



Women's Health *report*

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NUTRITIONAL GENOMICS: Implications for Dietetics

Ruth DeBusk, PhD, RD

Abstract

Nutritional genomics is emerging as central to dietetics. Nutrients, toxins, and other bioactive components within our environment communicate with our genes to increase or decrease the types and amount of proteins that are produced. Each of us has genetic variations that affect the “goodness-of-fit” between our proteins and these bioactive components and lead to each of us having a unique profile in terms of our nutritional needs and our risk for disease. These variations do not in themselves cause disease but can increase (or decrease) our susceptibility for developing disease. Many of the chronic disorders challenging us today are the result of interactions among genetic variations and environmental factors, including the bioactive components in food. Nutritional genomics enhances our ability to make diet and lifestyle choices that will minimize disease susceptibility and maximize our health potential. As genomics is increasingly integrated into health care and nutritional genomics into dietetics, the opportunities for dietitians to increase our effectiveness and thus our value to society are expanding. With that enhanced recognition will come opportunities for increased career satisfaction, income, and autonomy.

Expect these opportunities to steadily unfold over the next 5-10 years and encompass food and nutrition research, clinical nutrition, education, public health and food and nutrition policy. Dietitians are urged to begin now to build the foundation of knowledge and skills needed to succeed in this new era of dietetics.

and disease. The expectation is that making smart choices appropriate for our individual genetic makeup will prevent disease, promote health and ultimately maximize our genetic potential.

Accomplishing such lofty goals is unquestionably a challenge but achievable. In the process, nutritional genomics will impact every aspect of dietetics, from nutrition and food science research to food development and clinical applications to education, public health, and food and nutrition policymaking. This emerging field is based on the science of genetics grafted onto the breadth and depth of food and nutrition science, the backbone of dietetics. We need to begin now to ensure that our foundation in the biological and chemical sciences expands to include genetics and genetic technologies.

Nutritional Genomics, Nutrigenetics, Nutrigenomics

Nutritional genomics is an umbrella term that describes the application of genetic technology to food and nutrition and includes nutrigenetics and nutrigenomics. Nutrigenetics concerns the individual's genetic makeup and the proteins those genes produce and how well those proteins work, whether “work” is the ability to metabolize a nutrient, latch onto a receptor, or transmit a signal between the cell surface and the genetic material in the nucleus. Whatever the ability level of each protein, it translates into a unique characteristic of that individual. In contrast, nutrigenomics concerns how the environment acts upon us, by influencing the expression of the genes each of us has. An example would be the ability of omega-3 fatty acids to decrease the inflammatory response by decreasing the expression of genes that produce pro-inflammatory cytokines.

See the following overviews for various perspectives on nutritional genomics and its relevance to food and nutrition: Brown & Ouderaa, 2007; Genc and Schantz-Dunn, 2007; Joost 2007; Müller and Kersten, 2003; Mutch et al, 2005; Ordovas et al, 2007; Subbiah 2007; Ulrich 2005; Vakili and Caudill, 2007; Zeisel 2007.

Genes and the Genetics-Nutrition Connection

Deoxyribonucleic acid (DNA) is the genetic material and consists of a linear sequence of nucleotides distributed among 23 different pairs of chromosomes, with the exception of a single X- and Y-chromosome in males. Within each chromosome are stretches of nucleotides (“sequences”) that encode the information necessary for making the proteins that perform the work of our cells. Such sequences are called “genes” and there is one copy of a particular

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Introduction

The ultimate promise of nutritional genomics is early intervention in order to promote health and prevent disease. This field focuses on identifying and working with our genetically determined ability to taste, digest, absorb, and metabolize food for nourishment. How effectively we carry out these critical processes depends upon our unique genetic makeup and the food and lifestyle choices we make. Inappropriate matching of our choices to our genes promotes dysfunction

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from the chair Cathy Fagen, MA, RD

This spring issue of the Women's Health Report features a topic I find very intriguing. I traveled to the Lillian Fountain Smith conference for nutrition educators in Colorado last June to hear Raymond Rodriguez, PhD from the Center of Excellence in Nutritional Genomics present **Nutritional Genomics: The Next Frontier in Promoting Health?** In October I attended the FNCE session **Nutrigenomics, Inflammation and Obesity: A New Paradigm for Personalized Prevention** presented by Ruth DeBusk, PhD, RD and Colleen F. Draper, MS, RD. Our role as registered dietitians has countless possibilities and opportunities in the promotion of health and prevention of disease. Nutrigenomics is an upcoming field that I wanted our members to know more about. Thank you to the authors and our Publications Committee for making my dream for this issue come to fruition!

My year as your DPG Chair is coming close to term. But my devotion and dedication to this group will never end. We have several past Chairs continuing to participate in WH DPG projects or alliances. Claire Dalidowitz, MS, MA, RD, CDN, serves as our IBLCE liaison. Alyce Thomas, RD, CDN, is heading up a committee to pursue the development of an OB/GYN specialty RD certification through CDR. Jeanne Blankenship, MS, RD, leads the WH DPG Breastfeeding Task Force and serves as our alliance representative with the United States Breastfeeding Committee. I will follow suit with involvement in some project when I decide where I want to focus my energy. Our DPG has grown and developed in so many different aspects thanks to all of you! Each of us has a different interest, expertise, or strength. And when we put them altogether we make beautiful music!

Nutrition and health are my first loves and I would say music is my second. My husband, Jim, is a musician. And I need to thank him as well as my employer for their support of my extracurricular professional activities. I would also like to thank the Women's Health Executive Committee, Marianne Smith-Edge, MS, RD, LD, FADA, and Susan DuPraw, MPH, RD, for all their assistance in helping us develop a 3-5 year Strategic Plan for the WH DPG. This first year of implementing our strategic plan was very exciting for me. We are well on our way to reaching our goals!

I look forward to our next year with Jamillah Hoy-Rosas, MPH, RD, CDN, CDE as our Chair. Jamillah is the epitome of new ideas and synergy. These are exciting times. Our future is bright and full of opportunities!



from the editor Olivia Bletsos, MPH, RD, CLC

It is with great pleasure that I take over as the Editor for the Women's Health Report. I have spent the last year as the Assistant Editor learning the ropes from your previous editor Krista Neal, MS, RD, LD. As a newer member to the Women's Health DPG, I am continually amazed at how much I get out of this publication. From the timeliness of articles on emerging issues; to the latest and most up-to-date research on topics once thought to have been put to rest; to fresh ideas on expanding and building a career in the realm of dietetics, I find myself returning to them time and again. It is my hope that as your new editor, we can continue to bring you thought-provoking, relevant and helpful articles on a wide range of topics.

With springtime at finger's reach and the dietetic world perched on the edge of thrilling new frontiers, it seems only appropriate that we bring you this edition dedicated to Nutrigenomics – a word I could hardly pronounce let alone define before I first heard our lead author Ruth DeBusk, PhD, RD, speak at FNCE in Philadelphia last year. Needless to say, many of us walked away from that presentation wowed!

Our lead article written by Dr. Ruth DeBusk brilliantly outlines the blossoming field of nutrigenomics and its implications for the future of dietetic practice. Annette Haban Bartz, MS, RD, LD, CLC gives a fascinating overview of the gene and nutrition connection during lactation while an article by Richard M. Watanabe, PhD, Associate Professor at the Keck School of Medicine at USC speaks to the promise of genetic research into the treatment and prevention of type 2 diabetes.

Mark Messina, PhD, MS, and Virginia Messina, MPH, RD, are back with an article on *Soy Foods and Breast Cancer. Mentor Me!* discusses reaching your career potential by finding and fostering mentoring relationships. I am proud to say this newsletter is all about new horizons and new perspectives.

gene on each chromosome member of the pair. A gene's information is "expressed" in a stepwise fashion that leads to the assembly of amino acids in the correct sequence to yield a functional protein.

As we saw with the omega-3 example, gene expression can be influenced by environmental factors, such as nutrients and other bioactive components in food as well as by a variety of environmental toxins. For example, a derivative of vitamin A is essential for the expression of genes regulated by the peroxisome proliferator-activated receptor transcription factor, that in turn control numerous genes (Brown & Plutzky, 2007). Toxic molecules, including many prescription drugs, increase the expression of the phase I detoxification reaction in the liver and lead to increased free radical production (Jeffery, 2007). Glucosinolates in cruciferous vegetables can increase the expression of glutathione-S-transferase enzymes that carry out phase II detoxification reactions and help reduce the free radical load (Higdon et al, 2007; Lampe 2007; Lampe and Chang, 2007). Other bioactives decrease gene expression, such as the modulation of pro-inflammatory cytokine gene expression by polyunsaturated fatty acids and also by flavonoids (Watkins et al, 2007).

For overviews of various gene-diet interactions, see: Corella & Ordovas, 2005; Corella, 2007; Davis & Milner, 2007; Na & Surh, 2006; Ordovas & Corella, 2007).

Changes can occur within the genetic material, which may alter the information in a gene and thereby, the function of the encoded protein. In many cases the outcome is severe and manifests as disease. The inborn errors of metabolism, such as phenylketonuria and galactosemia, are examples of this type of outcome and represent the classical application of genetics to nutrition practice. In other cases the change or "genetic variation" alters the function of the protein but not to the extent that disease results. Instead, the genetic variation may increase the susceptibility that the individual will develop a particular disease but is not sufficient in and of itself to cause disease. Only when the combination of genetic variations and specific environmental factors interact does disease ensue. Today's chronic diseases result primarily from this type of gene-environmental interaction.

Unlike the rare inborn errors, genetic variations are quite common. When a variant occurs in >1% of a population, it is called a single nucleotide polymorphism (SNP, pronounced "snip"). SNPs can affect virtually any aspect of a protein's function. Much of nutritional genomics research is presently focused on identifying associations between SNPs and susceptibility and on the impact of SNPs on nutrient requirements. When the nucleotide change affects the protein's affinity for binding with its substrate, cofactor, or regulatory molecule, the amount of a nutrient required by that individual is often affected. This topic has been extensively reviewed by Ames and colleagues (2002). Given the direct connection between genes and nutrients, it is not surprising that each of us requires the same nutrients but in differing amounts depending on the interactions between our unique genetic makeup and the specific environment in which we are functioning.

Gene-environment interactions of particular relevance to women's health include: folate and cardiovascular disease, cancer, and neural

tube defects; dietary fat and vascular disease and adipocyte metabolism; dietary salt and hypertension, caffeine and heart disease and osteoporosis. Numerous studies have been reported in the scientific literature for each of these examples. The folate-MTHFR gene interaction is one of the best understood gene-environment interactions and has implications for multiple disorders. The 2005 review by Kauwell explains the system well in addition to placing it within the broader context of nutritional genomics and its relevance to dietetics.

An additional aspect of nutritional genomics that is being explored is epigenetics. The genetic variations previously discussed involve changes to the nucleotide sequence of DNA and can influence gene expression. Epigenetic changes can also influence gene expression but do so without altering the DNA sequence. By modifying the chemical structure of DNA and its associated histone proteins, such as by attaching methyl groups, gene expression can be silenced. There are several epigenetic strategies that cells use to selectively silence or activate particular genetic regions. A key point here is that although each cell contains the full complement of DNA in its nucleus, it does not use all of its information all of the time and upon differentiation, may only use a small, select portion of the information it possesses. Epigenetic "markings" serve to flag regions of DNA for silencing. Inappropriate silencing or activation can result in a myriad of outcomes ranging from abnormal cellular function to overt disease. One obvious connection to nutrition is the role of folate as the key methyl donor. Genetic variations that affect folate metabolism will likely affect epigenetic marking. Is it possible to over-methylate, at least for some genotypes, and inadvertently silence genes that should be expressed? Clearly understanding genetics will be important to nutrition policymaking.

In order to make diet and lifestyle choices appropriate to our genes, we need to know which genetic variations we have. Nutrigenetic testing involves analyzing a DNA sample, typically obtained from swabbing the inside of the cheek, to identify specific genetic variations associated with particular diseases. This information is integrated into the overall nutrition assessment, and diet and lifestyle recommendations are made for disease management or prevention purposes. Be aware that, although such genetic testing is a hot topic right now, it is simply a tool that enhances the nutrition assessment by providing information not otherwise available. It is not an end in itself.

*For additional overviews of nutritional genomics and background information, see the excellent reviews by Kauwell (2005), Ordovas & Corella (2004), the books **Genetics: the Nutrition Connection** (DeBusk, 2003) and **It's Not Just Your Genes!** (DeBusk & Joffe, 2006). Various authors have published reviews of nutritional genomics in the *Journal of the American Dietetic Association (JADA)*. Also watch *JADA* for upcoming articles on epigenetics and its relevance to dietetics.*

Where are we now?

Nutritional genomics requires a substantial research base from which to develop applications. Much of that base is being developed in earnest now, with numerous laboratories worldwide engaged in research relating to various aspects of nutritional genomics. The primary issues being addressed concern identifying the genes that are

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responsive to environmental factors; determining the impact of changes in these genes on protein function and nutritional requirements; identifying the bioactive components that influence gene expression; understanding the mechanisms by which these factors communicate with the genetic material; developing and validating diagnostic tests that detect variants in genes responsive to diet and lifestyle; surveying various populations to determine the frequency of key variants within those populations, which in turn affects the prevalence of particular diseases; and ultimately, translating these tools and genetic knowledge into diet and lifestyle choices that lead to effective disease prevention and disease management strategies.

Clearly, nutritional genomics is a science in development. In addition to the need for research and the education of practitioners in this area, there are issues of concern to health professionals and consumers alike. In order to make choices appropriate to our genes, we must know which genetic variations we have and their linkages to health and disease. Such knowledge requires that genetic testing assays be validated and conducted in appropriately credentialed laboratories. In response to the Personalized Health Care initiative introduced in March 2007 by the Secretary of Health and Human Services, a major report to be released in 2008 is presently being developed that provides a detailed roadmap for establishing such a credentialing program (www.hhs.gov/myhealthcare). Other issues concern the handling of genetic data. Will our privacy be maintained and will we be protected against misuse of the information, particularly for insurance and employment purposes? Will people be stigmatized if they're known to have particular genetic variations? Should children be tested? It makes sense to determine early in life what one's genetic makeup is and tailor life choices accordingly. Currently it is legally permissible for children to undergo genetic testing, but the standard of practice is not to test individuals below the age of consent.

As an encouraging note, current and proposed legislation appear to protect us from many of these possibilities (see Reilly & DeBusk, JADA 2008, for a discussion of these types of concerns). Nutrigenetic testing identifies genetic variations that increase susceptibility to developing a disease, not genetic changes that virtually guarantee we will develop a disorder. Further, it targets only those variations for which diet and lifestyle modification can significantly decrease or even prevent disease from developing. There is reason to be optimistic that nutrigenetic testing data will ultimately be seen as essential information for assessing health status, in the same way that we view height, weight, and blood pressure.

Professional Opportunities

The impact of nutrigenomics will extend far beyond the obvious clinical nutrition applications. Equally important will be the applications to food science and the development of health-promoting foods, the need to educate consumers and professionals alike in this new science, and the development of nutritional standards for different populations. Thus, virtually every aspect of dietetics will be affected. Dietitians are urged to begin now to add competency in genetics in general and nutritional genomics in particular to their knowledge base. Although the impact will not be fully felt for another decade or so, health-savvy clients are already interested in

nutrigenetic testing and in adjusting their diet and lifestyle choices in ways that could extend life and promote health. It is time to embark on the exciting journey that is ahead for the dietetics profession. Inappropriate matching of therapeutic interventions to clients' genetic makeup is suspected of being a major limiting factor in the success of such interventions in health care today. As interventions are increasingly tailored to our genetic variability and efficacy is enhanced significantly, diet-gene interactions will emerge as central to disease management and prevention and the role of the dietitian will change dramatically. When that opportunity knocks, dietitians, please be ready to throw wide the door and capture this exciting field from every vantage point!

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THE GENE AND NUTRITION (NUTRIGENOMIC) CONNECTION IN THE LACTATION PERIOD

Annette Haban Bartz, MS, RD, LD, CLC *Nationwide Children's Hospital, Columbus, Ohio.*

Forty some years ago, James Neel proposed in the Thrifty Genotype Hypothesis that poor nutrition led to changes in the actual DNA. Years later, David Barker refined this theory into the Thrifty Phenotype Hypothesis, sometimes called the Barker Hypothesis, by suggesting that it is not the actual DNA that changes, but what the DNA is told to do. Barker proposed that there are epidemiological associations between poor fetal and early postnatal growth to nutrition and subsequent development of adult chronic diseases. This is sometimes called Fetal Origin of Adult Disease. The basic premise involves the genes. They need instructions for what to do, and when and where to do it. The proteins around the genes themselves are the chemical switches that lie along the length of the DNA double helix and switch on or off the expression of that particular gene, telling it what to do. With regard to this hypothesis, an insult or stimulus occurring during the fetal and neonatal period can change the gene expression. Barker is credited with saying that "The genes supply the general recipe to make a human being, but the human being is the product of the recipe plus the specific ingredients that the mother supplies."

The theory behind both the Thrifty Genotype and the Thrifty Phenotype is the Developmental Plasticity Theory which suggests that an organism adapts to its environment during its life course. The results appear to be long-term consequences of how the body will function. The adaptation may be a positive thing in that the mother is cueing the fetus or neonate about what kind of environment it will experience and the fetus or neonate is making adaptations so that it can survive. There are three proposed ways that early malnutrition may affect cellular mechanisms: 1) a permanent decrease in the actual number of cells in the specific body structure which could lead to a smaller-sized organ; 2) a permanent change in the gene expression (epigenetic effect on the phenotype); and 3) a change can be duplicated so that not only can this change affect the gene expression of an organism itself, but it appears to be able to be passed on to subsequent generations. Any change should not be viewed as pathological, but as appropriate to the conditions in which the individual is growing.

Prenatally, the changes are adaptations to the poor uterine environment. In an attempt to preserve the brain in the poorly nourished environment, and possibly to adapt the organism to the extra-uterine environment, the other organs may not develop as expected, leading to

altered functioning of those organs. Postnatally, the changes that may have occurred during the prenatal period can be amplified. This may depend upon what is fed or what stressors the infant is or is not now exposed to. The fetus or neonate is predicting what the environment will be. This is called Predictive Adaptive Response. But it only works if the "forecast" of what the extra uterine life is going to be, actually occurs. This is the key of the whole theory of fetal or neonatal programming. If, as a fetus or neonate you make adaptations for a life that you expect and the life that you get is indeed the life that you adapted for, life is good. If you made those adaptations as a fetus or neonate, but the life that you actually GOT was different, you may have problems, i.e., adult diseases.

An example is the infant who is low birth weight. There is a reported relationship of the infant with Intrauterine Growth Restriction (IUGR) and adult hypertension. The infant with IUGR has set up to have a life of less than optimal calorie and protein intake and slowed growth and appears to alter the functioning of the kidneys and cardiovascular system in preparation for that life. The infant with IUGR who then has accelerated growth as a child, as an adult seems to be more susceptible to many adult chronic diseases related to kidney and cardiovascular functioning. If the less than optimal life does come to fruition, the person does not appear to suffer from those adult chronic diseases, although he/she may have other issues as a result of continued poor nutrition. In other words, less than optimal prenatal nutrition + slower rate of growth as a child or lack of catch up growth = less chance of adult chronic disease. Less than optimal prenatal nutrition + accelerated growth as a child = greater chance of adult chronic disease.

Many parts of the human body have been suggested to be affected by the poor fetal or neonatal nutrition and are thought to be related to this adaptive response during the fetal or neonatal period. Included in the list are: brain development, including visual processing; bone development and health including a relationship to future osteoporosis; development of muscle fibers and long term grip strength; cardiovascular health including the relationship to blood pressure; appetite control especially related to leptin; and glucose/insulin homeostasis, including a relationship to metabolic syndrome. Other areas being studied are breast cancer development, and development of schizophrenia. The list continues to grow as scientists delve further into this arena.

How does this all relate to breastfeeding the

neonate? The scientific evidence on what can be done in the neonatal period to change what has happened in the fetal period is still sketchy. Barker identifies the period from birth to 24 months of life as a "Critical Window of Development" and challenges researchers to take a look at what can be done to change the course set prenatally to a new course during the neonatal period. He has even set up a foundation to encourage the research. A limitation of many of the studies on maternal nutrition during pregnancy and lactation is that the prenatal period and lactation period are considered together, making it difficult to differentiate the effects of maternal nutrition during the prenatal period from those effects during the lactation period. Recent research by Caitlin Wyrwoll and her associates in Scotland studying neonatal sheep that were made hypertensive because of changes made to the mother during the prenatal period found that feeding the lambs a postnatal diet high in omega 3 fatty acids could actually reverse the programmed effects on the cardiovascular system. These results have also been found to be true in a study using rats. It is a long way from extrapolating data from sheep and rats to humans, but given that omega-3s appear to be beneficial in other respects such as brain development and visual processing, it seems to be prudent to encourage lactating women to consume diets rich in omega-3s. The fat in human milk is reflective of the fat content of the mother's diet during pregnancy and lactation. Consuming higher levels of omega-3s will increase their concentration in the mother's milk.

Human milk is the best source of nutrition for human infants. As the area of nutrigenomics becomes better understood and the research on Fetal Origins of Adult Disease continues, we may find ways to tweak the diets of the mother to improve the long-term adult health of the breastfeeding infant. In the meantime, encouraging a healthy diet for the mother and promoting breastfeeding for the infant should be our goal. Stay tuned.

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SOYFOODS AND BREAST CANCER: Beneficial or Harmful?

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Introduction

The possibility that soyfoods reduce cancer risk, and especially cancer of the breast, first attracted widespread attention in 1990.¹ Several factors sparked this initial interest. The most notable were the historically low breast cancer rates in Asia² and early research in mice showing that the naturally present isoflavones in soybeans held the potential to exert antiestrogenic effects.³

During the past 20 years, the soy-breast cancer relationship has been rigorously investigated but no clear consensus on the preventive properties of either soyfoods or isoflavones has emerged.⁴ Furthermore, there is concern, based primarily on in vitro and rodent data, that because soyfoods contain isoflavones, they may actually be contraindicated for breast cancer patients and for women at high-risk of this disease.

These concerns have led to questions about the value of incorporating soy into the diet despite data suggesting that soyfoods may reduce risk of coronary heart disease⁵ and osteoporosis,⁶ and may help alleviate hot flashes.⁷⁻⁹

The purpose of this article is to briefly examine the evidence that soyfoods reduce breast cancer risk and to assess whether these foods pose a risk to some women. Before addressing those issues, brief background information on isoflavones will be presented since these soybean constituents are at the center of both issues.

Isoflavones

Isoflavones are diphenolic compounds with a chemical structure similar to estrogen; they bind to both estrogen receptors alpha (ERa) and beta (ERb). The two primary soybean isoflavones are genistein and daidzein; a third isoflavone glycitein, is present in relatively small amounts. In soybeans and unfermented soyfoods, isoflavones are naturally present as the glycosides (that is, with a sugar molecule attached) genistin, daidzin and glycitin.

Isoflavones exhibit estrogen-like properties but bind much more weakly to estrogen receptors (ERs) than estrogen. However,

circulating levels of isoflavones in people who consume soyfoods are approximately 1000-fold higher than estrogen.¹⁰ For several reasons, including their preferential binding to ERb in comparison to ERa, isoflavones are often classified as selective estrogen receptor modulators (SERMs).¹¹ SERMs have tissue selective effects, exerting estrogen-like effects in some tissues but having either no effect or antiestrogenic effects in other tissues. An ideal SERM would, for example, have estrogenic effects on the skeletal system but antiestrogenic effects on the breast. A number of commonly-used drugs, such as tamoxifen and raloxifene, used to treat breast cancer and osteoporosis respectively, are also SERMs.

While there is some debate about the classification of isoflavones as SERMs, it is abundantly clear that there are many differences between isoflavones and estrogen. For example, conjugated equine estrogens and/or estradiol are known to increase levels of C-reactive protein, sex hormone binding globulin, triglycerides, high-density-lipoprotein, and thyroid stimulating hormone, and to increase endometrial thickness.¹²⁻²² In contrast, isoflavones have no impact on these biological measures.^{11, 22-31} Therefore, isoflavones cannot be equated with estrogen.

Breast Cancer Prevention

Rodent studies have generally shown that, when isoflavones or soy protein are given prior to the administration of chemical carcinogens or the implantation of cancer cells, mammary tumor development and/or growth is inhibited, although not all studies show this to be the case. In regard to the epidemiologic data, generally the findings have been viewed as relatively unimpressive.

For example, a meta-analysis published in 2006 by Trock et al.³² that included 18 studies (12 case-control and 6 cohort or nested case-control) found that among all women, high soy intake was modestly associated with reduced breast cancer risk (odds ratio [OR] = 0.86, 95% confidence interval [CI] = 0.75 to 0.99). However, the association was not statistically significant among

women in Asian countries (OR = 0.89, 95% CI = 0.71 to 1.12). It is the Asian studies that are most relevant since soy intake among Western women is typically very low and, therefore, of questionable biological significance.³³

However, in contrast to the above findings, a recently published meta-analysis by Wu et al.³⁴ found that isoflavone intake from soyfoods was associated with a 29% (95% CI, 0.60-0.85) reduction in risk when comparing high (≥ 20 mg/day) versus low (≤ 5 mg/day) intake. Intermediate (~ 10 mg/day) intake was associated with a 12% (95% CI, 0.78-0.98) risk reduction. This analysis included 7 case-control studies and 1 cohort study, all of which involved women of Asian ethnicity.

Importantly, the inclusion criteria by Wu et al.³⁴ also required that soy intake be comprehensively assessed and appropriate consideration given for potential confounders. Many studies, especially those published early on, recorded only the intake of select soyfoods, not total soy intake. Finally, this analysis found that soy/isoflavone intake was equally protective against both pre- and postmenopausal breast cancer, but in the 11 studies involving non-Asians with very low soy intake, no protective effects were observed.

In addition to this important meta-analysis, a recently published study of women in China suggests that the epidemiologic data may not be the most sensitive means by which to evaluate potential biological effects of isoflavones. This research

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involved 196 women with breast cancer, 304 with benign fibrocystic breast conditions and 1,002 age-matched controls. The study found that there was a marked inverse relationship between serum isoflavone concentrations and risk of both breast disease and breast cancer risk.³⁵

However, a previously published analysis involving this same group of women (this study was included in the meta-analysis by Wu et al.³⁴) found that soy intake was not related to breast cancer risk.³⁶ One explanation for these apparently discrepant findings is that soy and isoflavone intakes are difficult to accurately assess. A more likely explanation is that serum isoflavone levels are a much more accurate reflection of tissue exposure than either soy or isoflavone intake because isoflavone metabolism varies markedly among individuals.¹⁰ Thus, epidemiologic studies assessing only intake may not be a sensitive means by which to assess the biological effects of isoflavones.

Despite the supportive animal and epidemiologic data, clinical studies – those which involved both pre- and postmenopausal women and evaluated the effects of either isoflavones or soyfoods on markers of breast cancer risk, including breast tissue density and breast cell proliferation – fail to demonstrate chemopreventive effects. These data are discussed below. Without clinical support, it will be difficult to reach a firm conclusion that soy intake reduces breast cancer risk. Furthermore, questions remain about the effects of soy exposure at different periods of life and when such exposure is likely to have the most benefit.

Evidence indicates that the greatest benefit of soy is achieved when exposure occurs during childhood and/or adolescence. The hypothesis that early soy intake reduces later risk of breast cancer is supported by both epidemiologic and animal data and is consistent with mounting evidence that early life events greatly impact breast cancer risk.³⁷ The first 20 years of life appear to be particularly important.³⁸

Specifically in regard to soy, research from the University of Alabama has shown that, when rats are given genistein for just a few weeks early in life and then put on a typical laboratory diet, they develop 50% fewer

tumors than rats not given this isoflavone.³⁹ In agreement with these findings, women from Shanghai who consumed the equivalent of about 1 1/2 servings of soyfoods daily when they were 13 to 15 years of age were 50% less likely to develop breast cancer as adults compared to Chinese women who consumed little soy during adolescence.⁴⁰

More recently, researchers from the National Cancer Institute reported that in comparison to low-soy consumers, women who were classified as high-soy consumers during the ages of 5-11, 12-19, and 20+ years of age were 58%, 21% and 29% less likely to develop breast cancer, respectively.⁴¹

Finally, results from the first case-control study of its kind showed that the odds ratio of developing breast cancer for women who were fed soy infant formula exclusively during the first four months of life or during months 5 through 12 were ~0.42 (95% CI=0.13-1.40) and 0.59 (95% CI=0.18-1.90), respectively.⁴² The reference group in this study consisted of women fed only breast milk and/or cow's milk formula. These findings are provocative but also very preliminary.

Questions Regarding Contraindications to the Use of Soy

The estrogen-like effects of isoflavones form the basis for concern that soyfoods could theoretically be contraindicated for women at increased risk of breast cancer or for breast cancer patients with estrogen-sensitive tumors.⁴³⁻⁴⁷ Some argue that, in the low-estrogen environment of postmenopausal women, isoflavones will act as estrogen agonists in the breast.

There are two points that are germane to this line of thinking. First, although circulating estrogen concentrations are markedly lower in post- compared to premenopausal women,⁴⁸ estrogen levels in breast tissue and breast tumors are actually similar in these two groups of women due to *de novo* synthesis within the breast and uptake from the circulation against a concentration gradient.⁴⁹⁻⁵²

Second, there is relatively little evidence that exposure to exogenous estrogen (in contrast to exposure to estrogen plus progestin) increases breast cancer risk. In fact, in the estrogen-alone arm of this trial, after an average of 7.1 years of follow-up, women

assigned to conjugated equine estrogens (CEE, 0.625 mg/d) were 20% less likely ($P = 0.09$) to develop invasive breast cancer compared to women in the placebo group.⁵³

Nevertheless, in certain types of experimental rodent models, isoflavones do stimulate the growth of existing estrogen-responsive mammary tumors.^{54, 55} In the most commonly cited model, tumors are stimulated in athymic ovariectomized mice implanted with MCF-7 cells, an estrogen-receptor positive (ER+) human breast cancer cell line. Stimulation appears to result primarily from exposure to genistein. But despite containing similar amounts of this isoflavone, more highly processed soy products stimulate tumor growth to a greater extent than less processed ones like soy flour. In fact, soy flour does not stimulate tumor growth.⁵⁶ Whether these processing effects are relevant to humans is speculative.

In contrast to the results in mice, the human data suggest that isoflavones do not exert stimulatory effects on breast tissue. For example, in three trials in postmenopausal women and one in premenopausal women, in which biopsies were taken before and after exposure to isoflavones, there was no increase in breast cell proliferation (a marker of increased cancer risk) and no effects on other measures of estrogenicity related to isoflavone exposure.^{28, 57-59} In agreement, four studies also found isoflavone exposure does not increase breast tissue density.^{4, 60-62} Importantly, estrogen plus progestin therapy increases breast tissue density and breast cell proliferation,^{63, 64} and increases breast cancer risk.⁶⁵

Finally, the epidemiologic study most relevant to the soy-breast cancer controversy found that neither soy nor isoflavone intake was related to disease free survival over the 5.2 year follow-up period.⁶⁶ In this study, of the 1001 (total cohort included 1459 subjects) Chinese breast cancer patients for whom data on receptor status was available, approximately 63% were estrogen-receptor-positive.

Summary and Conclusions

There is evidence that soy intake reduces breast cancer risk, particularly when isoflavone exposure occurs during childhood

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and/or adolescence. In regard to concerns that soy may stimulate the growth of existing estrogen-sensitive breast tumors, although there are in vitro and animal data suggesting this possibility, the clinical and limited epidemiologic data suggest otherwise.

Further assurance of safety comes from evidence that exposure to the much more potent estrogen and conjugated equine estrogens do not increase breast cancer risk, either substantially or at all. Thus, overall, there is little reason to conclude that isoflavone-rich soyfoods are contraindicated for women at high risk of breast cancer or for breast cancer patients. This conclusion is consistent with the current position of the American Cancer Society.⁶⁷

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WAKING UP FROM THE DIABETES GENETICS NIGHTMARE

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Dr. James V. Neel, the father of human genetics, wrote a chapter entitled “Diabetes Mellitus – A Geneticist’s Nightmare” in 1976 (1). At that time, little was known about diabetes genes; the first successful identification of a disease gene using the positional cloning approach, the gene for cystic fibrosis, would not occur for another 13 years (2,3). However, the possibility of genetics playing a role in diabetes risk was generally known. People would talk about diabetes “running” in their families, which was essentially describing the passing of diabetes genes from one generation to another. However, what those genes were and how they worked to confer risk were mysteries and Dr. Neel’s chapter served as a warning of the challenges that lay ahead.

The benefits of identifying diabetes genes seemed tremendous. The discovery of these genes would allow: accurate prediction of who might develop diabetes, creation of therapies tailored to each individual, identification of new drug targets, and maybe development of gene therapies to correct the genetic defect(s) and cure the disease. Between 1976 and 2006, tremendous effort was expended to identify diabetes genes. There were success stories, like the identification of peroxisome proliferator-activated receptor (PPARG) as a diabetes gene, but in general Dr. Neel’s predictions rang true. The reality was that the individual effect of diabetes genes was small and the technology and methods of the 1980s and ‘90s were not powerful enough to detect these effects.

However, conditions changed rapidly as we entered the new millennium. The Human Genome Project (<http://genome.ucsc.edu>), an effort to decode our DNA, completed a first draft of the human genome in 2000 (4). The HapMap Project (<http://www.hapmap.org>), an effort to identify genetic difference across human populations, was initiated and has identified over six million differences across the human genome to date (5,6). Finally, improvements in technology now allow scientists to measure hundreds of thousands of genetic differences across the human genome in several thousand individuals in the span of weeks, whereas such a feat was deemed impossible a mere decade ago.

These breakthroughs, coupled with improved study designs and statistical methods, have led to a new era in human genetics. Prior to 2006, only two genes for type 2 diabetes had been identified and universally accepted by the scientific community. In 2006, DeCode Genetics, a private venture studying the population of Iceland to identify genes for a variety of diseases, reported the identification of transcription factor 7-like 2

(TCF7L2) as a susceptibility gene for type 2 diabetes (7). Then, in the first six months of 2007 seven new genes for type 2 diabetes were reported by five independent research groups (8-12). What was important about these recent findings was the significant overlap in results across the studies and that many of the genes had hypothesized effects on the pancreatic beta cell. Four more genes will appear in the literature by the time this article is published. However, individually each of these genes only raised the odds for diabetes by 5-10% and together they accounted for only a small fraction of diabetes cases. The specter of Dr. Neel’s diabetes nightmare seemed to rise again.

So far, our knowledge of these ten diabetes genes has not allowed us to predict diabetes, strongly suggesting that the identification of genes is merely the first step. We still need to understand how these genes work to increase risk for diabetes and if they also confer risk for gestational diabetes mellitus (GDM). This has been the focus of the research at the Keck School of Medicine of University of Southern California (USC). Previous research by Dr. Thomas Buchanan of USC showed that Latinas diagnosed with GDM are at elevated risk for type 2 diabetes (13-15); an observation that has been extended to other ethnic/racial groups (16). In his studies, over half these women developed type 2 diabetes within five years after delivery (13), and their risk increased with each subsequent pregnancy (14). His research also noted these Latinas did not appropriately increase their insulin secretion compensate for insulin resistance in peripheral tissues (15,17), but their risk for type 2 diabetes could be significantly reduced by reducing insulin secretory demand by treating them with a thiazolidinedione (18,19).

Given these observations, five years ago the Keck School initiated the BetaGene Study with the goal of identifying genes underlying this inability to secrete insulin and to understand how those genes worked to increase risk for type 2 diabetes. They are collecting data on families of a Latina with a previous diagnosis of GDM, collecting their DNA, measuring their body fat, and doing detailed metabolic measurements. To date, over 2,500 individuals in over 200 families have been studied. The study is investigating whether the genes identified for type 2 diabetes are also associated with GDM and the specific measurements from the BetaGene participants. More importantly, they are also studying whether the effect of these genes is altered by other factors like body fat, diet, or physical activity. For example, they recently showed that the effect of TCF7L2 on insulin secretion differed by the

amount of body fat (20). This observation is important, because it is generally known that a 7-10% loss of body weight can reduce type 2 diabetes risk by an average of ~50%. However, individual risk reduction with weight loss may depend upon the specific genes, like TCF7L2, that interact with body fat. Efforts like the BetaGene study should provide new insights into how these diabetes genes interact among themselves and with environmental factors to alter biology and increase risk for diabetes, one of the complicating factors noted in Dr. Neel’s nightmare scenario.

It has been thirty years since Dr. Neel warned us of the “geneticist’s nightmare”. Dr. Neel recognized the challenge in identifying diabetes genes, even in the age where the genes were unknown. But with the identification of type 2 diabetes genes and the likely discovery of additional genes in the near future, we are just now starting to awaken from Dr. Neel’s “nightmare”. The next few years should begin to see translation of some of the promises of genetic research into diabetes treatment and prevention.

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MENTOR ME! *How to select and use a mentor to enhance your career success*

By Jean R. Caton, MS, MBA, RD

Chances are we have all had a mentoring relationship of some type; many of us are mentors to others. In a recent survey by CareerWomen.com, 64% of the respondents indicated they have a formal or informal mentor. Successful women frequently credit a mentor for helping them advance in their career.

Who needs a mentor? Aspiring entrepreneurs, successful business people, those just starting out, and everyone who wants to be a career confident woman or man can benefit from having a mentor.

Personally, I have benefited from several mentors throughout my career. Most recently, several informal and formal mentors helped facilitate my transition from a 20-year career in a large organization to owner of a small business. I am fortunate to have identified mentors who generously share with me business contacts, resources, tools, and tips that enhanced the success of starting my business.

From the Greek Classics to the modern business world: The descriptions of Mentor, a character in Homer's *Odyssey*, from which we derive the term mentor, give insight into the mentoring relationship. Homer's character Mentor is a person of deep trust, a wise old man who instills knowledge and guides one to find the answers he seeks. In the modern world, a mentor does essentially the same thing. A mentor can provide advice, facilitate networking with key contacts, accelerate progress on the learning curve, help navigate the organization's culture, guide decision-making, and be a source of feedback and motivation. Perhaps some of the most beneficial roles of a mentor are to empower, inspire, and believe in you and your success potential.

Finding a mentor who is right for you: The first thing to consider when seeking a mentor is your goals. Define what you are looking for and where you need a mentor's support. Then, observe success. A good mentor is someone who has *been there and done that*. She/he is someone who has the knowledge and insight to provide valuable information consistent with your goals. Observe the strengths of your managers, peers, and colleagues in your network. Consider those who have strengths in the areas you most need to develop. Ask them to be a mentor for you. If you want to be part of the formal mentoring program in your organization, don't wait to be noticed. Asking for what you want often is one of the

most daunting things for many mentor-worthy individuals. If you are uncomfortable speaking up, speaking out and asking for the support of a mentor, then this becomes your number one goal for your work with a mentor or a coach.

Another way to find a mentor is to participate in volunteer and professional organizations both within and outside your profession's organizations. You may even consider a long-distance mentor with whom you can speak regularly by telephone.

Tap different expertise: You need not limit yourself to one mentor. Have an entire "Advisory Board of Mentors" if you are fortunate enough to find people who are willing and capable to serve in that capacity.

Female or male mentor: Selecting a female or male mentor again depends upon your goals. Cross-gender mentoring can have many benefits. Men and women typically have different strengths. Males can provide guidance in developing the skills to work in a male dominated field; men often have good negotiating skills and are usually the majority of the senior leaders in an organization so they can help with advancement of your career by facilitating your relationships with other senior leaders. Women's strengths include dealing with the gender bias, building a network of business relationships and team management.

Be mentor worthy: Use your mentor's time productively. Be sure to acknowledge appreciation for the time and effort your men-

tor is offering you. Complaining or whining is not a constructive use of your time with your mentor. Here are ten tips for a successful mentoring relationship:

- Know what you want and have clear goals for the relationship.
- Communicate your expectations to your mentor and discuss feasibility. You, not your mentor are responsible for your results.
- Be open, honest, and direct with your mentor.
- Establish priority issues for action or support.
- Come prepared to each meeting with issues to discuss.
- Don't expect your mentor to be an expert in every facet of business.
- Ask for and use resources and contacts for other members in and outside of the organization.
- Solicit feedback from your mentor. Don't take constructive upbraids personally. Recognize the feedback of a good mentor is in your best interest.
- Take action. Even when your mentor asks you to stretch outside your comfort zone.
- Acknowledge your mentor's help. A formal thank you note describing how the mentor has been of benefit is a way to say thanks.

Continued on page 13

Get Connected at the Food & Nutrition Conference & Expo

Check out your Program Preview to be included with the June issue of *Journal of the American Dietetic Association*



Network with colleagues, become informed and get energized while earning CPE at this year's Food & Nutrition Conference & Expo. An exciting array of sessions have been developed to keep you current in your field. Here is a just a sampling of topics that will be featured:

- Celebrating Culinary Trends to Meet Consumer Taste and Nutritional Needs
- Child Nutrition Initiatives: Less Talk, More Action
- The Science of Organics: Nourishing the Land, Animals and People
- Success Strategies for Unlimited Career and Income Growth

NEW! Hot Topic Sessions showcasing cutting-edge research, policy and advocacy issues, and newsworthy topics.

Learn more about these and over 140 educational events that will be offered by visiting www.eatright.org/fnce2008.

American Dietetic Association
Food & Nutrition Conference & Expo
McCormick Place West / October 25-28, 2008 / Chicago, Illinois

WOMEN'S HEALTH ANNOUNCES OUR NEW DPG LEADERS!

We are pleased to introduce to you the new, outstanding leaders of our DPG:

Chair Elect, 2008-2009, Chair 2009-2010

Denise Andersen, MS, RD, LD

Clinical Nutrition Manager
United Hospital, St. Paul, MN

"...the goals of the Women's Health DPG provide us with the opportunity to evolve in all areas that will impact the health and well being of women."

Denise Andersen participated in the State President's forum at FNCE and attended ADA's Leadership Institute. She has served as President of the Minnesota Dietetic Association.

Secretary, 2008-2010

Laura J. Couillard, MS, RD

Kingwood, TX

Laura served as the FNCE Coordinator for Women's Health DPG in 2003, 2005, 2006 and 2007.

"The evolution of this DPG is emerging into an all encompassing Women's Health group. The leadership and dedication of its Board and Coordinators has inspired me to move towards a deeper commitment."

Nominating Committee Chair-elect, 2008-2009, Chair 2009-2010.

Suzanne Haydu, MPH, RD

Public Health Nutrition Consultant III (Specialist)
Maternal, Child & Adolescent Health Program
California Department of Public Health

"...My interest is having the WH DPG 1) identify emerging issues, science-based assessments and science-based interventions in women's nutrition, 2) promulgate the role of the RD in reducing health disparities, and 3) market the WH DPG as the leader and authority in perinatal nutrition."

WOMEN'S HEALTH LEADERSHIP CONTACT INFORMATION

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MEMBER SPOTLIGHT Interviews by Allison Starr

*This year we have been delighted to feature our very own members, their career paths, their accomplishments and their favorite resources. This spring, April Rudat, author of **Oh Yes You Can Breastfeed Twins** joins us to share her own trajectory through dietetics guided in part by chance, passion and hard work.*

The wide and varied paths of dietetics from clinical to corporate, community health to self-employment, researcher to public speaker is a true testament to the many talents we each bring to our profession and our creativity in shaping dietetics to meet our own needs and passions.



April Rudat, MS, Ed, RD, LDN

Where have you worked? What was your first job?

Like many others, I began my dietetics career in clinical dietetics, though my true love has always been nutrition counseling. After a short time working clinically, my husband's job changed, requiring relocation. And the job of my dreams was open in the area where

we were moving! I began working in public health, providing free nutrition counseling at a city health department. In this position, I had the opportunity to work with overweight children and adults, create a fitness program for overweight kids, and establish and implement a prenatal nutrition counseling program for pregnant teens.

Following another relocation due to my husband's job, I began graduate school and continued outpatient dietetics work at a large teaching hospital in Virginia. During this time, I also became pregnant with and bore twins, which would eventually open the door to a new career!

After having experienced the challenges of breastfeeding and parenting twins, I wrote and published a book titled, *Oh Yes You Can Breastfeed Twins!...Plus More Tips for Simplifying Life with Twins*. While writing the book, I established my own business, which includes selling the book, freelance writing, and speaking (See: www.ohyesyoucanbreastfeedtwins.com). In addition, I also teach part-time as adjunct faculty at Marywood University in Scranton, PA and hope to teach elsewhere as opportunities come available.

What articles/books/publications, if any, have you written?

As mentioned, my book, *Oh Yes You Can Breastfeed Twins!* published in July 2007. In addition, I have written several features in "Today's Dietitian" magazine including articles titled, "Breastfeeding Multiples—Promoting 'Breast Is Best' to Mothers of Two, Three, or More" and "Tips for the Stay-at-Home RD." I have also written multiple articles for DPGs and my state and district dietetic associations.

What are your favorite tools and resources on women's health?

The WH DPG listserv and website is my favorite resource on women's

health! And since my current career focus is on breastfeeding, infant feeding, and childhood nutrition, I utilize many breastfeeding and childhood nutrition websites including www.lli.org, www.4woman.gov/breastfeeding, and www.sbanutrition.com. Of course, I also rely heavily upon www.mypyramid.gov and www.eatright.org.

What do you consider a highlight of your career?

The highlight of my career and my life was having twins – without them, I could not have learned about breastfeeding multiples and therefore would not have been able to write as effectively about it. In addition, breastfeeding and parenting twins created within me a new passion: active promotion of breastfeeding as the first way to prevent childhood obesity.

What are your recommendations for WH DPG?

My only recommendation for the WH DPG is to continue the great work it does – striving to keep its members aware of the latest in women's health issues and making great strides to improve nutrition related to women issues.

Do you have a great presentation on breastfeeding or lactation management? The WH Breastfeeding Task Force is looking for YOU!

With the unveiling of our new WH website in late September, the WH Breastfeeding Task Force has taken on the task of putting together a library of presentations on the topic of....you guessed it...BREASTFEEDING. The idea behind this is to have each presentation go through a series of peer reviews and eventually load these presentations onto the new website so that viewers can not only view them, but also download and use them.

Here is what we need from everyone interested: Please send your presentations directly to Lisa Hamlett at liser13@hotmail.com. Please include the estimated length of the presentation, the target audience, any links or resources, and your contact information should anyone viewing the presentation want to contact you. All presentations must be wholly owned or copyrighted by a WH member. Presentations produced by WH members for a non-profit organization (e.g., health care facility) that holds the copyright, must have written permission from that organization for a presentation to be included on the Web site. Permission must be submitted with the presentation.

We need presentations for a variety of audiences, so if you have anything that you would like to showcase, please submit it!!

Thank you in advance for your help!! If you have any questions please feel free to e-mail Lisa Hamlett at liser13@hotmail.com.

Oh Yes You Can Breastfeed Twins! Plus More Tips for Simplifying Life with Twins

By April Rudat, MS, Ed, RD, LDN

Whether you are a healthcare professional, an expectant mom, or a mom experiencing the challenge of breastfeeding twins, you will find April Rudat's *Oh Yes You Can Breastfeed Twins* to be a truly valuable guide. It is even a good read for dads!

Oh Yes You Can Breastfeed Twins! is divided into three parts:

Part I: What You Need to Know Now

Part II: All about Boobs

Part III: What I Did and What I Learned

The beginning of the book establishes why breastfeeding is best for both mom and babies. Part I of *Oh Yes You Can Breastfeed Twins!* Rudat focuses not only on what breastmilk will do for babies, but - equally important what it can do for mom. Everything from saving time to prolonging the inevitable return of one's period is discussed in the first chapter of the book. She also educates readers on what is needed before the birth; how to design the nursery to make nursing easier; tracking techniques; how to breastfeed; money savers; and a variety of her favorite resources, websites, and magazines that provide additional information on breastfeeding, parenting, and medical information. The tracking section is especially practical as it provides templates of logs and charts for keeping records of feedings, changings, and introduction of solids.

All About Boobs, Part II, educates readers on the anatomy and physiology of the breast; pains associated with breastfeeding; pumping; breastfeeding in public; and clothing tips. Especially informative is a discussion of the symptoms, prevention, and remedies for a variety of ailments including sore nipples, thrush, engorgement, plugged ducts, and mastitis.

Rudat incorporates much of her personal experience to conclude the book in Part III, What I Did and What I Learned. She discusses everything from labor through her twins first year and beyond.

April Rudat, a registered dietitian and successful breastfeeding mother of twins, has incorporated her scientific knowledge with her personal experience to develop a fabulous guidebook on breastfeeding twins. This is a must have for every expectant mother of twins! This book will surely increase the success rate of nursing mothers.

April uses a humorous, yet educational and resourceful tone throughout her book. It is a great read offering an array of advice. As a dietitian and mother I wish I had read this a few years ago when I was breastfeeding!! I will certainly recommend this to my clients and friends.

MENTOR ME! Continued from page 10

Have a mentor and be a mentor: Become a mentor and enhance the journey of another by sharing your time, wisdom, insight and experience. Don't doubt that you have something to offer another in a mentoring relationship – you most likely do. Here are 12 tips to help you be a good mentor:

- Establish clear expectations and boundaries for the relationship
- Create a positive constructive atmosphere.
- Allow mentee choice to accept, reject, or ponder advice.
- Listen and hear both what is and is not being said
- Identify opportunities and offer decision-making guidance as well as solutions.
- Use a problem-solving, coach-approach, asking questions and soliciting mentee input.
- Offer honest and direct feedback. Don't avoid tough conversations.

- Share stories, including mistakes from personal experience- your career “do-overs.”

- Request mentee do ‘homework’ to enhance growth. Only mentor those who demonstrate they are ‘mentor worthy.’

- Serve as a connector to other business colleagues by inviting to lunches, meetings, etc.

- Be open and solicit feedback from mentee

- Be fully present in mentor meetings.

Divorcing your mentor/mentee: Mentoring relationships will eventually end. One reason is that expectations of the mentor or mentee are not met. Other times there are clashes in personalities or styles. Pitfalls around “fit” are not uncommon. It is also possible to outgrow the mentoring relationship. Establishing a mentoring relationship with an ‘until death do us part’ attitude is not the best approach. A good way to avoid a problem ending a mentoring relationship is to establish time boundaries and expectations at the beginning of the relationship. A three-month period of weekly

meetings is a good starting point. Either party has the choice to request an extension of the relationship at the end of the expected period. Moving on and finding new and different mentors is worthwhile. A new mentor can further expand your knowledge skills and perspectives. You are never too experienced to benefit from the support of a mentor.

JEAN R. CATON, MS, MBA, RD, is a speaker, business/lifestyle coach and marketing strategist. She is the owner of McKinley Coaching and Consulting L.L.C. Jean is the 2007-2008 Chair of the Nutrition Entrepreneur Dietetic Practice Group. Jean has been a pioneer in the world of women in corporate America and learned what it takes to succeed in the business world during 22 years working as a marketing executive for four Fortune 500 companies. In 2004, Jean entered the entrepreneurial world and started McKinley Coaching & Consulting LLC. She is a graduate of the Advanced Coach Training program of Coach U, a leading global coach training school. Jean is a coach for women who work. Her niche includes professional women in corporate America, the legal profession, women in health-care, and entrepreneurs. She can be reached at Jean@JeanCaton.com

CALENDAR OF EVENTS

Institute of Medicine and National Research Council Food and Nutrition Board and Board on Children, Youth, and Families Implications of Weight Gain for Pregnancy Outcomes: Issues and Evidence

June 5, 2008

8:00 am - 5:00 pm

The National Academies

Keck Center, Room 100

500 Fifth Street, NW

Washington, D.C.

68th Scientific Sessions - American Diabetes Association

June 6 -10th 2008

San Francisco, California, United States

Website: <http://scientificsessions.diabetes.org>

Contact name: Maricela Arias-Cantu

National Maternal Nutrition Intensive Course

July 30 – August 1, 2008

University of Minnesota – West Bank Campus

Hubert Humphrey Center

Website: <http://www.sph.umn.edu/publichealthplanet/events>

After the Diet – Hormones, The Brain, Appetite and Chronic Disease, Monika Woosley

August 8-9th 2008

Columbus, Ohio

Website: <http://www.afterthediet.com/AftertheDietWorkshop.htm>

6th Annual World Congress on the Insulin Resistance Syndrome

September 25 – 28th, 2008

Los Angeles, CA - Hilton Universal City, California, United States

Website: <http://www.insulinresistance.us>

2008 Cardiometabolic Health Congress

October 15 -18th, 2008

Boston, Massachusetts, United States

Website: <http://www.cardiometabolichealth.org>

ADA Food & Nutrition Conference & Expo (FNCE)

October 25-28th

Chicago, IL

Website: <http://www.eatright.org/fnce2008>

GOALS OF THE WH PRACTICE GROUP

WH DPG promotes the development of dietetics professionals in the specialty area of nutritional care in women's health which includes preconception through pregnancy and lactation and expanded to late menopause.

The objectives of the Women's Health DPG are:

- 1. Build an aligned, engaged and diverse membership.**
- 2. Proactively focus on emerging areas of women's health.**
- 3. Impact the research agenda in women's health and nutrition.**
- 4. Identify and influence key food, nutrition and health initiatives specific to women.**
- 5. Increase demand, utilization and reimbursement of services provided by WH members.**

"WH members are the most valued source of nutrition expertise in women's health"