term effect on the patient. Other causes of B12 deficiency include excessive long-term use of antibiotics and other drugs to mask symptoms without learning and correcting their cause; oral antibiotics destroy the trillions of “good” bugs in the gut as well as the “bad,” ruining absorption. (77)

Many vegan (total) vegetarians have for decades consumed few if any foods containing B12, so that their body stores of the vitamin have gradually diminished. Forty-seven of 78 adult vegan vegetarians had levels below 200 pg/ml; when they chewed a 100 ug (microgram)

B12 tablet once a week, their levels promptly rose to normal. (78) Some depend on sea vegetables such as arame, wakame and some varieties of Kombu, or on algae; (79) the B12 in these, although absorbed, may not be fully bioavailable. (80) A study in *Townsend Letter* provided strong evidence that a commonly consumed seaweed known as *nori* does in fact contain bioavailable forms of B12. (81) But whether that substance is available to large numbers of vegans and whether its use would lift serum B12 levels enough, is not known.

In an AD mouse model, a diet rich in omega-3 essential fatty acids, specifically docosahexaenoic acid (DHA) has now been shown to potentially slow or even prevent AD. (82) DHA can be ingested in fish oil or cod liver oil.

Dr. Dommissé does not reveal the proportion of his patients who are thyroid deficient, drink fluoridated water, have extensive dental amalgams, take Ibuprofen, (83) etc (some NSAIDs lower AD risk by about 50 percent (84) according to J.S. Bland in *Functional Medicine Update 2004*). He doesn't consider the number of patients who have been exposed occupationally to electromagnetic fields, through promoting formation of beta amyloid, a protein common in the brains of AD patients. (85, 86) Or, the number showing high content of aluminum in the brain (87) from consumption of aluminum-treated drinking water (88, 89) and from a variety of everyday sources (see also below on the combination of aluminum-containing alum with fluoridated water). Or the extent of use of melatonin as brain antioxidant to counteract accumulation of free radical-creating iron. (90) Or the extent of potentially brain-damaging homocysteine in patients' brains. (91, 92)

Possible mechanisms for spinal cord and peripheral nerve effects of B12 deficiency include axonal degeneration and demyelination of the insulating nerve sheaths. (93) Deana found low levels of neurotransmitters in the brains of B12-deficient rats. (94) A University of Kentucky study found impaired G-protein sig-

naling and proposed a feed-forward cycle of progressive neuronal dysfunction, related to phosphoinositide signaling. (95) Spector has hypothesized that idiopathic (Alzheimer's) dementia is a brain-vitamin-deficiency state due to inadequate transport of vitamins from the blood across the choroid plexus (the 'blood brain barrier') and into the cerebrospinal fluid - the only source of these nutrients for the brain. (96) McCaddon and Kelly, using sources they cite, (97, 98, 99, 100, 101, 102) have proposed a somewhat different mechanism. (103)

Chris Reading, BSc, DipAgSc., MBBS, FRANZCP, FACNEM (reading@tpg.com.au) of Australia concurs with McCaddon and Kelly. He now thinks in most cases of "intractable depression," a subtle deficiency of B12, or other nutrient, or thyroid hormone, has been missed. (104)

At the start I said the B12 therapy is perfectly safe, and the risk of overdose is virtually nil. Patients of Dr. H.L. Newbold in New York City injected themselves three times daily with triple-strength doses of B12 (9,000 ug per day) indefinitely. Their serum B12 levels reached 200,000 pg/ml and more, but none suffered any significant ill effects. (105)

Dr. Dommissé prefers hydroxocobalamin to cyanocobalamin. A person taking the latter might over a long period of years accumulate a toxic amount of cyanide and possibly damage vision. Methylcobalamin, widely used in Japan, is increasingly popular in the U.S. because it is reputed to be better absorbed; a compounding pharmacist should be able to provide methylcobalamin after a doctor prescribes it.

Little research is published about all this because of the "heavy pharmaceutical industry sponsorship of research and teaching in medical schools. Career-track academicians have realized, if they want to fulfill their ambitions, they have to eschew nutritional research for that of drugs." (3) The volume of published research on drugs to fight AD is overwhelming. In order to continue their careers, all the authors of these

studies have a powerful financial incentive to report positive results.

II. Low-dose lithium for prevention and even reversal of early AD. The following summary is from page 1 of the April 1, 2004 edition of *Total Wellness*, a monthly newsletter by Dr. Sherry Rogers, M.D. "Health tips. Lithium to block AD. When the autopsy pathologist looks at a slide from the brain of someone who died from AD, he sees clumps of abnormal proteins called amyloid. These are scattered randomly throughout the brain and connected by tangled nerve fibers that literally strangle normal brain tissue to death. Lithium can inhibit the growth of these plaques and suffocating tangles and may even reverse them. It has been used for decades in higher doses for manic depression. Lithium orotate (Vitamin Research Products, 1-800-877-2447) is 20 times more bioactive than other forms, and available in 120 mg capsules providing 4.8 mg of elemental lithium. The dose for AD could be one twice a day. Obviously, you also need membrane nutrient assays and corrections as detailed in *DETOXIFY OR DIE*, Rogers Sherry A., M.D. and *Total Wellness* 2003 issues." (106)

These low doses of lithium can promote brain cell regeneration and enlarge brain cell mass. (107) Also, taking 50 mcg of natural Li at meals, 13 manic-depressive patients all improved in 10 days and there were no side effects with such a low dose. Lithate 5 mg is available (NEEDS 1-800-634-1380). (108, 109) I myself take one 5 mg capsule of Li aspartate from a health food store daily for the major physical and mental benefits disclosed this year in Dr. Jonathan Wright's *Nutrition & Healing* newsletter and in *Townsend Letter* for March and April 2004.

III. Review of 40 trials of ginkgo biloba in patients with cerebral insufficiency. This review demonstrated improvement in symptoms such as dizziness, difficulties of memory and concentration, headaches and depressive mood. In most trials the dosage was 120 mg Ginkgo stan-

dardized 24% extract daily for at least 4–6 weeks. (110)

Ginkgo biloba has been shown to be effective in prevention and treatment of AD. In a double blind study involving 40 patients, half were given 80 mg of extract three times daily for a period of three months. Those patients improved significantly in memory, attention, reaction time and cognitive skills throughout the period, compared to those on placebo, and no side effects were noted. (111) A meta-analysis of four studies found a significant benefit against dementia from ginkgo biloba at 120 to 240 mg daily. (112) A placebo-controlled trial of Ginkgo biloba EGb 761, 40 mg three times per day showed significant improvement in cognitive performance. (113)

Also, a Dr. Huguet and colleagues in Poitiers, France discovered that Ginkgo biloba extract could reverse the loss of serotonin receptors in the brain. They found, further, that it could protect and restore the damage to serotonin receptor sites in the brain within 21 days. (114) (Dr. Earl Conroy, my DC/ND friend in New Zealand strongly concurs with this finding. Results are augmented by periwinkle and hawthorn, he added.) Care is required in selecting a pure ginkgo extract; since wild claims are made for certain inferior products. (115)

London researchers will soon undertake a study of 250 patients over 55 in a local setting as treated by their own physicians. Scientists believe ginkgo causes blood vessels to dilate, thins blood and improves its flow to the brain; some believe it also has antioxidant effects such as protecting nerve cells. (116)

IV. Vitamin B6 ("pyridoxine") as preventive of AD. John Marion Ellis, MD, of Mount Pleasant, Texas, told me on the telephone May 3, 2004, "To my knowledge, none of my thousands of patients taking vitamin B6 [at 50–300 milligrams daily for a long time] developed AD." Dr. Ellis conducted this B6 supplementation from 1962 to 1996, when he retired.

Moses M. Suzman, MD, of Johannesburg,

South Africa, also, knew of no patient of his who developed AD. He treated tens of thousands of people who had been referred to him as a cardiologist and internist. From 1950 into 1992, his advice to all of them was, "Take 100 milligrams of vitamin B6 a day for the rest of your life." Other than those two doctors, no one in the world is known to have treated large numbers of patients with megadose B6 over a period of decades, and so these two doctors' patients constitute my database. For both large groups there were no cases of AD.

Brian Balin, DO, and others have proposed a mechanism that would likely explain this result. *Chlamydia pneumoniae*, an infectious organism, instilled into the nostrils induces Alzheimer-like amyloid plaques in the brains of BALB/c (highly susceptible) mice, and these plaques grow with time. (117, 118) About 98 percent of DNA sequences in mice and in people are identical, making clear the discovery's significance for humans. *C. pneumoniae* appears to cause growth of arterial blockage in humans as well. I set forth the mechanism, fully referenced, elsewhere. (119)

A very large number of, if not indeed most, heart attacks appear to result from infection. Researchers found the infectious organism *C. pneumoniae* in the coronary arteries of 93 percent of a large group of cardiac patients. (120) Dangerous forms of *Helicobacter pylori* (121, 122) and others, which can derive from allergies, (123) from tainted foods, and from the cavitations (holes in bone) that lurk under most dentist-installed root canals, (124, 125, 126) also show up there. These disease organisms, as well as *C. pneumoniae*, appear to be not only observers along for the ride, but to be at least a part, and probably a major part of the problem. (127)

Endotoxins, poisons emitted by some periodontal disease bacteria, can also enter the bloodstream via the mouth. (128) A 1997 study of 1,372 Native Americans found that the risk of heart attack was 2.7 times higher in individuals

with periodontal gum disease than in those with healthy gums. (129, 130)

A large but generally ignored body of research shows that vitamin B6 in ample quantity possesses important infection-fighting capability. (131, 132, 133, 134, 13, 136) This strong resistance to infectious diseases other than heart disease itself probably explains in large part why Dr. Ellis's B6-taking patients "felt better" than abstainers, and why Dr. Suzman's "enjoyed better health" than their neighbors and friends. Further, a systemic inflammatory condition explains many heart attacks, of whatever origin, in clean arteries. Antioxidant vitamins appear to ameliorate these through stimulating increased generation of nitric oxide (NO). (137) B6 appears to be far more effective an antioxidant than others, as I show elsewhere.

And so, as a new hypothesis I propose that much the same mechanism, although functioning in separate parts of the human anatomy, may cause both heart attacks and dementia. And that might explain why the same preventive technique, long-term mega dose vitamin B6 supplementation, succeeded against both conditions. Drs. Suzman and Ellis's success against both is thus explained.

From 1950 to 1965 while number of heart attacks ballooned in Western industrial countries, arterial damage did not increase; only clotting and inflammation grew. (138, 139) Both of these accompany every infection. (140) Stress, smoking, alcohol, caffeine promote clotting; enough B6 resists it. (141)

In depth, vitamin B6 appears to function as an antithrombotic agent, i.e., clot preventive. According to Subbarao et al, (142) concentrations greater than 0.8 mcg/L pyridoxal phosphate (PALP) completely inhibited the second wave of aggregation in platelet-rich plasma containing ADP, thrombin, or adrenalin. The inhibitory effect of PALP seems to be due to its specific binding to the platelet-surface proteins that may be involved in various platelet functions. In healthy volunteers intravenous PALP

also prolonged the whole-blood clotting time and thrombin clotting time. (143) These observations strongly suggest that PALP is an antithrombotic agent that not only alters platelet function but also prolongs clotting time. (144)

Subsequently, Zahari and Kakkar found that pyridoxal 5'-phosphate acts as a phosphodiesterase inhibitor in platelets; this in part explains its anti-platelet activity. (145) They suggested also that PALP influences internal calcium flux required for aggregating and release-inducing effects. (146)

Low vitamin B6 levels in inflammatory bowel disease (IBD) may be a link between inflammation and thrombosis. (147) Bowel infection with *H. pylori* can increase risk of heart attack. *H. pylori* infection may increase susceptibility to folate deficiency, which increases homocysteine levels and atherosclerotic risk. Early infection with *H. pylori* has also been reported to cause a transient hypochlorhydria as the bacteria colonize the gastric mucosa. There is a reduction in folate bioavailability due to this increased gastric pH. (148)

Infections also promote risk of myocardial infarction indirectly by raising levels of fibrinogen, a component of blood that forms the structure of clots. Fibrinogen levels rise with high serum lipid levels, diabetes, age, stressful lives, obesity, and contraceptive pills. (149)

V. The place of mercury (Hg) in AD. Professor Boyd Haley established that Hg is probably largely responsible for both autism and AD. (151) Mercury was found in the brains of 83 randomly chosen accident victims with amalgam fillings (which are about 50 percent Hg); (152) and the concentration of Hg is four times higher in autopsies of AD brains than in controls. (153) No other trace element tested was significantly elevated in those brains, not even aluminum. Also, Hg the only element that stimulates formation of the infamous neurofibrillary tangles in AD brains, (151) which lithium orotate, as we saw, may destroy or prevent. Patients

well on the way to a full-blown AD diagnosis have improved dramatically when their amalgam fillings were taken out and the Hg removed from the body by a detoxification program following established protocol. (152)

I propose modification of Dr. Haley's proof: Hg from amalgam fillings can cause AD in people who are vitamin B12-deficient. In people who are B12-repleted, if he repeated the test I suggest that he would probably not find the neurofibrillary tangles.

Flu shots can load the brain with Hg. Adults who had five yearly flu shots between 1970 and 1980 (the years studied) had ten times higher risk of AD than if they had one, two, or no flu shots. (153) Is that why AD is expected to quadruple? (154) Thimerosal-containing childhood vaccinations, as well as flu shots, contain both Hg and aluminum. Then isn't the campaign to get everyone to take a flu shot every year also a campaign to spread AD!

Mental stimulation, such as traveling, learning to play an instrument or doing crossword puzzles, is associated with a decreased risk of AD. Researchers suspect that mental challenge helps to build up the brain, which can include creating new neurons, as J. S. Bland pointed out in his Functional Medicine Update tape in 2003, making it less susceptible to the lesions associated with AD. However, that may not qualify for our list of probable preventives.

VI. Fluoridated water. J.A. Varner, PhD, and associates studied laboratory rats to learn whether alum (aluminum sulfate) would combine with fluoride and penetrate the blood-brain barrier. (157, 158) Water supply facilities frequently add alum to clarify drinking water; aluminum is neurotoxic to humans and animals. And there is no substance as biochemically active in the human organism as fluoride." (159)

In Varner's study eighty percent of the test rodents died; they also suffered significant kidney and brain damage. The respiratory infection, which is common in rodents, was always much more virulent in the animals receiving the

AlF₃ or NaF; there appeared to be a general impairment in the immune capacities of the treated animals. Those treated with aluminum fluoride were most affected. For the animals receiving AlF₃, "there was a reduction in the number of cells in the hippocampus, together with a disorganization of the hippocampal pyramidal cells. Changes were observed in both the phosphorylated and nonphosphorylated neurofilaments in the neocortex which are usually considered to be related to cell dysfunction." (160)

The dose of fluoride and alum used was similar to that in artificially fluoridated drinking water—even without the excessive levels of fluoride getting into people from toothpaste, fruit juices, the air, and other sources (161) and without the extraordinary toxicity of the witches' brew used in most American fluoridation systems.

Pharmaceutical grade sodium fluoride (NaF) is used in about 10% of American fluoridation, and likely close to 100% in the few places overseas that fluoridate. Ninety percent of US and probably Canadian fluoridation uses fertilizer or aluminum plant wastes rich in arsenic, uranium²³⁸, Hg, dioxins, etc., and often up to 400 milligrams of violence-promoting lead per liter. About 20 percent of the mixture is highly acidic silicofluorides. (162) None of this has ever been tested for safety; tests reported have dishonestly substituted pure NaF. The dental profession profits greatly from performing the expensive repairs that ensue.

Striking parallels were seen between aluminum-induced alterations in cerebral blood vessels associated with AD and other forms of presenile dementia. The Varner study confirmed results of their two previous studies. Inexcusably, it may take 2–3 years for the National Toxicology Program to act upon the USEPA (Environmental Protection Agency) request for further research. (163)

AD is observed in 30- to 40-year old Canadians who have long consumed fluoridated water.

(164) A number of people in Seattle, WA, between the ages of 40 and 60 with symptoms associated with early-onset AD (memory loss, confusion, difficulty concentrating) and pains in kidneys and back—symptoms of chronic fluoride poisoning—after two to three months of avoiding all sources of fluoride (particularly water and toothpaste), were restored to full functioning. (165)

VII. Many assume that the genetic risk of AD, particularly in patients who inherited the genetic allele APO-lipoprotein-E4 from both parents, is "hard-wired," making the disease inevitable for them. But recent research found that strongly boosting the body levels of vitamin B12 improved patients' condition in spite of the genes. (166) To learn more about the relationship between nutrient intake and our genetic code, consult <http://nutrigenomics.ucdavis.edu/>.

And any inherited disease or trait that has a serious impact on fitness must fade over time, because the genes that spell it out will be passed on to fewer and fewer surviving individuals. Therefore, common illnesses with severe fitness costs—such as AD—are unlikely to have a genetic cause. (167)

VIII. When vitamin B12, lithium orotate, ginkgo, and vitamin B6 are so effective in preventing and B12 in even reversing AD, how likely are these simple measures to actually be widely enough used to "turn the tide"? Arthur B. Robinson, PhD, is not optimistic and borders on cynical. "We have, unfortunately, a vast industry that thrives on human suffering. This industry is composed of government regulators, public and private lawyers, academic and scientific bureaucrats, and other similar components of our increasingly fascist and socialist medical system [notice that he doesn't include the doctors]. They make sure that no progress in medical science goes forward unless it involves them—this requirement being far more important than actual medical progress."

Later, he writes, "Simple therapies involving common substances are not wanted. Ordinary

substances that are in the public domain cannot easily be turned into proprietary products, lucrative liability litigation, subjects for endless safety studies and magic pills available only through your doctor and pharmacist. Therefore simple therapies that could be delivered at very low cost are not interesting to this unprincipled, government-controlled industry.” (168)

The effort to get such effective, safe, yet very low cost therapies into wide use will have two parts:

(1) Educate the families, the “man on the street,” those who are at risk of heart disease, diabetes, cancer and AD, as well as infectious illnesses. Show them how to stay out of the clutches of cardiologists and other specialists who assume there is no way to prevent these catastrophes and understandably have little or no interest in such methods. No one can profit from manufacturing a natural substance, and so drug makers do all they can to play down their potential. Show people how to protect themselves, and they can then try to influence the doctors. This part of the program needs to be presented in language these people will readily understand.

(2) Get the information convincingly in more technical form to hundreds of health-oriented scientists worldwide who for various reasons are not likely to be frightened by pressure from sources such as the FDA. More than 500 of the world’s top scientists have joined Dr. Peter Duesberg in challenging the hypothesis that HIV causes AIDS. (169) The 500 can afford to be brave because they are retired or close to retirement, self-employed, live abroad, or work in unrelated fields. (For another view, see *What Really Causes AIDS*, by Harold Foster, PhD. Trafford Publishing, 2002, www.trafford.com: Dr. Foster shows that HIV does indeed cause AIDS.)

Get the accurate information to these scientists in depth and in detail, and organize them into a society that might hold periodic meetings. Then individually and as a group, exert pressure

on government health agencies and legislators to spend the relatively small amount of money to run appropriate tests. Critically important: make sure those tests employ appropriately large doses, appropriately long test periods, and fulfill the requirements of honesty.

More and more people are becoming disillusioned with conventional medicine, now shown to be America’s worst killer of people. (170)

IX. Why don't the doctors use these effective techniques, asked a friend innocently.

1. The techniques using nutritional measures were not developed by an allopathic doctor, tested double-blind, and published in a mainline medical journal. They are not taught in medical school. And they are not promoted and advertised as a pharmaceutical drug.
2. No prospective double blind, placebo controlled, randomly assigned trials have been performed, and there is no prospect of such tests. No one can modify a natural molecule into a patentable form from which to make a lot of money.

The products here discussed have no adverse side effects, except outside a very wide range of safety, compared to the narrow range of safety for many drugs.

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LITERATURE BRIEFS

Literature briefs are designed only to provide a concise impression of the contents of each article. If you are interested in the topic, you should read the entire article for a clearer description of results.

Food supplements and cognitive aging

An observational study of subjects born in 1936, whose mental ability was tested in 1947 and followed up in 2000–2001, showed that fish oil users had higher cognitive function. Fish-oil supplement users also consumed more vitamin C and vegetable and cereal fiber than did non-supplement-users. Erythrocyte omega-3 fatty acids and the ratio of docosahexaenoic acid to arachidonic acid was associated with better cognitive function in late life before and after adjustment for childhood IQ.

Whalley LJ, Fox HC, Wahle KW, Starr JM, Deary IJ. Cognitive aging, childhood intelligence, and the use of food supplements: possible involvement of n-3 fatty acids. *Am J Clin Nutr.* 2004;80:1650–7.

Benefits of food fortification

Deficiency in intake of essential micronutrients is severe and widespread in many parts of the world. Studies by World Bank have shown that countries whose populations suffer from micronutrient deficiencies encounter economic losses as high as 5% of gross domestic product. The solution is to implement programs to fortify commonly eaten foods with the missing micronutrients or provide nutrient supplements through targeted distribution.

Mannar MG, Sankar R. Micronutrient fortification of foods—rationale, application and impact. *Indian J Pediatr.* 2004;71:997–1002.

Maternal zinc supplements and morbidity in infants

Maternal micronutrient deficiency has been related to adverse fetal effects and it is believed that micronutrient supplementation during pregnancy may improve fetal and neonatal outcome. Single micronutrient supplementation interventions have not consistently shown an effect on size at birth. However, preliminary data suggests that zinc supplements may cause reduction in later diarrheal and infectious morbidity in infants.

Shah D, Sachdev HP. Maternal micronutrients and fetal outcome. *Indian J Pediatr.* 2004;71:985–90.

Food supplements for weight loss

More than 50 individual dietary supplements and more than 125 commercial combination products are available and promoted for weight loss. Chromium is a popular weight-loss supplement, but its efficacy and long-term safety are uncertain. Guar gum and chitosan appear to be ineffective and use of these products should be discouraged. Because of insufficient or conflicting evidence regarding the efficacy of conjugated linoleic acid, ginseng, glucomannan, green tea, hydroxycitric acid, L-carnitine, psyllium, pyruvate, and St. John's wort in weight loss, physicians should caution patients about the use of these supplements. Although evidence of modest weight loss with ephedra-caffeine ingestion exists, the U.S. Food and Drug Administration has banned the sale of these products.

Saper RB, Eisenberg DM, Phillips RS. Common dietary supplements for weight loss. *Am Fam Physician.* 2004;70:1731–8

Omega-3 fatty acids, cancer and heart disease

Epidemiological evidence has established that ingestion of long-chain polyunsaturated omega-3 fatty acids, abundant in fish oils, have profound effects on many human disorders and diseases, including cardiovascular disease and cancer. There are a number of products available to supplement omega-3 fatty acid dietary intake, including eggs, bread, and cereals. While the beneficial effects of omega-3 fatty acids can no longer be doubted, their molecular mechanism of action is complex and apparently involves a number of integrated signaling pathways.

Siddiqui RA, Shaikh SR, Sech LA, Yount HR, Stillwell W, Zaloga GP. Omega 3-fatty acids: health benefits and cellular mechanisms of action. *Mini Rev Med Chem.* 2004;4:859–71.

Botanical medicines

The growing costs of discovering new drugs may again reconnect plants and human health at a new level of technological sophistication. Multi-component botanical therapeutics represented by functional foods, dietary supplements and botanical drugs hold several advantages over conventional drugs and may earn them a more prominent place in the medicine of the future. Technologies that address the needs of discovery, development and manufacturing of multi-component botanical therapeutics are emerging. Nevertheless, multi-component botanical therapeutics face problems of acceptance by the medical community and pharmaceutical industry – safety and efficacy validation, poor standardization and quality control, and difficulties in identifying active ingredients and determining their complex mode(s) of action.

Raskin I, Ripoll C. Can an apple a day keep the doctor away? *Curr Pharm Des.* 2004;10:3419–29.

Calcium supplements and adenoma of the distal colon

Calcium can reduce the risk of colorectal tumors by binding bile and fatty acids. Comparing the supplemental and dietary calcium intakes of 3696 participants with adenoma of the distal colon with the calcium intakes of 34,817 control subjects, adenoma risk was lower by 12% for participants in the highest quintile of total calcium intake than for participants in the lowest quintile. The protective association between total calcium and colorectal adenoma was largely due to calcium supplement use.

Peters U, Chatterjee N, McGlynn KA, Schoen RE, Church TR, Bresalier RS, Gaudet MM, Flood A, Schatzkin A, Hayes RB. Calcium intake and colorectal adenoma in a US colorectal cancer early detection program. *Am J Clin Nutr.* 2004;80:1358–65.

Safety of selenium-enriched yeast

Selenium-enriched yeast (Se-yeast) is a common form of Se supplement. However its availability within the European Union is under threat amid concerns that Se-yeast supplements are poorly characterized and could potentially cause the toxic buildup of selenium in tissues. This review indicates that in about one dozen supplementation studies, none has shown evidence of toxicity even up to an intake level of 800 mcg Se/day over a period of years, compared to the tolerable upper intake level of 300 mcg/day according to the European Community Scientific Committee on Food. Selenomethionine is the largest single form of Se in Se-yeast, accounting for 54–74 % of total Se.

Rayman MP. The use of high-selenium yeast to raise selenium status: how does it measure up? *Br J Nutr.* 2004;92:557–73.

L-Arginine and creatine and exercise capacity

L-Arginine is a conditionally essential amino acid. In some clinical circumstances (e.g., burn

injury, sepsis) in which the demand for arginine cannot be fully met by de novo synthesis and normal dietary intake, supplemental arginine has been shown to facilitate the maintenance of lean body mass and functional capacity. However, the evidence that supplemental arginine may also confer an ergogenic effect in normal healthy individuals is less compelling. In contrast to arginine, numerous studies have reported that supplementation with the arginine metabolite creatine facilitates an increase in anaerobic work capacity and muscle mass when accompanied by resistance training programs in both normal and patient populations.

Paddon-Jones D, Borsheim E, Wolfe RR. Potential ergogenic effects of arginine and creatine supplementation. *J Nutr.* 2004 Oct;134(10 Suppl):2888S-2894S.

Fortification of foods with folate.

Median dietary folate equivalents in adolescents who are non-consumers of folate-fortified foods are 50–70% of dietary folate equivalents in consumers.

Sichert-Hellert W, Kersting M. Fortifying food with folic acid improves folate intake in German infants, children, and adolescents. *J Nutr.* 2004 Oct;134(10):2685–90.

Folate supplements during pregnancy

Periconceptional consumption of the B vitamin folic acid reduces the occurrence of neural tube defects (NTDs) by 50%-70%. Fortification of the U.S. food supply with folic acid has resulted in a 26% reduction in NTDs. However, even with fortification, not all women receive adequate levels of folic acid from their diets. Therefore, increasing the use of vitamins containing folic acid remains an important component of NTD prevention.

Centers for Disease Control and Prevention (CDC). Use of vitamins containing folic acid among women of childbearing age—United States, 2004. *Morb Mortal Wkly Rep.* 2004 Sep 17;53(36):847–50.

Alpha carotene and cancer

Although supplements of beta-carotene alone increased cancer incidence in smokers, alpha-carotene and vitamin A may protect against cancer recurrence in nonsmokers and nondrinkers.

Steck-Scott S, Forman MR, Sowell A, Borkowf CB, Albert PS, Slattery M, Brewer B, Caan B, Paskett E, Iber F, Kikendall W, Marshall J, Shike M, Weissfeld J, Snyder K, Schatzkin A, Lanza E. Carotenoids, vitamin A and risk of adenomatous polyp recurrence in the polyp prevention trial. *Int J Cancer.* 2004 Nov 1;112(2):295–305.

Zinc and osteoporosis in men

Low zinc intakes and reduced blood zinc concentrations are associated with osteoporosis in women. A study of zinc status in men indicates a similar relationship. Dietary zinc intake and plasma zinc concentrations were lower in men with osteoporosis at the hip and spine than in men without osteoporosis

Hyun TH, Barrett-Connor E, Milne DB. Zinc intakes and plasma concentrations in men with osteoporosis: the Rancho Bernardo Study. *Am J Clin Nutr.* 2004;80:715–21.

Essential fatty acids and autism

A health questionnaire based on parental observations of clinical signs of fatty acid deficiency showed that patients with autism and Asperger's syndrome had significantly higher deficiency scores than controls. Patients diagnosed with both autism and Asperger's showed increased levels of EPA and DHA, and significantly reduced levels of arachidonic acid when supplemented with EPA-rich fish oils. Patients with autism/Aspergers who had taken EPA supplements had significantly reduced phospholipase A2 concentrations compared to un-supplemented patients.

Bell JG, MacKinlay EE, Dick JR, MacDonald DJ, Boyle RM, Glen AC. Essential fatty acids and phospholipase A2 in autistic spectrum

disorders. *Prostaglandins Leukot Essent Fatty Acids*. 2004;71:201–4.

Vitamins C and E and free radicals

Cigarette smoking has been associated with impaired endothelium-dependent flow-mediated dilation, and increased oxygen-derived free radicals have been suspected of being one of the major causes. Vitamins C and E are widely used antioxidant vitamins that have also been reported to effectively improve the endothelial function in several conditions. Oral supplementation of vitamin C (1.0 g/day) and vitamin E (500 mg/day) for 25 days significantly improved endothelium-dependent flow-mediated dilation. The effect disappeared 4 weeks after the vitamin supplementations ended.

Takase B, Etsuda H, Matsushima Y, Ayaori M, Kusano H, Hamabe A, Uehata A, Ohsuzu F, Ishihara M, Kurita A. Effect of chronic oral supplementation with vitamins on the endothelial function in chronic smokers. *Angiology*. 2004;55:653–60.

Vitamin D and osteomalacia

Overt clinical osteomalacia is usually treated with a loading dose of vitamin D, followed by a regular supplement. However, little is known of the time taken to reach a stable biochemical state after starting treatment. A 2-year follow-up study of 42 patients with biopsy proven osteomalacia treated with a standard replacement regimen and general nutritional support showed that although normocalcaemia was attained within 4 weeks the mean values continued to rise, to a mid-range plateau at 52 weeks. The phosphate and alkaline phosphatase values also took at least a year to reach a stable mean, with a slight further trend towards the mid-range for the entire 104 weeks. The dynamic relationship between calcium, phosphate and bone requires at least a year, and probably longer, to reach an equilibrium.

Allen SC, Raut S. Biochemical recovery time

scales in elderly patients with osteomalacia. *J R Soc Med*. 2004;97:527–30.

Nutrient intake and orofacial clefts

It has been suggested that nutrients other than folic acid prevent orofacial clefts (OFC). The energy-adjusted intakes of vegetable protein, fiber, beta-carotene, ascorbic acid, alpha-tocopherol, iron, and magnesium were significantly lower in mothers of OFC children compared with controls. The preconceptional intake of the nutrients predominantly present in fruits and vegetables reduces the risk of offspring affected by OFC.

Krapels IP, van Rooij IA, Ocke MC, West CE, van der Horst CM, Steegers-Theunissen RP. Maternal nutritional status and the risk for orofacial cleft offspring in humans. *J Nutr*. 2004;134:3106–13.

Antioxidants and oxidant stress

Antioxidants may alleviate the oxidative stress associated with photochemical oxidant pollutants such as ozone. Antioxidant supplements (Vitamin C 250 mg/day and vitamin E 50 mg/day) were given to atopic asthmatic children in Mexico City who were exposed to ozone. The supplements significantly reduced the production of interleukin-6 as measured in nasal secretions and non-significantly reduced IL-8.

Sienra-Monge JJ, Ramirez-Aguilar M, Moreno-Macias H, Reyes-Ruiz NI, Del Rio-Navarro BE, Ruiz-Navarro MX, Hatch G, Crissman K, Slade R, Devlin RB, Romieu I. Antioxidant supplementation and nasal inflammatory responses among young asthmatics exposed to high levels of ozone. *Clin Exp Immunol*. 2004 Nov;138(2):317–22.

Characteristics of tablet/capsule preference

Patients over the age of 50 years ranked the importance of eight physical characteristics of vitamin pills (ease of swallowing, size, shape, color, smell, coating, texture, and taste). When

given test products and asked which they preferred and why, ease of swallowing was the most important characteristic influencing preference.

Brotherman DP, Bayraktaroglu TO, Garofalo RJ. Comparison of ease of swallowing of dietary supplement products for age-related eye disease. *Am Pharm Assoc* (Wash DC). 2004 Sep-Oct;44(5):587–93.

Supplements of arginine, vitamin C, zinc and protein heals pressure ulcers

Nutritional intervention in the form of an oral nutritional supplement rich in protein and enriched with arginine, vitamin C and zinc resulted in a significant reduction in wound area and an improvement in wound condition in patients with grade III and IV pressure ulcers. Within three weeks the amount of exudate and the incidence of necrotic tissue reduced significantly.

Frias Soriano L, Lage Vazquez MA, Maristany CP, Xandri Graupera JM, Wouters-Wesseling W, Wagenaar L. The effectiveness of oral nutritional supplementation in the healing of pressure ulcers. *J Wound Care*. 2004 Sep;13(8):319–22.

Vitamin A supplements and incidence of diarrhea and acute respiratory infection in children.

Regularly providing a high-dose (200,000 IU) capsule of vitamin A to children in Nepal aged 6 to 60 months, including those who eat vitamin A-rich foods, reduced the prevalence of diarrhea and acute respiratory infection compared to children who received the vitamin A supplement only once or who received no vitamin A supplement.

Grubestic RB. Children aged 6 to 60 months in Nepal may require a vitamin A supplement

regardless of dietary intake from plant and animal food sources. *Food Nutr Bull*. 2004 Sep;25(3):248–55.

Supplements of cod liver oil and a children's multivitamin/mineral including selenium reduces number of upper respiratory tract pediatric visits.

Use of nutritional supplements supplying cod liver oil and a multivitamin-mineral supplement containing selenium was acceptable to the inner-city Latino families and over time was associated with a decrease in upper respiratory tract pediatric visits.

Linday LA, Shindledecker RD, Tapia-Mendoza J, Dolitsky JN. Effect of daily cod liver oil and a multivitamin-mineral supplement with selenium on upper respiratory tract pediatric visits by young, inner-city, Latino children: randomized pediatric sites. *Ann Otol Rhinol Laryngol*. 2004 Nov;113(11):891–901.

Geophagia and mineral absorption

Geophagia, the intentional and repeated ingestion of soil, is a perplexing eating behavior. It is generally assumed that a large proportion of mineral nutrients in soil is available for absorption in the body and that geophagia may help supplement mineral nutrients. Using an in vitro soil ingestion simulation test, it was determined that geophagia can potentially reduce the absorption of already bioavailable nutrients, particularly micronutrients such as Fe, Cu and Zn, although the ingested soil may be a source of Ca, Mg and Mn.

Hooda PS, Henry CJ, Seyoum TA, Armstrong LD, Fowler MB. The potential impact of soil ingestion on human mineral nutrition. *Sci Total Environ*. 2004 Oct 15;333(1–3):75–87.

BOOK REVIEWS

Natural Alternatives to Vioxx, Celebrex & Other Anti-Inflammatory Prescription Drugs: What to Use to Help Relieve Arthritis Pain and Inflammation Carol Simontacchi. Square One Publishers, Garden City Park, NY. Paperback, 2005, 120 pages, \$5.95. ISBN 0-7570-0278-1

It is hard to imagine a clinical nutritionist without several clients who are suffering from some sort of inflammatory complaint. And, thankfully, most clinical nutritionists know a variety of nutritional interventions that will go a long way to alleviating their problems. So why is there a need for this small book of roughly 100 pages outlining the “safer solutions to COX-2 inhibitors?”

One good reason is that it packs a lot of information in those 100 pages, information that does not always come quickly to mind when we are counseling a client. It is really handy to be reminded of the dozens of options available to help people with arthritis and other inflammatory conditions. The author has given us the whole story, including the nutrients, the dietary changes, the herbal remedies and even the physical therapies that can relieve pain.

What may be even better for the busy practitioner, at only \$5.95, this little book is inexpensive enough to give to clients, which may save a good bit of your time by explaining why you are recommending what you recommend. The explanations are detailed and accurate but not so technical that a reasonably intelligent person would have a hard time grasping the concepts. After all, clinical nutritionists are or should be educators, instructing our clients in the fundamentals of health. What better way to serve our clients than to train them in ways to avoid the scourge of our present existence, the inflammatory diseases?

James Heffley, Ph.D., CCN, DANLA
Austin,

We're Killing Our Kids: How to End the Epidemic of Overweight and Sedentary Children Todd Hollander, Worthy Press, Atlanta, GA 30356. 2004, Paperback, 192 pages, \$19.95. ISBN: 0-9753166-4-8

Even though you probably know a lot of what is in this unpretentious little book, it is nice to have it all collected in one place for easy reference. The author does not profess to be a trained nutritionist, but a market research consultant who is interested in keeping his own and other American kids healthy. He has not tried to bite off more than he can chew, and without analyzing what the biochemical/physiological mechanisms are, he merely describes what is happening in America. From simply reading popular publications, he is convinced that eating too much non-nutritious food, sugar in particular, coupled with 21st century temptations, video games and TV in particular, have resulted in a rise in obesity among both children and adults. The remedy, which is only 5 steps instead of 12, begins, as you would expect, with acknowledging the problem. In fact, the chapters on “What you need to know” and “What you need to do” may be the best parts of the book.

In addition, there are plenty of statistics that start out the book, and some helpful charts to encourage you to implement your plan. You don't need to keep giving your kids “breakfast candy,” aka the cereal advertised on children's TV shows, or allow their school to continue making money from selling “liquid candy,” aka soft drinks, at the expense of your child's health.

James Heffley, Ph.D., CCN, DANLA
Austin, TX

BOOKS RECEIVED FOR REVIEW

Your Body's Sign Language: Clues to Nutritional Well-being James W. McAfee, CCN. Image Awareness Corp, Auburn, CA 95603. Paperback, 2005, 294 pages, \$24.95. ISBN 0-9604592-1-9

Chemistry and Safety of Acrylamide in Food Eds Mendel Friedman and Don Mottram. Springer Science + Business Media, NY. Hardback, 2005, 476 pages. \$169.00. ISBN 0-287-23920-0

THE JOURNAL OF APPLIED NUTRITION

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