Therapies...  
Translating Nutrition and Genetic Information Into Clinical Practice  

Vyoone T. Lewis PhD, MPH, RD

The Human Genome Project provides us with the information necessary to understand how hereditary differences make each of us unique. The project highlights new approaches in molecular medicine to the diagnosis, treatment, and management of disease. Genes, which are short segments of DNA, are instructions that tell cells how to behave. As information continues to unravel on the role of genes and how they affect nutrient requirements and/or individual responses to dietary interventions, nutrition practitioners will need to incorporate this information into their nutrition counseling practices.

The interaction of genes, nutrients, and the environment determines phenotype and the development of an individual. Although many chronic diseases have a genetic component, changes in environmental factors, like diet and lifestyle, contribute to the development of these diseases. Genetic variation impacts human nutrition and can account for individual nutrient requirements for therapeutic benefit.

Genetic diseases are usually characterized as single gene defects, chromosomal disorders, congenital malformations, disorders of mitochondrial DNA (cytoplasmic inheritance), disorders due to somatic cell mutations, and multifactorial inheritance, which is the basis for many common diseases.

Some diseases are inherited in Mendelian fashion, which include autosomal recessive, autosomal dominant, X-linked recessive, and X-linked dominant. A dominant gene is one that manifests its

Supplements...  
Hemochromatosis

Susan Moore, MS, RD

Description and Prevalence

Hereditary hemochromatosis (HH) is a type of primary iron overload characterized by a genetic mutation that causes increased intestinal absorption of iron. The excess iron is deposited in the parenchymal cells of the liver, pancreas, heart, and joints and causes inflammation and subsequent fibrosis and destruction. If undetected and untreated, HH can result in cirrhosis, hepatoma, diabetes, cardiomyopathy, arthritis, hypogonadism, and death. Previously considered to be rare, recent estimates place the prevalence of the homozygous genotype at 1 in 250 persons—roughly 1 million people—and about 1 in 9 people are carriers (people with one defective and one normal gene), making HH more common than cystic fibrosis, sickle-cell anemia, or phenylketonuria.

Diagnosis

HH is frequently under-diagnosed, primarily because its sequelae are not specific to iron overload. Consequently, the underlying cause is not recognized or treated and organ damage progresses. The clinical manifestations of HH usually do not appear until a person reaches 40 to 60 years of age, when sufficient iron has accumulated to cause organ damage. By age 40 about 50% of men and 13-20% of women with untreated HH will have clinical manifestations of iron overload such as gray or bronze skin pigmentation, diabetes, and chronic abdominal pain. However, early signs and symptoms of HH include impotence, amenorrhea, irritability, depression, and fatigue. Abnormal laboratory values suggestive of progressive iron overload are an asymptomatic elevation of the liver enzymes alanine aminotransferase and aspartate aminotransferase. Liver disease—particularly cirrhosis—is present in 30-94% of patients
Today I picked up this week’s edition of *Time* magazine. And yes, “Times—they are a changin’”. This issue of *Time* entitled “The Future of Technology” contains an article written by the infamous Bill Gates called “Will Frankenfood Feed the World?” Fittingly, this issue of the NCC newsletter focuses on genetics and biotechnology and the changes ahead for us as dietetics professionals. The world of dietetics as we know it will never again be the same. As the profession changes, so must we change.

The leadership of NCC and the editorial board of the newsletter are constantly working to keep pace with these changing times and provide cutting edge information for our members. You will agree that NCC’s editorial staff is extra-ordinaire when producing a great newsletter covering the latest happenings of nutritional complementary care. Other changes are coming to NCC. Rick Hall, NCC’s Webmaster, and Ruth DeBusk, our Publications Chair, are planning exciting new features for NCC Web site in the near future. Be sure to bookmark www.complementarynutrition.com and visit us often. Make it a priority also to be on line with NCC’s electronic mail list by contacting Gretchen Forsell at gisan RD@aol.com to enroll. On line you will discover colleagues willing to answer questions and share a wealth of information.

The American Dietetic Association is changing as can be seen in the new face of the 2000 annual meeting in Denver-The Food and Nutrition Conference and Exhibition (FNCE). Efforts are being made to keep the ADA in the forefront by providing an annual meeting and exhibition that has something for everyone. Be sure to read about NCC activities being planned for the FCNE in Denver in this issue. We hope to see you in Denver October 16-19, 2000.

Finally, I would like to recognize the change in leadership of NCC. New officers in several positions are eager to help NCC keep pace with the times. Welcome to Rebecca, Felicia, Diane, Geeta, Margaret, Sarah, and Esther (see contact information on the back page). And a very special thank you to those who are saying adieu. Pam, Leslie, Mari and Theresa. Lisa, of course, is still providing NCC with valued assistance in her position as Past Chair.

As a new year begins for NCC, I say welcome if you are new and thanks for joining again if you are returning. Please contact us to let us know how we can help you “keep up with the changing times”. Here’s to a great third year for NCC.

**Editor’s Notes . . .**

Sarah Harding Laidlaw, MS, RD, MPA

As Cheryl so aptly stated, “The times, they are (definitely) a changin’! With this issue you will observe the first major changing of the guard for NCC. This is our third year as a DPG, and what a memorable one it will be. Welcome to an exciting DPG and changing profession!

This past summer most of us could not turn on the news or open a newspaper without hearing about the mapping of the human genome. NCC was ahead of the game, planning this issue on genetics months before the “big” announcement. I am sure that after you have read this issue you will know that we, as a DPG, are in the forefront. We understand that the relationship of clients’ genetics and how we counsel them will determine whether their nutrition prescriptions will work or not.

For a novice in the rapidly changing area of genetics, I have already applied, with success, concepts that I have read about in preparation for this issue. One size diet prescription does not fit all. As the profession changes, yes, we too must change with an open mind. I am sure that many of you will experience what I have as you approach change with an open mind.

A heartfelt “thanks” goes to Ruth DeBusk, RD, PhD the Publications Chair and first Editor of the NCC newsletter. She has been supportive and helpful during this transition, and I look forward to continuing my work with her in the future. Thanks also to those who assisted in preparation of this issue, Ruth, as a major contributor, and all of the Section Editors. And also to Esther Trepal, RD who will be assuming the role of Associate Editor; her help has been invaluable in the past year!

I look forward to working with all 2100 plus members of NCC, individually whenever possible. I encourage each of you to provide suggestions for how we can keep the newsletter and Web site working for you. Your support of the DPG and newsletter is what has made us, in a short three years, what we are today. I am certain that I can speak for all of the Executive Committee when I say that we want this trend to continue.

I hope to see you in Denver. Look for NCC Executive Committee members and make yourself known! Your input is appreciated and wanted.

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Carotenoids, Cancer, and the Connexin Gene

John S. Bertram, PhD

In Western societies, it is now generally accepted that lifestyle, rather than genetics, is the major contributory factor to cancer risk. In a landmark review, Doll and Peto estimated this risk to be approximately 70%, with 30% attributable to use of tobacco products and the remainder to diet and lifestyle. 1 A consistent finding has been that populations with low cancer risk consume significantly higher amounts of fruits and vegetables than populations with high cancer risk. Analysis of the types of fruits and vegetables and of their constituents has reliably implicated the carotenoids as being associated with decreased cancer risk. These findings have been confirmed in studies measuring plasma carotenoid concentrations used is not toxic to human cells. Any changes thus result from changes in expression of the engineered gene. We selected carcinoma cells that did not express this gene and transferred the connexin gene to these carcinoma cells, which then began to express large quantities of functional gap junctions. When these cells were injected into immunodeficient athymic mice, tumor growth was strongly inhibited in the mice in which connexin gene expression was induced by tetracycline as compared with mice in which the connexin gene was not expressed. 6 A search is now on to identify and characterize the nature of the functionally transmitted signals involved in tumor growth inhibition.

Proliferation is central to the production of cancer. 7 Dietary carotenoids, obtained from foods or herbal supplements, counter the loss of junctional communication that occurs in preneoplastic cells. By encouraging cell/cell communication, carotenoids can help suppress abnormal proliferation and, thereby, act as cancer preventive agents. Because all cells are exposed to DNA damage, all individuals probably contain many cells that have a mutation in a gene that, when the cell proliferates, the sequence of events that leads to cancer is initiated. By preventing, or at least decreasing, abnormal proliferation, carotenoids can decrease the rate at which these “initiated cells” are able to progress to malignancy. At present, carotenoids appear to act preventively rather than therapeutically. As in other diseases, however, it is better to aim for prevention than cure. 8

References
3. Zhang L-X, Cooney RV, Bertram JS. Carotenoids under defined cell culture conditions, we found that neither activity correlated with their ability to inhibit experimentally induced cancer. Instead, all active carotenoids were found to cause upregulated (increased) expression of a gene, connexin 43, which codes for a trans-membrane protein that is critical in the formation of gap junctions. 3

Gap junctions are necessary for cell-to-cell communication. Adjacent cells in a tissue communicate with each other through gap junctions in their cell membranes, water-filled pores through which small molecules in the cytoplasm of one cell can pass into the cytoplasm of the adjacent cell, forming a communication network among the cells in a tissue. Each pore is ringed with connexin proteins, six molecules per pore. The connexins on one cell dock with those on an adjacent cell to form a tunnel linking the two cells. Molecules and ions pass through this tunnel. This process is called “junctional communication.” Connexin 43 is the most widely expressed connexin protein and its genetic expression is regulated by carotenoids.

This type of communication occurs in virtually all cell types and is the most direct and most rapid form of intercellular communication known. 4 These findings were exciting since in other studies we had found that growth-inhibited normal cells could inhibit proliferation of tumor cells when cells were in communication through gap junctions. 5

Interestingly virtually all human tumors lack junctional communication. Even in pre-cancerous dysplastic regions of the uterine cervix, connexin 43 is poorly expressed in comparison with normal tissue. 6 Lack of communication in dysplastic cells would lead to enhanced proliferation due to isolation from surrounding normal cells; by enhancing gene expression, dietary carotenoids could restore communication, decrease proliferation and decrease progression to malignancy. How carotenoids stimulate the expression of this gene is not known.

To determine the significance of increased communication, we directly tested effects in human cervical cancer cells. We genetically engineered these cells so that they would express connexin 43 only when treated with tetracycline, which at the concentrations used is not toxic to human cells. Any changes thus result from changes in expression of the engineered gene. We selected carcinoma cells that did not express this gene and transferred the connexin gene to these carcinoma cells, which then began to express large quantities of functional gap junctions. When these cells were injected into immunodeficient athymic mice, tumor growth was strongly inhibited in the mice in which connexin gene expression was induced by tetracycline as compared with mice in which the connexin gene was not expressed. 6 A search is now on to identify and characterize the nature of the functionally transmitted signals involved in tumor growth inhibition.

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The Apolipoprotein Variants
Genetic variants in apolipoprotein E (Apo E2, 3 or 4) can predict response to a low cholesterol diet. One person in 50 has the Apo E2 variant that is associated with hypertriglyceridemia secondary to increases in energy, trans fatty acid, or carbohydrate intake. This variant is expressed in the presence of obesity, diabetes, and hypothyroidism. The Apo E4 variant is found in 15% of Caucasians, 35% of African (30% of Nigerian) and Asian populations, 22.7% of Finns, 20.3% of Swedes and 9.4% of Italians.

Women with Apo 3/2 genotypes have a reduction in the more protective high-density lipoprotein (HDL) cholesterol and would benefit from a high polyunsaturated diet. However, a general recommendation of a low fat diet would not benefit this genotype. Men with the Apo 4/3 genotype show an improvement in LDL/HDL ratio on a low fat diet. In individuals with the 3/3 genotype, a decrease in serum cholesterol when consuming oat bran at four weeks is noted compared to no changes in the E4/4 or 4/3 genotypes.

In the variant Apo A-IV-1/2, found in 1 in 7 persons in the United States, a decrease in the response of plasma cholesterol concentration with dietary cholesterol restriction is reported. However, no changes in triglycerides or HDL cholesterol are demonstrated. Thus, not every individual with these variants will respond to a general recommendation of a low fat, low cholesterol diet.

The Variant Associated With Sodium
The angiotensinogen gene that has been suggested to influence salt sensitivity has been described. Individuals with the A4 and AG genotypes respond to a sodium restricted diet with decreases in blood pressure. GG genotypes are less salt sensitive and will have decreases in blood pressure in response to a low sodium diet but not so significantly as the A4 and AG genotypes.

A polymorphic variant (Gly 460 Trp) is associated with changes in blood pressure and can be used to identify which patients will respond to sodium restriction. Patients with this variant allele have a greater decrease in blood pressure with sodium restriction.

Vitamin D Receptor Variant
In postmenopausal women with the BB vitamin D receptor genotype, it has been shown that they absorb less dietary calcium than the bb genotype. Thus the BB genotype women prescribed the current Dietary Reference Intake (DRI) for calcium, 1000-1200 mg/day, may not absorb adequate amounts of calcium and would typically require more than the DRI.

Translating to Clinical Practice
Nutritional recommendations that are universal do not take into account genetic variations that determine an individual’s response to nutrition intervention. The following are tips for integrating this information into counseling practices:

• Prior to counseling patients on general recommendations for disease management or RDAs/DRIs for prevention of common nutrition deficiencies, evaluate their family histories.
• Make family history a part of your nutrition assessment tool. Obtain important demographic and ethnic information to identify individuals at risk for genetic variants.
• Determine if individuals have undergone genetic/biochemical testing to identify risk categories for chronic disease development. Ascertaining this information can help to target dietary recommendations and nutrient requirements for that person.
• Keep up to date on advances in the Human Genome Project at www.nhgri.nih.gov or www.balancehealthsolutions.com.

References
Functional Foods...

Food Biotechnology
Lori Holladay, MPH, RD and Felicia Busch, MPH, RD, FADA

Do you eat cheese? If so, you’re a consumer of the benefits of biotechnology. Over half the rennet used in cheese making worldwide is produced through a biotech fermentation process that eliminates the “old-fashioned” method. (Laura Ingalls Wilder describes the method in her Little House on the Prairie books: sacrificing a baby calf for the rennet found in its stomach lining in order to make cheese). Rapid-rise yeast products that speed up bread-making were developed in England by rearranging and duplicating certain yeast genes. On the horizon are foods that contain higher amounts of health-promoting phytochemicals or possess disease- or drought-resistance.

What Is Biotechnology?
For centuries, farmers have bred crops and animals to develop foods with greater yields, better drought resistance, or faster ripening. The objective of biotechnology is to apply biologic methods to improve various characteristics of plants and/or animals. Biotechnology allows for the transfer of genetic information in a more precise manner. Traditional breeding involves the transfer of large blocks of genes, whereas biotechnology entails the transfer of only a limited number of genes.

Genetics – A Quick Review
A brief review of genetics can be useful in evaluating the role of biotechnology in our food supply. In 1953 James Watson and Francis Crick discovered the structure of DNA. This double helix molecule has two strands of DNA composed of pairs of chemicals: adenine (A) with thymine (T); and guanine (G) with cytosine (C). These are the base pairs. A segment of DNA that encodes enough information to make one protein is called a gene. It’s the specific order of DNA’s base pairs that determines which specific genes code for specific proteins, which determine individual traits.

By 1973, scientists were able to isolate individual genes, and by the 1980s could transfer genes from one organism to another. With biotechnology, a single gene may be added to the DNA strand in a precise manner without having to transfer large blocks of genes that are unrelated to the traits sought (see Genetics and Nutrition: The Future is Now on p. 15 for an overview).

Biotechnology vs. Traditional Agriculture Methods
Farmers have routinely used their knowledge of genetics to improve food production. Corn, for example, looks nothing like it did one hundred years ago because of plant breeding, which has helped U.S. farmers produce almost 600% more corn in 1985 than in 1930. Biotechnology is the latest development in the evolution of farming practices. Ideally, present day corn is used as the base and then is further enhanced by adding to it another desirable characteristic, such as an improved amino acid composition. Biotechnology allows for this precise insertion of genetic material. Traditional breeding using conventional technology, in contrast, will lose characteristics of the original corn as part of the genetic reshuffling that occurs during the attempt to add the new trait. The difference between these two techniques is dramatic.

Food, Nutrition and Health: Implications for Functional Foods
Many experts predict that the next wave of biotechnology will be in the area of efforts to develop functional foods. As additional healthful food components are identified, biotechnology will provide ways to incorporate these components into plants and animals.

Nutrient profiles of certain foods are already improved by biotechnology. A soybean that produces oil with a “heart healthy” fatty acid profile targeted to improve cardiovascular health is on the market. Rice and oils have been produced to express high carotene levels, with the potential to reduce the incidence of vitamin A-related blindness found in many parts of the developing world.

Safety Issues
The Food and Drug Administration (FDA) provides primary oversight for the safety of all foods. Foods are judged by whether they are safe, not by the process by which they have been developed. Foods developed through biotechnology are evaluated to assess their equivalence to those foods developed through traditional methods. Equivalent in this context means there is no meaningful change or difference in the nutrient composition or allergy potential of a food.

The FDA, the United States Department of Agriculture (USDA), and the Environmental Protection Agency (EPA), along with individual state governments, have been working together to ensure that crops produced through biotechnology are safe to eat. In 1992 FDA resolved that crops produced using biotechnology must meet the same rigorous standards as those created by traditional means. While there is no such thing as zero risk for any food, consumers can be confident that foods produced using biotechnology meet the stringent food safety standards enforced by the government. In fact, biotechnology has been studied and researched significantly more than common food manufacturing techniques such as freezing, canning, and dehydration.

Concern has been expressed about the transfer of genes from animals to plants. Such transfer between species is possible because plants and animals carry out similar metabolic reactions and the genes for these proteins are similar. Technically, an individual gene is neither a plant nor an animal gene. To date, no cross-species foodstuffs have been developed – despite media headlines to the contrary.

Who Benefits From Biotechnology?
Consumers and producers benefit from food biotechnology. Direct benefits to consumers include enhanced flavor and freshness and improvements in taste, quality, and nutritional value. A number of indirect benefits to consumers include reduced use of pesticides; more sustainable tillage practices, which address costly environmental problems like water pollution; less potential exposure to chemical residues by farmers and groundwater; and increased food yields (for developing countries this could help to address food shortages and hunger).

FDA Food Labeling Policy
Foods produced through biotechnology will need to have a special label if a known food allergen has been introduced, continued on page 11
Use of Genetically Engineered Foods

Edited by Rebecca Ephraim, RD

COUNTERPOINT
Lori B. Taylor, MEd, MS, RD

Genetic engineering is one of the most intriguing and powerful tools developed by science to manipulate the natural world. This technology has allowed major advances in the treatment of human disease. Scientists have been able to synthesize drug treatments that more closely approximate human proteins, such as erythropoietin, human insulin, t-PA and rituxan. The side effects of these drugs are minimal compared to those of their predecessors and offer real hope for the treatment of chronic disease.

Biotechnology has also been used extensively in the plant kingdom. Genetically engineered (GE), also called genetically modified, foods such as soybeans and corn have been designed to have herbicide tolerance, pest resistance, and altered nutritional profiles. Initially, these new foods offered great promise: they would be safer for the environment, have greater yields and be more nutritious. However, scientific evidence does not support these promises.

Assumed—not proven—safe.

The regulatory process for assessing the safety of GE foods assumes that recombinant DNA (rDNA) movement of genetic material between organisms is equivalent in risk to hybridization or naturally occurring movement of genetic material. The National Academy of Sciences (NAS) upheld this principle in an April 2000 report stating that “there is no evidence that unique hazards exist either in the use of rDNA techniques or in the movement of genes between unrelated organisms.” This does not mean that GE foods are safe; it means nothing harmful has been discovered or reported yet.

Many scientists predict dire consequences when it comes to GE foods. John Fagan, PhD, noted molecular biologist and NCI cancer researcher, states “splicing foreign DNA into an organism’s genome can cause unpredictable disruptions in the behavior of one or more active genes. The foreign genes...are routinely fused to promoters from viruses or bacteria, which cause them to function independently of the host organism’s intricate regulatory system. Moreover, the presence of these powerful promoters can alter the expression levels of the native genes. Due to such factors, the resulting food could be rendered toxic, allergenic, or otherwise harmful.”

Dr. Fagan is not alone in his concern about the safety of GE foods. Some Food and Drug Administration’s scientists themselves believe that there are different risks posed by GE foods that need to be assessed and that a more stringent regulatory process should be put into place.

GE foods may pose health risk.

Recent studies have shown the potential for increased allergenicity and toxicity in GE foods. When genes encoding a brazil nut protein sequence were engineered into soybeans to increase amino acid content, the allergenic properties of the brazil nut were transferred as well. Rats fed GE potatoes developed both immune and organ damage, as well as a viral infection of the GI tract.3

GE foods can be of inferior nutrition.

Genetically engineered foods have altered protein content and thus have a different nutritive value than conventionally grown crops. Recent research discovered that GE soybeans had decreased levels of beneficial phyto-estrogens when compared to conventional soybeans. In what may be an attempt to control research outcomes, the researchers have been refused further seed to repeat their experiments.

No environmental gains offered.

In laboratory studies, scientists have discovered that GE crops producing the Bacillus thuringiensis (Bt) toxin cause lethality of non-target, beneficial insects (Monarch butterflies and corn-borer eating lacewings). Bt toxin present in crop foliage has also been found to have a persistent, negative effect on soil microecology. Since Bt crops produce toxin systemically, one wonders how continuous consumption of Bt toxin will affect humans?

Although the NAS report admits to the potential for crop resistance to GE herbicides and inadvertent gene transfer that could produce new plant viruses and super weeds resistant to herbicides, the report recommended only that the effects be monitored, not that rigorous, pre-market testing be required to prevent these effects.

Increases in crop yield are inconsistent. In a study by the USDA, yields of GE soybeans were not substantially greater than conventional soybeans.11,12

GE foods: who benefits?

Given the health and environmental risks and lack of sustained, measurable benefit of GE foods to farmers, whose interests are GE foods serving? Is it large chemical and agricultural businesses that integrate their seed and chemical packages and forbid farmers from keeping or selling seed to ensure further economic benefit to the corporation?

Until rigorous pre-market safety testing and labeling happens, we are the experimental group for human safety. Unfortunately, there is no control group. Without labeling, even retrospective research will be impossible to conduct, as food origin will not be traceable.

Alternatives to GE foods

In all the rhetoric, few look beyond the two major options of farming conventionally with pesticides or using GE foods and herbicides. Long-term sustainable practices employing smaller farms and “agroecological” technologies are being used throughout the world with much success. Perhaps it’s time to look at restructuring our agricultural infrastructure to support smaller, more sustainable forms of farming that improve the physical and economic health of farmers and consumers.

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3. Key FDA documents revealing (1) hazards of genetically engineered foods and (2) flaws with how the agency made its policy. Available at http://www.bio-integrity.org/list.html. Accessed 5/12/00.

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POINT

Channapatna S. Prakash, PhD

I suspect that the average American, hearing or reading something about biotechnology, is at a loss to understand all that is involved. For many, the discussion of genes and proteins boils down to: Whom am I going to believe, those who oppose biotechnology or those who support it?

Without question, the scientific community is overwhelmingly supportive of the technology as evidenced by a series of recent reports:

• On April 5, 2000 the National Academy of Sciences (NAS) issued a statement that there is no evidence suggesting foods produced through biotechnology are any less safe than conventional crops.1 In fact, the scientific panel concluded that growing such crops could have environmental advantages over other crops. NAS recommended a few regulatory changes, mostly to improve public acceptance.

• Another report, issued on April 13, 2000 by the Basic Research Subcommittee of the House Committee on Science summarizes the testimony of leading scientific experts who had appeared before the subcommittee. It makes a very strong case for the safety of biotechnology and warns against needless over-regulation, which could delay development of a technology with great potential for good.

• On April 14, 2000 the National Center for Food and Agricultural Policy released a report about the benefits of transgenic soybeans, which have been improved to withstand a certain herbicide. The report, funded by the Rockefeller Foundation, concludes that farmers, who had been using three to four herbicides to control weeds in soybeans, were able to use just one herbicide, eliminating 16 million acre treatments of herbicide per year. The annual savings to farmers was estimated to be $220 million. www.ncfap.org

• Around the world, more than 2,000 scientists, including two Nobel Prize winners, have signed a petition in support of genetically modified crops. Overwhelmingly, scientific organizations, professional societies, and international bodies such as the World Health Organization agree that oversight of biotech crops should focus on the characteristics of the plant, its intended use and the environment into which it will be introduced, not on the method used to produce it. In other words, the scientific community is strongly united in the view that there is nothing inherently risky about biotech crops than crops developed through conventional breeding methods.

Three agencies of the federal government – the Environmental Protection Agency, the Food and Drug Administration, and the Department of Agriculture – are responsible for the regulation of biotechnology.2 Hundreds of field studies and specifically designed toxicology studies have demonstrated that biotech crops pose no greater risk than any other crop.

Opponents of biotechnology ignore these data and continually cite the same handful of studies that have been done in artificial laboratory settings and have not been duplicated in actual field situations. The most notable example is a study with Monarch butterflies, which showed that larvae of the popular insect could be harmed if they ate enough pollen from corn improved to resist harmful insects. The author of the study cautioned that it was only a laboratory finding,3 but activists seized upon it to stir emotions. The following summer, 20 scientists from several major universities conducted many field studies, which showed that Monarch larvae are rarely exposed to the corn pollen and, therefore, are not threatened.4

Scientific reviewers have soundly criticized other studies cited by biotech opponents. The American Soybean Association (ASA) criticized one study, which purported to show that biotech soybeans differ in nutritional content from conventional soybeans. ASA said there is more variability among various conventional soybean varieties than was seen when biotech and conventional soybeans were compared.5 Another study, which alleged that genetically modified potatoes harmed rats, was criticized by the Royal Society, Britain’s independent science academy, as seriously flawed.6 Furthermore, rather than testing a commercial product, the researcher used potatoes that had been altered to contain a gene from a known toxin, which never would make it through existing regulatory processes.

An April 2000 report from a U.S. House subcommittee, chaired by Rep. Nick Smith of Michigan, is an outstanding opportunity to read and understand in clear terms how biotechnology works and how it can help solve agricultural problems. It discusses the risks and benefits, reviews the regulatory system, and shows why many of the emotional issues raised by opponents have little scientific support. The discussion of the alleged threat to Monarch butterflies, described as “overblown and probably insignificant”7, is an excellent example. Testimony from 17 scientists, including some critics of biotechnology, was the basis for the report, which can be viewed at www.house.gov/science.

The report makes several findings, including: biotechnology is reducing chemical pesticide use and will continue to do so; there is no greater risk of introducing allergens into biotech crops than with traditionally bred crops; the risk...
of biotech crops becoming weedy pests is no greater than that for other crops. And perhaps most comforting to those who have heard the activist claims, the statement: “The risks associated with plant varieties developed using agricultural biotechnology are the same as those for similar varieties developed using classical breeding methods.”

References

Channapatna S. Prakash is professor of plant molecular genetics and the director of the Center for Plant Biotechnology Research at Tuskegee University. He serves on The USDA Agricultural Biotechnology Advisory Committee. Contact Dr. Prakash at prakash@tuske.edu.

Supplements . . . (cont.)

continued from page 1

with HH. Therefore, HH should be considered in the evaluation of any liver abnormality. HH should be considered a possibility based on laboratory values indicating abnormal iron metabolism. The College of American Pathologists (CAP) developed an algorithm that recommends re-testing of trans-ferrin saturation (in the fasting state) in the event of an elevated initial value, > 60% in men and > 50% in women; if results remain high, measure serum ferritin. If serum ferritin concentration is > 400 mg/L in men, > 200 mg/L in women of childbearing age, and > 300 mg/L in post-menopausal women, consider a liver biopsy. Biopsy of the liver evaluates the accumulation and extent of iron infiltration into tissues. A definitive diagnosis of HH can be made if hepatic iron concentration is greater than 80 mmol/g dry weight.6

Treatment
The primary treatment of HH is phlebotomy or the removal of 1 U (500 mL of blood) once or twice weekly until iron deficiency anemia develops. Thereafter, normal iron status is maintained by periodic phlebotomy, typically 3 to 5 U of whole blood per year. The frequency of phlebotomy is unique to each patient and should be guided by monitoring serum ferritin and maintenance of a normal hemo-globin level.

Diet Therapy
Strict dietary restrictions are not indicated—patients don’t have to “go iron-free.” Reducing intake of iron-containing and iron-fortified foods and avoiding cast-iron cookware can avoid excess dietary iron. Multivitamins without iron are preferable and no more than the DRI for vitamin C is recommended. Avoid foods and beverages that place an added stress on the liver, such as alcohol and raw seafood.

Role of the RD
Because many physicians are not aware of the prevalence of HH, the dietitian can be an invaluable resource. Investigating a patients’ family history for clues can help identify the patient as a carrier. A dietitian who is alert to the clinical signs and symptoms of HH can recommend the appropriate lab tests to help the doctor make the correct diagnosis. Detecting and treating HH early can make a vast difference in the patients’ quality of life.

Screening
CAP recommends screening for iron overload with a transferrin saturation test in all persons 18 years of age or older as part of routine medical care. Screening is also advised in all persons, regardless of age, who have one or more of the following risk factors: a family history of HH, any of the clinical manifestations of iron overload, and laboratory abnormal-ities.

Genetic Testing
HH is an autosomal recessive disorder. To develop the disorder, an individual must inherit the defective gene on both chromosomes. In 1996 the defective gene, dubbed the HFE gene, was discovered. Since the discovery of the HFE gene, DNA testing has been done on an experimental basis. While some experts argue in favor of universal screening, several important concerns exist. As with all screening, the benefits of screening for HH must be balanced against its adverse effects, which may include complications of diagnostic procedures (such as liver biopsy) and legal, social, and psychological problems (such as discrimination, loss of insurance benefits for a person with a known genetic condition, and increased costs of health care or insurance). These risks need to be weighed against the psychological benefit of finding an explanation for symptoms, alleviating symptoms with treatment, and preventing disease progression.

References

Susan Moore, MS, RD is an educator and technical/consumer writer in the dietary supplement industry and currently is the Western Regional Trainer for Health From The Sun/Arkopharma, an East Coast-based dietary supplement company. Contact Susan at (714) 528-5936.
Homocysteinemia

Judy Shabert, MD, MPH, RD

Biochemical Physiology
Homocysteine is a sulfur-containing amino acid derived only from the essential amino acid methionine. Abnormally elevated blood homocysteine occurs from genetic abnormalities of enzymes that recycle methionine, such as nutritional deficiencies of specific vitamins and, frequently, a combination of genetic and nutritional interactions.

The production of homocysteine is a necessary step in the metabolic formation of methionine and cysteine. See Table on page 10 for the enzymes and nutrition cofactors for these various reactions.

Homocysteinuria was proposed as a risk factor for atherosclerotic vascular disease in 1969 by Kilmer McCully. He published a report of an infant who had an autosomal recessive gene (two copies of the faulty gene are required to express the disease) that led to abnormally high concentrations of homocysteine in blood and urine and who died of extensive atherosclerosis. The cholesterol theory of heart disease was popular at the time, and Dr. McCully’s hypothesis was met with sharp criticism. However, over the next 20 years, other investigators confirmed his association of homocysteinuria and early death from vascular disease.

Genetics
In the rare, classical presentation of homocysteinuria, individuals who are homozygous (have two faulty genes) for a defect in any of the methionine-regenerating enzymes produce excessive quantities of homocysteine. These enzymatic deficiencies result in severely elevated blood homocysteine and homocysteinuria with a characteristic syndrome of mental retardation, detached lens of the eyes, osteoporosis and atherosclerotic and/or thromboembolic events. Individuals who are heterozygous (only one faulty gene) for cystathionine synthase deficiency will have mild hyperhomocysteinemia. In people who are heterozygous for the classic 5,10-methylene-tetrahydrofolate reductase deficiency, homocysteine will not be elevated.

Pathology
Mild hyperhomocysteinemia as a risk factor for coronary heart disease in young people was recognized in 1976. The adverse effect of homocysteine on blood vessels appears to be a graded response and is similar to that seen with increasing concentrations of cholesterol. Those individuals with increasing plasma levels of homocysteine have more severe vascular disease. In fact, in men with a homocysteine concentration of 15.8 mcg mol/l compared to those with a concentration within the normal range, the relative risk for vascular disease increases three-fold.

Diagnosis
There are two methods for diagnosing hyperhomocysteinemia. One method is to obtain a fasting homocysteine level. The other method is to give an oral dose of methionine (0.1 g/kg body weight, called “methionine loading”) prior to obtaining the serum homocysteine level. Approximately 50% of individuals who demonstrate elevated homocysteine levels following methionine loading have normal fasting homocysteine levels. So, a normal serum homocysteine without a methionine-loading test does not rule out this risk factor for cardiovascular disease. Individuals who do have post-loading elevations in homocysteine have a relative risk for cardiovascular disease of 13, compared to those with no elevation.

Variant MTHFR mutation
Most recently a common genetic defect in 5,10-methylene-tetrahydrofolate reductase (MTHFR) has been recognized. This mutation leads to a heat-labile form of MTHFR. Individuals homozygous for this mutation do not have severe hyperhomocysteinemia but may demonstrate mild elevations in blood homocysteine. Mild hyperhomocysteinemia is associated with significant morbidity, however, and in the general population the homozygous state for heat-labile MTHFR occurs in 1-in-20 (1:20) individuals. Its frequency increases in people with occlusive vascular disorders: coronary artery disease 1:5, peripheral vascular disease 1:3, and cerebrovascular disease 1:4. Individuals who are homozygous for MTHFR and also have low serum folate concentrations are at highest risk for elevated homocysteine and, presumably, for occlusive vascular disease.

Nutritional Expression of Genetic Defect
Heterozygous individuals with heat-labile MTHFR deficiency may have normal serum homocysteine. However, if serum folate is inadequate, hyperhomocysteinemia occurs. The relationship between low serum folate and elevated homocysteine demonstrates the unmasking of a genetic mutation when genetic individuality requires nutrients greater than the Recommended Dietary Allowances. The genetic defect leading to hyperhomocysteinemia is unmasked when folate intake is inadequate. Numerous epidemiological studies have demonstrated this relationship for both folate and vitamin B6.

Other research demonstrated that in a group of elderly people who had normal serum concentrations of B6, B12 and folate, homocysteine was elevated. When the participants of the study received eight intramuscular injections of B6, B12 and folate over three weeks, serum homocysteine normalized.

Dietitian’s Role
The registered dietitian has a significant role to play in this condition by helping physicians understand the incidence and impact of homocysteinemia and how it can be treated. The measurement of blood homocysteine concentrations is not routine practice and the methionine-loading test less so. Many physicians are unaware that genetic defects of methionine are common in the general population. Furthermore, they sometimes do not appreciate the impact of specific vitamins in lowering homocysteine levels.

References

References continued on page 10
References continued from page 9


Judy Shabert, MD, MPH, RD, trained in obstetrics, gynecology and public health, is a clinical instructor at Harvard Medical School in Boston, MA. She has, over the past 10 years, returned to her first love—nutrition—where the basis of all health resides. Contact Dr. Shabert at js@dietrehab.com.

Overview of Homocysteine Pathway:

1. Serine hydroxylase (requires B6)
2. 5,10-methylene tetrahydrofolate reductase
3. 5-methyltetrahydrofolate homocysteine methyltransferase (requires B12)
4. S-adenosylmethionine synthase
5. Methyltransferase
6. S-adenosylhomocysteine hydrolase
7. Cystathionine b-synthase (requires B6)
8. Cystathionase (requires B6)
9. Betaine:homocysteine methyltransferase

Judy Shabert, author of the article Homocysteinemia in this issue, was awarded the 2000 John M. Kinney International Award for Nutrition and Metabolism for the paper entitled: Glutamine-Antioxidant Supplementation Increases Body Cell Mass in AIDS patients with Weight Loss: a Randomized, Double-Blind Controlled Trial. Nutrition 1999;15:860-4. Judy and other ADA members who contributed to the article, Charmaine Winslow, RD (Florida), Janet Lacey DrPH, RD (Boston) and Doug Wilmore, MD (honorary member (Boston)) will be presented their award in Madrid Spain on Sunday, Sept 10.
the nutritional content of the food has been changed, or the product’s composition has been substantially changed. Otherwise, these foods will have the same labels as all other foods.

FDA evaluates all new foods for the presence of allergens. There are no foods currently on the market containing allergens transferred via biotechnology. Because virtually all crop plants have been modified through plant breeding or biotechnology, the FDA does not require labeling stating any declaration of process that produced the food.

The American Dietetic Association’s (ADA) Position On Biotechnology

It is the position of ADA that biotechnology has the potential to be useful in enhancing the quality, nutritional value, and variety of food available for human consumption and in increasing the efficiency of food production, food processing, food distribution, and waste management.

ADA has just released the Biotechnology Resource Kit, which is a comprehensive peer-reviewed package of information on the background, applications, safety and other issues related to food biotechnology. Educational resources and opposing views are also included. This kit is available from ADA’s Customer Service (800-877-1600 ext. 5000) or through ADA’s Web site at www.eatright.org.

Resources

Food Biotechnology: The International Food Information Council Foundation; October 1998 (teaching tool).

Lori Holladay, MPH, RD is a consultant and writes articles and curriculum on a variety of nutrition topics for community education programs and print media for Felicia Busch & Associates, Nutrition Communications Consultants.

Felicia Busch has been in private practice since 1986 and is the author of The New Nutrition: From Antioxidants to Zucchini. She is treasurer of the NCC DPG and media spokesperson for ADA. Contact Lori or Felicia at Felicia.Busch-1@tc.umn.edu or 651-645-4621 (fax).

Future Biotechnology Benefits

In the near future expect food biotechnology to provide for:
• reduced levels of natural toxins in plants;
• simpler and faster methods to locate pathogens, toxins, and contaminants;
• longer time before spoilage;
• safer foods through reduction of allergenic proteins;
• drought and flood tolerance;
• heat and cold tolerance.

Food Crops currently produced through biotechnology:
• soybeans
• corn
• canola
• tomatoes
• squash
• potatoes
• rice

New products that are currently being developed for release in the future include:
• Oils, such as soybean and canola oils developed with more stearate to make margarine and shortening more healthful
• Sweeter peas
• Smaller, seedless melons for use as single servings
• Bananas and pineapples that ripen more slowly
• Peanuts with better protein balance
• Fungus-resistant bananas
• Tomatoes with more antioxidants
• Potatoes with higher starch content so that they will absorb less oil when fried
• Fruits and vegetables with more vitamin C and E
• Garlic cloves with more alllicin
• Rice with more protein (using genes from pea plants)
• Strawberries with more ellagic acid

Although food biotechnology is the hot topic today, consider some other benefits of the science of biotechnology:

• Medicines created from proteins that are naturally produced by the human body. These include FDA-approved medicines to treat diabetes, anemia, leukemia and many others.

• Vaccines consisting of the antigen only (unlike conventional vaccines that use a weakened form of the virus) and therefore cannot transmit the virus itself. FDA has approved a biotech vaccine for Hepatitis B.

• Diagnostics used to detect many diseases and conditions including HIV, hepatitis, and pregnancy.

• Gene therapy, which uses the genes themselves to treat inherited genetic disorders, has been used to treat severe combined immunodeficiency disease (SCID) or the “bubble boy disease.”
Food Production Time Line 10,000 years

8000 BC • The nomadic lifestyle was replaced by geographically stable communities that grew plants as crops. Seeds are saved for planting in the next season.

1800 BC • Yeast is used to make beer, wine and to leaven bread. This is the first time people used microorganisms to create new foods.

1865 • Mendel concludes from his experiments with pea plants that unseen particles pass traits from generation to generation.

1922 • Hybridized corn seed created by crossbreeding two corn plants. This new corn helps to account for a 600% increase in corn production in the US between 1930 and 1965.

1953 • Structure of DNA defined showing how cells in all living things duplicate and pass genetic information from generation to generation.

1986 • Biotechnology is used to create herbicide resistant soybean plants. Approved by USDA, FDA and EPA by 1995 and commercialized in 1996.

1990 • Approval granted to the first food product modified by biotechnology- an enzyme used in cheese making.

1994 • FlavrSavr tomato the first food product enhanced by biotechnology available to consumers (slower ripening and longer shelf life).

1997 • 18 crops improved through biotechnology have been approved by the US government.

Web site Resources
The American Dietetic Association
www.eatright.org

Biotechnology Industry Organization
www.bio.org

Council for Biotechnology Information
www.whybiotech.com

Environmental Protection Agency
www.epa.gov

Food and Drug Administration
www.fda.gov

International Food Information Council
http://ificinfo.health.org

United States Department of Agriculture
www.usda.gov

Interested in contributing to the NCC Newsletter?
The following themes will be covered in upcoming issues.

Spring 2001-Allergies and food intolerances

Summer 2001-Anti-Aging

Fall 2001-Interaction of dietary supplements with medications

For more information contact Sarah Harding Laidlaw at peaknut@wic.net.

Approved US Crops
Since the mid-1990s many different crops that have been modified for a variety of agronomic and functional traits have completed the regulatory process in the United States. Here is a partial listing of those crops.

CROP
Canola
Cherry Tomato
Corn
Papaya
Potato
Soybean
Squash
Sugarbeet
Sunflower
Tomato

TRAIT(s)
Herbicide tolerance, high laurate oil
Taste, color, texture
Insect protection, herbicide tolerance
Virus protection
Insect protection
High oleic oil, low saturated fat oil, low
linolenic oil
Virus protection
Herbicide tolerance
High oleic oil
Altered ripening, thicker skin, modified
pectins
Book Review


One diet does not fit all because each of us has our own genetic blueprint. This is the basic message the authors want to convey in this book. Although intended for consumers, dietetics and other health professionals can benefit from the information provided. And, despite that it was published 7 years ago, the information remains relevant. With a greater understanding of genetics resulting from the Human Genome Project, dietetics professionals will need to have at least an overview knowledge of genetics and its effects on health.

Chapter one, “Your Genes and Your Health,” provides just that type of overview. The authors guide you through cell replication, review DNA in the cell nucleus and its role in genetic expression, and discuss types of genetic expression and genetic transmission of diseases and disorders. Our genetic heritage positions us anywhere from being merely predisposed to a medical condition to frankly expressing a disorder. This chapter also provides a review of how our human history led to ethnic variations in the incidence of certain chronic diseases. One study cited discusses the environmental and genetic factors that cause some Finnish men to have higher heart disease death rates than Japanese men.

Chapter two details how to chart a family medical history. A questionnaire and a diagram for charting this history are provided.

Chapters three through ten discuss feeding your genes if you or your family history includes obesity, heart disease, hypertension, diabetes, alcoholism, cancer, or food allergy. One chapter discusses the cautions of excessive iron in dietary intake. Because this book was published in 1993, the chapter on obesity does not contain information on the link between insulin resistance and obesity. Interestingly, the hypertension chapter reads like a forerunner to the DASH (Dietary Approaches to Stop Hypertension) diet.

Chapter eleven provides suggested menus for various genetic tendencies. The menus developed by several registered dietitians are based on USDA publications and the Food Guide Pyramid. Also offered are the 1989 dietary guidelines and RDAs, both of which have been updated since the book’s publication.

The greatest benefit dietetics professionals can get from this book is an understanding of how genetics influences our health. With this understanding, we can become more thorough in our patient assessments by investigating genetic history and increase our effectiveness with appropriate medical nutrition therapy. Patients with higher levels of education would be able to benefit from the book’s information. Check your local library for this book. You can then determine if you want to add it to your own resource base.

One recommendation for this book would be to include the references for the studies cited.

Reviewed by René Norman, RD, in private practice with Nutrition Consultants of Tulsa. Contact René at (918) 749-9077 or by fax (918) 749-4041.

Book Review


Each individual has unique genetic characteristics. Some of these characteristics place a person at increased risk for specific diseases. The premise of Dr. Bland’s book is that inherited traits can be modified through diet, nutritional supplements and lifestyle to allow people to live longer, healthier lives.

Dr. Bland has spent his career educating professionals on, and researching, this approach to disease management and health. He holds a PhD in biochemistry from the University of California where he was a faculty member and worked with Linus Pauling, PhD at the Linus Pauling Institute.

Whereas this book was written for the lay reader, it is an excellent first pass for a wide spectrum of health professionals in understanding the complementary and alternative health approach to nutrition that is being practiced in this country.

Chapter one explores the relationship of one’s genes and the environment in determining health. It introduces the reader to Roger Williams, PhD who first described the concept of biochemical individuality over 50 years ago.

Chapter two reviews the basics of genetic inheritance and, in a simple way, explains how genes work. In chapter three the book reports on foods that alter the genetic expression of disease, such as phytochemicals in fruits and vegetables and their ability to modulate the immune system.

The following chapter includes reports that illustrate the relationship of an individual’s insensitivity to a food and disease outcome. For example, lactose intolerant people are more likely to develop cataracts if they continue to drink milk in their adult life. The concept of liver detoxification is introduced. The ability of the liver to detoxify compounds is determined genetically but functionally expressed through toxins that are presented to it.

Chapters five through eleven discuss nutrition and genetic factors that influence aging, diabetes, arthritis, heart disease, brain aging and cancer. The book cites references to substantiate the ideas presented in these chapters. However, there is a lack of large scale, double blind, placebo-controlled trials to support the recommended nutritional supplementation. Despite this limitation, Genetic Nutritioneering is an important, readable book that builds on our understanding of dietary choice and disease expression.

Reviewed by Judy Shabert, MD, MPH, RD. Dr. Shabert can be reached at js@dietrehab.com.
The Human Genome Project: Implications for Dietetics Practitioners

David H. Holben, PhD, RD

Introduction

How fitting that the 82

nd Annual Meeting and Exhibition of The American Dietetic Association (ADA) held in October, 1999, had the theme entitled, THE FUTURE IS NOW—DIETETICS 2000. It is. Scientists, medical professionals, and individuals from the media talk about genetics, biotechnology, and molecular biology on a daily basis. “Understanding the basics of genetics, how nutrients and nutritional status influence gene expression, and the relationship between genetics and nutrition in chronic disease will become increasingly important for dietetic professionals as the discoveries of the Human Genome Project (HGP) unfold....” Several authors have discussed genetics issues related to dietetics practice. This article summarizes the HGP and the Human Genome Education Model Project II (HuGEM II) and discusses their relevance to dietetics professionals.

What Is the Human Genome Project?

Coordinated by the National Institutes of Health (NIH) and the Department of Energy, the HGP is an international research effort that formally began in 1990 to fully define the human genetic makeup (our “genome”). The specific goals of this project are to: 1) characterize the genomes of human and selected model organisms (Escherichia coli, the fruit fly, and the laboratory mouse) through complete mapping and sequencing of their genetic material, i.e., DNA (there are approximate 100,000 genes in human DNA, which contains 3 billion chemical bases); 2) store this information in databases; 3) develop tools and technologies for genomic analysis; 4) train scientists who will be able to utilize the tools and resources developed through the HGP to pursue biological studies that will improve human health; and 5) examine the ethical, legal, and social implications (ELSI) that may surface from this research into the human genome. The HGP is the first large scientific undertaking to address the potential ELSI that may arise from the characterization of all of the genes in humans.

What Is the Human Genome Education Model?HuGEM II is an initiative funded by the NIH to provide educational training and resources to increase the knowledge of and sensitivity to human genetics, the Human Genome Project, and the ELSI of genetic testing and research for members of seven collaborating professional organizations:

• the American Dietetic Association,
• the American Occupational Therapy Association,
• the American Physical Therapy Association,
• the American Speech-Language-Hearing Association,
• the American Psychological Association,
• the Council on Social Work Education
• the National Association of Social Workers

The educational training includes several seminars covering an orientation to HuGEM II and an overview of the HGP and its ethical, legal, and psychosocial issues provided for board members and national staff members of the seven collaborating professional organizations. “Educate the Leaders to Educate Others” workshops are held at national, regional, and state conferences of the collaborating organizations (basic human genetics and priority topics of ethical, legal, and social issues identified by the HuGEM II survey). And, a five-day continuing education course, “Incorporating Genetics into Clinical Practice and Teaching” that provides 30 hours of instruction in human genetics and genetic issues is held for health professional educators at Georgetown University.

How Will the HGP Affect Dietetics Practice?

Genetic-related issues such as gene-environment interactions, gene therapy, and genetically engineered foods will have profound implications for the dietetics profession in the future and impact how we provide medical nutrition therapy. The HGP will undoubtedly change how we detect, understand, and treat human disease. Dietetics professionals must be proactive and seek out genetics resources to be effective practitioners. Gilbride has reviewed how to find and use genetics resources. Finally, Camp summarized that dietetic professionals need to:

• have a basic knowledge of genetics,
• be comfortable with genetics terminology,
• have the knowledge to distinguish between genetic and environmental factors of diseases when making specific recommendations about nutrient needs or changing behaviors,
• understand how an individual's genetic heritage influences requirements,
• be able to effectively work with families with genetic conditions, and
• be an effective member of a multidisciplinary team.

References


David H. Holben, PhD, RD is an assistant professor in the School of Human and Consumer Sciences at Ohio University. Formerly a pediatric dietitian who specialized in caring for individuals with cystic fibrosis, he was selected in 1999 as one of ADA’s participants in the HuGEM II. Contact Dr. Holben at holben@ohio.edu, 740/593-2875.
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• Lifestyle Therapies: Mind-Body Interventions
  Trish Neel, RN, Program Manager for the Center for Wellness and Prevention at the Ohio State University Medical Center and American College of Sports Medicine-certified

• Power Food for the Health Heart
  Cathy Kapica, PhD, RD, senior scientist at The Quaker Oats Company and former faculty member of The Chicago Medical School, with expertise in functional foods

• Lifestyle therapies: Mending a Broken Heart
  David Grotto, RD, Director of Nutrition for Block Medical Center for Integrative Cancer Care and Optimal Health, Evanston, Illinois

• Case study practical applications led by Ms. Neel

• Summary and Overview of Complementary Care and Cardiovascular Disease by Dr. Kris-Etherton

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Preparing for the Genetics Revolution

Teresa Carithers, MHS, RD

For dietetic students and those just entering their professional careers, genetics should be viewed as an essential area of study. More and more opportunities are being integrated into educational experiences due to the “diet-gene” or “nutrition-genetic” relationships that this rapidly evolving science is bringing to light. Students should take advantage of every educational opportunity to enhance their knowledge of genetics, especially genetic-nutrition relationships, and consider specialization within an area of genetics, possibly becoming a genetic counselor.

All this probably sounds exciting to those just beginning their professional pursuits, but what about those of us who are not students any more, those of us who are already “established” in our nutrition roles? Should we be concerned about the impact of genetics on our careers as well or is this just futuristic thinking? If you don’t already know the answer to this question, read the article recently published in the Journal of The American Dietetic Association (J Am Diet Assoc. 1999; 99:1412-1420) that describes the relationships between nutrition and genetics and provides an overview of the impact genetics will have on our clinical practice and our personal lives. This ReaDer Reports column is provided in an effort to relieve the panic that many professionals experience when they first encounter genetic information. Although most find it intriguing, gaining a working knowledge and becoming comfortable with the information takes time and effort.

All of us are familiar with the statement “and duties as assigned.” My opportunity to work in genetics came suddenly and was an addendum to a job assignment as an administrative consultant to a public health department. Since then my work in the genetics area has evolved into a truly rewarding faculty level position within the division of medical genetics at the University of Mississippi Medical Center.

Although the initial years were challenging and sometimes stressful, the opportunity to work with individuals with inborn errors of metabolism and train under the close direction of a skilled geneticist provided a great opportunity for my professional development and growth. Mutual respect for the role both the geneticist and nutritionist could bring to the table created a strong and successful MD/RD team that has had a dramatic impact on our state and greatly surpassed our initial expectations. My goal for this column is to share several “lessons learned” that will help other dietetics professionals faced with similar professional challenges.

• **Become computer literate.** This advice may sound elementary, but it’s amazing how many skilled professionals avoid becoming proficient with the computer. Genetic information can be accessed rapidly, but only if you know how to operate computer search engines with ease. Professionals not associated with computer networks can have significant access just through their personal home computer.

• **Find a genetics mentor.** Ask to observe a genetic clinic. Most genetics professionals are quite willing to help educate and can direct you to opportunities that just don’t exist in the library or local clinic. Many states are beginning to offer genetic education courses for multidisciplinary groups. Most colleges and medical schools offer introductory genetics or molecular biology courses. Taking the course for credit (being graded) really forces you to learn the complex principles. Self-study continuing education offerings are exciting, but it usually takes a great deal of self-discipline to review and retain the information over time.

• **Focus initially on basic genetics concepts and principles.** Many people make the mistake of focusing on specialized diagnostic information. Your understanding of concepts such as the principles of inheritance, pedigree analysis, and variability of expression will be much more valuable in the long term than learning specialized diagnostic information or a few genetic facts.

• **Focus on what’s of immediate use to you.** Because of the complexity of the information, pace and focus your learning on what will help you the most day-to-day. If you work with patients who have diabetes or cardiovascular disease, begin to study multi-factorial diseases in general and diabetes or cardiovascular disease in particular. If you work with patients with inborn errors of metabolism, then a study of autosomal recessive disease inheritance may be more worthwhile. If you don’t stay focused, you can waste a lot of time on material that you may never need.

Below are resources that will be helpful in expanding your exposure to genetics. From one who has “been there and done that,” I encourage you to take the plunge. Best of luck!

**Recommended Resources**


**Recommended Web site**

www.complementarynutrition.org

Teresa Carithers, MHS, RD is a doctoral candidate and Instructor at the University of Mississippi Medical Center, Department of Preventive Medicine, Division of Medical Genetics. She is the State Nutrition Coordinator for individuals with inborn errors of metabolism and the Nutrition Investigator for the NHLBI-funded Jackson Heart Study. Contact Teresa at (601) 984-1900 (Secretary) or tcarithers@prevmed.umsmed.edu
Genetics and Nutrition: The Future is Now
Ruth M. DeBusk, RD, PhD

OVERVIEW

The central role of genetics in health care is rapidly emerging. Dietetics professionals need to understand how genetics relates to health and disease and the many connections among genes, nutrients, and disease development. Genes contain the information for making proteins. Proteins, in turn, determine the structure and function of the body and are the basis for human individuality. Each person’s genes contain the same basic information but with enough individual variation to result in unique differences in how the body is put together and carries out its activities. Metabolism, that complex protein-based biochemical machinery, underlies our functional capability by providing the vast number of metabolic products needed to sustain life. Mistakes in the genetic material result in mistakes in the metabolic machinery that result in mistakes in the production of metabolites. If a mistake occurs in a critical process, disease, and even death, can result.

Food plays a central role in this interrelationship of metabolism and function. It supplies the raw material for making critical metabolic products, can supply many of the products preformed, and can result in genes turning on or off so that more or less of a product is synthesized. The genetic information must be expressed as a workable protein component, and nutrition is a powerful regulator and modulator of this whole process. Nutrition, whether from food or supplements, can potentially fill functional gaps created by an individual’s genetic information and wire around genetic limitations.

A simplistic view of this concept is the absolute requirement of humans for vitamin C. We are genetic mutants for vitamin C; our genes do not produce the enzyme gulonolactone oxidase that’s needed for vitamin C synthesis. Failure to supply vitamin C results in death. Thus, by supplying vitamin C, nutrition fills the biochemical gap created by our genes and enables us to live. Similarly, genetic limitations result in requirements for certain amino acids and fatty acids, biochemical gaps that must be filled by our diet. All humans share these genetic limitations and nutritional requirements. In addition, each individual has his or her own unique genetic limitations and nutritional requirements. It is these unique differences that will form the basis for gene-directed nutrition therapy.

Examples of the Genetic-Nutrition Connection

Many examples exist where the genes contain the potential for disease but diet and other lifestyle choices prevent the expression of this potential.

- The Pima Indians in Arizona and in Sonora, Mexico share the same genetic makeup, but the Arizona population has the highest prevalence of type 2 diabetes in the world whereas their Mexican counterparts are lean and healthy. Bouchard and colleagues showed with identical twins that, although the genetic predisposition existed for obesity, development of obesity was not a foregone conclusion. Further, genetic makeup appears to determine which obese individuals benefit from particular diet therapies and even from physical activity.

- Individuals with gluten-sensitive enteropathy have the genetic predisposition but do not manifest the disease if they do not eat gluten-containing foods. They still have the genes that cause this disease, but nutrition prevents the expression of the disease.

- Individuals with a common genetic variation in the 5,10-methylentetrahydrofolate reductase gene are predisposed to increased levels of homocysteine, a known risk factor for cardiovascular disease, yet do not develop elevated homocysteine levels if folate is adequate. Genetic variability in components in the renin-angiotensin-aldosterone system have been correlated with hypertension and its response to low-salt diets.

These are just a few examples. We can expect many more as research continues. Clearly, failure to understand the underlying genetics/ biochemistry/ nutrition connection leads to a mismatch between our genes and the foods we eat, with potentially serious health consequences.

Genetic individuality leads to biochemical individuality, which leads to nutritional individuality. This inter-relationship is so straightforward, why has it taken so long to come to light? All the parts have been there; we simply haven’t understood how they fit together: which genes are involved, what their gene products are, and where these gene products fit into the metabolic machinery. The Human Genome Project will help to fill in these knowledge gaps. In time, we will be able to look into the genes and figure out which ones aren’t working in a particular individual and what the biochemical ramifications are. With this knowledge, we will have the basis for customizing nutrition therapy according to genetic limitations and for developing rational biochemical approaches that address these limitations. We need to begin now to incorporate these principles into the therapies we develop for our clients and the public policies we develop for populations. Science does not support a one-size-fits-all approach nor the extrapolation of data from one population...
as the basis for policy development for another population.

**The Challenge for Dietetics Professionals**

Dietetics professionals need to learn key genetic concepts and practice applying them. We also need to understand that science has only begun to uncover specific examples of how nutrition can be applied in this way. The pace will soon snowball as a result of the rapid progress of the Human Genome Project, which is predicted to fundamentally change the practice of medicine. Understanding which diseases individuals are predisposed to, how genes interact with nutrients and medications, and what we can do to prevent future illness will allow health care practitioners of all disciplines to target their therapies to the genetically-determined unique needs of the individual. We need to see personal health as a continuum that ranges between wellness and illness. Genetics sets the upper and lower limits of our potential, but where we fall on that continuum depends on our unique genetic makeup and the lifestyle choices we make, with nutrition being possibly the most critical choice.

In the forward to their book, *Genetic Nutrition*, Artemis Simopoulos, MD, and her colleagues captured the key relationship between genetics and nutrition succinctly: “Genetic factors determine susceptibility to disease, and environmental factors—of which nutrition is one of the most important—determine which genetically susceptible individuals will be affected.”

The take-home message here is profound: *Genetics is not destiny*. Having “bad” genes does not necessarily doom us to a life of disease and disability. The information in our genes serves only to make us more or less susceptible to developing disorders that range from inconvenient to deadly. Nutrition therapy has the potential for intervening at strategic points, which can alter our genetic course and prevent the information in our genes from spelling disaster for us.

**FUNDAMENTAL GENETIC CONCEPTS**

**DNA: The Genetic Material**

DNA contains information. This information directs the synthesis of all the life-sustaining activities of the organism. DNA is made up of 4 chemical compounds called nucleotide bases: adenine (A), guanine (G), cytosine (C), and thymine (T). The structures of these bases allow for weak hydrogen bonding between specific pairs, A pairs with T and G with C, to form a double helix. Picture a right-handed spiral staircase with the sugar-phosphate backbone of the DNA forming the parallel railings and the paired bases forming the steps that connect the railings.

DNA is a language. It tells a story. As the text is read, an organized, logical description unfolds as to how to make the myriad of proteins needed to build the organism and support its functions. Like any language, there’s an underlying organization: an alphabet that gets assembled into words, sentences, chapters, books and, ultimately, a complete encyclopedia of information. A, G, C, and T are the alphabet. These bases are arranged side-by-side to form a linear molecule. Words are formed by reading 3 bases at a time, a unit called a codon. Sets of three bases ultimately direct the positioning of a particular amino acid into the protein molecule being assembled. Each codon specifies one of the 20 amino acids that make up proteins. Thus, DNA is “read” 3 bases (1 codon) at a time.

A gene is the set of sequential codons required to synthesize a protein and is analogous to a sentence. Like a sentence, a gene has a characteristic anatomy. Instead of a subject, verb, and predicate, genes have start, coding (informational), and stop sequences. DNA has many genes arranged along its length. Just as the total sentence content in a book is divided into chapters, DNA is subdivided into units called chromosomes. And, just as a book is the sum total of all its chapters, the genome (“gene” + “chromosome”) is the sum total of all the chromosomes containing all the genes.

A fundamental principle of genetics is that each cell’s nucleus contains the complete genome for the organism, even though all the information may not be used by a particular cell type. Liver cells use different subsections of genetic information than do heart cells or brain cells. Think of the genome as a book; the book in this case is an encyclopedia. For us to learn something, we don’t need to read the whole encyclopedia; we just need to go to the section of interest and retrieve that specific information. Similarly, for a cell to carry out a particular function, it needs to “read” (translate) only certain sections of the total “book” (genome).

Another fundamental principle is that the language of DNA is common to all organisms, whether microbes, plants, or animals. Species differ in terms of what’s in their encyclopedias. The content is different, but it’s all in the same language and is decoded using the same basic methods, which is why scientists can study the fruit fly and uncover fundamental principles that also apply to humans.

If all organisms within a species have basically the same DNA, why don’t we all look alike? Returning to the book analogy, think of individual differences as resulting from individual authors attempting to write the same chapter for the encyclopedia. If we give ten authors the same outline for a chapter, they’ll write the sentences for that chapter slightly differently to express the same basic concepts. The meaning of many concepts will still be clear in spite of slight variations. Other concepts will get lost in the translation. Similarly, many genes can accommodate slight variations and still produce a protein that’s functional. It may
be as functional, less functional, or even more functional than the original version. Other genes cannot accommodate even a slight change. In molecular terms, changes are called “mutations” and are the cornerstone of evolution. Genes that cannot accommodate change are said to be “highly conserved” and usually code for proteins that are critical to the functioning of the organism. Those genes that can accommodate change exist in many variations (called “polymorphisms”). Polymorphisms generate the differences between individuals within a species. Everyone in the species uses the same basic blueprint; they simply go about building and maintaining their houses slightly differently so that the end result reflects their individual characters.

The Molecular Biology of DNA

The information in DNA is in code and needs to be decoded. Molecular biology is the study of the decoding of the DNA, how the sequence of bases is converted into protein components such as enzymes, hormones, messenger molecules, receptors and so on that are useful to the cell. Essentially the point of the Human Genome Project is to figure out what each gene codes for and how its expression is regulated.

Transferring the information in the DNA sequence into a protein requires two major steps: transcription and translation. Transcription “transcribes” the DNA sequence into an RNA sequence, which is in turn “translated” into an amino acid sequence. In the nucleus the DNA double helix “unzips” and the enzyme RNA polymerase moves along one strand matching bases in complementary pairing fashion to the linear sequence of bases in the DNA. The RNA molecule that is formed is called messenger RNA or “mRNA”. RNA is similar to DNA except that it pairs adenine with the base uracil (U) rather than with thymine.

Genes have a standard anatomy. Upstream from the informational region is a regulatory region where RNA polymerase binds (the “promoter region”) and where a variety of factors, including nutrients, can bind to turn on and off the expression of that gene. Then comes the informational region (“coding region”), followed by a stop region. Like reading an encyclopedia, RNA polymerase doesn’t “read” the entire DNA just to make a particular protein; it transcribes only the genes needed by the cell at a particular time.

Within the coding region of a gene, interspersed in the information sequences, are sequences that are non-coding, that don’t translate into proteins. In molecular biology terminology, DNA sequences that contain information that translates into the amino acid sequence of a protein are called “exons.” The intervening sequences are called “introns.” RNA polymerase transcribes the entire gene into mRNA, both the coding (informational) and non-coding regions. Predictably, post-transcriptional processing has to occur. The introns are removed from the message, a run of adenines is attached, and the mRNA is transported to the cytoplasm for translation into protein.

In the cytoplasm, the mRNA attaches to ribosomal RNA and one codon (sequential sets of 3 RNA bases) at a time systematically directs the assembly of amino acid molecules into a protein. The protein may be ready to go at this point or may require “post-translational processing” before it is an active molecule, such as the addition of sugars or cleavage into a smaller molecule. The finished protein then spontaneously assumes the conformation needed for that molecule to function. Familiar examples include the post-translational cleaving of proenzymes such as pepsinogen and trypsinogen and the addition of a carbohydrate moiety to glycoproteins.

Thus, changes (called “mutations”) in the DNA translate into changes in the protein product. Changes in the coding region are more likely to have harmful effects on function and lead to a disease state than changes in the intervening sequences.

Inheritance: Transmitting Genes to New Cells, New People

Cells are the foundational unit of the human body. Within a cell’s nucleus is the complete genome. To create new cells, genetic information must be passed from the original cell to the new or “daughter” cell. To continue a species, the information in the DNA must be passed from parent to child. From the pioneering genetic experiments of Gregor Mendel, we understand the mechanics of passing genes from one generation to the next. DNA is not one long, continuous molecule in humans. It’s divided into segments, called “chromosomes.” Each segment contains thousands of base pairs and, among those base pairs, are the informational sequences that make up the many genes. Humans are estimated to contain approximately 80, 000-100,000 genes.

Humans have 23 distinct types of chromosomes, which can be distinguished by their physical characteristics, a process called “karyotyping”. Each chromosome has a partner, one member of the pair coming from our mother and one from our father. Of the 23 pairs, one pair contains the genes that determine our sex and is called the “sex chromosomes”: X and Y chromosomes for males, two X’s for females. The other 22 pairs are called “autosomes” and are numbered 1-22.

During growth and repair, cells divide to form new cells (a process called mitosis). Both partners of a chromosome pair are duplicated and distributed to the new cell so that each has a copy of all 46 chromosomes. When an egg or sperm cell is formed, however, a special division process (called meiosis) insures that the genetic material is not doubled when the egg and sperm combine. Only one member of each chromosomal pair goes into an egg or sperm cell (23 chromosomes total) so that, when these two unite to form the fetus, the normal number of 46 chromosomes is restored. Which member of each pair, the one donated by the mother or the one from the father, ends up in the egg (or sperm) is random and explains why children of the same parents are each unique, their “phenotype”—how they look, act, function—is distinct from the parents and from each other because their underlying genes, their “genotypes”, are different. Further, meiosis has a special feature where both chromosomal partners physically pair and can exchange portions of their genetic material, which further increases genetic diversity. Genes that are physically located near each other tend to stay together during this genetic recombination and are said to be “linked”.

Genotype vs. Phenotype/Dominance vs. Recessiveness

The genes are arranged along the length of the chromosomes at specific
locations, called “loci”. There are two copies of each gene, one on each member of the chromosome pair and located at the same position on the two chromosomes. The base sequence of the two copies is the genotype for that particular gene; the expression of those base sequences (translation into protein) is the phenotype for that gene. Phenotypes can be dominant or recessive—genes themselves are not dominant or recessive; the effects they produce are.

The concept of dominance and recessiveness used to be straightforward: one copy’s effects dominated the other, in an all-or-none fashion. Disease was thought to result only when both copies of the gene were mutated (a “homozygous individual”) and function was severely impaired. Genetic disease was, thereby, considered to be quite rare. So long as one normal copy of that gene was present, some active protein was produced, severe dysfunction was prevented and the disease, as it had been defined, was not evident. As our level of technological sophistication has increased and we’ve learned what the gene product is and how to detect it, it’s become obvious that, in the carrier individual (a “heterozygous individual”), one normal gene product and one mutant gene product are present and that such individuals have a functional level less than normal but not severely dysfunctional. Increasing focus is now on detecting the carrier individual and assessing whether having less than 100% normal (fully functional) gene expression impairs function and increases the risk of developing disease.

Mendelian Inheritance

A trait may be inherited in 1 of 6 ways, which corresponds to whether a gene is on an autosome, a sex chromosome, or on mitochondrial DNA and whether a mutant gene’s effect is dominant or recessive. The 6 possibilities are: autosomal dominant, autosomal recessive, X-linked dominant, X-linked recessive, Y-linked dominant, or mitochondrial. Y-linked transmission from one generation to the next is straightforward and seldom elaborated on: males inherit whatever genes are on the Y-chromosomes of their fathers For those genes that are carried on mitochondrial DNA, inheritance is strictly maternal (see Maternal Inheritance below).

Autosomal dominant means the gene is on one of the 22 autosomes and that the trait is observable even though the second copy of the gene may be normal. Autosomal recessive means the trait is not observed unless two abnormal copies of the gene are present. X-linked means the gene is on the X-chromosome. Whether a trait on the X-chromosome is dominant or recessive is determined by its expression in women, where two copies of the gene are present. Traits on the X-chromosome will be expressed in men since only a single copy is present.

Clear detection of the mutant individual can be difficult even when there’s a double dose of the mutant allele because the dysfunctional phenotype may not be expressed fully. You would expect 100% “penetrance”, that is, 100% of the time when the double dose of the mutant allele is present, you would expect the disease to be present. You don’t always observe 100% penetrance, however, for reasons we don’t yet understand but suspect may be due to environment filling in the genetic gaps. A related concept is “expressivity”. If different members of a family all have the mutant gene but their expression of that gene takes different forms, perhaps varying from mild to severe disease, the gene is said to exhibit variable expressivity.

Clearly, gene expression is a complex process that varies with the particular gene and the environment in which it’s expressed. Fully understanding diseases that are due to changes in a single gene is an ongoing challenge. Chronic diseases such as atherosclerosis, obesity, diabetes, and cancer are even more challenging because there are a number of genes involved (multigenic) and their expression is strongly influenced by a number of lifestyle factors (multifactorial). At this early stage of understanding, scientists talk about “genetic susceptibility” and “modifiable risk factors” in lieu of identifying which genes are involved, what they do, and how nutrition can influence their impact on health. Unraveling the complexity of chronic disease genes and the factors that influence their expression is a major goal of human genetics researchers, however, and the next decade is expected to yield considerable information.

Maternal Inheritance

An exception to Mendel’s laws regarding how traits are inherited is the inheritance of the DNA contained in the mitochondria of each cell. Like the DNA in the cell’s nucleus, mitochondrial DNA is double-stranded but, unlike nuclear DNA, it’s circular and only a single copy exists so the concept of dominance/recessiveness does not apply. This DNA codes for “housekeeping” proteins needed for protein synthesis and for some of the proteins involved in energy metabolism, the key metabolic function of the mitochondria. Changes in mitochondrial DNA can cause diseases in the same way changes in nuclear DNA affect function. What’s unique about mitochondrial DNA is that, when the egg and sperm unite, the egg donates the mitochondria. Our mitochondrial DNA is inherited from our mothers, a mode of transmission called “maternal inheritance.”

APPLICATIONS

Genetic Techniques

From genetic research have come technological advances that have broadened the impact of genetics on dietetics beyond just diet therapy. The discovery of restriction endonucleases (aka restriction enzymes) provided the basis for the development of genetic engineering techniques, which in turn gave rise to biotechnology and its diverse clinical and food science applications.

Restriction enzymes are produced by bacteria as a defense against foreign DNA invading the cell and act like “molecular scissors” to cut the foreign DNA into fragments. Incubating an organism’s DNA with a particular restriction enzyme results in a set of fragments that is characteristic and reproducible for that particular DNA and that enzyme. These fragments are called restriction fragment length polymorphisms (RFLPs). Each person, except for identical twins, has a unique RFLP pattern that identifies them. RFLPs form the basis for the identification technology known as DNA fingerprinting.

In a broader context, restriction enzymes allow DNA to be cut-and-pasted and for pieces of DNA to be moved from one organism to another. Since all organisms share the same DNA language, you can move words, sentences,
gene therapy whereby genetic limitations can be repaired by replacing the defective gene with a copy that restores normal function (ex. severe combined immunodeficiency disease, hemophilia, growth of new blood vessels for the treatment of cardiovascular disease).  

agricultural applications whereby crop yield and nutrient content can be improved.

TAKE-HOME MESSAGE

Genetics is central to nutrition and is impacting dietetics professionals in every aspect of dietetics, from clinical nutrition to public policy development to food service. The aspect that provides the greatest leverage to us as a profession is in clinical nutrition and public policy. Genetics is the base; it determines the limits of our personal health continuum between disease and wellness and determines whether we will be susceptible to disease or to wellness. Nutrition is potentially THE most powerful tool for tipping the balance to the wellness end of the continuum because it has the potential for filling the metabolic gaps caused by our genetic limitations. The dietetics professional who understands the implications of the client’s genetic history and the underlying biochemistry is in a position to develop effective nutrition therapies and to possess a competency that will be in high demand within health care and that no other health care practitioner can provide. The potential for the profession is enormous if we will take action now and prepare for the future, which is upon us.

Watch for further exploration of these key concepts in future issues of the newsletter and on our Web site www.complementarynutrition.org.

References
22. Thompson MW, McInnes RR, Huntington FW. Individualized nutrition therapies for her clinical patients. Dr. DeBusk can be reached at 850/562-3261 or RDeBuskRD@aol.com.
Sunday, October 15
8:00 am - 5:30 pm
NCC Executive Committee Meeting
Adams Mark Hotel, Plaza Court 8
1:00 - 5:00 pm
Skill Building Workshop: Botanical Medicine 101 for the Health Care Professional presented by Tieraona Low Dog, MD, AHG
Colorado Convention Center, Room A214

Monday, October 16
1:30 – 3 pm
Health Benefits of Soy Protein Throughout the Life Span; Pediadrics, Soy and Chronic Disease Management, and Adult Health and Wellness.
3:00 - 3:30 pm
Symposium organized by The Office of Dietary Supplements of the National Institutes of Health.
Colorado Convention Center, Room A101-7
5:30 - 8 pm
NCC Business Meeting/Reception
Adams Mark Hotel, Plaza Ballroom D&E/Foyer

Tuesday, October 17
9:30 am – 1:30 pm
DPG Showcase
Exhibit Hall

Other Activities of Interest to NCC Members at FNCE
Tuesday, October 17
2:00 - 3:30 pm
Biotechnology: The Science and the Issues
4:00 - 5:30 pm
Biotechnology: Point/Counterpoint

Tuesday October 17
4:00 - 5:30 pm
Complementary Care in the Treatment of Mood Disorder: Homeopathy and Exercise Intervention (sponsored by the DDPD-DPG) presented by Amy Rothenberg, ND and Lisa Dorfman, RD, MS

Wednesday October 18
4:00 - 5:30 pm
Herbal Medicine and Vitamin Supplements Relevant to Multiple Sclerosis and other Neurological Diseases

Thursday October 19
10:00 - 11:30 am
Pills and Potions: Evaluating Dietary Supplements
Choline and Canotenoids: The Next Generation of Nutrition

THANK YOU to the reviewers of the CPE article for this issue:

Dr. Stephen C. Hedman
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Other Activities of Interest to NCC Members at FNCE

NCC at FNCE

* Note: Schedule is subject to change.

Denver, Colorado

For more information on upcoming conferences on biotechnology and Natural Pharmacy go to: http://www.bioconferences.com
CPE Questions - True or false

1. _____ Genetics is important for understanding health and disease and is becoming a useful tool for dietetics professionals.

2. _____ DNA is the genetic material for humans and is found in the nucleus of each cell and in the mitochondria.

3. _____ DNA is a double helix consisting of a sugar-phosphate backbone and paired nucleotide bases that has encoded within it the information that directs the synthesis and function of the human body.

4. _____ The DNA code must be translated into proteins used to construct the framework of the body and its metabolic machinery.

5. _____ The DNA code consists of four nucleotide bases and it’s the linear sequence of these bases that determines the protein product information encoded within.

6. _____ A gene is a set of sequences that contains the information for a protein.

7. _____ The information in the gene is transcribed directly into protein.

8. _____ The base sequence of the messenger RNA is translated in units, sets of 3 consecutive bases, that correspond to one of the 20 amino acids that are used to synthesize a protein.

9. _____ Proteins are used by the body for structural purposes and for functional (metabolic) purposes.

10. _____ Examples of metabolic proteins are enzymes, hormones, receptors, carriers and pumps, which play critical roles in cellular metabolism.

11. _____ Changes in the genetic material are transferred to the protein for which that gene codes and may affect the function of the protein.

12. _____ If function is negatively impacted, a disease may result.

13. _____ Each cell with a nucleus contains the full genome for that individual.

14. _____ The human genome consists of 23 pairs of chromosomes: 22 pairs of autosomes and 1 pair of sex chromosomes, with one member of each pair originating with the individual’s mother (the maternal copy) and one member with the father (the paternal copy).

15. _____ Each chromosome contains many genes along its length, genes have a specific location on a specific chromosome, and the location is the same for both members of the chromosome pair.

16. _____ Two copies of each gene are present, one on each member of a chromosome pair, including the sex chromosomes.

17. _____ The two copies of a gene may be polymorphic, that is, they may have slightly different base sequences and, thereby, code for slightly different proteins that may differ in ability to carry out the function of that protein.

18. _____ Variations in genes, particularly polymorphisms, are responsible for the functional variation among individuals.

19. _____ New cells only originate from existing cells, and mitosis is the process of cell division that duplicates the 46 chromosomes in the original cell and distributes them to each new cell.

20. _____ Meiosis is a special division process whereby the chromosomes are duplicated from the original cell but only one member of each chromosome pair is distributed to the gamete (egg or sperm).

21. _____ It’s during meiosis that genes are recombined into new combinations because which member of the chromosome pair, the maternal or paternal copy, ends up in a particular gamete is random.

22. _____ Genes that are physically close together are “linked” and tend to stay together when chromosomes are distributed into gametes.

23. _____ “Genotype” refers to the DNA sequence of the gene; “phenotype” refers to the trait (function) that’s produced upon translation of that DNA sequence.

24. _____ Dominance and recessiveness refer to phenotype and are not absolute terms but used to describe which allele prevails functionally over the other in heterozygotes.

25. _____ Most genes are actually co-dominant at the DNA level: both alleles are expressed but the protein produced from one allele may be able to get the job done and mask the inadequacies of a less functional allele.

26. _____ The phenotype may not be “fully penetrant”, meaning that an individual may have the genotype that should manifest as a disease state but may not actually have disease symptoms.

27. _____ Further, family members that have the disease genotype may show a great deal of variability in how the disease is expressed.

28. _____ Lifestyle choices, including nutrition choices, may be impacting the penetrance and expressivity of the phenotype.

29. _____ Nutrition, through its ability to influence the expression of the genes and its ability to supply needed metabolites, has the potential to compensate functionally for faulty genes.

30. _____ Genetic technology has given rise to new medical and agricultural applications, which have potential implications for the dietetics professional.