

Review Article

# The Genetic Tango: Genes in Your Body, Genes in Your Food

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## Abstract

This review article highlights a range of new research from the realm of genomics that crosses traditional disciplines to address the genetic relationships of food and health. Research examples include the interactions of food and diet to obesity, heart disease, cancers, diabetes, and other risk-related symptoms. Interdisciplinary research from agriculture, medicine, nutrition, public health, behavioral sciences, food safety, public policy, and other disciplines must combine to better address the devastation to families and to the U.S. healthcare budget from diet-related chronic diseases. Food is a choice, thus the chemicals in these foods is secondary to that choice. There are numerous types or varieties of soy, corn, grains, legumes, vegetables, fruits, etc. Not all are commercial success; but may provide healthful benefits. What if foods were financially valued on the basis of their health ingredient potential rather than their yield in the field (bushels per acre) potential? Clearly the genes that control yield may not be the same genes that control for production of beneficial food chemicals or ingredients, or their amounts? We should know how to choose those foods that contain beneficial agents for our health and our children's health. Genes in your food as a phrase is used throughout this article as a euphemism for meaning ultimately the 'chemicals in your food'.

**Keywords:** Genomics; Genetics; Diet-related chronic disease; Nutrition; Food

## Introduction and Background

Food is medicine, medicine is food.

The sound bite was first used by Hippocrates in 500 B.C.E. The philosopher's elegant package of comprehensive knowledge is now being bolstered by research in the realm of genomics and its scions: Nutrigenomics, proteomics, metabolomics, and numerous others -omics. Genomics is the study of the entire set of genes found in a species. It has opened a frontier of research that reinforces Hippocrates' ancient understanding of the relationship between food and health – or ill-health, increasingly, the issue of the day.

The daily news headlines highlight incidence of heart disease, the personal and societal losses from cancer, the complexities of diabetes, policy issues surrounding healthcare coverage for families, and the related skyrocketing costs. The United States has a gross annual domestic product larger than \$12 trillion. Nearly \$2 trillion US is spent on healthcare annually. Of these healthcare expenditures, diet-related chronic diseases – diabetes, heart disease, stroke, cancer, obesity, and asthma – consume more than 75 percent of the nearly \$2 trillion U.S. healthcare budget, according to Department of Health and Human Services statistics, Centers for Disease Control and Prevention [1]. Type 2 diabetes alone, for example, is 11 percent of U.S. health care expenditures reported by the Centers for Disease Control and Prevention [2]. Chronic disease is increasing. Health care costs are increasing. The numerous labor strikes and contentious negotiations that consistently boil down to health care benefits signal that the country is in the midst of a crisis.

Here's the kicker: Diet-related chronic diseases are preventable.

From 60 percent to 90 percent of these diseases are preventable with modifications to diet and lifestyle according to the Centers for Disease Control and Prevention [1]. Consumer behaviors, physical activity and other lifestyle choices are significant factors in the diet and health equation. In this paper we focus on people and the food they eat. More specifically, we focus on the interrelationships of the genes in food and the genes in the body.

As a starter to this review some definitions are in order. These authors use the catch phrase 'genes in your food' not to be wrongly confused with chemicals in your food, which we all understand. The words are meant more to be clever and illustrative full well knowing that genes don't transfer by eating them. But I wish to illustrate we do have choices in our food. Differing soybean plant varieties offer better protection for cancer, due to their differing genes in those varieties. Differing tomato plants produce redder tomatoes, or more yellow or purple colors; they might be more healthful. While it is true the DNA which codes for this affect has no useful value to the person eating the tomato, it nevertheless has produce a more valuable reds, yellows, purples, which is due to yellow or red carotenoids, which are the healthful agents to the person eating the tomato. The same can be said of dozens of plant varieties of all types of plants. The adage that 'genes in your food' is meant to be provocative to think about your food choices insofar as choosing which chemicals you wish to ingest.

## The power of genes: on Again, off Again

The interaction of genes in the body and the genes in the foods people eat is a choreographed dance, a tango of various steps, figures and poses. Before describing the interaction of the body's genes with

the food that is consumed, we highlight key information from basic genetics and genomics.

In brief, genes are composed of DNA; they are the software instructions for all of the body's life functions. To further the computer analogy the memory capacity, or software instructions, that are contained within the DNA of a gene is immense. That massive DNA memory contains the massive information, which drives all bodily processes. Roughly that same quantity of DNA information is contained in the genes of many common house plants, also in the genes of animals such as a, fish, dog, cat, mouse, cow, pig, horse, or the genes of the next person you meet on the street. What can influence the genes in the body? chemicals. Some are environmental chemicals like cigarette smoke, pollutants, drugs, hormones, food ingredients, flavors, fragrances, among many more. Some are synthetic and many are naturally occurring synthetic. We will try to focus on chemicals ingested with the food that enters our bodies.

Genes control body functions by a variety of techniques. Mendelian inheritance, or the characteristics that are passed to an individual from the parents, is one way that genes control an individual's functions. Genes also influence functions through mutation. Genes can be regulated by natural processes, and they can be mutated at an individual level, a species level, and over time. The environmental influences on genes and gene expression are particularly relevant to this discussion.

Mendel's publication of his classic work on segregation of the genes of the pea plant [3] paved the way for us to understand Mendelian inheritance and ever since geneticists also discovered other inheritance patterns. Mendelian genetics shows that a father with blue eyes and a mother with brown eyes have a 1-in-4 chance of producing a blue-eyed child. These inheritance genetics are well understood, based on Mendel's publication of his classic work on segregation of the genes of the pea plant from the little known pea study not published for 65 years after it was completed.

A second type of regulation, gene expression, is key to the understanding of genomics. Genomics is the study of all the genes of a species as they interact in concert. Essentially, an individual infant child supposedly has all the genetic machinery and biological software and genetic programming skills to make a fully functional adult. Some changes will be needed, to be sure. Sex hormone genes need activation at just the right age; bones must grow for a while and then stop. Brain development continues for a long time then wanes after many decades perhaps well into later life. Organs grow and mature, teeth are replaced at just the right intervals, etc. New genes or new gene functions arise as the individual ages, partly by changes in gene function or sometimes called changes in gene expression but also by other changes such as selected programmed mutations. The functional changes might be epigenetic changes, some of which might be influenced by diet-related chemicals, hormone production, organ maturation, environment related or others perhaps unknown. The adult human is visibly different in form and function from the infant. Since the adult body expresses different genes than it did in infancy, it can be partly described an example of changing gene expressions, epigenetic changes, or gene mutation, or more likely, combinations of all three. Another striking example is the metamorphosis of the caterpillar into butterfly. The monarch caterpillar and the monarch

butterfly look completely different, yet they are basically genetically identical; same genotype but different phenotype! At various stages of life, certain genes are turned on; others are turned off. On and off translates visibly as wings in the monarch and legs in the caterpillar. On and off can mean cancer or heart disease in humans, spinal defects or normal spinal development using the well-known example of folic acid consumption by a pregnant mother to protect against neural tube defects in offspring. Folic acid dietary supplementation is perhaps the most widely accepted and practiced genetic modulation therapy ever tried in mass. The folic acid changes are to the conformational structure of DNA and not to the primary DNA sequence.

Third, the environment exerts powerful influences on genes and likely gene expression. For example, genes are influenced in profound ways by the foods that an individual consumes. Food choices by pregnant women may influence the fetus developing in the uterus. Food choices (and the chemicals therein) continues to influence genes through the stages of youth to old age, but with different outcomes at different stages. Some food choices (chemicals) are better choices than others. Not all chemicals from foods are good choices at certain times in life. Just recently [4] published a paper showing men who are obese can contribute specific methylation status to their offspring predisposing them to increased risks of chronic diseases in adulthood. This is directly translated as newborns of obese parents have altered DNA methylation patterns at imprinted genes [4]. Certain animal models are well documented for having transgenerational effects due to diet or environmental factors from inducing various epigenetic changes [5,6]; but as show above, there is now evidence for similar affects in humans.

Knowledge of the genome is critical as researchers look back into the evolutionary history of life on Earth, and as they look forward to the alleviation of disease and improved quality of life. Scientists tackled the worm *Caenorhabditiselegans* as the first complex organism to be sequenced. The 1998 publication of the work from a five-year collaboration of the *C. elegans* Sequencing Consortium in 1998 revealed more than 19,000 genes in the genomic sequence of *C. elegans*, with over a 40 percent match of the protein products to those of other organisms – including humans[7]. Two years later, the genome of *Drosophilamelanogaster* was published [8]. The species of fruit fly is the classic model for laboratory studies of genetics. The identification of the significant percentage of genes of the fruit fly that are shared in common with other organisms and humans opens the way for potential understanding of aging behavior, neurodegeneration, Parkinson's disease, and pharmaceutical development.

The nematode and the fruit fly diverged in evolutionary time by hundreds of millions of years. Humans diverged from the two species around 600 million years ago. Basic core genes, held in common across species, are important and have been conserved over evolutionary time. The homeotic hox (HH) gene family, for example, performs many of the essential body functions that occur in all non-plant species: the orientation of head to toe; the formation of spinal cord; the formation of head, heart, chest, abdomen; and the control of basic metabolism. University of Minnesota graduate Edward B.

Lewis received a Nobel Prize in 1995 for his contributions to the understanding of the genetic control of early embryonic development [9-11].

### Fat worms, Fat people

Obesity in humans results from the interaction of genetics with lifestyle and other environmental factors. The macroscopic worm noted above, *C. elegans*, illustrates the universal opportunities to study and treat obesity in humans, based on new knowledge derived from the genomic maps of other species. Ashrafi and colleagues in 2003 studied the processes in the brain that regulate feeding and the storage and use of body fat in laboratory worms [12]. Researchers identified the genes – active or inactive, on or off – that are responsible for reducing body fat and for increasing fat storage. To make worms thin, 305 genes must be down regulated. Another 112 genes control the increase in obesity. Importantly, many of these genes are the same as those in animals and humans. Further research will have major implications for obesity and diabetes and for a better understanding of fat metabolism in the human body.

A group of Italian researchers led by Strazzulo in 2003 is credited with identifying the so-called beer-belly gene, studied the relationship of increases in body weight and blood pressure in aging individuals [13]. They found that angiotensin-converting enzyme (ACE), which helps regulate blood pressure in the body, also might play a role in the growth of fat cells. One variation of the relevant gene, in a study of healthy men, is linked to the deposition of fat around the stomach.

### Agriculture and genomics

New information from genomics research is bringing agriculture to the brink of a paradigm shift. Traditional agriculture was all about bushels per acre and commodities. The new agriculture is food for health – as well as biomass for renewable energy and bio-based products such as clothing, construction materials, medical sutures, bedding, and plastics replacements.

#### The grains

There is no better example to illustrate new opportunities in agriculture than the ‘stuff’ of life, the cereal grains. Corn, couscous, wheat, barley, rice, oats, sorghum, wild rice, and others are significant sources of nutrients for the world’s population, constituting about half of the calories in the human diet. These species share 99 percent of their genes with all other members of the grass family, including species found on the prairie and in urban parks. Even though the family of ‘grass’ plants (cereal grains) seem different in their appearance, they all carry the same few genes in common that control seed size, grain placement, grain fill properties, heat stress, germination properties, resistance to certain chemicals, herbicides, and other important harvest traits. The genome of rice, for example, elucidates the genetic structure and function of the other cereal grains reported by Paterson, Bowers, Peterson, Estill, & Chapman in 2003 [14]. If you look at pictures of all the cereal grains you cannot help notice they have similar shape, flowering, tassels, seed placement, kernel shapes, and many other phenotypic styles. Agronomic breeding over centuries or eons of time have allow for expression of the most favorable traits to become expressed at a higher level. The genetic ancestors of corn bore little resemblance to current corn

we eat as humans or feed to our livestock. The original apples from Kazakastan were bitter and good for making cider, but little else. The transformation of the tomato throughout history even has an iPad app about the history of tomato plants showing the myriad of varieties through time.

A well-publicized example of genomics research that pursues the goal of reducing malnutrition in several regions of the world is the development of nutrient-loaded dietary staples such as rice. Ye et al. in 2000 developed the so-called golden rice by genetically introducing the biochemical pathway for provitamin A ( $\beta$ -carotene) into white rice (*Oryza sativa*) [15]. White rice cultivars do not contain this provitamin, thus the genetically enhanced golden rice presents an opportunity to address vitamin A-deficiency blindness, a significant public health challenge in at least 26 countries in the developing world, through consumption of a dietary staple.

Potential benefits from the new knowledge of genomics can result in new options to address food safety and public health concerns as well as nutrition. For example, researchers at the University of Minnesota are developing corn varieties for livestock feed that have the potential to help reduce the human illness caused by the highly virulent bacterium *Escherichia coli* O157:H7. Cattle naturally harbor *E. coli* O57:H7 in the gut. *E. coli* contamination of beef products such as hamburger – and subsequent product recalls and illness – can result from carcass contamination during the slaughter process or through manure contamination of drinking water sources. Recent analyses show that an experimental corn variety that has been genetically enhanced to include a gene that produces an *E. coli* O157:H7-inhibiting protein, colicin E7, reduces this human pathogenic bacterium in the animal’s digestive tract and it was reported by [16].

### Fruits and vegetables

Developments in genomics in agriculture extend well beyond the cereal grains to fruits and vegetables. Human interaction with the tomato – beginning with the ancient, diminutive, and bitter-tasting cherry tomato that gave rise to all subsequent varieties of tomatoes – continues to evolve. Despite the trendy popularity of heirloom tomatoes – purple, orange, green, maroon and pink – the healthiest tomato for a man worried about prostate cancer might be the bright red varieties. Why? Because red varieties generally contain larger quantities of lycopene, a natural constituent that has potent antioxidant, or cancer-inhibiting, properties. In an evaluation of studies that included 2,481 individuals with prostate cancer, Giovannucci, Rimm, Liu, Stampfer, & Willett in 2002 concluded that frequent consumption of tomato products is associated with a reduced risk of prostate cancer in men [17]. It is rare that we see well controlled published human clinical studies of diet-disease relationships like the one mentioned above. Most often there is a rumor mill operating at the local gathering place about what is the latest food trend. Hopefully this is the beginning of a new field of study.

Taking yet another angle on the tomato and its relationship to human health, researchers are assessing edible plants – potato, banana, and cereals in addition to tomato – as vehicles for the production and delivery of vaccines. Ongoing research by Mason, Warzecha, Mor, & Arntzen about 2002 has been exploring the application of genomics

and proteomics as a potential lower cost alternative to traditional fermentation processes in vaccine production [18]. In brief, vaccine proteins are introduced into the DNA of the edible plant, and are carried in fruits as the plant reproduces. As the fruit grows, the inserted gene codes for production of the immunogenic protein, which can accumulate within the plant material, fruit or starch, or wherever it was placed in the plant. As the fruits are eaten, the vaccine proteins cause the individual to produce antibodies and hopefully immunity to the immunogenic protein(s) carried within the food produce. If the immunogenic protein is chosen correctly the immune responses will protect against the disease. To date, a number of edible plant vaccines have been assessed in humans. These approaches might be useful for places in the world needing inexpensive vaccines where they might be locally grown and utilized. Luxuries such as refrigeration might not be needed if you can pick the next batch of potatoes for your annual vaccination program.

Cabbage, cauliflower, kale, Brussel sprouts, and broccoli are derived from the same genetic ancestor, *Brassicaoleracea*, but were developed as distinct vegetables through traditional breeding and selection. They differ in appearance, but they share in common some of the most potent chemicals to prevent cancer. Glucosinolates, natural anti-cancer molecules, are produced by these species that derive from a common ancestor. Verhoeven, Goldbohm, van Poppel, Verhagen, & van den Brandt (1996) summarize results from studies on a number of species and conclude that a high consumption of *Brassica* vegetables is associated with a decreased risk of certain lung, stomach, colon, and rectal cancers [19]. The chemicals in the plant made under direction of the genes of this plant have a profound human health effect.

### Nutrition and genomics

The *Brassica* family is a classic example of ways that the genes in your food interact with the genes in your body. Genes in your food doesn't mean that the genes in broccoli actually change your own genes. But it does mean there are dozens or hundreds of differing varieties of broccoli, each strain of the plant produces slightly differing quantities of key chemical ingredients. In some cases different strains of broccoli may produce even different chemicals. So it really does matter what genes are in your food. The same is true for all the plant foods we eat. We can chose to eat soy with more cancer fighting proteins, corn with different healthier oils, cotton with better insect control needing less noxious pesticides used in our environment, tomatoes with more lycopene supposedly aiding male health. Simply, the way it matters is how we chose to farm and do modern agriculture. Agriculture needs to be a full partner with human medicine. Instead of agriculture focusing on bushels per acre; it needs to be focused on which genes to put in or turn on in the plant. This author believes that a bushel of produce that can prevent cancer in people will fetch more cash at the elevator than one bushel that feeds livestock.

What you enjoy eating matters, obviously. About one fifth of the population doesn't like the taste of broccoli, and so self selectively exclude themselves from potential health benefits through this vegetable in the diet. This segment of the population belongs to a unique group of people called the supertasters. Taste research holds significant insights into diet-related chronic disease when the bottom line comes into focus: We eat because of the pleasures of taste, among

other reasons. Taste is genetic published [20]. More specifically, all five tastes appear to be genetic – sweet, sour, salty, bitter, and umami, (or meaty flavor). While seemingly unrelated but very intriguing, taste responses might shed light on an interesting finding about the way our facial nerves are connected to our taste nerves. Most people have similar physical reactions to each of the key taste groups. In some cases these reactions are similar, even across species lines such as nonhuman primates. It seems hard to believe, but when a bitter taste is placed on the tongue, facial muscles function in a similar manner in reaction to taste whether the taster is an adult, a newborn infant or a chimpanzee or orangutan – another example of conservation of genetics over evolutionary time [21]. This author wonders at the conservation of taste genes across millions of years of evolutionary divergence. A drop of sugar water on the tongue elicits a smile; lemon juice elicits a wrinkled-nosed squinch. Furthermore, solutions can be titrated to sort tasters into three general categories: nontasters, medium tasters, and supertasters. The same can be done across species. Genomics bridges evolutionary time.

Because of their genetics, supertasters find broccoli, horseradish, and coffee too bitter. These bitter tasting foods, especially *Brassicas*, may reduce the risk of cancer. Supertasters find rich foods too sweet and fatty. One in four people are nontasters and they relish rich foods. Nontasters reach for the Tabasco™, and enjoy strong flavors, including broccoli and alcohol. Some research suggests or has questioned whether higher rates of certain cancers might be seen in the percentage of the population that avoids vegetables since they obviously miss out on the potential benefits of sulforaphanes, glucosinolates, and other molecules in these foods. Another intriguing question is; can an individual become an alcoholic if they find alcohol too distasteful; even if they are genetically predisposed to alcoholism[22]?

### Wine and cholesterol

Another focus of research on alcohol suggests that drinking a glass of wine a day can reduce the risk of heart disease. But there is a genetic catch; beneficial effects are imparted to individuals who are “slow” converters of alcohol dehydrogenase (ADH). Specific to cholesterol levels, people who metabolically convert this enzyme at a medium or fast rate receive little or no benefit. However [23] demonstrated that individuals who are slow genetic converters of alcohol had higher levels of high-density lipoprotein (HDL), or good cholesterol, and thus a significantly reduced risk of heart attack.

### Hedges against hypertension

Research related to blood pressure regulation concluded that a set of genes found in some people increases the risk of hypertension. Interestingly, [24] published that same set of genes appears to confer better responses to dietary choices that lower blood pressure and prevent hypertension (SNP Rs699). The researchers based their work on individuals who participated in a multi-center study that included a daily diet rich in fruits, vegetables, and low-fat dairy products, the Dietary Approaches to Stop Hypertension (DASH) diet. Further understanding of the genetic factors that regulate blood pressure, and its responses to other factors, including diet, can lead to options for management and prevention.

In addition to the genetic profile in an individual that responds



to the metabolism of fruits and vegetables in the diet, fiber also enters into the hypertension configuration [25]. Investigated the possibility that variations in the angiotensinogen gene can determine the effects of fiber in the diet on blood pressure. In a 12-month study of 40 individuals, the researchers found that, in people with one variation of the gene (AGT codon 235), blood pressure decreased when subjects ate insoluble fiber (wheat-based) and increased when they ate soluble fiber (oats, barley and beans, for example). Subjects with yet another specific variation of the gene had the opposite response. Their blood pressures increased when they ate insoluble fiber and decreased with soluble fiber. Although the study focused on a small group of individuals, the outcomes suggest the potential to address hypertension by designing diets specific to an individual, given detailed knowledge of his or her genes.

### Soy and cancer

Increased consumer interest in soy products has expanded too many parts of the world. For example, one retrospective case-controlled study based in China published [26] concluded that women who consumed at least 2 grams a day of soy prior to their teen years may have reduced their risk of breast cancer by half. The Shu study also claimed the benefit waned as the women aged passed adolescence. Since the Shu study, several other very recent reports have indicated a benefit over a longer lifespan than just the teenage years. Three subsequent and more recent studies reported by [27-29]. Added support for a broader effect over long lifetime periods. Certainly it could be the case that specific genes regulate for resistance in certain individuals or that the effect might be dose dependent, age dependent or combined effect. In fact the response might change over time. Men might choose to consume soy at age 50 and older to protect against prostate cancer. Recent animal research by [30] suggests that genistein, the primary isoflavone in soy, can protect against mammary and prostate cancers by regulating specific sex steroid receptors.

Figure 1 provides an example of how 'Genes in your food', may provide choices for you to improve health outcomes.

### Eating for two

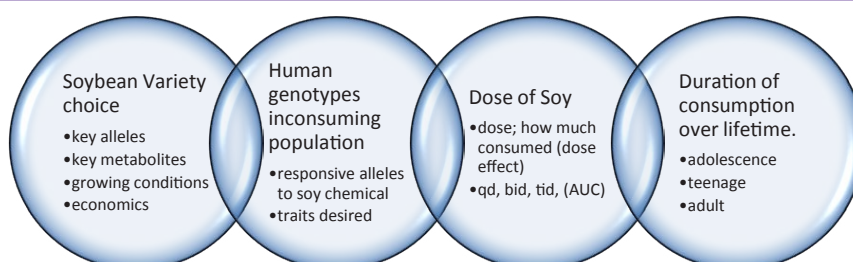
Food and drink can have profound and unique effects on an individual, depending on the interaction of genes. Further, the food (meaning actual food choices) consumed by pregnant mothers may have a significant effect on offspring. By that we mean the chemicals encoded by genes of specific varieties of crops. Research in mice with

gene traits where coat color is linked to healthfulness suggests that nutrient supplements (products of genes) in the mother's diet may increase health and lifespan in her offspring [31]. The key is the effect of a gene-encoded nutrient on the genetic mechanisms in developing embryos. This study, with visual impact of yellow and black mice, compares offspring from a mother that has received appropriate supplements of zinc, methionine, betaine, choline, folate, or vitamin B-12 with offspring from a mother who received no supplements. The parents are genetically identical. Appropriate supplements to the mother's diet result in a black mouse that is lean, healthy and longer-lived than its yellow siblings. Without supplementation, lab mouse mothers produce a yellow offspring that is large, obese, diabetic, susceptible to cancer, and short-lived. In this case, it appears that micronutrients interact with the individual's DNA to alter epigenetic expression; meaning changing the gene expression by methylation. Additional research suggests that supplementing the mother's diet with certain nutrients can also have unintended negative effects on the offspring as well [32,33]. Certainly, different nutrients can act at different intersections. Indeed, you are what you eat – and perhaps you are what your mother ate as well.

### Genomics and beyond

Today genomics, and all its branching -omics, have provided researchers with the ability to understand the elegant and complex steps of the genetic tango between the genes in the body and the genes (food choices) in the foods we eat. Clearly 'genes in your food' really means the specific chemicals in your food since we don't benefit from eating genes per se. But remember not all crops of the same species produce the same chemicals. Some varieties are more healthful than others, but may not be as commercially profitable as some farmers wish, at least not yet. Nutrigenomics provides a molecular understanding of how common dietary chemicals, or nutrients, affect health by altering function (not sequence) of the DNA by changing the 3-dimensional conformational structure of an individual's genetic makeup, called 'epigenetics'. To analyze how food interacts with the protein in the body, proteomics is employed. Metabolomics elucidates the process whereby consumption of sugar influences glucose metabolism, for example. Beyond these, toxicogenomics studies how genes respond to environmental stressors or toxicants. Pharmacogenomics blends pharmacology with genomics capabilities with the future goal of developing drugs customized for certain populations.

This knowledge about genetic information that is shared across



**Figure 1:** Example of 'Genes in your food'. How choosing the right genes in your food may improve interactions with your own personal genes. Visualization of an example of how choosing the right chemicals in your diet might work. Picking varieties known to contain the right metabolites and known to provide beneficial effects from controlled studies. Determine which patients (alleles or associations) might be responsive to the particular nutrient or dietary chemical; then determine by classical pharmacology how much to dose, how often and for what duration.

species and across time helps to bring researchers and disciplines together to achieve interdisciplinary synergies. The impact is being felt in agricultural breeding and the production of food plants and animals, in horticulture, food processing, nutrition, molecular and cellular biology, clinical medicine, human physiology, epidemiology, pharmacology, and many other traditional areas of academic research. Research on the interaction of genes in the human body and genes in the food we eat is a massive and important area of scientific pursuit. The report *Foods for Health* [34], based on a conference at the University of Minnesota in May 2002, provides a collection of papers on the integration of agriculture, medicine, food, and health (National Agricultural Biotechnology Council, 2002). The meeting was a national call to action for universities, the federal government, and the public to better support the ability of researchers to come together in an intentionally interdisciplinary way to address health, moving beyond treatment to prevention.

### Policies and politics

Importantly, other areas of research are essential for progress toward reducing the incidence of diet-related chronic disease and its devastation of families and of the national healthcare budget. Lifestyle choices – including exercise and physical fitness; consumer preferences for food, food products, meal preparation and dining out; and the interactions of behaviors such as tobacco and alcohol use – are significant factors that are being researched by sociologists, psychologists, economists, statisticians, historians, and public policy researchers.

The drivers that have been in place over the decades and have contributed to this state of ill-health cannot be forgotten. They include agricultural policies and subsidies, global trade agreements, health care coverage and policies, federal research institutions and funding streams, availability of professional work force, and institutional relationships (across federal and state entities, academia, foundations, the private sector and not-for-profit organizations, and citizen and community groups). The National Research Council of the National Academies brought together representatives of the U.S. Department of Agriculture and the National Institutes of Health, along with other public and private institutions in June 2003, “in the hope of sparking exploration of a more efficient and effective system for conducting food and health research geared to improving, maintaining, and promoting health” [35]. Such national policy focus on the integration of knowledge for food and health is an essential balance to the awesome power of the interactions among the genes in our bodies and the genes in our food.

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