Better Health in 120 Days

is all about

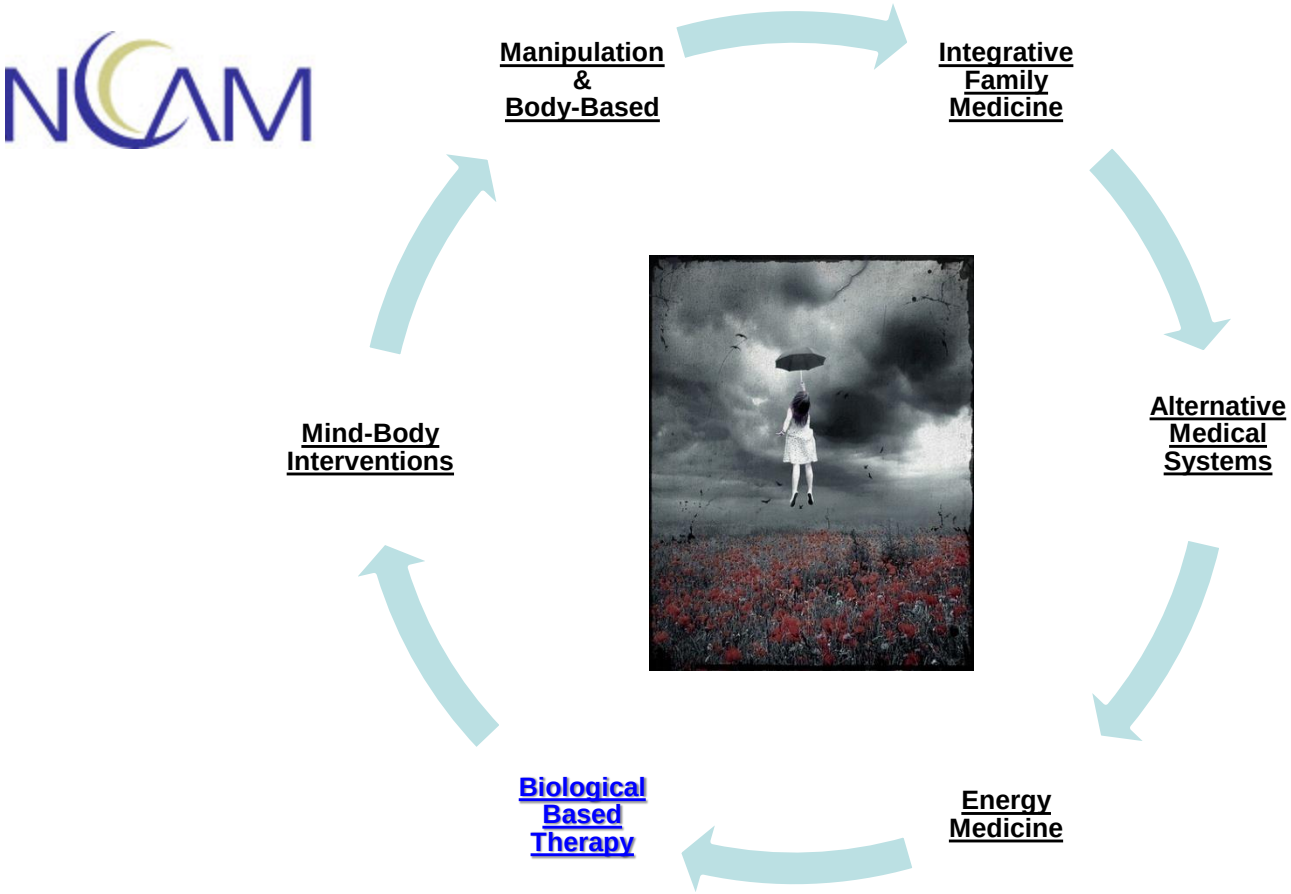
How Wee* NutriGenomics
Looks in All The Right Places,
Increases the Right Foods,
Eliminates the Toxic Foods,
Decreases the Food Excesses
to Reach Optimal Health +/- Nutrients

The Great Lakes Orthodontic Association
September 26, 2015
Glen Aukerman, MD, **Medical Director**
The Ohio State University Center for Integrative Medicine

Our Discussion in Next 60 Minutes

- Review Origin of NutriGenomics at OSUCIM
- Natural History of Nutrition and Health
- What is Toxic in Excess in our Foods
- What is Missing in Food and Water
- Uncovering New Patient Problems
- Recovering the New Patient
- Managing Patients over Time
- Wee Education for Patients
- Review and Questions

NutriGenomics is *Only* one of many services at *The Ohio State University* *Center for Integrative Medicine*



NutriGenomics started with One Patient

- My New Role was to Support CAM Providers' patient care;
  - Rx for Massage, Acupuncture, Chiropractic Treatments
  - Continue Primary Care Family Medicine practice
- Worked well for about 3 months;
  - ***Unique Experience Happened*** that Changed My Life and which
  - ***Can Uniquely Change Yours***
- A Woman Appeared who;
  - Took a year out of Chemotherapy for Breast Cancer to have a baby
  - At her 6 weeks Postpartum Visit the doctor told her
  - She had 60 days to prepare to leave her 6 week old and 1 year old
  - Her Surgeon Oncologist had done all they knew without success
  - She wanted some way to stay alive until Babies reached school age

NutriGenomics Was Her Only Option

- She was to Keep Positive for the time she had
- I to Research every Evening and Weekends
- 6 weeks into her 60 days, @ 3:30 a.m.
  - I found The Opportunity *if we did everything right*
  - Plant oil in her Foods *were* the Cause of her Cancer
  - Monocytes % *is the proxy for* Plant oil in the Body
  - KIM-2 is an NIH Tool to Identify Healthful Foods & to Eliminate Harmful Foods; Seeds, Nuts, Flax, Soy, Granola, Trail mix, Hummus, Avocado, + Poultry
- That afternoon, She *Committed to Try* the new program, which I later called *NutriGenomics*

The 3:30 a.m. Article Pointing to How *Omega-3 Fish Oil* NutriGenomicly Can Quiet Cancer

Dietary long-chain n−3 fatty acids for the prevention of cancer: a review of potential mechanisms[1–3]

Susanna C Larsson, Maria Kumlin, Magnus Ingelman-Sundberg, and Alicja Wolk

ABSTRACT

Increasing evidence from animal and in vitro studies indicates that n−3 fatty acids, especially the long-chain polyunsaturated fatty acids eicosapentaenoic acid and docosahexaenoic acid, present in fatty fish and fish oils inhibit carcinogenesis. The epidemiologic data on the association between fish consumption, as a surrogate marker for n−3 fatty acid intake, and cancer risk are, however, somewhat less consistent. This review highlights current knowledge of the potential mechanisms of the anticarcinogenic actions of n−3 fatty acids. Moreover, a possible explanation of why some epidemiologic studies failed to find an association between n−3 fatty acid intake and cancer risk is provided. Several molecular mechanisms whereby n−3 fatty acids may modify the carcinogenic process have been proposed. These include suppression of arachidonic acid–derived eicosanoid biosynthesis; influences on transcription factor activity, gene expression, and signal transduction pathways; alteration of estrogen metabolism; increased or decreased production of free radicals and reactive oxygen species; and mechanisms involving insulin sensitivity and membrane fluidity. Further studies are needed to evaluate and verify these mechanisms in humans to gain more understanding of the effects of n−3 fatty acid intake on cancer risk. *Am J Clin Nutr* 2004;79:935–45.

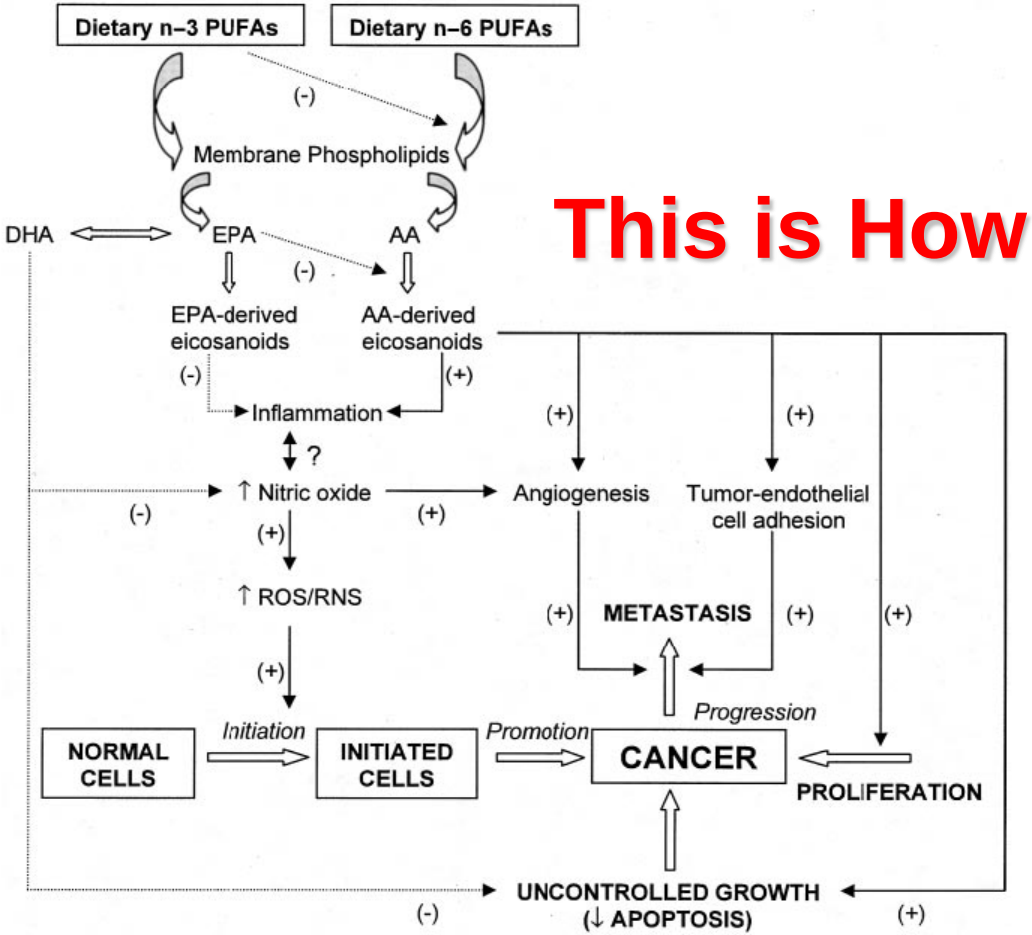
has been highlighted by animal experiments and in vitro studies showing that these PUFAs suppress the development of major cancers (26–31). These experimental findings are supported by results from clinical studies showing a reduction in intestinal hyperproliferation after consumption of fish oil–derived n−3 PUFAs in subjects at elevated risk of colon cancer due to sporadic colonic adenomas (32, 33). Although a few previous reviews have described some selected actions through which long-chain n−3 fatty acids may play a role in carcinogenesis, such as biosynthesis of eicosanoids (34, 35), lipid peroxidation (36–38), and some signal transduction pathways (34, 36), to our knowledge, no comprehensive review that puts all these pieces and further evidence together is available.

The present review focuses on several putative mechanisms whereby long-chain n−3 fatty acids may modulate the carcinogenic process. Furthermore, a potential explanation of why several case-control studies and large cohort studies failed to confirm a protective effect of long-chain n−3 fatty acids against cancer development is briefly discussed. Moreover, we discuss how knowledge of the mechanisms of action of PUFAs should be taken into account in epidemiologic analyses.

MECHANISMS OF POTENTIAL CHEMOPREVENTIVE

he American Journal of Clinical Nutrition

Seed, Nut, Flax, Soy Omega-6 Plant Oils Stimulate Cancers NutriGenomically



This is How

FIGURE 2. Hypothetical scheme showing potential mechanisms whereby n−6 polyunsaturated fatty acids (PUFAs) and n−3 PUFAs may promote and suppress carcinogenesis, respectively. In initiated tumor cells, phospolipase A_2, cyclooxygenase 2, and lipoxygenases are often overexpressed, which leads to overproduction of arachidonic acid (AA, 20:4n−6)–derived eicosanoids that augment inflammation. Nitric oxide, which is elevated in inflammation, is implicated in both the initiation and the progression stages of carcinogenesis. Nitric oxide may stimulate tumor growth and metastasis by enhancing the angiogenic and migratory abilities of tumor cells. Dietary n−3 PUFAs reduce the desaturation and elongation of linoleic acid (18:2n−6) to AA, the incorporation of AA into membranes, and the biosynthesis of AA-derived eicosanoids; suppress inflammation; stimulate apoptosis; up-regulate the expression of genes coding for antioxidant enzymes; and thus inhibit tumor growth and metastasis. + and solid arrows, stimulation; − and dashed arrows, suppression;

Seed, Nut, Flax, Soy Omega-6 Plant Oils Stimulate Cancer Cytokines NutriGenomically

Cytokines

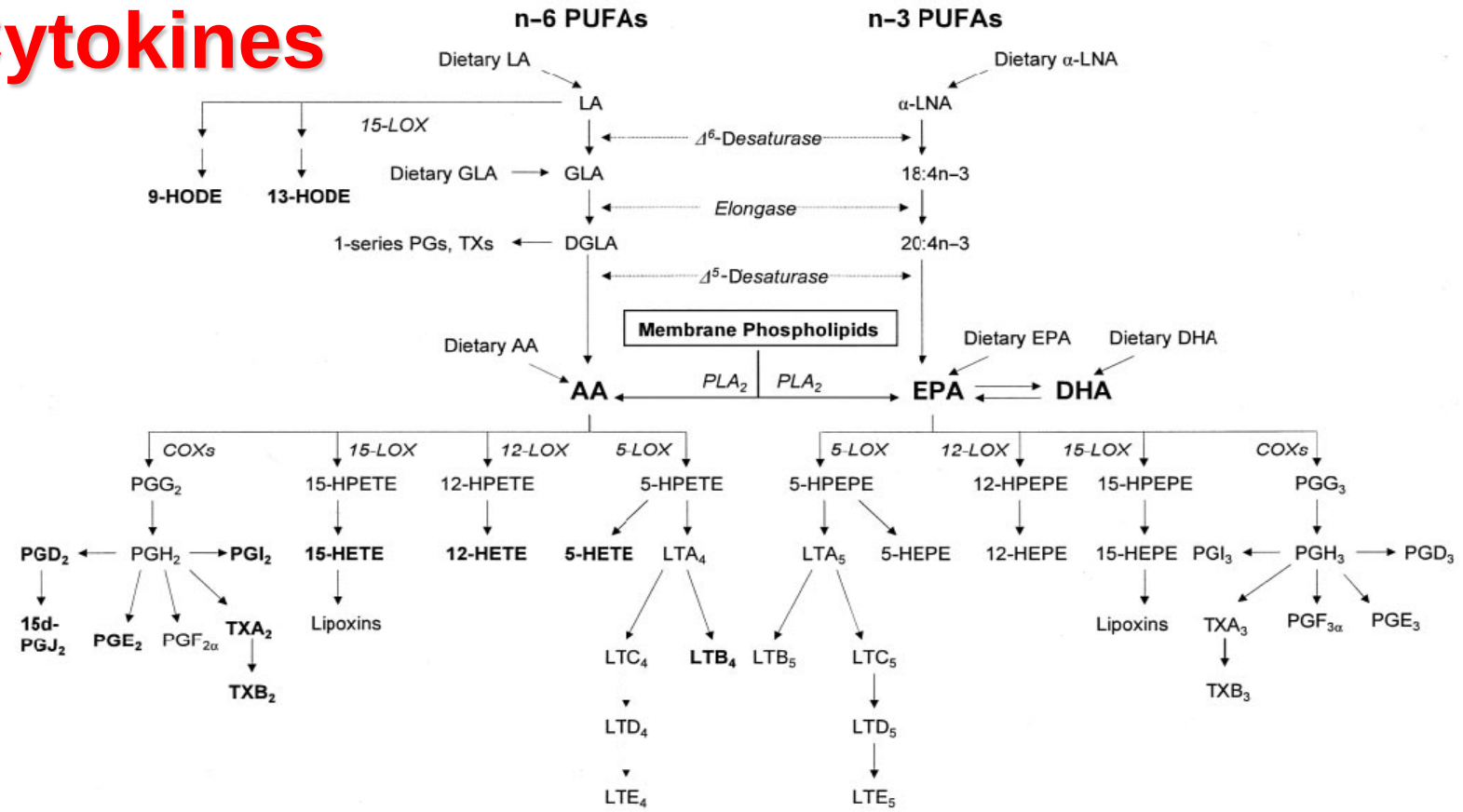


FIGURE 1. Overview of the metabolism of n−6 and n−3 polyunsaturated fatty acids (PUFAs) into eicosanoids involved in inflammation and carcinogenesis. The names of these eicosanoids are shown in bold. LA, linoleic acid (18:2n−6); α-LNA, α-linolenic acid (18:3n−3); GLA, γ-linolenic acid (18:3n−6); DGLA, dihomo-γ-linolenic acid (20:3n−6); AA, arachidonic acid (20:4n−6); EPA, eicosapentaenoic acid (20:5n−3); DHA, docosahexaenoic acid (22:6n−3); PLA_2, phospholipase A_2; LOX, lipoxygenase; COXs, cyclooxygenases (COX-1 and COX-2); 15-HETE, 15(*S*)-hydroxyeicosatetraenoic acid; 12-HETE, 12-hydroxyeicosatetraenoic acid; 5-HETE, 5-hydroxyeicosatetraenoic acid; HEPE, hydroxyeicosapentaenoic acid; HPETE, hydroperoxyeicosatetraenoic acid; HPEPE, hydroperoxyeicosapentaenoic acid; LT, leukotriene; HODE, hydroxyoctadecadienoic acid; PG, prostaglandin; TX, thromboxane.

KIM-2 Loaded with Snack & Meats

Fish Oil Needed To Fix

Snacks	Serving Size	grams	kcals	Servings	Short 6	Short 3	Long 6	Long 3
Nuts, walnuts, english	1 oz (14 halves)	28	185	1	10799	2574	0	0
Nuts, butternuts, dried	1 oz	28	174	1	9562	2472	0	0
Nuts, walnuts, black, dried	1 oz	28	175	1	9376	569	0	0
Seeds, sunflower seed kernels, dry roasted,	1 oz	28	165	1	9294	20	0	0
Seeds, watermelon seed kernels, dried	1 oz	28	158	1	7965	0	0	0
Seeds, safflower seed kernels, dried	1 oz	28	147	1	7962	31	0	0
Nuts, brazilnuts, dried, unblanched	1 oz (6-8 kernels)	28	186	1	6749	18	0	0
Seeds, pumpkin and squash seed kernels, dried	1 oz hulled (142	28	153	1	5869	51	0	0
Snacks, potato chips, made from dried potatoes,	1 oz	28	158	1	5562	102	0	0
Seeds, sesame seeds, whole, roasted and	1 oz	28	160	1	5855	103	0	0
Seeds, sesame butter, tahini, from raw and	1 oz	28	162	1	5855	103	0	0
Nuts, pecans	1 oz (20 halves)	28	196	1	5848	280	0	0
Nuts, pine nuts, pignolia, dried	1 oz	28	160	1	5865	185	0	0
Nuts, hickorynuts, dried	1 oz	28	186	1	5847	297	0	0
Nuts, beechnuts, dried	1 oz	28	163	1	5214	482	0	0
Chickpeas (garbanzo beans, bengal gram),	1 cup	200	728	1	5186	202	0	0
Snacks, potato chips,	1 oz	28	151	1	4916	23	0	0
Snacks, corn-based, extruded, chips, plain	1 oz	28	153	1	4318	357	0	0
Nuts, pistachio nuts, dry roasted, without salt	1 oz (47 kernels)	28	162	1	3866	74	0	0
Snacks, taro chips	1 oz	28	141	1	3634	17	0	0
Nuts, almonds	1 oz (24 whole	28	164	1	3463	0	0	0
Snacks, trail mix, regular, with chocolate chips,	1 oz	28	137	1	3178	23	0	0
Nuts, hazelnuts or filberts, dry roasted, without	1 oz	28	183	1	2382	17	0	0
Nuts, cashew butter, plain, with salt added	1 oz	28	166	1	2315	48	0	0
Nuts, cashew nuts, dry roasted, without salt	1 oz	28	163	1	2172	46	0	0
Snacks, tortilla chips, nacho-flavor, made with	1 oz	28	141	1	964	40	0	0
Nuts, macadamia nuts, dry roasted, without salt	1 oz (10-12	28	204	1	369	56	0	0
Chicken, broilers or fryers, light meat, meat and	1 unit (yield from	79	175	1	1564	71	71	47
Chicken, broilers or fryers, dark meat, meat and	1 unit (yield from	101	256	1	3070	141	141	101
Turkey, all classes, light meat, meat and skin,	1 unit (yield from	136	268	1	2230	136	218	82
Turkey, all classes, dark meat, meat and skin,	1 cup, chopped or	140	309	1	3654	196	336	112
Beef, short loin, t-bone steak, separable lean	1 lb	454	599	1	1179	91	181	0
Pork, fresh, loin, center loin (chops), bone-in,	1 chop, excluding	74	149	1	370	15	30	0
Beef, ground, 90% lean meat / 10% fat, patty,	1 serving (3 oz)	85	173	1	240	41	37	0
Lamb, Australian, imported, fresh, leg, sirloin	3 oz	85	160	1	199	94	37	0

Omega-3

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Plant Omega-3 are NOT as Healthful Nor Useable as EPA/DHA in Humans

Dietary Omega-3 Fatty Acids, *Cyclooxygenase-2* Genetic Variation, and Aggressive Prostate Cancer Risk

Vincent Fradet,[1,3] Iona Cheng,[2,3] Graham Casey,[4] and John S. Witte[1,2,3]

Abstract

Purpose: Dietary intake of long-chain ω-3 (LC n-3) polyunsaturated fatty acids may reduce inflammation and in turn decrease risk of prostate cancer development and progression. This potential effect may be modified by genetic variation in *cyclooxygenase-2* (*COX-2*), a key enzyme in fatty acid metabolism and inflammation.

Experimental Design: We used a case-control study of 466 men diagnosed with aggressive prostate cancer and 478 age- and ethnicity-matched controls. Diet was assessed with a semiquantitative food frequency questionnaire, and nine *COX-2* tag single nucleotide polymorphisms (SNP) were genotyped. We used logistic regression models to estimate odds ratios (OR) for association and interaction.

Results: Increasing intake of LC n-3 was strongly associated with a decreased risk of aggressive prostate cancer ($P_{trend} \leq 0.0001$). The OR (95% confidence interval) for prostate cancer comparing the highest with the lowest quartile of n-3 intake was of 0.37 (0.25-0.54). The LC n-3 association was modified by SNP rs4648310 (+8897 A/G), flanking the 3′ region of *COX-2* ($P_{interaction} = 0.02$). In particular, the inverse association was even stronger among men with this variant SNP. This reflected the observation that men with low LC n-3 intake and the variant rs4648310 SNP had an increased risk of disease (OR, 5.49; 95% confidence interval, 1.80-16.7), which was reversed by increasing intake of LC n-3.

Conclusions: Dietary LC n-3 polyunsaturated fatty acids appear protective for aggressive prostate cancer, and this effect is modified by the *COX-2* SNP rs4648310. Our findings support the hypothesis that LC n-3 may impact prostate inflammation and carcinogenesis through the COX-2 enzymatic pathway.

Prostate cancer is one of the most common cancers in men (1) and in 2008 is projected to account for ~30% of the

inflammatory atrophy that may transition to prostatic intraepithelial neoplasia and eventually prostate adenocarcinoma (3).

Seed & Nut Omega-3's Do Not Possess the EPA / DHA Effect in Humans of Fish Oil

The American Journal of Clinical Nutrition

Dietary supplementation with eicosapentaenoic acid, but not with other long-chain n−3 or n−6 polyunsaturated fatty acids, decreases natural killer cell activity in healthy subjects aged >55 y[1–3]

Frank Thies, Gerhard Nebe-von-Caron, Jonathan R Powell, Parveen Yaqoob, Eric A Newsholme, and Philip C Calder

ABSTRACT

Background: Animal studies showed that dietary flaxseed oil [rich in the n−3 polyunsaturated fatty acid α-linolenic acid (ALA)], evening primrose oil [rich in the n−6 polyunsaturated fatty acid γ-linolenic acid (GLA)], and fish oil [rich in the long-chain n−3 polyunsaturated fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)] can decrease natural killer (NK) cell activity. There have been no studies of the effect on NK cell activity of adding these oils to the diet of humans.

Objective: Our objective was to determine the effect of dietary supplementation with oil blends rich in ALA, GLA, arachidonic acid (AA), DHA, or EPA plus DHA (fish oil) on the NK cell activity of human peripheral blood mononuclear cells.

Design: A randomized, placebo-controlled, double-blind, parallel study was conducted. Healthy subjects aged 55–75 y consumed 9 capsules/d for 12 wk; the capsules contained placebo oil (an 80:20 mix of palm and sunflower seed oils) or blends of placebo oil and oils rich in ALA, GLA, AA, DHA, or EPA plus DHA. Subjects in these groups consumed 2 g ALA, 770 mg GLA, 680 mg AA, 720 mg DHA, or 1 g EPA plus DHA (720 mg EPA + 280 mg DHA) daily, respectively. Total fat intake from the capsules was 4 g/d.

Results: The fatty acid composition of plasma phospholipids changed significantly in the GLA, AA, DHA, and fish oil groups. NK cell activity was not significantly affected by the placebo, ALA, GLA, AA, or DHA treatment. Fish oil caused a significant reduction (mean decline: 48%) in NK cell activity that was fully reversed by 4 wk after supplementation had ceased.

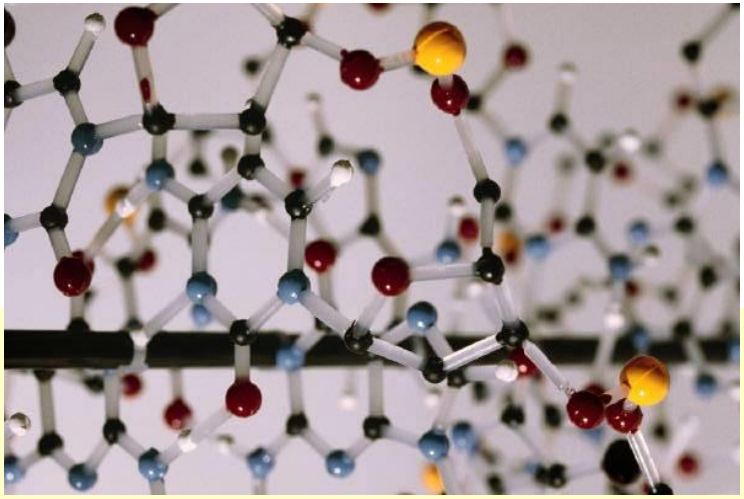
Conclusion: A moderate amount of EPA but not of other n−6 or n−3 polyunsaturated fatty acids can decrease NK cell activity in healthy subjects. *Am J Clin Nutr* 2001;73:539–48.

response to virus-infected and tumor cells and play a role in graft rejection (1). NK cell activity declines with age in both humans and laboratory animals (2, 3).

A reduction in fat intake (to <30% or 22% of total energy) is associated with a significant increase in the NK cell activity of human peripheral blood mononuclear cells (PBMCs; a mixture of lymphocytes including NK cells and monocytes) (4, 5), suggesting that high fat consumption suppresses NK cell activity. This is supported by some studies in laboratory rodents (6) but not by others (7). Animal feeding studies show that the type of fatty acid in the diet affects NK cell activity; n−3 polyunsaturated fatty acids (PUFAs) appear to have particularly potent effects. Increasing the amount of α-linolenic acid (ALA; 18:3n−3) in a rat diet lowers spleen NK cell activity compared with that measured after feeding diets rich in linoleic acid (8, 9). Feeding rats or mice diets containing large amounts of fish oil [FO; which is rich in eicosapentaenoic acid (EPA; 20:5n−3) and docosahexaenoic acid (DHA; 22:6n−3)] results in suppressed spleen NK cell activity compared with that measured after feeding low-fat diets or high-fat diets rich in saturated fat or n−6 PUFAs (10–15). Feeding rats diets rich in γ-linolenic acid (GLA; 18:3n−6) also lowers spleen NK cell activity compared with that measured after feeding some other diets (13, 14). These studies are supported by cell culture studies in which human NK cell activity was suppressed by culture of the cells with GLA (16, 17), EPA (17–19), and DHA (17–19).

Despite these cell culture and animal feeding studies, few investigations have been conducted on the effect of dietary fatty

[1]From the Department of Biochemistry, University of Oxford, Oxford, United Kingdom; Unilever Research Colworth Laboratory, Sharnbrook, United



In the Beginning,

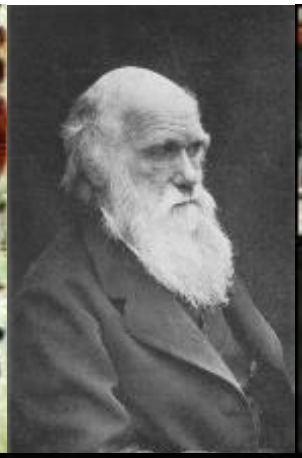
NutriGenomics

was All About

Omega-6 Plant Oil vs Omega 3

From the Beginning, It Appears that We Humans were Either

- ***Created in a Perfect Environment, or***
- ***Evolved in time in an Evolving Environment***



Nutrition *Ruled* **Genes from Start**

Stability Prevailed from Beginning

- **People Lived and Died in Same Area**
  - Movements with Herds
  - Ate with Seasons Each Year
- **Nutrient Stable Foods for Lifetime**
  - Foods were all Primary Foods
  - No Added-Ingredient Foods
  - B Vitamins = 100%
  - Minerals Came in Water /Foods
- **Essential Oils Omega 3:6:9**
  - @ 1:1:1 ratio
- **Eating was Essential,**
  - Cut Corners and We Died
- **Nutrition Healthy Mothers Conceive**
  - No Doctors, Drugs, or Surgeries

Until Civilization Changed in

- **1492 Lead to Need for**
  – People Transported to New Food
- **1909 to 1940**
  - Plant Oil (omega-6) 1st refined
  - 1943 = Multiple Sclerosis Dxd
- **1950** – 7 Nutrients Lost in Foods
- **1959 to 1975 to *Today***
  - Latex in foods ethylene ripened are Bananas, Kiwi, Avocados, Figs
  - Rubber Tires fumes = Latex inhaled from Rubber Tires
  - Latex-in-Food Sensitivity Disorder
- **1980** – Mg/Ca lost Water/Foods
- **1990** - Melanoma Scare (Vit D)
- **2005** = Begins ***Nutri*Genomics**

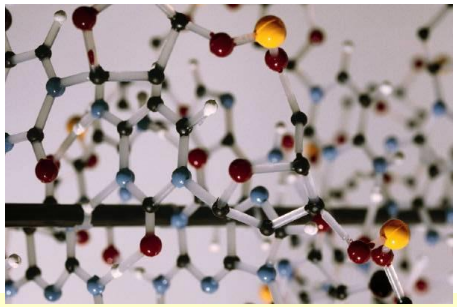
NutriGenomics = "Gene Nutrition"

***What We Eat* Impacts *Our Genes*;**

- **Optimal Health** by **Eating Genomically**
- ***Lab Tests Improve*** to Optimal Levels
  - Symptoms Decrease and Leave
  - Diseases Quiet Down and Disappear
- **Optimizing our Genes' Functioning** by
  - **Improving our Nutritional Food Quality**
  - Removing Toxic Foods,
  - Reducing Excess Foods
  - Restoring Healthful Balance in our Lives

NutriGenomic Personalized Health Care *is* *All About You*

- ***Personalized*** = **Based on *Your Genes***
- ***Participatory*** = ***Depends on You***
- ***Preventive*** = **Prevention *First***
- ***Predictive*** = **Know Outcomes *First***
- ***Precise*** = ***Need Accurate Diagnosis***



Very Quickly,
NutriGenomics went beyond
Omega-6 Plant Oil vs Omega-3
To Minerals Loss in Soft Water
To 6 Nutrients Loss in 43 Foods
To Vitamin D (-) Glass Windows

What Happened to our Western Food Supply in Last 10,000 Years

The American Journal of Clinical Nutrition

Origins and evolution of the Western diet: health implications for the 21st century[1,2]

Loren Cordain, S Boyd Eaton, Anthony Sebastian, Neil Mann, Staffan Lindeberg, Bruce A Watkins, James H O'Keefe, and Janette Brand-Miller

ABSTRACT

There is growing awareness that the profound changes in the environment (eg, in diet and other lifestyle conditions) that began with the introduction of agriculture and animal husbandry ≈10 000 y ago occurred too recently on an evolutionary time scale for the human genome to adjust. In conjunction with this discordance between our ancient, genetically determined biology and the nutritional, cultural, and activity patterns of contemporary Western populations, many of the so-called diseases of civilization have emerged. In particular, food staples and food-processing procedures introduced during the Neolithic and Industrial Periods have fundamentally altered 7 crucial nutritional characteristics of ancestral hominin diets: *1*) glycemic load, *2*) fatty acid composition, *3*) macronutrient composition, *4*) micronutrient density, *5*) acid-base balance, *6*) sodium-potassium ratio, and *7*) fiber content. The evolutionary collision of our ancient genome with the nutritional qualities of recently introduced foods may underlie many of the chronic diseases of Western civilization. *Am J Clin Nutr* 2005;81:341–54.

KEY WORDS Westernized diets, chronic disease, processed foods, genetic discordance, hunter-gatherers, human evolution

environment that their ancestors survived in and that consequently conditioned their genetic makeup (1–3). There is growing awareness that the profound environmental changes (eg, in diet and other lifestyle conditions) that began with the introduction of agriculture and animal husbandry ≈10 000 y ago occurred too recently on an evolutionary time scale for the human genome to adapt (2–5). In conjunction with this discordance between our ancient, genetically determined biology and the nutritional, cultural, and activity patterns in contemporary Western populations, many of the so-called diseases of civilization have emerged (2–12).

CHRONIC DISEASE INCIDENCE

In the United States, chronic illnesses and health problems either wholly or partially attributable to diet represent by far the most serious threat to public health. Sixty-five percent of adults aged ≥20 y in the United States are either overweight or obese (13), and the estimated number of deaths ascribable to obesity is 280 184 per year (14). More than 64 million Americans have one or more types of cardiovascular disease (CVD), which represents the leading cause of mortality (38.5% of all deaths) in the United

What Happened to The U.S. Food Supply;
Loss of Nutrients in the Last 60 Years?

Original Research

Changes in USDA Food Composition Data for 43 Garden Crops, 1950 to 1999

Donald R. Davis, PhD, FACN, Melvin D. Epp, PhD and Hugh D. Riordan, MD

Bio-Communications Research Institute, Wichita, Kansas (D.R.D., M.D.E., H.D.R.), Biochemical Institute, The University of Texas, Austin, Texas (D.R.D.)

Key words: Nutritive value, history, food analysis, agriculture

Objectives: To evaluate possible changes in USDA nutrient content data for 43 garden crops between 1950 and 1999 and consider their potential causes.

Methods: We compare USDA nutrient content data published in 1950 and 1999 for 13 nutrients and water in 43 garden crops, mostly vegetables. After adjusting for differences in moisture content, we calculate ratios of nutrient contents, R (1999/1950), for each food and nutrient. To evaluate the foods as a group, we calculate median and geometric mean R-values for the 13 nutrients and water. To evaluate R-values for individual foods and nutrients, with hypothetical confidence intervals, we use USDA's standard errors (SEs) of the 1999 values, from which we generate 2 estimates for the SEs of the 1950 values.

Results: As a group, the 43 foods show apparent, statistically reliable declines ($R < 1$) for 6 nutrients (protein, Ca, P, Fe, riboflavin and ascorbic acid), but no statistically reliable changes for 7 other nutrients. Declines in the medians range from 6% for protein to 38% for riboflavin. When evaluated for individual foods

Deficiencies Exist in All 43 Garden Crops

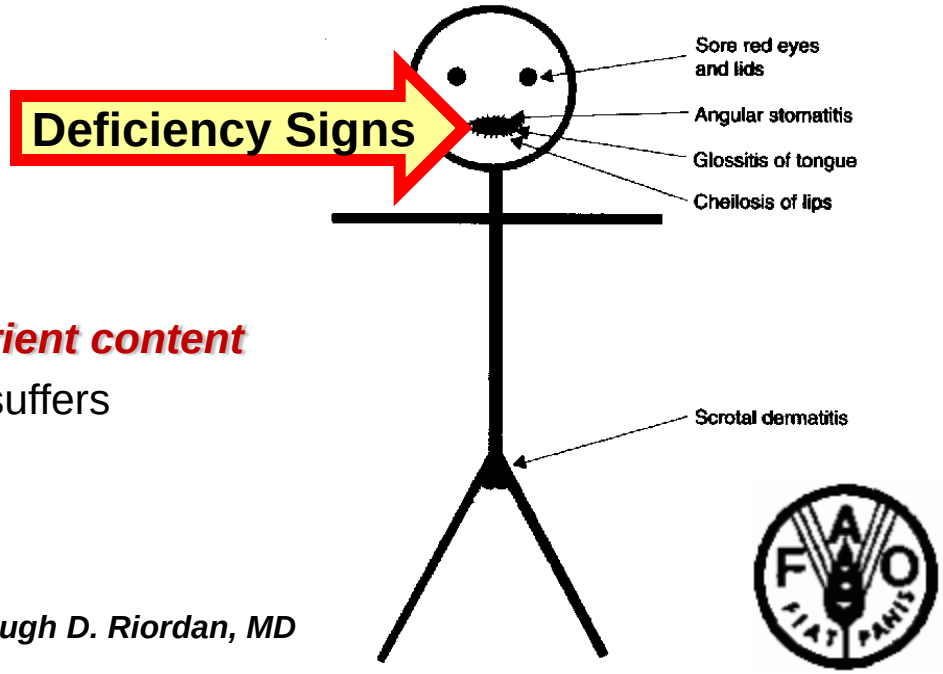
USDA Food Composition Data, 1950-1999

All 43 fruits / vegetables had declines for 6 nutrients:

- Calcium, **16%**
- Phosphorus, **9%**
- Iron, **15%**
- Vitamin C **16%**
- Protein, **6%**
- **Riboflavin 38%**

Conclusions:

- Declines changes in cultivated varieties between 1950 and 1999, ***trade-off between yield and nutrient content***
- Large percents of the population suffers from deficiency disorders and symptoms of B vitamin deficiency



Donald R. Davis, PhD, FACN, Melvin D. s, PhD and Hugh D. Riordan, MD

What B-Complex Deficiencies Look Like

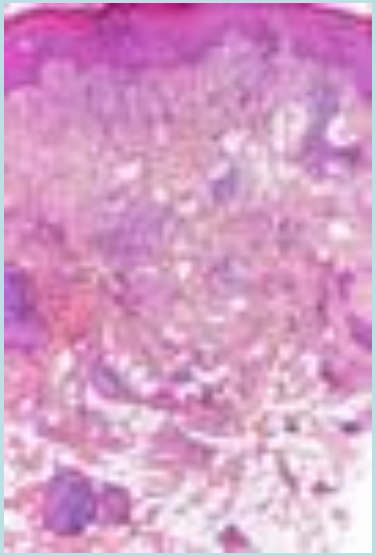
CHEILITIS, ACTINIC / CHEILITIS/RHITIDES / WRINKLES











epithelioid cell granulomas with focal accumulations of lymphocytes and plasma cells

http://dermatlas.med.jhmi.edu/derm/resultNoCache.cfm

What B-Complex Deficiencies Look Like

Glossitis, Geographic Tongue

What Happened to America's Water Supply in the Last 60 Years

Chemical Sensitivity Due to Latex in Fruits / Vegetables

after exposure to Auto-tire Inhalation

In Nature, since time began;

- **Fruits & Vegetables store proteins and carbohydrates as Latex = protection**
- **Latex is naturally converted by plant hormone, ethylene, to protein & carbohydrates when fruit or vegetable ripens on the plant naturally**

In 1959, A Lime grower in southern France noticed that all stages of fruit ripened when exposed to kerosene fumes (while protecting orchards from frost)

Since 1960, fruits (and vegetables) have been picked green to better stand shipping stress

- Latex is trapped in the fruits and vegetables when picked green to ship
- Fruits / Vegetables become hyper-sensitizing when treated with ethylene gas in grocery warehouse process since 1959,
- Dried, frozen, canned foods usually have less latex *since processed where they grow* ***Since 1979*** **NIEH reports >75% of US population have Auto Tire fumes Latex sensitivity** Symptoms of stiffness, fatigue, aches, redness on chin, cheeks, ears, forehead, chest, plus burning eyes and itchy non-healing skin ***(persons exposed to tire fumes)*** **worsened by eating foods *laden with naturally-occurring latex, such as Bananas, Avocado, Celery, Kiwi and Figs, plus stone fruits out of season which depress the immune system and magnifying pain and fatigue***

Reference:

Gluten Free Diet is Best for Most

HLA DQ 2,8 genetic screening is 97% accurate ***Screening and diagnosis for People with celiac disease:***

Complications of Untreated Celiac Disease:

- **Malnutrition** in spite of an adequate diet vital nutrients are lost in the stool rather than absorbed in the bloodstream, causing a deficiency in vitamins A, B-12, D, E and K, and folate, resulting in anemia, weight loss, and stunted growth and developmental delays
- **Loss of calcium and bone density** from continued loss of fat, calcium and vitamin D lost in the stool resulting osteomalacia, a softening of the bone also known as rickets in children, and loss of bone density (osteoporosis)
- **Kidney stones** (calcium oxalate stone)
- **Lactose intolerance** from damage to your small intestine from gluten foods and cause abdominal pain and diarrhea from other foods and lactose in dairy products. Once healed, you may be able to tolerate dairy products again
- **Cancers** in celiac disease patients not maintain a gluten-free diet, especially intestinal lymphoma and bowel cancer
- **Neurological complications** seizures (epilepsy), nerve damage (peripheral neuropathy)

Treatment of Celiac disease:

- Manage the disease by removing gluten from your diet, Vitamin and mineral supplements to correct deficiencies
- Avoiding gluten is essential, these grains contain gluten:
  - **Wheat (including farina, graham flour, semolina and durum), barley and rye**
  - **Barley, rye, bulgur, Kamut**
  - **Kasha, matzo meal**
  - **Spelt and triticale**
  - **Amaranth, buckwheat and quinoa**
  - **Corn and Oats** now know to contain Gluten-like prolamins

Basic gluten-free diet: lamb, pork, beef (not breaded or marinated), wild fish, omega-3 eggs, pure dairy products, Fruits, Vegetables, Rice, Potatoes, flours (rice, potato)

Avoid Breads, Cereals, Crackers, Pasta, Cookies, Cakes, Pies, Gravies, Sauces

Note: Rice cereals are usually OK, white cheddar cheese rice crackers are OK

Read food labels: adapt your favorite recipes and bake the food longer at a lower temperature.

For 1 tablespoon of wheat flour, substitute:	**For 1 cup of wheat flour, substitute:**
1 1/2 teaspoons potato starch	**5/8-cup potato flour**
1 1/2 teaspoons arrowroot starch	
1 1/2 teaspoons rice flour	**3/4-cup rice flour**
2 teaspoons quick-cooking tapioca	

- **Additional Information:** Celiac Sprue Association/United States of America http://www.csaceliacs.org and search for "recipes" The Mayo Clinic, http://www.mayoclinic.com and search for "celiac disease"

Gluten Free Diet is Now Oats Free

Open access, freely available online PLoS MEDICINE

The Molecular Basis for Oat Intolerance in Patients with Celiac Disease

Helene Arentz-Hansen[1], Burkhard Fleckenstein[1,2], Øyvind Molberg[1], Helge Scott[3], Frits Koning[4], Günther Jung[5], Peter Roepstorff[2], Knut E. A. Lundin[1,6], Ludvig M. Sollid[1*]

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LMS is a member of the editorial board of *PLoS Medicine*.

Author Contributions: ØM, KEAL, and LMS designed the study. HAH, BF, ØM, HS, FK, GJ, PR, KEAL, and LMS analyzed the data. FK and GJ contributed synthetic peptides for the study. HAH, BF, ØM, HS, FK, GJ, KEAL, and LMS contributed to writing the paper.

Academic Editor: Marco Londei, University College London, United Kingdom

Citation: Arentz-Hansen H, Fleckenstein B, Molberg Ø, Scott H, Koning F, et al. (2004) The molecular basis for oat intolerance in

ABSTRACT

Background

Celiac disease is a small intestinal inflammatory disorder characterized by malabsorption, nutrient deficiency, and a range of clinical manifestations. It is caused by an inappropriate immune response to dietary gluten and is treated with a gluten-free diet. Recent feeding studies have indicated oats to be safe for celiac disease patients, and oats are now often included in the celiac disease diet. This study aimed to investigate whether oat intolerance exists in celiac disease and to characterize the cells and processes underlying this intolerance.

Methods and Findings

We selected for study nine adults with celiac disease who had a history of oats exposure. Four of the patients had clinical symptoms on an oats-containing diet, and three of these four patients had intestinal inflammation typical of celiac disease at the time of oats exposure. We established oats-avenin-specific and -reactive intestinal T-cell lines from these three patients, as

Gluten Free Diet is Now Corn Free

Plant Foods Hum Nutr (2012) 67:24–30
DOI 10.1007/s11130-012-0274-4

ORIGINAL PAPER

Maize Prolamins Resistant to Peptic-tryptic Digestion Maintain Immune-recognition by IgA from Some Celiac Disease Patients

Francisco Cabrera-Chávez · Stefania Iametti · Matteo Miriani · Ana M. Calderón de la Barca · Gianfranco Mamone · Francesco Bonomi

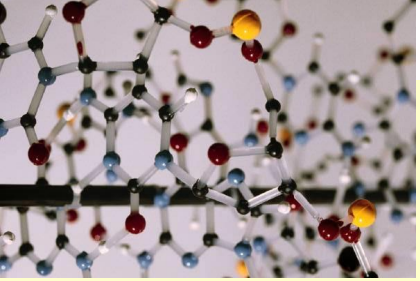
Published online: 2 February 2012

Abstract Maize is used as an alternative to wheat to elaborate foodstuffs for celiac patients in a gluten-free diet. However, some maize prolamins (zeins) contain amino acid sequences that resemble the wheat gluten immunodominant peptides and their integrity after gastrointestinal proteolysis is unknown. In this study, the celiac IgA-immunoreactivity to zeins from raw or nixtamalized grains, before and after peptic/tryptic digestion was evaluated and their possible immunogenicity was investigated by *in silico* methods. IgA from some celiac patients with HLA-DQ2 or DQ8 haplotypes recognized two alpha-zeins even after peptic/tryptic proteolysis. However, digestion affected zeins after relative abundance of these zeins, along with factors affecting their resistance to proteolysis, may be of paramount clinical relevance, and the use of maize in the formulation and preparation of gluten-free foods must be reevaluated in some cases of celiac disease.

Keywords Celiac disease · IgA-reactivity · Zeins

Abbreviations

CD	Celiac disease
HLA	Human leukocyte antigen
HRP	Horse radish peroxidase



Over Time,
Wee *NutriGenomics*
Focused on Identifying How
your symptoms were Caused by;

Omega-6 Plant Oil versus Omega 3

Calcium, Magnesium missing in Waters

B-Vitamins missing in 43 Fruits/Vegies

Vitamin D avoided/missing in Region

Gluten blocking Cal Mag D and Bs

Latex sensitization from Tire Fumes, plus

Bananas, Avocados, Celery, Kiwi, Figs, +8

How Wee Start Looking for Opportunity

- ***Integrative Medicine First Visit Intake by Dr. Aukerman***
  - Ancestral History, ***Where were Yours 500 years ago?***
  - Personal, Family Medical/Social History, ***Conditions?***
  - Current Medical Concerns, ***this Week, Month, Year?***
  - Behavioral Learning Style,
    - ***How do you learn best?***
    - ***What is your Readiness for Change?***
    - ***What Opportunities for Change do you have?***
  - ***Exploring Your Interests in Health Improvements***
    - Flax, Soy, Latex Foods, Loyalty Issues with Products
    - Essential Classes, Workshops, + Self-help Programs
    - Lab Tests Targeted for Improvements in Your Health
- ***Clinical Signs Assessment by Doctor/CNP***
  - **Past + Current Problems, Concerns and Issues ?**
  - **Nutritional markers** = ***Skin, Lips, Tongue, Reflexes, Abdomen?***
  - **Functional Fitness Level**, ***this month, year, decade ?***
  - **NutriGenomic Medicine Rx**, *approach that is evidence-based*

Where Wee Lab Tests Identify Opportunities

Looking for Health NutriGenomic 360 Labs:

- *Cal Mag D, PTH, Lipid Profile, CrProtein* to identify your nutrient deficit impacts lost from our water, foods and your carbohydrate excesses
- *CBC, differential* assess plant oil excess
- *Gluten-based lab tests* reflect impacts by
  - *Celiac Genes,TPO,Anti-gliadin antibodies*
  - *ANA, ESR, auto-immune lab profile*
- *Specific Labs* identified in your evaluation
- *Tumor Disease Markers* related to history

How Wee Works with You over Time

***6-8 weeks after 1st Visit**, Doctor Follows-up;*

- *To Get More From Your Efforts using Wee NutriGenomics*
  - **To better understand your personal plan for health**
  - **To better understand your personal lab results**
  - **To be certain you are taking the corrective nutrients**
  - **To improve your diet by removing gluten and plant oil**
  - **To improve your outcomes while reducing your costs**
- **Thereafter,**
  - **Each 6-8-12 weeks, Follow-up Labs show progress and options**
  - **Every 120 days, Follow-up with Doctor to keep on track**
  - **Doctor Visits as often as needed to correct your course**

(Genotype) = Your Unique Genes makes you Special !

However, Your Diet Makes You Average = (Phenotype)

NutriGenomic Performance Depends on the WEE Nutrition Provided to Your Unique Gene's

To Be Healthy and a Survivor Requires *The Wee Simple Diet as Wee Walk You Back in Time to Health*

- Need Lose the Gluten, Plant oil, Xs Carbs /Protein
  - If you are to be a healthy survivor
- Work with Us to go ***A Wee Simple Diet***
  - Until at least in a full remission
- ***Simpler is always Better, in all ways!***
  - No Cheating by the Body
  - No Remorse by the Patient
  - No Symptoms, No Disease, No Cancer

Wee Simple Diet = **4 Things** + 1 Rule

Four Wee Things:

- **Protein** <150% RDA / day by Using ***KIM-2 program***
  - Enough Dairy, Red Meat, Egg, Fish, Beans <u>to be Full</u>
- **Complex Carbs** PLUS **Cinnamon**
  - Enough Rice and Potato <u>to be Full</u> (No Corn, Oats, Soy, Flax)
- Fats = ***Triple Strength Omega-3 >*** *Omega-6 Plant Oil*
  - Enough Dairy, Red Meat, Eggs, Fish <u>to be Full</u>
- Avoid Gluten since Blocks Cal Mag D + B Complex
  - Avoid Like the Plague, Go to potato, rice, tapioca starches
- Body's One Rule is ***<u>No Cheating</u>***

Best Nutrigenomic Approaches

To remove Toxic Foods in Your

Diet and Environment

Natural Standard
The Authority on Integrative Medicine

The Real Facts About Flax

ConsumerLab.com

- **Flaxseed and flaxseed (linseed) oil are rich sources of the essential fatty acid, alpha-linolenic acid**
  - Alpha-linolenic acid is a building block in the body for omega-3 fatty acids in most animals, but ***only 0.2% to 8 % can be converted due to the human lack of elongase enzyme in the liver versus rats***
- **Safety**:
  Avoid flax or flax oil with prostrate cancer, breast cancer, uterine cancer or endometriosis.
  Avoid flaxseed (*not* flaxseed oil) if history of esophageal stricture, ileus, gastrointestinal stricture, bowel obstruction, acute or chronic diarrhea, irritable bowel syndrome, diverticulitis or inflammatory bowel disease.
- **Caution if history** of a bleeding disorder or with drugs that cause bleeding risk (like anticoagulants and Non-steroidal Anti-inflammatories (like aspirin, warfarin, Advil®), high triglyceride levels, diabetes, mania, seizures or asthma. Avoid if pregnant or breastfeeding.
  Side effects: Skin rash, itching, watery eyes, stuffy nose, sneezing, shortness of breath, low blood pressure, nausea, difficulty breathing, vomiting, stomach discomfort, diarrhea, increased bowel movements, mania and other moods, blocked intestines, changes in blood sugar levels, hormone changes, increased risk of bleeding, prostate cancer.
  Drug interactions: Drugs used to treat constipation, stool softeners, drugs that increase the risk of bleeding, thyroid drugs, drugs that lower cholesterol, mental illness drugs (like lithium), blood pressure drugs, drugs used to control blood sugar, and herbs or supplements with similar effects
- **Conversion of ALA to EPA in humans ranges from a low of 0.2% to a high of 8%.**
  - **40-fold difference in these conversion rates may be due to differences in study methods, study populations, and background diets when high in omega-6 plant oil**
- **Amount of ALA converted to DHA in humans remains controversial, with some studies showing small amounts converted to DHA, and other studies showing virtually no conversion to DHA**
- **Factors Affecting ALA Conversion.** Diet composition has long been known to affect ALA conversion. New research suggests that other factors also affect the conversion rate
  - **Gender, Smoking**
  - **Diets high in ALA decrease conversion by 40%, so the more omega-6 in the diet the less conversion**

Natural Standard
The Authority on Integrative Medicine

ConsumerLab.com

Soy and Soy Milk, NOT Safe in Women

Soy, the Good:

Soy protein reduces menopausal "hot flashes," total cholesterol and increases HDL ("good" cholesterol), and is a low fat source of protein

Soy, the Bad: **Soy isoflavones may not be safe for women with estrogen receptor-positive breast cancer or for pregnant or nursing women**

Researchers at the University of Toronto explored the effect of flaxseed and soy protein diets on human breast cancer tumor growth, while several epidemiological studies suggest that a phytoestrogen-rich diet containing lignans and isoflavones may reduced breast cancer risk

- In postmenopausal mammary cancer xenograft model, soy protein (SP), a rich source of isoflavones, enhanced breast cancer.
- **Intake of phytoestrogens is increasing particularly among postmenopausal women, emphasizing the importance of clarifying their interactive effects on breast cancer**
- Soy Protein initially regressed the tumors but **starting at week 13, the tumors regressed significantly less than in the control and 43 percent of the tumors were re-growing until the end of the experiment and were significantly larger in size than in the control**
- Long-term consumption of Soy Protein did stimulate the growth of estrogen responsive MCF-7 cancers in ovariectomized mice
- **Tumor growth stimulating effect of soy protein in ovariectomized athymic mice with MCF-7 human breast cancer xenografts. Saarinen NM, Int J Cancer. 2006 Aug 15;119(4):925-31**

Soy and Soy Milk, NOT Safe in Families

How this looks in a Modern **Soy Eating Family**

- **Dad L 54 year old male** Abnormal Stress Test Forearm Pain Knee Pain Leg Cramps Lipids abnormal Other abnormal blood chemistry Other and Unspecified Hyperlipidemia Family History of Coronary Artery Disease Family History of Stroke

ESTRONE, SEND OUT	No range found	**62... (H)**
GLIADIN AB, IGA	No range found	5.2...
GLIADIN AB, IGG	No range found	2.0...
Tissue transglutaminase, IgG	No range found	23.9... (H)

- **Mom F 34 year old female** Dysplasia of Cervix Suppressed periods Aches Hyperthyroidism Heart Murmur

MAGNESIUM,SERUM	Latest Range: 1.6-2.6 mg/dL	2.1
CALCIUM	Latest Range: 8.6-10.0 mg/dL	9.2
VITAMIN D 25 HYDROXY	Latest Range: 30-100 ng/mL	27.6 (L)
PTH INTACT	Latest Range: 14.0-72.0 pg/mL	61.7
TSH, HIGH-SENSITIVITY	Latest Range: 0.550-4.780 uIU/mL	14.268 (H)
THYROGLOBULIN (NEW)	Latest Range: 1.6-59.9 ng/mL	73.9 (H)
GLIADIN AB, IGA	No range found	1.6...
GLIADIN AB, IGG	No range found	1.0...

- **F, 12 year old female** Contact dermatitis and other eczema, czema Food allergy Pain in joint, ankle,foot Sleep related leg cramps

MAGNESIUM,SERUM	Latest Range: 1.6-2.2 mg/dL	2.0
CALCIUM	Latest Range: 8.6-10.2 mg/dL	9.5
VITAMIN D 25 HYDROXY	Latest Range: 30-100 ng/mL	20.7 (L)
ESTRADIOL	Latest Range: 0.3-20.6 pg/mL	**96.6 (H)**
ESTRADIOL, ENHANCED	No range found	117.9 (H)
IGE	Latest Range: 0-397 IU/mL	496.0 (H)

- **T, 13 year old female** Anxiety state, unspecified Eczema

MAGNESIUM,SERUM	Latest Range: 1.6-2.3 mg/dL	1.9
CALCIUM	Latest Range: 8.4-10.0 mg/dL	9.4
VITAMIN D 25 HYDROXY	Latest Range: 30-100 ng/mL	27.8 (L)
ESTRADIOL	Latest Range: 0.3-33.2 pg/mL	**82.9 (H)**
ESTRADIOL, ENHANCED	Latest Range: 0.3-33.2 pg/mL	**78.9 (H)**
ELIAC GENE PAIRS PRESENT?	No range found	Yes

- **F, 5 year old male** Autism Unspecified vitamin D deficiency
- **A, 7 year old male** Asthenia Reflux Tinea pedis Unspecified vitamin D deficiency

Why Omega-3 Eggs are NOT Healthful

- It is of interest to note that the egg enrichment with DHA, an important fatty acid for human health, was very effective in hens consuming canola seed and was only lower by 12 mg/60 g of egg (i.e., **82.9 vs. 95.0 mg/60 g of egg**) from that of flaxseed and Linpro (Table 5).
- This could indicate that DHA deposition reaches the plateau at a much lower concentration of dietary n-3 fatty acids than that corresponding to the 15% inclusion rate of flaxseed. This is further substantiated by the fact that no difference in DHA deposition was observed between the flaxseed and the Linpro diets, both products included in the diets at 15% but Linpro contributing only 7.5% of the flaxseed.
- This finding is consistent with earlier studies indicating that the increase in EPA and DHA was not proportional to the level of ALA, which increased significantly in the eggs from hens fed flaxseed (Caston and Leeson, 1990; Cherian and Sim, 1991; Aymond and Van Elswyk, 1995).
- The poor conversion rate of ALA to DHA probably relates to the complexity of DHA biosynthesis. Recent studies have shown that the biosynthesis of polyunsaturated fatty acids is more complex than previously recognized, because enzymes from more than one intracellular compartment are required for the synthesis of 22-carbon polyunsaturated fatty acid with their first double bond at position 4 in a partial degradation-resynthesis cycle (Sprecher, 2000).
- **Biochimica et Biophysica Acta (BBA) - Molecular and Cell Biology of Lipids Volume 1486, Issues 2-3, 19 July 2000, Pages 219-231** **Review** Metabolism of highly unsaturated *n-3 and n-6 fatty acids* **Howard Sprecher**, Department of Molecular and Cellular Biochemistry, The Ohio State University, 337 Hamilton Hall, 1645 Neil Avenue, Columbus, OH 43210, USA

The Effect of Diet and Enzyme Supplementation on Egg Saturated, Monounsaturated, n-3, n-6, and n-6:n-3 Ratio (mg/60 g of egg) IS Not Useful Omega-3 for Humans

Table 6. The effect of diet and enzyme supplementation on egg saturated, monounsaturated, n-3, n-6, and n-6:n-3 ratio (mg/60 g of egg)

Effect	SFA[1]	MUFA[2]	n-3[3]	n-6[4]	n-6:n-3
Canola seed	1,379.06	2,197.26	211.49	761.51	3.60
Canola seed + enzyme	1,379.58	2,254.14	203.12	751.50	3.70
Flaxseed	1,457.13	1,870.53	545.73^{b}	725.93	1.33
Flaxseed + enzyme	1,493.35	1,857.21	578.04^{a}	733.32	1.27
Linpro	1,482.16^{b}	1,932.28^{b}	415.44^{b}	720.97	1.74
Linpro + enzyme	1,547.82^{a}	2,013.78^{a}	438.48^{a}	734.49	1.67
Pooled SEM	18.21	21.78	6.69	15.00	0.03
Diet					
Canola seed	1,379.32^{b}	2,225.70^{a}	207.31^{c}	756.51	3.65^{a}
Flaxseed	1,475.24^{a}	1,863.87^{c}	561.88^{a}	729.63	1.30^{c}
Linpro	1,514.99^{a}	1,973.03^{b}	426.96^{b}	727.73	1.71^{b}
Pooled SEM	12.88	15.40	4.73	10.61	0.02
Diet	<0.0001	<0.0001	<0.0001	0.1425	<0.0001
Enzyme	0.0405	0.0371	0.0142	0.7717	0.7427
Diet x enzyme	0.2412	0.1193	0.0252	0.7246	0.0472

Looks good at first

$^{a–c}$Means within a column and within a source with no comm… *P* < 0.05).

[1]SFA = saturated fatty acids; include $C_{14:0}$, $C_{15:0}$, $C_{16:0}$, $C_{18:0}$, $C_{20:0}$, C_{2}…

[2]MUFA = monounsaturated fatty acids; include $C_{14:1}$, $C_{16:1}$, $C_{18:1}$, $C_{20:1}$, $C_{22:}$…

[3]n-3 fatty acids; include $C_{18:3n-3}$, $C_{18:4n-3}$, $C_{20:3n-3}$, $C_{20:4n-3}$, $C_{20:5n-3}$, $C_{22:5n-3}$, and $C_{22:6n-3}$.

[4]n-6 fatty acids; include $C_{18:2n-6}$, $C_{18:3n-6}$, $C_{20:2n-6}$, $C_{20:3n-6}$, $C_{20:4n-6}$, $C_{22:2n-6}$, $C_{22:4n-6}$, and $C_{22:5n-6}$.

The Effect of Diet and Enzyme Supplementation on Egg Fatty Acid Composition (mg/60 gm egg)

Table 5. The effect of diet and enzyme supplementation on egg fatty acid composition (mg/60 g of egg)

Effect	Palmitic ($C_{16:0}$)	Palmitoleic ($C_{16:1}$)	Stearic ($C_{18:0}$)	Oleic ($C_{18:1}$)	Linoleic ($C_{18:2n-6}$)	Linolenic ($C_{18:3n-3}$)	Arachidonic ($C_{20:4n-6}$)	EPA1 ($C_{20:5n-3}$)	DHA[2] ($C_{22:6n-3}$)	TFA[3]
Canola seed	1,043.11	89.15	318.41	2,085.96	674.83	115.76	68.97	2.17	81.87	4,549.32
Canola seed + enzyme	84.00	1,028.78	89.34	331.92	2,142.91	662.77	105.85	70.85	1.90	4,588.34
Flaxseed	1,046.53	112.71	391.86	1,742.29	667.14	419.88	44.20	8.46	91.82[b]	4,599.32
Flaxseed + enzyme	101.89[a]	1,068.46	112.16	405.08	1,729.81	672.56	438.06	45.97	8.87	4,661.91
Linpro	1,080.91	125.36	381.02[b]	1,792.36[b]	656.93	297.72	48.97	6.51[a]	89.43[b]	4,550.85[b]
Linpro + enzyme	96.80[a]	1,117.72	124.26	409.07[a]	1,875.72[a]	668.00	311.36	50.96	7.20[b]	4,734.56[a]
Pooled SEM	14.95	2.72	5.67	20.06	14.18	6.08	0.95	0.16	1.82	47.41
Diet										
Canola seed	82.94[b]	1,035.95[b]	89.25[c]	325.17[b]	2,114.43[a]	668.80	110.81[c]	69.91[a]	2.03[c]	4,568.83
Flaxseed	1,057.50[b]	112.44[b]	398.47[a]	1,736.05[c]	669.85	428.97[a]	45.08[c]	8.67[a]	96.85[a]	4,630.62
Linpro	1,099.32[a]	124.81[a]	395.05[a]	1,834.04[b]	662.47	304.54[b]	49.97[b]	6.85[b]	93.12[a]	4,642.71
Pooled SEM	10.57	1.92	4.00	14.19	10.03	4.30	0.67	0.11	1.29	33.52
Diet	0.0036	<0.0001	<0.0001	<0.0001	0.8551	<0.0001	<0.0001	<0.0001	<0.0001	0.2848
Enzyme	0.2485	0.8294	0.0019	0.0232	0.9005	0.1673	0.0328	0.0568	0.0009	0.0302
Diet x enzyme	0.1279	0.2523	0.9718	0.3580	0.0852	0.7041	0.0837	0.9930	0.0306	0.2982

[a–c]Means within a column and within a source with no common superscript differ sign

[1]EPA = eicosapentaenoic acid.

[2]DHA = docosahexaenoic acid.

[3]TFA = total fatty acids.

Useful to Rats

Useful to Humans

How Cal Mag D *Helps* Your Body

1. Where you are the Sickest
2. Cholesterol, LDL
3. Osteoporosis = intact PTH
4. Cal Mag D Levels
5. Cancer Protections
6. Other 321 functions of Cal Mag D

How Cinnamon *Helps* Your Body

1. Where you are the Sickest
2. Triglycerides
3. HgbA1c and post-meal blood sugars
4. Cancer growth protection
5. Obesity
6. Energy

Triple Strength **Fish Oil** *Helps* Body

1. Where you are the Sickest
2. All 4 Cholesterols
3. Obesity
4. Mood
5. Cancer quieting down
6. Many other areas of body

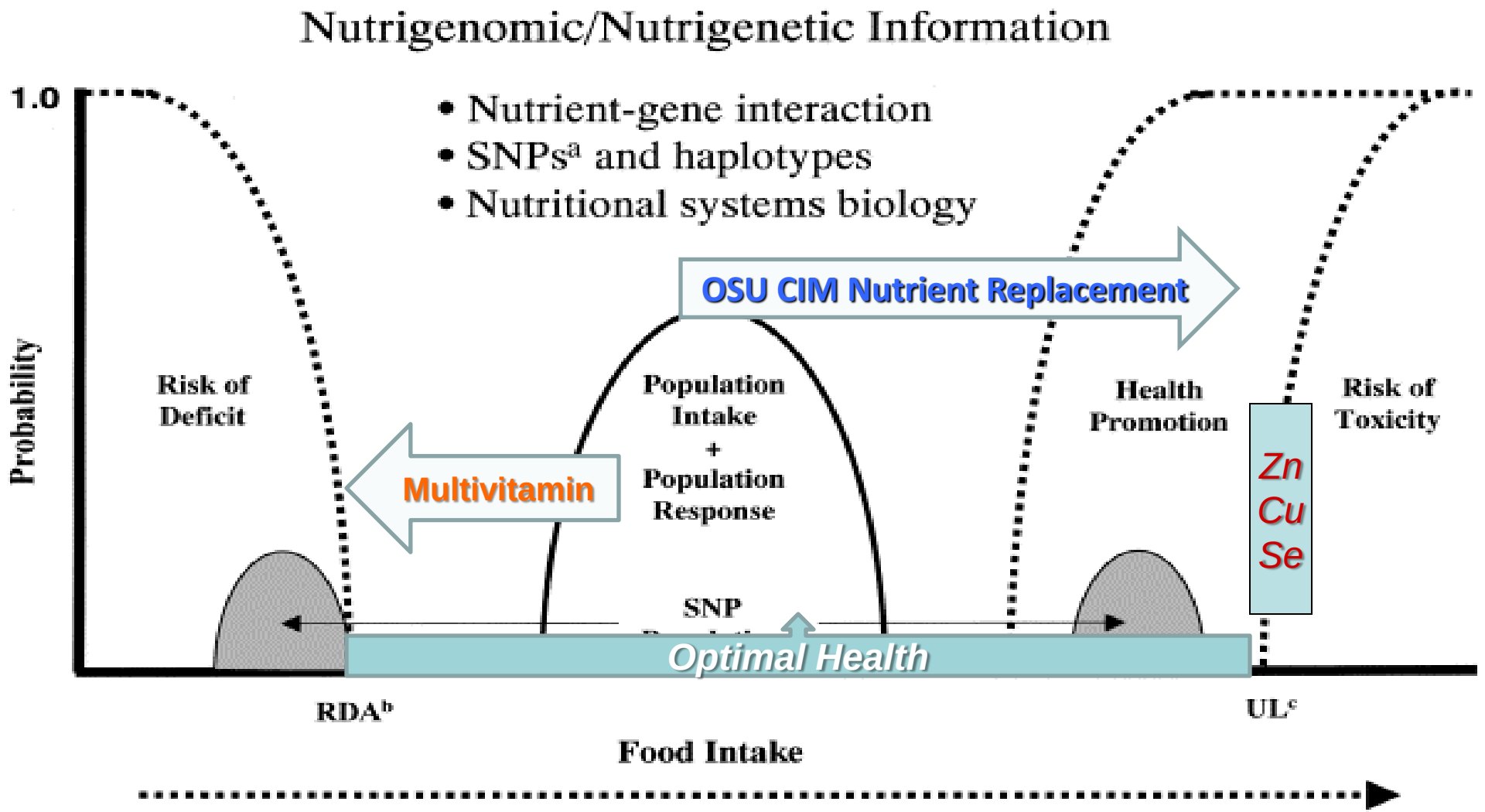
How B-Complex TR *Helps* Your Body

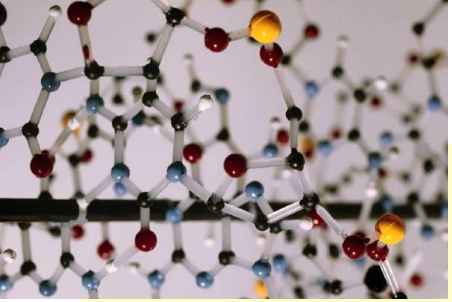
1. Where you are the Sickest
2. Getting to Sleep and Snoring
3. Nocturia = night time urination
4. Mood and Memory
5. Cheilitis, Gingivitis, Stomatitis
6. Avoid Chemo Damage to Body

Gene-based Information Used in

Development of *New* Food-based Dietary Guidelines

SNP = Single Nucleotide Polymorphism,
RDA = Recommended Dietary Allowance, UL = Upper Limits of Safety





In Summary,

NutriGenomics focus is Improving your Genes' Responses to;

Omega-6 Plant Oil versus Omega 3

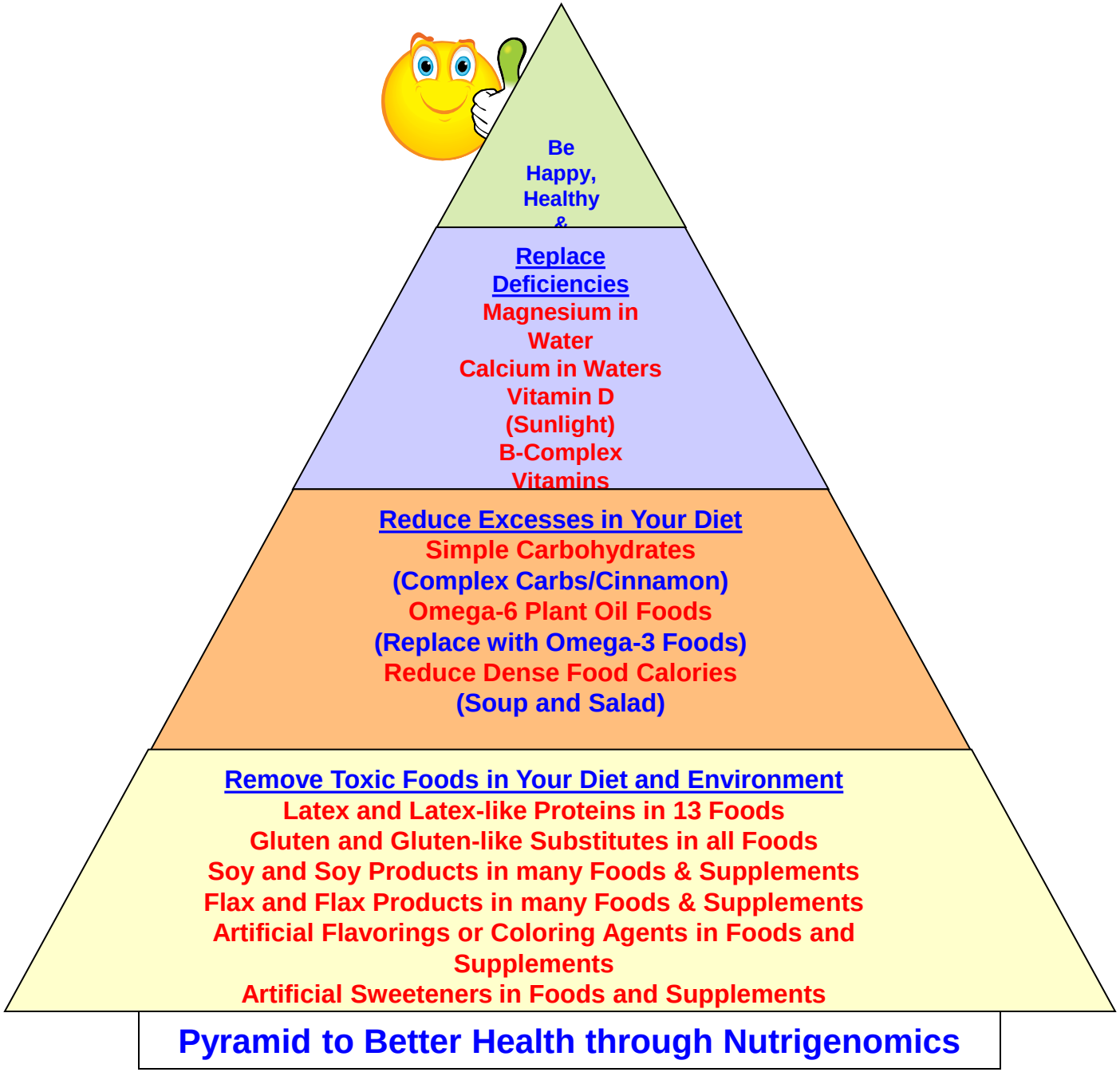
Calcium, Magnesium missing in Waters

B-Vitamins missing in 43 Fruits/Vegies

Vitamin D avoided/missing in Region

Gluten blocking Cal Mag D and Bs

Latex sensitization from Tire Fumes to Banana, Avocado, Celery, Kiwi, Figs, +8


Be
Happy,
Healthy
&
Replace
Deficiencies
Magnesium in
Water
Calcium in Waters
Vitamin D
(Sunlight)
B-Complex
Vitamins
Reduce Excesses in Your Diet
Simple Carbohydrates
(Complex Carbs/Cinnamon)
Omega-6 Plant Oil Foods
(Replace with Omega-3 Foods)
Reduce Dense Food Calories
(Soup and Salad)
Remove Toxic Foods in Your Diet and Environment
Latex and Latex-like Proteins in 13 Foods
Gluten and Gluten-like Substitutes in all Foods
Soy and Soy Products in many Foods & Supplements
Flax and Flax Products in many Foods & Supplements
Artificial Flavorings or Coloring Agents in Foods and
Supplements
Artificial Sweeteners in Foods and Supplements
Pyramid to Better Health through Nutrigenomics

Diagnoses for which NutriGenomics Helps

- **Allergies**
- **Anemia, Sickle-cell, Iron Deficiency, Pernicious**
- **Arthritis,** Joint Pain, Spine
- **Asbestos**
- **Aspirin for Prevention, Heart and Stroke**
- **Asthma**
- **Autism**
- **Auto-immune disorders**
- **B-complex, safe dose**
- **Cholesterol and HDL,**
  - **Cholesterol, LDL, small particle**
  - **Low HDL,**
  - **Triglycerides,**
  - **Treatment; Cinnamon, Magnesium ox, Fish Oil, Statins, Zetia, Lopid, Tricor**
- **Chronic Pain Syndromes,**
  - Fatigue, Tired muscles
  - Fibromyalgia, TMJ/TMD,
- **Fish Oil caps versus Fish Oil Liquid, conversion**
- **Dermatology** from Sun Damage to
  - Leukocytoclastic Vasculitis
- **Diabetes, Cinnamon, action and onset**
- **Digestive problems**
  - **Celiac Disease needs gluten-free diet**
  - **Colitis, ulcerative**
  - **Constipation = magnesium deficiency**
  - **Crohn's Disease**
  - **Diverticulosis, Diverticulitis**
  - **GERD reflux disorder**
  - **Irritable Bowel Syndrome, IBS**
- **Foods**
  - **Acid Alkaline Diet**
  - **Coconut, meat and oil concerns**
  - **Flax concerns**
  - **Free-range, grass-fed, NOT grain-fed**
  - **Grilling, Smoking, Microwaving Meats**
  - **Labels and Additives**
  - **Latex in foods concerns**
  - **Latex garments and products**
  - **Meats and Fish, best and worse**
  - **MSG, monosodium glutamate syndrome**
  - **Oatmeal, Brown Rice need omega-3**
  - **Organics, Pesticides, Nutrition**
  - **Popcorn, corn product concerns**
  - **Shellfish Allergies is to the meat**
  - **Soy and soy products concerning**
  - **Sweeteners are Toxic versus Sugar**
  - **Veganism health concerns**
  - **Vegetarianism health concerns**
- **Health, Definition**
  - **Medications, how, when to get off**

Diagnoses for which NutriGenomics Helps

- Heart
  - Arrhythmias, Atrial Fibrillation
  - Attacks
  - Edema, Failure
  - Hypertension
- Infections, Inflammation
- Insomnia, Sleep
- Kidney and Bladder
  - Frequency
  - Incontinence
  - Interstitial Cystitis, IC
  - Nocturia (getting up at night)
  - Poly-cystic
  - Stones
- Life Expectancy
- Magnesium oxide, versus magnesium-chelates
- Malignancy Cancer
  - Brain Tumors, glioblastomas, glioma, astrocytomas
  - Hairy-cell Leukemia
  - Leukemia
  - Lymphoma
  - Lynch Syndrome
  - Multiple Myeloma
  - Multiple Syndrome
- Mental
  - Anxiety Disorders
  - Bipolar syndromes
  - Depression
  - Dysfunction
  - Headaches, Migraine, Histamine
  - Memory loss
  - Mood Disorders
  - Panic Disorder
  - Premenstrual Disorders
  - Schizophrenia
- Moncytes, what are they, why important?
- Neurological Disorders
  - Alzheimer's Dementia
  - Dementia
  - Multiple Sclerosis
  - Muscular Dystrophy
  - Myasthenia Gravis
  - Parkinson's disease
  - Seizures, epilepsy
  - Stroke Rehabilitation
- Osteoporosis, Osteopenia
- Perimenopausal, hormones
- Thyroid
  - Goiter
  - Graves Disease
  - Hashimoto's Autoimmune Thyroiditis, Nodules
  - Hyperthyroid,
  - Hypothyroid
- Weight Loss or Gain
- Zinc Deficiency or Toxicity

Our Discussion in Past 60 Minutes

- Review Origin of **NutriGenomics** at OSUCIM
- Natural History of **Nutrition and Health**
- What is **Toxic in Excess** in our Foods
- What is **Missing** in Food and Water
- **Uncovering** New Patient Problems
- **Recovering** the New Patient
- **Managing Patients** over Time
- **Wee Continuing Education** for Patients
- **NutriGenomics** Improves Conditions >46,000+

Food Components Bio-actively Influence Those Genetic and Genetic-related Events Associated With *Your Disease Processes*

What We Eat is Always

> Just Food

>Proteins,

> Fats

> Carbohydrates

Nutrigenomics (Nutrition-Genomics) Identifies How

Dietary Factors Contribute to Establishing a Condition

WHAT WE EAT AFFECTS
HOW WE LOOK and FEEL
AT THE END OF THE DAY

MUST EAT THE CORRECT FOODS
TO RESTORE GENES DAILY

Human Disease-Gene Network

No Matter What Disease We Approach

All Diseases are Affected

By What We Eat and What We Supplement

Loscalzo J. Molec Syst Biol 2007

http://www.pnas.org/content/104/21/8685.full

Network Medicine = Relationships Count

Can Our Social Networks Generate Our Next Disease?

RESULTS: Discernible clusters of obese persons were present in the network at all time point
and the clusters extended to three degrees of separation.
Not solely attributable to the selective formation of social ties among obese persons.
A person's chances of becoming obese increased by 57%
if he or she had a friend who became obese in a given interval.
Among pairs of adult siblings, if one sibling became obese,
the chance that the other would become obese increased by 40%.
If one spouse became obese,
the likelihood that the other spouse would become obese increased by 37%.
These effects were not seen among neighbors in the immediate geographic location.
Persons of the same sex had greater influence on each other than those of the opposite sex.
The spread of smoking cessation did not account for the spread of obesity in the network.
CONCLUSIONS:
Network phenomena appear to be relevant to the biologic and behavioral trait of obesity,
and obesity appears to spread through social ties.
These findings have implications for clinical and public health interventions.
 http://www.ncbi.nlm.nih.gov/pubmed/17652652

Looking for Answers?

Who is the WEE Protocol for?

The terminally ill?

The chronically ill?

Those in less than perfect health?

The answer is YES! The WEE Protocol is for everyone who wants to experience optimal health.

In this book, you will meet Dr. Glen Aukerman and a few of his patients, learn about the work he is doing, and how you can use his WEE Protocol to find your way back to optimal health.

The WEE Protocol is not a diet, although it will change the foods that you eat. It is a whole-body approach to wellness. It is based on research, both traditional science and evidence-based research, and it can save your life.

WEE Protocol
for a Genomic-Specific Nutritional Plan

WEE™

Work by the physician to Examine and Evaluate | **Work by the patient to Educate themselves and to Eat accordingly**

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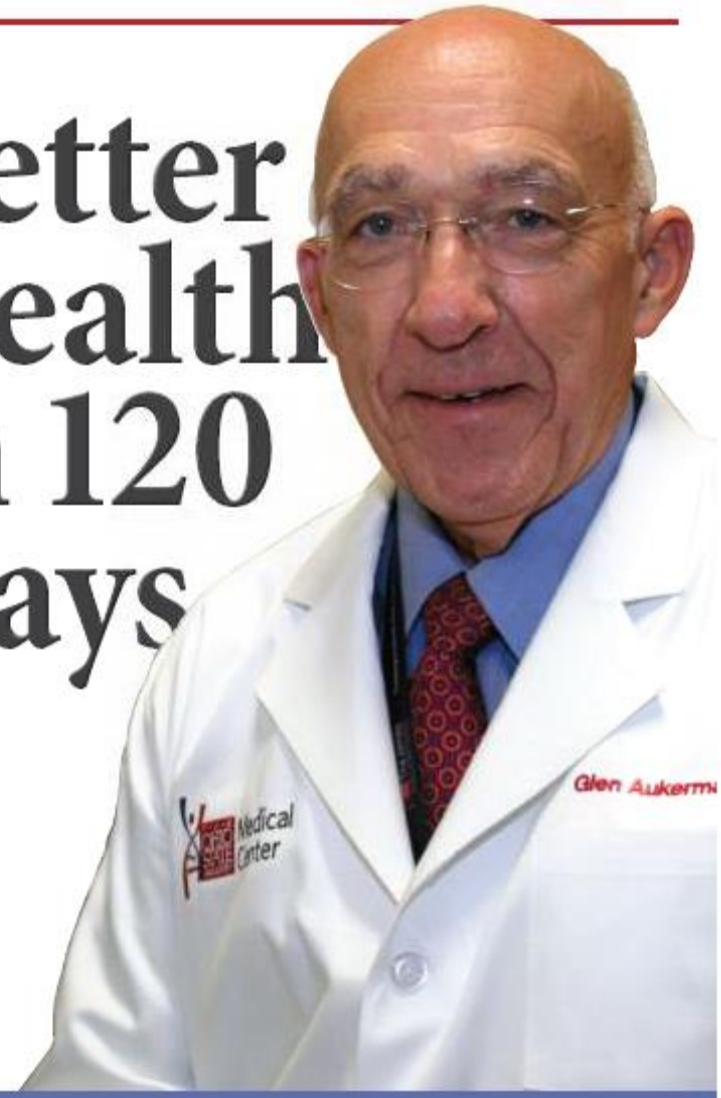
Dr. Glen Aukerman

Better Health in 120 Days



Finding Answers with the WEE Protocol

What is New since Publication

- New Research by searching @ nih.question
- DHEA to restore Hormonal Balance, Libido and ED at nih.dhea
- MTHFR at nih.mthfr
- Homocysteine control for Dementia and mind at nih.homocysteine

Go Bucks!