**THE USE OF COMPLEMENTARY MEDICINE FOR HEALTHY AGING**

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By the year 2020, twenty percent of the US population will be aged 65 years or older. The greatest growth in numbers will be among those aged 85 years or older. If the healthcare demands of this group match those of their parents, it will place an extraordinary burden on funding for medical services. By promoting healthy aging, complementary medicine practitioners can improve the cost-effectiveness of healthcare delivery. A scientifically based complementary medicine program to promote healthy aging includes (1) diet and nutritional tailoring, (2) nutrient enhancement to meet specific individual needs, (3) exercise training, (4) stress management, (5) promotion of structural integrity, (6) environmental mental adjustment, (7) counseling on purposeful living, and (8) normalizing intercellular communication. The program described in this article incorporates these features and focuses on the following modifiable factors of unhealthy aging: altered mitochondrial function and oxidative stress, increased protein glycation, chronic inflammation, defects in methylation, reduced detoxification ability, and altered immunity. (Altern Ther Health Med. 1998;4(4):42-48)

Medicine is at a crossroads. Although it has been very successful in evolving the science of disease diagnosis and treatment during the past 4 decades, it has not been as successful in the promotion of healthy aging. The failure to achieve healthy aging has left us with burgeoning healthcare costs as the number of unhealthy older people increases. The response has been to implement a cost-containment approach toward healthcare delivery called “managed care,” which has not been well received by patients or their healthcare providers. This system reduces service as well as quality of care, and results in an increasing number of medically disadvantaged individuals, particularly among the older members of the population.

The healthcare system can either continue on this path of rationing healthcare services, or it can modify this approach to focus more resources on the promotion of healthy aging. This latter option will assist in extending the health span of the individual and facilitate natural death rather than the high-technology–supported death many now experience. Promotion of healthy aging represents a domain in which complementary medicine can make a significant contribution.

Olshansky et al calculated that within the next 20 years, the mean life expectancy might approach 85 to 90 years. Demographic projections indicate that 20% of the US population by the year 2030 will be aged 65 years or older. If not healthy, older individuals will demand more medical services. Beyond the impact of the developed world’s changing demographics is the recognition that the aging baby boomer population has a rising expectation of good health. In the past, people felt that if they could retire at age 65 and have 10 years to interact with their grandchildren, engage in senior activities, and be involved in community affairs, they would experience their golden years. Today, however, the expectation of aging baby boomers is that they will never retire and will continue to engage in multiple activities, travel the world, be physically active, engage in exciting new challenges, and be available as catalysts for social change as they grow into their 70s and 80s. This rising expectation of good health provides opportunities for increasing disillusionment with a healthcare system that is focused on the diagnosis and treatment of disease, and that historically has provided limited emphasis on health promotion and disease prevention.

As Fries’ states, much of the loss of function associated with disease among older individuals is a consequence of the progressive loss of “organ reserve.” When we are young, there is a reserve of organ function beyond that which is necessary for the baseline requirements of most organ systems. As we age, however, we lose organ reserve, and stresses that we could have accommodated while younger may now exceed our resilience, resulting in health crises. Fries emphasizes that organ reserve is related to biological age. As we lose organ reserve, our biological age increases, making us more susceptible to disease. We can modify how quickly we lose organ reserve and undergo biological aging through changes in lifestyle, environment, and nutrition. It is now recognized that 75% of our health and life expectancy after age 40 is modifiable on the basis of such choices.

Health after age 40 is a consequence of the interaction of genetic inheritance factors and environmental modifiers. Diseases that are most common among the older segments of the population (eg, coronary artery disease, stroke, cancer,
maturity-onset diabetes, arthritis, Alzheimer’s disease, Parkinson’s disease, kidney disease, and liver disease) in part result from the interaction of unique genetic susceptibilities with lifestyle, environmental, and nutritional factors.

Medicine traditionally has been built on the notion that one is well until proven sick. This concept is incorporated in the differential diagnosis and treatment model that frames the structure of healthcare in its present form. We normally assume that diagnosis of a disease in those aged over 40 years is a consequence of faulty genes, for which little or nothing can be done. Following this assumption, medicine’s role would be to rescue the individual from the results of bad genes and bad lifestyle through appropriate interventions such as the management of symptoms, the correction of pathology, or even the replacement of a poorly functioning organ. Although this model has been successful in extending the mean life expectancy, it has increased the number of unhealthy, older individuals who require significant medical services as they age.

The Mendelian view that health after age 40 is determined principally by genes has been challenged recently because of the advancing understanding of the structure of our genome. As Bishop and Waldholz write in *Genome*, "[u]nmasking the identity of the genes in and of itself does not determine the inevitability of disease. Rather, it defines the risk of disease when an individual is plunged into a harmful environment." Medicine is built around the principle of the deterministic nature of genes and how they control health. We are now beginning to recognize, however, that there is considerable plasticity in the way genes are expressed. The modification of genetic expression as a consequence of how we treat the genes plays a principal role in controlling our health as we age.

There are two general types of genes: constitutional and inducible. Constitutional genes encode messages that are expressed in a constant way and are not significantly modified by environmental factors or lifestyle choices. Inducible genes, however, are sensitive to environmental, lifestyle, and nutritional factors, and can be either up- or down-regulated in their expression. The expression of inducible genes is modified over the course of aging—by environmental, diet, and lifestyle choices—plays a significant role in determining health after age 40.5 It is as though we are all involved in a nonblinded, non-placebo–controlled, noncrossover experiment known as our "life experiences," which wash over our genes and give rise to different expression based on the decisions we make. The expression of these inducible factors, which are locked in our genetic inheritance, controls how quickly we lose organ reserve and how vulnerable we are to age-related diseases.

Genetic research has indicated that there is considerably more polymorphism at the physiological and cellular biochemical levels than previously recognized.3 Although we may look similar—2 eyes, a nose, 10 fingers, 10 toes—our genes code for vast functional differences at the biochemical level. From one person to the next, the enzyme functions of the liver that are related to detoxification may vary 4- to 7-fold.4 Whereas one individual exposed to a potentially toxic chemical may detoxify easily, another—due to unique genetic inheritance factors—may be susceptible to that substance’s toxic effects through a reduced ability to detoxify.

According to Steventon et al,2 those who develop Parkinson’s or Alzheimer’s disease often have genetic impairment in several of their detoxification pathways, rendering them more susceptible to the neurotoxic effects of certain chemicals. Ambrosone et al12 found that women smokers who got breast cancer had defects in their detoxification status, making them more at risk to carcinogenic exposures. Lin12 notes that 30% of enzymes are polymorphic (ie, they differ from individual to individual based on genetic inheritance) and 7% of all individuals have two forms of a specific enzyme. One form is expressed under certain environmental conditions, and another is expressed under a second set of conditions. This variation of phenotype, coming from a different expression of our genotype, results in pleomorphism. A person’s lifestyle plays a significant role in determining which genes are expressed and how the genotype is translated into the phenotype.

Environment modifies not only the expression of inducible genes, but also the posttranslational cellular function. After the genes have been expressed and their message has been translated into the manufacture of protein and other cellular materials, the structure and function of these substances can be further altered as a consequence of processes such as oxidation or glycation. Both of these posttranslational influences can affect cellular function in ways that are associated with unhealthy aging.

The combination of environmental effects on both gene expression and posttranslational modification of cellular materials gives rise to symptoms of aging that are well recognized in clinical medicine: those who smoke heavily appear to age faster and have higher risk of age-related diseases such as cancer and heart disease; those who consume excessive alcohol also appear to age more quickly and are exposed to increased risk of liver- and heart-related problems; and those with poor-quality diets that are high in calories and low in essential nutrients show signs of overconsumptive undernutrition through obesity, poor health patterns, and more prevalent age-related diseases. These examples demonstrate how environment and lifestyle influence gene expression and posttranslational modification of cellular function, giving rise to increased risk of age-related diseases.

Medicine has focused principally on the diagnosis of these diseases once they occur, and physicians have usually placed less emphasis on understanding genetic susceptibilities and gene expression modifiers. Complementary medicine provides tools for assessing and improving functional status before the onset of pathology. By its epistemology, complementary medicine focuses on functional aspects of medicine, providing the assessment of and early intervention for the improvement of physiological, cognitive/emotional, and physical functioning of the individual.

**BIOMARKERS OF AGING**

Evans and Rosenberg7 describe various biomarkers associated with decreased function and unhealthy aging, including the following:

- loss of strength
- reduced flexibility
- decreased cardiovascular endurance
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Williams defines genetotrophic disease as the faulty expression of AND NUTRIENT MODULATION

Genetotrophic disease as aging as well as an unhealthy aging process. This loss is associated with increased biological aging as well as an unhealthy aging process.

The biomarkers of aging represent a loss of organizational energy that results in a loss of the ability to maintain structure and function. Although in the past people commonly believed that loss of structure was a natural consequence of aging, research now indicates that the rate at which this organizational structure is lost can be modified on the basis of gene exposures. The DNA in our chromosomes, which represents the basis of our genetic inheritance, is an energy information map in which are encoded the “attractors” of matter to build the organized structure of the body. The matter making up the atoms, molecules, biomolecules, cells, tissues, organs, and organ systems of the body is concentrated and organized through an energy-requiring process based on the genetic template called genes. The maintenance of this highly organized structure depends on the efficient use of energy. In the absence of proper control of metabolic energy, the natural tendency of the universe toward disorganization—entropy—will prevail. In a sense, unhealthy aging is the inability to maintain organizational structure of the organs. Factors that accelerate the loss of organizational structure include chronic infections, poor-quality diet, reduced aerobic competency, distress, toxic exposure, trauma, and lack of attention to individual genetic needs.

If the organizational energy driven by metabolism is incapable of overcoming these contributions to disorganization, loss of organ reserve will occur. This loss is associated with increased biological aging as well as an unhealthy aging process.

**GENETOTROPHIC DISEASE AND NUTRIENT MODULATION**

The interaction of diet and nutrition with genes contributes to the maintenance of cellular energy required for organization. Williams defines genetotrophic disease as the faulty expression of genes resulting from the inadequate intake of specific nutrients as determined by one’s inheritance factors.

The concept of genetotrophic disease and biochemical individuality was further amplified through the pioneering work of Pauling. In 1949, Pauling and colleagues reported that sickle cell anemia was caused by a single genetic mutation resulting in an altered structure of the globin component of the hemoglobin molecule. They proposed that future procedures might be found to modify the genetic expression of this characteristic to prevent sickle cell crisis.

In 1993, Perrine and colleagues reported that administration of butyrate could stimulate the gene expression of fetal hemoglobin, which dilutes the sickle hemoglobin and thereby helps to prevent sickle cell crisis. In a multicenter study of hydroxyurea in sickle cell anemia, Charache and colleagues reported that hydroxyurea could also stimulate gene expression in the sickle cell, causing the individual to increase the level of fetal hemoglobin and preventing the painful sickle cell crisis. These remarkable studies indicate that even a significant genetic mutation that produces a disease affecting many organs, such as the crisis of sickle cell anemia, can be prevented through intervention with specific substances that modulate gene expression and prevent the phenotype associated with disease.

Nutrients can influence gene expression and the activity and function of gene products such as enzymes. For instance, the consumption of cruciferous vegetables (eg, broccoli, brussels sprout, cauliflower, and cabbage) has been found to increase the gene expression of certain detoxification enzymes, increasing first-pass drug detoxification and elimination of environmental xenobiotics. Grapefruit juice, which contains the flavonoid naringenin, has been found to inhibit the activity of the detoxification enzyme cytochrome P-450A. This fact has been found useful in sustaining the blood levels of cyclosporine, a drug used to help prevent organ transplant rejection. Naringenin from grapefruit juice is now recognized for its ability to modify blood levels of specific drugs through its impact on gene expression and detoxification enzyme activity. Diet and specific nutrients can therefore modify gene expression as well as promote and improve phenotypes—factors associated with healthy aging. Complementary medicine practitioners are skilled in applying these principles to promote healthy aging.

The science of medicine is perhaps the most frequently cited case of increasing specialization seeming to follow inevitably from increasing knowledge as new cures and better treatments for more diseases are discovered, but as medical and biochemical research comes up with deeper explanations of disease processes (and healthy processes) in the body, understanding is also on the increase. More general concepts are replacing more specific ones as common, underlying molecular mechanisms are found for dissimilar disease in different parts of the body. Once a disease can be understood as fitting into a general framework, the role of the specialist diminishes. Physicians can look up such facts as are known. But more importantly, they may be able to apply a general theory to
Mitochondrial DNA is therefore approximately 20 times more like-
ing superoxide, hyroxal radical, and hydrogen peroxide. similar to a bacterial DNA. circular and unprotected by histone and nonhistone proteins—susceptible to oxidative injury than is nuclear DNA, because it is 50% of our genetic makeup. Mitochondrial DNA is much more mother, which suggests that our mothers influence more than nucleus. Mitochondrial DNA is inherited exclusively from the USE OF COMPLEMENTARY MEDICINE REDUCING RISK OF DISEASE THROUGH THE cellular repair, defense mechanism, nervous system and muscle system function, and any other energy-requiring process that maintains the organization of the body and helps resist aging. SIX MODIFIABLE FACTORS OF UNHEALTHY AGING Six modifiable factors that contribute to unhealthy aging have emerged as a result of research conducted over the last decade: 1. Aging related to altered mitochondrial function and oxidative stress 2. Aging disorders as a consequence of increased protein glycation 3. Unhealthy aging as a consequence of chronic inflammation 4. Contributions of defects in methylation to the aging process 5. Compromised detoxification ability and the risk of disease 6. Altered immunity related to aging All six factors and their courses can be modified through the application of complementary medicine. Wallace23 has described how the loss of mitochondrial function may be associated with unhealthy aging. The mitochondria are the energy powerhouses of the cell, in which nutrients (potential energy) are broken down into metabolic energy for cellular repair, defense mechanism, nervous system and muscle system function, and any other energy-requiring process that maintains the organization of the body and helps resist aging.

REDDUCING RISK OF DISEASE THROUGH THE USE OF COMPLEMENTARY MEDICINE Mitochondria are known to have their own genetic information apart from the DNA of the chromosomes located in the nucleus. Mitochondrial DNA is inherited exclusively from the mother, which suggests that our mothers influence more than 50% of our genetic makeup. Mitochondrial DNA is much more susceptible to oxidative injury than is nuclear DNA, because it is circular and unprotected by histone and nonhistone proteins—similar to a bacterial DNA. Mitochondrial DNA also is found within the organelle of the cell, in which are produced the greatest number of oxidants including superoxide, hydroxal radical, and hydrogen peroxide. Mitochondrial DNA is therefore approximately 20 times more likely to be injured by oxidative reactions in the cell than is chromosomal DNA.28 Because mitochondrial DNA is injured by adverse free radical reactions, it can accumulate mutational injuries resulting in less efficient energy production. Ultimately, when enough mitochondrial function is lost, the whole organism loses energy for organization, and the symptoms of fatigue, memory loss, poor cardiac function, and general compromised physiological function occur. A number of mitochondrial disorders are now associated with accelerated aging. Among such conditions are fibromyalgia, cardiac problems, immune deficiencies, and central and peripheral nervous system problems that include both Alzheimer’s and non-Alzheimer’s dementia.29 Significant differences in mitochondrial genetics exist from person to person; some individuals are much more susceptible to mitochondrial injuries as a consequence of oxidative stress. In these cases, increased nutritional intake of specific nutrients that support mitochondrial function may be desirable. These nutrients include N-acetylcysteine, ubiquinone (coenzyme Q10), lipoic acid, N-acetyl carnitine, creatine, and vitamin E.30 The concept of oxygen free-radical–induced aging was first set forth by Harman31 in 1952. He proposed that excessive production of free radicals such as lipid peroxides or superoxides could result in injury to the cell, which can accumulate over time and result in the aging diseases. Mitochondria have been recognized as the site within the cell where excessive free radicals might be produced. Certain nutrients may play a role in modulating gene expression associated with this oxidative process.

GLYCATION A second characteristic associated with aging is increased protein glycation.32 Glycation is the process by which sugar (glucose) in the blood or tissues is combined with the epsilon-amino group of lysine within cellular proteins to produce glycosylated proteins. These are proteins whose structure and function are altered. Glycation is a process that is analogous to the browning reaction of baking that produces the crust on bread. In a sense, glycosylated proteins like glycohemoglobin are “crusty proteins.” The glycosylated protein hemoglobin can be used to follow the control of blood sugar in the diabetic by measuring glycosylated hemoglobin A1c. An increased percentage of glycosylated hemoglobin in the total hemoglobin is indicative of poor glycemic control in the diabetic.

All proteins in the body (not just hemoglobin) are glycosylated during conditions of poor control of insulin and glucose metabolism. The glycosylation of proteins results in the production of advanced glycosylation end products (AGEs). Accumulation of AGEs in the bloodstream results in activation of the receptors for AGEs (RAGEs) on the membranes of cells, increasing mitochondrial oxidative damage and inflammation.33 The accumulation of AGEs and the resulting oxidative stress from the activation of RAGEs result in diverse age-related conditions such as periodontal disease and the loss of teeth in the adult,34 skin aging and accelerated wrinkling,35 and increased risk of heart disease.36 Glycation of proteins and the formation of AGEs can be modified through dietary and lifestyle manipulation. A diet that is high in unrefined complex carbohydrates and fiber, adequate in protein, and enriched in ω-3 fatty acids (eg, fish oils) can promote proper insulin sensitivity and blood sugar control.37 Micronutrients that have been found helpful in improving control of insulin and blood sugar include magnesium,38 vitamin E,39 vanadium,40 and chromium.41 CHRONIC INFLAMMATION The third factor of aging that is modifiable by complementary medicine is chronic inflammation. Chronic inflammation42 is associated with age-related diseases as diverse as dementia and...
heart disease. Chronic inflammation and the activation of the immune system may be initiated by chronic infection or alteration in the immunological function of the gastrointestinal tract. Infection with parasitic bacteria in the intestinal tract as well as exposure to food antigens through diet can give rise to local and systemic immune reactions with the gut-associated lymphoid tissue (GALT), which can result in systemic immunological responses associated with inflammation. The GALT comprises more than 50% of the body’s immune system and is the principal site in which antibody proteins are produced. It is also capable of manufacturing proinflammatory cytokines such as interleukin-1 (IL-1), IL-2, IL-6, and tumor necrosis factor α (TNF-α) upon exposure to allergens or toxins.

Nitric oxide is also produced in increased quantities by the GALT on immunological activation. Increased output of nitric oxide can break down the intestinal barrier function and result in permeability of the lumen of the small bowel, increasing the diffusion of larger molecular weight molecules across the gastrointestinal tract into the portal circulation and challenging the functions of liver detoxification and the immune system. Removing foods to which one is sensitive can lower the inflammatory reactions at the GALT and reduce systemic markers of inflammation.

Hunter notes that the inflammatory reactions initiated by activation of the GALT are not due solely to food allergens, but also result from what he calls “enterometabolic disorders.” These are a class of proinflammatory and immune responses that occur as a consequence of the GALT exposure to parasitic bacterial metabolites and bacterial cell wall lipopolysaccharides. These challenges up-regulate the immune response of the GALT and produce increased levels of inflammatory cytokines, which modulate the function of the Kupffer’s cells in the liver. The Kupffer’s cells then produce their own inflammatory markers, which are delivered to systemic circulation and may transmit a general state of chronic inflammation to the body.

The susceptibility of bacteria and other parasites to attach to the intestinal lumen, as well as the response to various food antigens, is genetically determined and can vary remarkably from person to person. Adherence of certain types of bacteria to the intestinal lumen and their proliferation in the gut can result in the induction of inflammatory cytokines that aggravate arthritis. This may explain findings that show arthritis can be treated successfully with antimicrobial medications in some individuals.

Specific diets tailored to the genetic needs of the individual have been shown to reduce disease activity in rheumatoid arthritis by altering bacterial flora and reducing inflammatory markers. It has also been found that osteoarthritis pain and inflammation can be amplified by activation of the GALT and by increased intestinal permeability.

Modification of the intestinal environment to reduce the activation of the GALT and the subsequent decrease of inflammatory markers may be achieved among genetically susceptible individuals by applying the four-R program or a biotherapeutic approach toward the normalization of intestinal flora. The four Rs in this approach—remove, replace, reinoculate, repair—involves (1) removing the offending organisms (parasite or bacterial origin), (2) replacing digestive enzymes and stomach acid where necessary, (3) reinoculating the bowel flora with oral supplementation of symbiotic bacteria (such as Lactobacillus acidophilus and Bifidobacterium bifidum), and (4) providing adequate levels of nutrients for repairing the damaged gastrointestinal lumen (including L-glutamine, pantothenic acid, zinc, and antioxidants). As Elmer et al have written: “In an effort to decrease the reliance on antimicrobials, the time has come to carefully explore the therapeutic applications of these biotherapeutic agents in both treatment and prevention of selected intestinal and vaginal infections.”

A variety of phytonutrients derived from botanical supplements also have been found useful for modulating the immunological activation of the GALT and inflammation. Herbal concentrates including standardized extracts of curcumin, quercetin, and boswellic acid have helped to reduce the inflammatory cascade among genetically susceptible individuals. Jonas et al reported that increased intake of niacinamide (vitamin B3) can help reduce inflammation associated with osteoarthritis. This finding confirms the observational studies reported by Kaufman on the use of niacinamide therapy in osteoarthritis in 1953.

Among genetically susceptible individuals, chronic inflammation has been associated not only with gastrointestinal and liver-related disorders, but with the risk of Alzheimer’s disease and heart disease as well. Those who are the most genetically susceptible to nervous or cardiovascular system damage as a consequence of chronic inflammation appear to carry the APOE4 single or double allele genotype. Screening for APOE genotype should alert the individual to be more concerned about chronic infection and inflammation and of the risk of damage to the nervous and cardiovascular systems resulting in dementia and cardiovascular disease. The APOE4 genotype is much more sensitive to dietary intake of saturated fat that increases cardiovascular and oxidative stress risk; therefore, APOE screening may be a useful complementary medical tool for evaluating the need for diet, lifestyle, and nutrient modulation.

ALTERATIONS IN HOMOCYSTEINE METABOLISM

The fourth modifiable factor associated with aging relates to defects in methylation and the management of the homocysteine pathway. Lindenbaum and colleagues report that many neuropsychiatric disorders diagnosed as senile dementia are actually caused by insufficiencies of vitamin B12, folate, and vitamin B6. According to a recent study, “[t]he response rate of improved cognitive function to vitamin supplements in an older population supports the notion that metabolic evidence of vitamin deficiency is common in the elderly, even in the presence of normal serum vitamin levels.” Functional insufficiencies of vitamin B12, folate, and vitamin B6 are associated with defects in the metabolism of the amino acid homocysteine and with impairment of proper methylation through the tetrahydrofolate cycle.

Because a number of genes control the metabolism of homocysteine, the percentage of those in the population who may need enhanced levels of these nutrients to promote proper metabolism...
of homocysteine is greater than previously recognized.

McCully was one of the first investigators to actively study the adverse effects of homocysteine on vascular function. His work pioneered the understanding of the role of excess homocysteine in increasing the risk of heart disease, stroke, and dementia among genetically susceptible individuals. Herbert and Bigaouette have petitioned the Food and Drug Administration to require that flour be fortified with 25 µg of vitamin B12 per 100 g (a significantly higher level than the recommended dietary allowance) as well as with folic acid to help protect genetically susceptible people against injuries due to homocysteine. According to these authors, “this combined supplement will also prevent millions of Americans from getting vascular toxic hyperhomocysteinemia with its enormous costs in heart attack, stroke, and other vascular toxic morbidity and mortality and billions more healthcare dollars.”

Alterations in the expression of the folate cycle also can result in increased risk of cancer. Blount et al note that a significant proportion of the US population have low dietary and blood levels of folate in the range associated with elevated nucleic acid misincorporation and chromosome breaks. This occurrence is found more frequently among genetically susceptible individuals who have defects in folate and homocysteine metabolism. Such breaks could contribute to the increased risk of cancer and cognitive defects associated with folate insufficiency.

COMPROMISED DETOXIFICATION AND IMMUNITY

A fifth modifiable factor of aging is reduced detoxification ability among those who are nutritionally compromised and thus more susceptible to xenobiotics. Numerous studies have indicated that enhanced levels of certain nutrients in the diet can improve detoxification. Glycine supplementation has been used to increase the ability to detoxify aspirin for those who suffer from aspirin overload, N-acetylcysteine has been used to improve the detoxification of acetaminophen, and green tea polyphenols have been found to increase the detoxification of various carcinogens. These are just a few of the many nutrients that have been used under certain metabolic demands to help improve detoxification and prevent diseases associated with toxic exposures.

Finally, it has been found that a central feature of aging is altered immunological function, with reduced cell-mediated immunity and increased immune intolerance characterized by increasing levels of autoantibodies in the endocrine glands. One characteristic that differentiated healthy nonagenarians from unhealthy younger people was the absence of autoantibodies in the endocrine glands of the older population. By designing an individual’s proper diet based on his or her genetic needs, and by reducing antigens to which the person is sensitive—such as gluten in wheat or casein in dairy products—reduction in antigen exposure will result, helping to improve immunological function. It also has been found that certain nutrients may be useful to support cell-mediated immunity among older individuals. Those nutrients include essential fatty acids from the ω-3 family and antioxidants such as vitamin E.

Stress management also has been found to play a significant role in modulating neurotransmitter receptor sites and altering neuroid immune function. Programmed exercise among older individuals has been found not only to increase strength and endurance, but to improve immunological function. Exercise represents another approach of complementary medicine toward the promotion of healthy aging.

THE COMPLEMENTARY MEDICINE APPROACH

In this article I have focused on new ways healthcare providers can promote healthy aging. This perspective moves us from the deterministic view that genes control the incidence of disease in the aging process to the recognition that genetic expression is polymorphic and that our phenotype can be modified through diet, environment, and lifestyle choices. Complementary medicine has a significant opportunity to contribute to the delivery of services that promote healthy aging. It does so through the functional assessment of biological age prior to the onset of a diagnosed pathology and through the introduction of specific modifiers of gene expression and physiological function that improve health span. Tools available to the complementary medicine practitioner—those that have not been applied traditionally in disease-focused medicine—include the following:

• diet and nutritional tailoring for the individual
• nutrient enhancement for the genetic needs of the individual
• exercise and aerobic competency training
• stress management
• promotion of structural integrity and muscular skeletal function
• environmental adjustment
• counseling on purposeful living and spiritual approaches to healing
• techniques focused on normalizing intercellular communication, such as acupuncture

By using the complementary medicine resources of the past to understand an individual’s functional capacity, as well as how diet, lifestyle, and environment modify this function, the complementary medicine practitioner can meet patients’ needs, promoting healthy aging. The payoffs for this developing relationship between the complementary medicine practitioner and his or her patient are reduced expenditures in disease care throughout the aging process as well as improved quality of life and functional health span. The emerging science that describes the origin of age-related diseases is moving rapidly toward the recognition that “don’t fix it until it’s broken” is no longer viable—either from a humanistic perspective or from the perspective of conserving disease care expenditures. The healthcare provider who focuses on complementary medicine will be able to contribute to the medicine of the future, particularly to the promotion of healthy aging.

References
