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William R. Ware, PhD - Editor

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I hope readers are not tiring of our excursion into psychiatry and radiation. It is about to end. First, there is radiation. The apparent misconceptions concerning the dangers of low- or moderate-dose radiation so completely saturate the thinking of many experts, scientists, physicians and the media that the situation cries for someone to address the other side of the picture—the one with evidence and data rather than dogma and conjecture. Thus in this issue a brief update and commentary are presented on what the mainstream view as abject heresy, i.e. radiation hormesis (benefit) and the suggestion, outlandish, absurd and radical as it may seem, that one of our problems as 21st century humans is a deficiency of radiation, not too much. Problems associated with the mainstream view of radiation risks were discussed at length in a Research Review in the Newsletter in 2008 where the hormesis hypothesis was explored. It opposes the conventional view, unsupported by any epidemiological evidence, that no dose of radiation is safe and the risk of cancer has no threshold. This dogma completely dominates mainstream thinking concerning the risk of radiation and as well dominates the approach to estimating risks of diagnostic and screening exposure such as mammography and CT scans. It is the basis of the almost universal fear of any radiation that is promoted by authors of papers projecting millions of death from CT scans and as well, by the media commentators and their "experts."

Last month we discussed Chernobyl, but aside from thyroid cancer resulting from childhood exposure and just a hint of leukemia in heavily exposed emergency and clean-up workers, there was no excess cancer to be found and instead, there was some evidence of hormesis. One could not ask for a better study population. Where were the cancer cases?

In this issue we look at the aspect of the conventional wisdom that involves belief in the absence of a threshold for radiation-induced cancer. As discussed, one finds thresholds almost every place one looks and almost always, below this threshold there is hormesis.

The second area, the rise of psychopharmacotherapy and the decline in the various forms of psychotherapy, is an irresistible topic since it offers in one neat package examples of what is wrong with modern medicine. In this issue we add to the material presented in the two-part research review that appeared in February and March and again direct reader's attention to the books of Dr. Peter Breggin for a new perspective.

This May issue appears early in April for reasons having to do with scheduling and traveling.

Finally, if you need to restock your supplements, please remember that by ordering through the on-line vitamin store you will be helping to maintain the web site and the publication of IHN. You can find the store at http://www.yourhealthbase.com/vitamins.htm.

Wishing you and your family good health,

William R. Ware, PhD, Editor

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RADIATION HORMESIS (BENEFIT) REVISITED

"The exact opposite of what is generally believed is often the truth."
Jean de la Bruyere, French Philosopher (1645-1696)

The Research Review published in this Newsletter in 2008 examined the cancer risks of low- and moderate-level radiation exposure and also introduced the reader to the concept of radiation hormesis and evidence for its existence available at that time. Radiation hormesis refers to a beneficial effect at low-doses of radiation whereas higher doses produce harm. Subsequently, there have been a number of new studies, reviews and critical perspectives which have significantly reinforced the already strong foundation associated with the radiation hormesis hypothesis and at the same time significantly weakened the foundation for the linear-no-threshold hypothesis (LNT), which has achieved the status as a true dogma characterized by almost no supporting evidence. The LNT hypothesis assumes that the excess risk of cancer from radiation has no threshold and simply increases in a straight line manner at doses above natural background. In fact we appear to have reached the point where it can reasonably be considered falsified due to the many observations of hormesis and the observation of numerous thresholds. One can predict that in a decade expert opinion may change. These new results, some of which are discussed below, provide additional perspective concerning low-dose radiation exposure, a particularly timely subject considering the reactor disaster in Japan, the ever-increasing use of radiation-based diagnostic tools and protocols deemed risky by the experts and the inevitable renewal of intense debate and controversy concerning the future of nuclear power generation.

A study appeared in 2010 that made use of the background radiation variation with altitude in order to examine the impact of radiation on mortality from all causes, cancer, heart disease and diabetes.[1] The first three were selected because they had been linked to radiation exposure whereas the latter had no such association and thus could be used for comparison. Six states were selected with a mean elevation above sea level of about 560 ± 210 feet. The six states used for the high elevation group had a mean altitude of 2340 ± 900 feet. The comparative radiation levels were estimated to be 6.3 mSv, and 7.9 mSv per each 10 year exposure (see the May Newsletter for a comment on units).

Populations living at high elevations were found to have significantly lower all cause mortality and cancer mortality. Both of these results had a large size effect, which from the statistical point of view enhances the biological relevance of the results. Smaller protective effect was seen for heart disease mortality and the results for diabetes were not statistically significant. It is possible that this study was confounded by the lower atmospheric oxygen concentrations at high elevations.

A related study by the same investigator looked at cancer mortality in three areas in Texas differing by altitude from sea level to 3000+ feet above sea level. Three population groups representing residence in low, medium and high elevations were studied. Statistically significant differences in cancer mortality were found between the low and high elevation groups and the medium and high groups.[2] High elevation was consistent with radiation hormesis.

Another study examined the association between lung cancer incidence and residential radon exposure as measured by the concentration of radioactive radon in the air of subject's homes.[3] The radon activity was measured in Becquerel units, where 1 Bq is equivalent to one nuclear decay per second. In the following we will for simplicity use Bq with the understanding that it implies the decay rate observed for all nuclear disintegration in this case in a cubic meter of air. When a reference of 25 Bq was used, reduced lung cancer rates were observed for exposures from 25 to < 250 Bq. Odds ratios

were adjusted for smoking, residency, job exposure, income and education. For the best mathematical dose model of exposure, lung cancer risk decreased over 75 to 250 Bq exposure to a maximum of 69% reduction with all confidence limits indicating statistical significance of protection starting in the range of 25-50 Bq. The authors discuss other radon studies which also provide evidence of hormesis.

In a review published in 2010, Vaiserman cites a number of studies where hormesis was associated with background radiation not only from radon but from soil, cosmic rays and building materials.[4] Even when evidence of hormesis does not reach statistical significance, there is considerable evidence suggesting that low-dose continuous exposure due to abnormally elevated background radiation, which in some areas can exceed average exposures by a factor of 10, does not cause adverse health effects including cancer. Earlier studies concerning hormesis and background radiation were discussed in the 2008 Research Review.

To these results can be added the data presented in paper published in 2008. Luckey examined the health benefits experienced by atom bomb survivors from both Nagasaki and Hiroshima.[5]. Exposure in Nagasaki was due primarily to gamma rays and a few fast neutrons whereas in Hiroshima there was heavier exposure to neutrons from the uranium bomb. Luckey shows a number of graphs of mortality rates vs. dose for leukemia, non-leukemia cancer, and total cancer. All show strong evidence of hormesis and a definite threshold. For these atom bomb survivors, the average threshold where hormesis ends and positive risk commences was around 500-800 mSv. Thus this summary of 14 studies provides convincing evidence for the presence of a protective mechanism which was overwhelmed only at higher dose rates to produce a positive association between cancer mortality and dose. It is these higher rates that the advocates of the LNT model use, although some LNT practitioners do adjust at some point below 1000 mSv and arbitrarily use a factor of ½ to generate a new linear plot. They still deny the existence of thresholds and hormesis which are so clearly seen in Luckey's plots of risk against dose.

In summarizing the atomic bomb survivor studies, Vaiserman[4] points out that there was no increase in the number of deaths due to cancer for those who received doses lower than 200 mSv and mortality caused by leukemia in the population exposed to < 100 mSv was lower than age-matched control cohorts (hormesis). Also, no significant genetic or hereditary effects have been found. In addition, as Luckey also points out, there is some evidence of increased health in the atom bomb survivors including decreased mutations and an increased life span.

Luckey also reviews the 1954 fallout incident from a H-bomb test at Bikini Island which severely exposed 23 young Japanese fishermen.[5] All had radiation sickness but none died from cancer over a subsequent 40 years of observation. One died about 8 months after exposure from anaemia, hepatitis and leucopenia. One died 21 years later with liver cirrhosis. The rest recovered. He compares this result with that observed for the Chernobyl workers who were hospitalized with radiation sickness during the weeks after the accident. None who received < 2000 mSv died of cancer or other disorders during a 10 year follow-up.

The mainstream rejection of hormesis, and this includes the U.S Environmental Protection Agency, is inconsistent with animal and cell culture studies which provide a firm foundation for biological plausibility and back up the extensive epidemiological evidence. The radiation induced beneficial responses include elimination of preneoplastic and other aberrant cells, induction of DNA repair pathways, activation of immune functions, production of stress proteins, scavenging of free radicals, activation of membrane receptors, secretion of cytokines and growth factors, compensatory cell proliferation, and the up-regulation of adaptive response genes.[4] This all makes sense if one considers that as living, reproducing organisms evolved, the background radiation was considerably higher than now, and survival depended in part on defence mechanisms against radiation induced chromosomal damage caused either directly or indirectly (via reactive oxygen species for example). Modern studies appear to be consistent with the evolution of these mechanisms.[4,6]

Areas of active research concerning radiation hormesis relate to increased longevity and to the beneficial effect of low-dose radiation on the effectiveness of conventional cancer therapy, including high dose focused radiation. It has been suggested by a number of observers that perhaps current humans suffer from a deficiency of low levels of ionizing radiation exposure which puts them at higher risk of cancer. Such a notion would no doubt elicit ridicule from mainstream experts, but it is a logical conclusion based on the epidemiological data concerning hormesis and the many cell culture and animal studies which find both hormesis and biological mechanisms by which it could function. It also seems very clear that the high level of fear of low-dose and moderate-dose radiation is not evidence based and causes unnecessary psychological distress. In the case of accidents like Japan and Chernobyl it can be acute and have long lasting associated health problems. There are reports that after Hurricane Katrina, the rate of heart attacks among those affected tripled, and that increase persists three years later!

NEW STUDY ON THYROID CANCER INCIDENCE AFTER CHERNOBYL

This new study has just appeared in the journal *Environmental Health Perspectives*.[7] It attempted to evaluate the dose-response for incident thyroid cancer using measurement-based radioactive iodine-131 thyroid dose estimates in a prospective follow-up study. The cohort consisted of individuals who were < 18 years of age on the day of the accident who resided in three contaminated states of the Ukraine. The authors do not make clear the range of doses, but from a figure the highest was about 4.6 SV (4600 mSv to be consistent with the unit used in the Chernobyl discussion in the April Newsletter). Given the fact that some experienced very low doses, this is a huge dose range with an upper end capable of producing acute radiation sickness if whole-body rather than concentrated in a gland. The cohort underwent four thyroid screening sessions between 1998 and 2007.

This study demonstrated that thyroid cancers attributable to iodine-131 exposure, which lasted only for a few months due to the short half-life of this isotope and the assumption that there was no significant residual fission in the disabled reactor, continued to occur two decades after the exposure with the hallmark papillary thyroid cancer the main histological type (94%). There was no indication of diminishing risk per Sv dose with increasing time since exposure. The study also found a large dependence on age at exposure with the risk per Sv for the 0-<4 age group being ten times that of the 12-<18 age group. No consistent variation, however was found for gender. The authors cite the major strengths of this study as the availability of individual iodine-131 exposure estimates, low losses to follow-up and near complete ascertainment of cases. Finally, they present a plot which they propose indicates a linear dose dependence. However, this plot lacks critical low-dose points but shows a sharp increase at high dose after a fairly long moderate-high dose plateau, although the plateau could extent to 1400 mSv. The risk for exposure below 100 mSv was not discussed nor data provided.

This study is interesting because, as mentioned in the discussion in the April issue of the Newsletter, pediatric thyroid cancer cases started showing up only 4-5 years after exposure. Nevertheless, it seems clear that there can be a very long latency period after childhood exposure, and that very young age makes a large and significant difference in the probability of an eventual adverse outcome. However, some perspective can be gained by considering that of the 12,514 individuals followed, there were only 65 incident thyroid cancers diagnosed during the screenings, and this involved over 73,000 persons-years of observation. This represents an overall incidence rate of only about 0.5% even though these children and adolescents were living in three contaminated states.

IS THERE A CRISIS IN MODERN PSYCHIATRY?

Thirty or more years ago many patients with depression, anxiety, insomnia or other non-acute mental health problems generally received psychotherapy or what is frequently simply referred to as talk therapy. Psychiatrists devoted considerable time to each patient during each visit, typically around an hour, and got to know their patients and their problems very well. This was the traditional approach that goes way back to, for example, Sigmund Freud. Drug choices were limited and psychopharmacy did not play a major role aside from dealing with acute situations. However, electric shock treatment was common.

The revolution came with insured care and the evolution of insurance reimbursement policies which allowed physicians in some jurisdictions, and in particular the U.S., to bill much more for a 15 minute visit than for a 45 minute talk therapy session. Part of the rationale appears to be that insurance providers rated the value of talk therapy by the fact that psychologists and social workers provided the same service at a much lower rate. After all, they did not have to attend medical school. Furthermore, they assumed that the quality of talk therapy was similar for these three providers, which may be close to the truth.

The simultaneous growth of the available drug therapies and their exceedingly aggressive promotion provided financial salvation since they were ideal for the 15 minute visit. This situation has evolved to the point where the use of services of psychiatrists who offer talk therapy has become mostly restricted to patients who can afford the un-reimbursed cash payments or tolerate partial reimbursement. The media has documented very high fees collected by elite psychiatrists who provide talk therapy to the rich and successful or their children. Those who have adapted to the new regime mostly refer patients who want or need talk therapy and are forced to a large extent to withhold this therapy entirely simply for financial reasons. It appears that many patients do not follow through with the advice to seek talk therapy. In fact, even with a system which enables the modern psychiatrist to run what amounts to a mill, some demand up front co-payment.

This state of affairs was described very recently in a much discussed article in the *New York Times* by Gardiner Harris (March 5, 2011). The title was blunt: “Talk doesn’t Pay, so Psychiatry Turns Instead to Drug Therapy.” Two discussions of this article quickly appeared in the online professional magazine *Psychiatric Times* which attempt to provide a more realistic picture. In the first to appear, Dr. Ronald Pies[8] suggests that some of the views expressed in the NYT article came from a paper by Mojtabai and Olfson. This study found that the number of psychiatrists who provided psychotherapy declined from 19% to 11% between 1996-7 and 2004-5 and this coincided with the changes in reimbursement, increases in managed care and increased use of prescription drugs. Pies then points out that this same article found that almost 60% of psychiatrists continued to provide some psychotherapy to some of their patients. However, this point seems too vague to be informative. What Pies did not mention is that if one looks closely at the Mojtabai and Olfson paper, when the two periods are compared with respect to medication prescribed, the change was from 68% to 83% and thus in the later period only 17% left the office without a prescription. If the same survey were made today this number might even be lower. Pies also attempts to convince the reader that changes have occurred such that there are now “brief therapies” that work and fit into the 15 minute window. Considering that not all of the 15 minutes is devoted to the new talk therapy, this still represents a huge change from the classical 50-minute psychotherapy session.

In the second commentary, Dr. James Knoll shows a much deeper and fundamental concern for what has been happening. He poses two questions psychiatrists should ask themselves. These reflect his concern for what is happening.

1. Are your patients’ best interests being served?

2. Are you content practicing medicine according to the oath [Hippocratic] you took?

Knoll outlines the philosophical and moral dilemma that characterizes modern psychiatry. However, his commentary is short on specific workable solutions if the answer to either of the above questions is no.[9]

COMORBIDITIES IN CHILDREN DIAGNOSED WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

In a study just published in the journal *Pediatrics*, the prevalence and impact of so-called comorbidities in children diagnosed with attention-deficit/hyperactivity disorder (ADHD) are discussed.[10] Readers will perhaps recall that in the February 2011 Newsletter, mention was made of a "confession" by a well known psychiatrist, Dr. Allen Frances, who was chairman of the task force that produce the psychiatric diagnostic manual (DSM-IV) now universally used. Among the false epidemics for which he claimed responsibility was ADHD. This is a serious matter. As many as 2-4 million American children have been diagnosed with this disorder with 1 to 3 children in every classroom viewed by mainstream medicine as having ADHD. According to Dr. Frances, a significant fraction of these 2-4 million children have been overdiagnosed and thus a significant fraction also unnecessarily treated. In light of this confession, consider the new study.

The study published in *Pediatrics* identified 10 comorbid disorders found in children with ADHD and examined the issue of prevalence. The top 4 comorbidities with the highest prevalence were learning disability (46%), conduct disorder (27%), anxiety (18%), and depression (14%). Furthermore, 16% of the study group had two disorders and 18% had three or more. By applying these percentages to the number 2 or 4 million, the magnitude of the problem is evident. The increased risk of having the 4 most prevalent disorders ranged from about 8 fold to 13 fold and there was a steep increase in impairment and support service use by those with multiple comorbidities. This study was essentially cross-sectional in nature. It obtained snapshots of the current situation and thus was unable to compare time lines of ADHD and the studied comorbidities.

What is interesting about this set of comorbidities is that if one looks in Dr. Peter Breggin's 2008 (second edition) medical monograph *Brain-Disabling Treatments in Psychiatry,*[11] one finds these disorders listed as *side effects* of the pharmaceutical treatment of ADHD with stimulants such as Ritalin, Concerta, Dexedrine, Adderall, Concerta and other stimulants (Table 11.2). Other comorbidities discussed in the article that are also side effects of ADHD medication are vision problems and Tourette's syndrome, and there is overlap with autism spectrum disorder. Thus most of the comorbidities can potentially be explained as resulting from medication for ADHD. The paper does not give the percentage of the children studied who were medicated, but it can be safely assumed that it was high. Typical figures on sees cited are around 80%-90%.

The authors discuss the need for studies on effective care for these children. Indeed! But there is no mention that the approach involving gradually stopping all medication and trying non-drug therapy. Of course, it is possible that these comorbidities were present when ADHD was first diagnosed. However, the results of most studies of concurrent comorbidities which have provided similar results refer to lifetime or prospective incidence which would be open to influence by medication.[12] Furthermore, if one examines a sample of comorbidity studies, medication is almost never even mentioned. In this context, medication appears to be viewed as benign. Also, some comorbidities may be the result of medications other than stimulants, which might be used for example to treat depression and subsequently result in other comorbidities. A common theme in Breggin's case histories is a downward spiral characterized by ever increasing doses and variety of psychiatric drugs used. Breggin's view is reinforced by a recent UK study indicating a very poor cure

rate and increase in severity in ADHD associated with drug treatment.[13] If medication makes ADHD worse, this may include causing or making comorbidities worse. This is consistent with the common observation that some patients proceed from stimulants to antidepressants to antipsychotics, frequently in combination, and finally end up disabled.[11]

The reader is referred to the Research Review in the February 2011 Newsletter for a lengthy discussion of the effects of the conventional treatment of ADHD and successful alternative treatment approaches. The recommendation from the Research Review: Any parent with a child on ADHD medication or considering approving drug intervention, or in a position where no choice is provided aside from litigation, should purchase the above-cited book by Dr. Peter Breggin and read chapters 10 and 11. The book, being a medical monograph, is very expensive but worth it. The text is easily accessible and understandable even in the absence of medical training. Consider also Breggin's book *Medication Madness: The Role of Psychiatric Drugs in Cases of Violence, Suicide and Crime*. This book is for the layman and has a case history format. The ADHD case histories are truly shocking. Breggin is widely regarded as the conscience of American Psychiatry.

ELIMINATION DIET APPROACH TO TREATING ATTENTION-DEFICIT HYPERACTIVITY DISORDER

A study has just reported in the journal *Lancet* which examined the impact of an elimination diet and subsequent challenge phase on the symptoms of ADHD.[14] Aside from the various forms of psychotherapy and behavior modification, elimination diets and enhanced micronutrient intake represent the two major non-drug approaches to the treatment of ADHD (see Part II of the Research Review in the March Newsletter). In this study 100 children were randomly assigned to either a healthy diet according to guidelines in the Netherlands or to an elimination diet from which major components were eliminated to ascertain beneficial effect (phase 1). Briefly, the elimination diet consisted of rice, meat, vegetables, pears and water to which were added specific foods with potential for adverse impact on ADHD. At the end of the second week the absence of parent reported improved behavioral changes prompted the gradual elimination until only the basic diet remained. Children who experienced behavioral improvement were entered in the challenge phase where foods specific to the individual were reintroduced (phase 2). Serum immunoglobin (IgG and IgE) levels were measured weekly during the study, and helped dictate choices of foods during the challenge phase. The challenge phase involved a randomized crossover from low IgG to high-IgG foods.

Between baseline and the end of phase 1 there was a large and significant improvement in behavioral scores between the diet and controls groups. In phase 2, the validity of the results was confirmed by relapse, but IgG was not a reliable indicator. It was concluded that a strictly supervised elimination diet was a valuable instrument to determine whether ADHD is induced by food. In fact, the authors view their results, taken with other studies of elimination diets, to support the implementation of dietary intervention in the standard care for all children with ADHD.

Given the remarkable benefits associated with micronutrient supplementation of ADHD as described in the Research Review Part II, the combination of this intervention and a diet dictated by the results of an elimination protocol might produce outstanding results without any risk of side effects or the classical downward spiral associated with psychopharmacotherapy in some patients.

ALCOHOL AND CARDIOVASCULAR DISEASE

A large meta-analysis just published in the *British Medical Journal* addresses again the question of alcohol consumption and cardiovascular disease.[15] Out of 4235 possible prospective cohort studies the researchers selected 84. The criteria were adults over 18, no pre-existing cardiovascular disease (CVD), and consumption data and with comparison groups of non-drinkers as controls. The studies were required to have as outcomes overall CVD mortality, incident coronary heart disease (CHD), CHD mortality, and incident stroke or stroke mortality. For the 84 studies, 34 reported on all-male cohorts, six on women only, and 44 on both. The mean follow-up was 11 years with a range of 2.5 to 35. The studies used had a mean of six confounders used in adjustment with a range of none to 18.

The pooled relative risk reductions that were statistically significant were 25% for cardiovascular mortality, 29% for incident CHD, and 25% for CHD mortality. There was no significant association with stroke incidence or mortality. As mentioned above, these results have abstainers as a reference. For consumption of 30-60 g/day CVD mortality risk reduction was 15%, for CHD incidence 24%, and for CHD mortality 25%. Consumption of 60 g of alcohol is equivalent to about 4 drinks. Consumption was not stratified by gender. For consumption of > 60 g/day no statistically significant conclusion was possible for CVD disease mortality and CHD incidence, but for CHD mortality, the risk reduction remained at 25%. The results were robust when judged in terms of the effect of adjustment for confounding or median follow-up time. Finally, an analysis of mortality from all causes showed a significantly lower risk for drinkers compared to non-drinkers (risk reduction 13%) According to the authors, this latest study significantly updates the picture based on systematic reviews. One of its strengths was the consideration of several meaningful clinical outcomes.

In a second article published simultaneously in the same journal, this research group presented a systematic review and meta-analysis of the effect of alcohol consumption on markers associated with the risk of CHD.[16] From 4690 articles, 63 relevant articles were selected. The overall picture that emerged indicated moderate alcohol consumption (one drink or 15 g alcohol/day for women and two drinks or 30 g/day for men) produced favourable changes in levels of HDL cholesterol, adiponectin and fibrinogen. These changes suggest an indirect pathophysiological mechanism for the protective effect of moderate alcohol use on CHD. These results were consistent with interventional studies that also found alcohol consumption favourably influences various biomarkers associated with the risk of CHD. The authors regard the results of their analysis as strengthening the argument for a causal link between alcohol intake and reduced risk of CHD. They conclude, on the basis of these two studies, that additional observational studies will have limited value except to elucidate more precisely the association of alcohol and stroke. Rather, the issue now becomes how to integrate the evidence into clinical practice and public health. As they emphasize, issues revolve around optimal patient selection, and the presentation of risks and benefits during counselling with regard to incorporating moderate amounts of alcohol in to diets.

For those who already engage in moderate alcohol consumption and are able to successfully control intake, these two studies add a strong element of comfort. For the non-drinker, the matter obviously remains complex.

VITAMIN E AND RISK IN THE ELDERLY OF DEVELOPING ALZHEIMER'S DISEASE

There is increasing evidence that oxidative stress is implicated in the pathophysiology of Alzheimer's disease (AD). This observation is based in part on the finding of oxidative damage in diseased brains of patients with AD. Also, compared to cognitively intact controls, individuals with AD or mild cognitive impairment have reduced concentrations of

circulating antioxidants. Thus there is considerable interest in the preventive and therapeutic use of antioxidants in this context. However, cohort and intervention studies have been inconsistent.

A recent study has further addressed this issue by examining the association of the risk of incident AD with the various forms of vitamin E, of which there are two classes of four forms each, called congeners. The study involved a dementia-free group of 232 subjects aged 80+ years, which was followed for 6 years to detect incident AD. Blood levels of vitamin E (α-, β-, γ-, and δ-tocopherol and α-, β-, γ-, δ-tocotrienols) were measured at baseline. Only a few percent of the subjects took supplemental vitamin E and thus the distribution of congeners was determined by diet. During the following 6 years, the group was followed to determine the incidence of AD. The participants received a full dementia work-up which included clinical examination by physicians and neuropsychological assessment by psychologists.[17]

Risk reduction was found when the highest vs. the lowest tertile of total tocopherols, total tocotrienols and total vitamin E were compared. The reductions were 45%, 54% and 45% respectively and all were statistically significant based on 95% confidence intervals. When the results were stratified by individual congeners, the most active congener was β-tocopherol. While all but the δ-congeners were similar in reducing risk, the results lacked statistical significance. The β-forms occur in low amounts in the diet and show weaker antioxidant power compared to the other forms and thus these results are hypothesis generating and suggest the need for much more research.

Foods rich in vitamin E include sunflower seeds, almonds, olives, papaya, blueberries and various vegetables greens including mustard, turnip, and collard. Both natural mixed tocopherols and tocotrienols are available as supplements. Given the large statistically and clinically significant risk reductions observed in this study, these become quite interesting even in the absence of intervention studies.

FRUIT, VEGETABLES AND OLIVE OIL AND RISK OF CORONARY HEART DISEASE IN WOMEN

Another study of diet and coronary heart disease has just reported.[18] This study involved a cohort of almost 30,000 women who were followed for about 8 years. Baseline dietary, anthropometric and lifestyle characteristics were collected and major events of coronary heart disease (CHD). i.e. fatal and nonfatal heart attack and coronary revascularization (angioplasty or bypass) were identified from clinical records. At baseline, subjects were without diagnosis of stroke, heart attack, diabetes or elevated blood lipids and were viewed as healthy. Data were adjusted for hypertension, smoking, education, menopause status, physical activity, anthropometric measures, non-alcohol energy intake, alcohol, total meat, vegetables in the analysis for fruit and fruit in the analysis for vegetables.

For the entire cohort, when the highest quartile of leafy vegetable intake was compared with the lowest, a 46% reduction in risk of CHD endpoints was observed. The same comparison for olive oil intake gave a 44% risk reduction. Both results were statistically significant. The leafy vegetable intake for the highest quartile was > 51 g/day whereas the figure for olive oil was > 31 g/day, which is about 34 mL or 3-4 tablespoons. When just postmenopausal women were considered, the risk reduction for leafy vegetables was 54% and for olive oil 45% when the highest quartile was compared with the lowest. This result for olive oil just barely missed being statistically significant. The dietary components included in the analysis that failed to yield statistically significant results were total vegetables, tomatoes raw and cooked, root vegetables, cabbages, other vegetables, total fruit, citrus and non-citrus fruit and combined fruits and vegetables. The Mediterranean tradition of using olive oil as the main dressing fat for leafy vegetables made it difficult, according to the researchers, to disentangle the effect of olive oil and vegetables.

The results for leafy vegetables were consistent with those from the Nurses' Health Study, but the results for leafy vegetables were stronger than that found in the Woman's Health Study. Mechanisms suggested concentrated on micronutrients such as folate, B vitamins and vitamin E, C and beta-carotene and as well, the antioxidants in olive oil and leafy vegetables.

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