KNOWLEDGE AND PERCEPTION OF COLLEGE STUDENTS TOWARD GENETIC TESTING FOR PERSONALIZED NUTRITION CARE

A dissertation submitted to the Kent State University College of Education, Health, and Human Services in partial fulfillment of the requirements for the degree of Master of Science

By

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Nutrigenomics is a rapidly developing field of study involving the relationship between genetics and nutrition. Multiple companies are now offering personalized dietary advice based on the results of genetic testing. College students, who are educated and more familiar with new technology may provide valuable information about perceptions toward nutrigenomic technology while it is still in its early stages of development. The purpose of this study was to examine the knowledge and perception of college students toward genetic testing for personalized nutrition. Participants in this study were college students from Kent State University who completed an online survey administered through Qualtrics. The survey assessed perception toward nutrigenomics along with basic genetics knowledge. Analysis of the data revealed a general lack of genetics knowledge among college students. In addition, only 25% of participants had ever heard or read about nutrigenomic testing. The overall perception toward these developments was more positive than negative. There were significant differences in genetics knowledge and perception of nutrigenomics among various class ranks and majors. In addition, findings indicate a significant relationship between participation in college level nutrition and/or genetics courses, higher genetics knowledge and more positive
perceptions toward nutrigenomics. Individuals who scored higher on the genetics knowledge assessment also displayed a more positive perception toward nutrigenomics. More research is needed to understand how college students perceive nutrigenomics and what factors affect their attitude toward these scientific developments. Future studies with a valid and reliable questionnaire are needed to confirm the findings of this study.
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CHAPTER I
INTRODUCTION

Diet related chronic diseases including obesity, type 2 diabetes, cardiovascular disease, and some types of cancer are the most common and deadly health concerns worldwide, posing a significant financial burden on society of more than $659 billion in medical expenses and lost productivity per year (Dietz, Dougals, & Brownson, 2016; World Health Organization, 2009;). It is known that nutrition is a major determinant of these diseases, which often arise from disrupted biological networks involving multiple genetic polymorphisms, which lead to a state of metabolic stress (metabolic syndrome) (Afman & Muller, 2006). For this reason, dietary interventions to prevent chronic diseases are complex, requiring knowledge of how a mixture of nutrients (diet) will interact to change biological functions (Mutch, Wahli & Williamson, 2005).

To better understand the interaction between genes and diet, the field of nutrition has begun to capitalize on new technologies and supporting analytical software used to unravel the relationship between nutritional components, genetic polymorphisms, and the biological system as a whole (Mutch, et al., 2005). Nutrigenetics and nutrigenomics are two scientific fields using distinct approaches to decode the relationship between genes and diet with a common goal of optimizing health (Mutch et al., 2005). While nutrigenetics and nutrigenomics are still in their infancy with respect to targeted disease prediction, prevention, and treatment, advances in these disciplines will allow for personalization of nutrition, the ultimate paradigm of responsible medical nutrition.
therapy (Fenech et al., 2011; Merched & Chan, 2013). However, in order to use genetic blueprints (genotypes) for dietary prevention of disease, the mechanisms driving the connection between diet and the outward expression of our genes (phenotype) must be identified first. An important aim of nutrigenomics research is to study genome-wide influences of diet, specifically focusing on the role of metabolic stress in the creation of metabolic syndrome, which includes inflammation, insulin resistance, and diabetes (Afman, & Muller, 2006).

Evidence supports the notion that early diet and lifestyle modification reduce the risk of developing diet related chronic diseases, therefore prolonging and enhancing healthy years of life (Nelson, Story, Larson, Neumark-Sztainer & Lytle, 2008). While it is becoming increasingly accepted that not all people respond to diet equally, research in the developing field of nutrigenomics is lacking. Causal relationships between bioactive dietary components and prevention or disease outcomes can only be assessed by long-term intervention trials, which are time-consuming and costly (Kaput, 2008). While expectations are high, ethical challenges and unanswered questions have slowed progress for personalized applications of nutrigenomics in health care.

Adequate knowledge regarding the genetic component of diseases, as well as perceived benefits and risks are major determinants for successful implementation of nutrigenomic technology (AFFECT ; Capelli et al., 1999; Gudde, 2009). Since nutrigenomics is complex, involving advanced technological developments, many individuals, including healthcare professionals, do not yet feel comfortable applying this science (San-Cristobal, Milagro & Martinez, 2013). Given the unfamiliar and
controversial nature of nutrigenomics, perhaps the best place to start with introducing these concepts is in a population of younger, more educated, and more influential individuals. College students make up a diverse and monumental population whose current choices, viewpoints, and behaviors have the potential to impact the future health, productivity, and financial status of our nation. A more clear understanding of current perceptions of college students toward nutrigenomics may provide a unique opportunity to examine public opinion of this technology across a broad range of cultural and socioeconomic backgrounds, therefore providing insight into ways for optimizing implementation of this technology while it is still in early stages of development.

Statement of the Problem

Experts believe there is great potential for nutrigenomics to reduce the onset and impact of complex diseases including non-communicable conditions that currently account for nearly two-thirds of the world’s deaths (Afman & Muller, 2006; Bauer, Briss, Goodman, & Bowman, 2014). Many scientists predict that new developments will soon be unfolding, promoting the health and well-being of the general public, genetic subgroups, and individuals based on their genetic make-up (Afman & Muller, 2006; Fenech et al., 2011). However, public support of genomic technology, especially by emerging adults, will impact successful implementation of personalized nutrition care and it remains uncertain whether enough groundwork has been laid to warrant the use of nutrigenomics in nutrition care (Gudde, 2009).

Many barriers must be addressed for nutrigenomics to make a positive impact on global health. For example, most Americans are unaware that nutrigenomic technologies
exist (Goddard, Mooere, Ottman, Szegda, Bradley & Khoury, 2007), even though several companies now offer these tests (San-Cristobal et al., 2013). Another concern is the complexity of the field, which requires substantial knowledge in nutrition, genetics, and biochemistry, making it difficult, even for highly educated individuals to understand (Görman, Mathers, Grimaldi, Ahlgren & Nordstrom 2013; Lapham, Kozma, Weiss, Benkendork & Wilson, 2000). In addition, there are concerns whether the goal of matching specific foods to individual genotypes is within reach, or whether tests will be misleading to the public. Some professionals believe that claims cannot yet be scientifically proven and raise considerable ethical dilemmas including concerns about cost, privacy, and misuse of genetic information (Görman et al., 2013; Pavlidis, Patrinos & Katsila, 2015).

Considering the depth of knowledge necessary to comprehend nutrigenomic processes, perhaps college students, who are more educated (Ryan & Bauman, 2016), more exposed to new scientific and technological advances (Smith, Rainie & Zickuhr, 2011), and at a more influential stage in their lives (Nelson et al., 2008), might find these concepts to be especially appealing. As the largest generation is U.S. history (U.S. Census Bureau), college students are also a highly diverse population, who represent the future of the U.S. and, therefore, could play a significant role in the development of this field. As the youngest population of educated adults, perhaps the college population is a good starting point for assessing baseline knowledge and perception toward nutrigenomics.
Post-secondary institutions, which serve a population of approximately 17 million college students, accounting for nearly half (47%) of Americans between the ages of 17-24 years old (U.S. Census Bueau), may be well suited for addressing unanswered questions related to nutrigenomics. However, few studies have examined how college students perceive the possibility of targeted recommendations based on their personal genetic make-up (Angelakis, 2004; Horne, Madill & Connor, 2016). Thus, examining the knowledge and attitudes of college students could help researchers and health care providers develop strategies, realize future opportunities, and overcome potential challenges in this rapidly evolving scientific field.

**Purpose Statement**

The purpose of this study was to examine the knowledge and perception of college students towards genetic testing for personalized nutrition care.

**Hypotheses**

Hypothesis 1: There is a lack of general genetics knowledge in college students.

Hypothesis 2: Genetic testing for personalized nutrition is perceived to have benefits that out-weigh risks in college students.

Hypothesis 3: There is a difference between males and females in perception of genetic testing for personalized nutrition.

**Operational Definitions**

**Awareness:** having knowledge that something exists or being familiar with a subject at the present time based on information or experience; Nutrigenomics awareness measured through survey question
Knowledge: facts, information, and skills acquired through experience or education; genetics knowledge measured through a series of survey questions

Perception: a way or regarding, understanding, or interpreting something; a mental impression; perceptions toward nutrigenomics measured through a series of survey questions

College Students: students enrolled in a college or university of higher education who are over the age of 18 years old, including both undergraduate and graduate students.

Genetic Testing: the sequencing of human DNA in order to discover genetic differences, abnormalities, or mutations that may provide information about an individual’s susceptibility toward developing a certain disease

Nutrigenomic Test: a genetic test that examines an individual’s genome, allowing for personalization of their nutrition care

Personalized Nutrition Care: the practice of adapting diet to meet specific nutritional needs or prevent chronic disease in individuals or genetic subgroups based on the results of genetic testing

Nutrigenomics: The scientific study of the molecular interaction between genes and nutrients

Nutrigenetics: The scientific study of the relationships among genes, diet, and health outcome

Risk: Exposure to the chance of injury, loss, or undesirable outcome
CHAPTER II
REVIEW OF LITERATURE
Diet Related Chronic Diseases

Throughout the 20th century, nutritional science has focused on identifying vitamins, minerals, and other essential nutrients to prevent life-threatening deficiencies (Silveira, Martínez-Piñeiro, & Carraro, 2007). Over time the health risks faced by populations have shifted from infectious disease and undernutrition to risks for chronic disease and overnutrition (World Health Organization, 2009). Chronic diseases, such as cardiovascular disease, cancer, obesity, and diabetes, are characterized by their slow progression, long duration, and negative health outcomes. Currently, they make up the most common, deadly and costly of all health problems worldwide (World Health Organization, 2009; Baur, Briss, Goodman, & Bowman, 2014).

Globally, chronic diseases account for two thirds of all deaths (Baur et al., 2014). Similarly, in the United States, chronic diseases are the main cause of poor health, premature death, and disability (Baur et al., 2014). More than half of Americans suffer from at least one chronic diseases (Devol et al., 2007), and in 2014, approximately one in four Americans were diagnosed with multiple chronic condition (Ward & Black, 2016). Each year, millions of Americans are diagnosed with a chronic disease, while even more die from their condition (Devol et al., 2007). Furthermore, chronic diseases account for most health care expenditures in the United States (Baur et al., 2014). Currently, it is
estimated that chronic diseases are costing the economy $3 trillion per year and this number is expected to double to $6 trillion by 2050 (Devol et al., 2007).

High blood pressure, high blood glucose, physical inactivity, overweight and obesity, and tobacco use are now considered the five leading global risks for mortality (World Health Organization, 2009; Mokdad, Marks, Stroup & Gerberding, 2004) and are directly linked to an increased risk of chronic disease. Hyperlipidemia, and alcohol consumption are also considered prevalent risk factors (Mokdad et al., 2004). The current rate of chronic disease prevalence along with its predicted growth over the next years argue the need for establishment of a more preventative orientation in the U.S. healthcare and public health systems (Mokdad et al., 2004).

Obesity is associated with multiple chronic conditions including type 2 diabetes, heart disease, metabolic syndrome, hypertension, stroke, and certain forms of cancer, which is why there is a tendency for rates of these conditions to increase simultaneously with obesity prevalence (CDC, 2003; Flynn et al., 2006; Kopelman, 2007). Risks of Coronary Artery Disease (CAD) ischemic stroke, type 2 diabetes, and cancer all grow steadily with increasing body mass index and globally, the burdens of 44% of diabetes, 23% of ischemic heart disease, and 7-41% of certain cancers can be attributed to overweight or obesity (World Health Organization, 2009). A chronic low-grade inflammatory state may play a direct role in early stages and progression of these diseases and could be a determinant of the pathological impact of excess adiposity (Minihane et al., 2015; Harford & Reynolds, 2011).
**Overweight & Obesity**

Obesity, or excess adipose tissue is a preventable chronic condition that results from a combination of environmental and genetic factors. Over an extended period of time, a high caloric diet and decreased levels of physical activity are significant contributors to the state of energy imbalance, which leads to overweight and obesity (Rodgers, Tschöp & Wilding, 2012). Typically, obesity is diagnosed through body mass index (BMI) greater than 30. BMI is the ratio between weight (in kilograms) and height (in meters squared), and can be used along with body composition, waist circumference, and blood tests to quantify adiposity and diagnose metabolic syndrome or comorbidities associated with excess body weight. Overweight is defined as BMI between 25 and 29.9. Obesity is considered to be severe when BMI is greater than 40 (Youdim, 2016). However, it should be noted that BMI cannot make the distinction between an elevated weights due to high levels of adipose tissue vs. lean body mass, and generally, it is excess adipose tissue, opposed to lean tissue, that is frequently associated with metabolic abnormalities and a higher risk of developing chronic diseases (Bastien, Poirier, Lemieux, & Després, 2014).

**Global and national prevalence.** The prevalence of obesity has increased dramatically worldwide over the last decades, reaching epidemic proportions. It was estimated that 1.5 billion people were overweight or obese in 2015 (World Health Organization, 2009; GHO, 2013), and these conditions are now more deadly than under-nutrition in 65% of the world’s population. Globalization has now pushed obesity
beyond the borders of the United States, with rates expected to increase in all countries (World Health Organization, 2009) in the next 10 years.

Prevalence of overweight and obesity is particularly high in the United States. According to NHANES data from 2011-2014, about 40% of men and 30% of women are overweight in the United States and 36% of American adults are obese (GHO, 2013; Ogden, Carroll, Fryar, & Flegal, 2015; Yang & Colditz, 2015). Even more alarming is the associated epidemic of obesity in young adults, adolescents, and children, affecting about 17% of these populations (McPherson, Marsh & Brown, 2007; Flyn et al., 2006; Ogden et al., 2015).

The distribution of the population’s weight status has increased in the past 20 years and is expected to continue to rise in almost all countries (Yang & Colditz, 2015; World Health Organization, 2009). However in the U.S., data from the last decade (199-2010), suggests that prevalence of obesity may have plateaued among Americans (Flegal, Carroll, Ogden, & Curtin; 2010). Between 2003-2004 and 2011-2014, no significant change in prevalence has been seen among youth, with estimates plateauing at about 17% and the observed change in adult prevalence between 2011 and 2014 was not significant, with estimates increasing from 34.9% to 37.7% (Ogden et al., 2015). More concerning, however, is the disproportionately higher increase in morbid or class III obesity (BMI ≥ 40), which now totals more than 3% of the US population (Bastien et al., 2014; Lavie et al., 2016;). With some data indicating that the epidemic of obesity may have begun to recede in some populations, high rates of obesity and severe obesity continue to threaten
the health and future of many American children and adults (Ogden et al. 2015; Robbins, Mallya, Polansky & Schwartz, 2015).

**Contributing factors.** There are multiple risk factors associated with obesity including both environmental and genetic influences (Frayling et al., 2007; Mahan & Escott-Stump, 2008). Many of these risk factors are modifiable, which makes this condition both curable and preventable (Bauer et al., 2014; Mahan & Escott Stump, 2008).

**Environmental factors.** There are numerous environmental factors involved with the development and propensity of weight gain and obesity. Many of these factors are secondary to the commercialization of the food and marketing techniques that promote consumption of energy dense foods.

**Diet.** The biology of food intake is complex, involving many psychological and physiological factors (Torres & Nowson, 2007). Diets high in calories, especially those coming from high fat, high sugar, and low fiber foods are shown to promote weight gain and obesity over time (Kopelman, 2007). Overconsumption of energy from carbohydrates, fats, or proteins can all lead to excess storage of adipose tissue and obesity (Harper & Murray, 2008; Mahan & Escott-Strump, 2008).

Animal and human studies have shown that dietary composition may play a significant role in the development of obesity with evidence linking high fat diets to weight gain (Salmon & Flatt, 1984). More recent investigations indicate that the type of dietary fat may be more significant than total fat intake in development of weight gain and hepatic steatosis (De Wit et al., 2012). Several investigations suggest that saturated
fat may significantly worsen insulin resistance and metabolic abnormalities, while monounsaturated and polyunsaturated fatty acids may improve these conditions (Due et al., 2008; Riccardi, Giacco & Rivellese, 2004). This is supported by another study, where rats fed saturated (animal) fats generated obesity and insulin resistance, and those fed fish oil did not (Buettner, Scholmerich & Bollheimer, 2007). In humans, total energy intake is shown to increase in individuals with relatively higher fat diets (Hill & Peters, 1998) but this increased energy intake may be due to the energy density of the diet rather than the fat content alone (Stubbs, Harbron, Murgatroyd, & Prentise, 1995). Overall, it appears that energy dense diets exceeding the recommended intake levels for calories from solid fats, added sugars, refined grains, sodium, and saturated fat may contribute to weight gain and development of obesity (Aubrey, 2011; Dong, Bilger, van Dam, & Finkelstein, 2015; Austin, Ogden & Hill, 2011).

Environmental influences such as larger portion sizes at restaurants, access to low-cost fast food, and cultural traditions encourage overconsumption of energy and contribute to an obesogenic environment, which promotes weight gain and its associated complications. Developments in food processing and changes to the food supply in efforts to reduce production costs, extend shelf life, and increase palatability has adversely impacted dietary composition patterns in the U.S. and worldwide (Austin, Ogden, & Hill 2011; Drewnoski, 2004; Zobel, Hansen, Rossing, von Scholten, 2016). Technological advances in food production and profound improvements in the distribution systems supporting the food supply have made ultra-processed foods rich in sugars and saturated fats and lower in fiber increasingly available and more affordable to
the world’s population (Crino, Sacks, Vandevijvere, Swinburn & Neal, 2015; Drewnowski, 2004; Kac & Perez-Escamilla, 2013; Kearney, 2010; Popkin & Adair, 2012). In addition, globalization has opened borders and allowed for a “nutrition transition,” involving the adoption of diets high in energy, fats, sugar, and salt among low and middle-income nations (World Health Organization, 2005). For this reason, obesogenic environments have expanded beyond the borders of the United States creating a global epidemic (Caballero, 2007).

Overconsumption of energy through eating too much or too often may both contribute to excess weight gain or obesity risk (Syrad et al., 2016). In the United States, there is a trend toward larger portion sizes at restaurants, which market their highly palatable foods as being more value for a lower cost (Hill & Peters, 1998). According to NHANES and National Health Interview Survey (NHIS) data, between 1987 and 1992, 36% of the population reported, eating three or more commercially prepared meals per week. Between 1999 and 2000, this proportion increased to 41%, indicating increased exposure to commercially prepared meals, and likely higher caloric intakes (Kant & Graubard, 2004). Between 2003 and 2010, American adults’ daily consumption of fast food showed a modest decline from 13% to 10% of total calories coming from fast food (Fryer & Ervin, 2013). However, global estimates indicate that the average energy supply has increased from 2,250 calories per person per day in 1961, to 2,750 calories per person per day in 2007 (Alexandratos & Bruinsma, 2012). Eating at fast food and full-service restaurants is associated with a net increase in total daily intake of energy, total fat, saturated fat, cholesterol, and sodium. Fast food consumption is also associated with
decreased intakes of fiber, vitamin A, vitamin C, vitamin D, vitamin K, copper, and magnesium (An, 2016).

Since the passage of the Patient Protection and Affordable Care Act in 2010, all restaurants and food establishments with more than 20 locations are mandated to display calorie information on menus (Gable, 2011). Since then, it appears that menu labeling has had little impact on consumer purchases (Kiszko, Martinez, Abrams & Abel, 2014), but may encourage restaurants to develop choices with fewer calories (Bleich, Wolfson & Jarlenski, 2016). A recent meta-analysis investigating nutrient composition in menu offerings in fast food restaurants in the U.S. reported a mean caloric content of 456 calories for all food items, with no significant decrease in calories from sugars or saturated fat (Jarlenski, Wolfson & Bleich, 2016).

Recent data indicates that Americans have decreased their percentage of energy consumed from fat from 36.6% to 33.7% between 1971 and 2006. However, this decrease in dietary fat resulted in an increase in carbohydrate intake, and ultimately an increase in total energy intake (Austin, Ogden & Hill, 2011). During this period Americans have increased the percentage of total energy from carbohydrates from 44% to 48.7%. Simultaneously, there has been an increase in carbonated soft drink consumption, raising the concern for additional carbohydrates in the form of sucrose or high-fructose corn syrup (Austin et al., 2011; Nielsen & Popkin, 2004).

Sedentary lifestyle. Physical activity recommendations of 30 minutes per day, five days per week are associated with improved fitness and protection against development of obesity and associated chronic diseases (Thompson et al., 2003).
However, recent research has shown that 45-60 minutes per day is even more effective at reaching and maintaining healthy body weight (Kesaniemi, Danforth, Jenson, Kopelman, Lefebvre, & Reeder, 2001; Thompson et al., 2003). In addition to aerobic exercise, individuals are likely to achieve additional benefits from engaging in resistance training and flexibility exercises at least twice per week, which promotes maintenance of lean body mass, endurance, preservation of function, and improved quality of life (Blair, LaMonte, Nichaman, 2004).

Along with a transfer of dietary habits, as nations develop economically, many individuals begin working in employment situations that limit physical activity. Urbanization has created an environment involving exposure to new products, new technology, and the marketing of unhealthy food options. Through this nutrition transition, it is common to see the development of increased levels of obesity and chronic disease (World Health Organization, 2005). Overall total physical activity in the U.S. population is decreasing, and according to Behavioral Risk Factor Surveillance System (BRFSS) data, only 26.2% of US adults are engaging in recommended levels of physical activity (Brownson, Boehmer, Luke, 2005).

Reasons for this total decline in physical activity include trends of decreased levels of work-related activity, decreased transportation activity, and decreased activity at home. Agricultural employment, typically associated with high activity levels, has declined from 12.5% to 2% between 1950 and 2000 (Brownson et al., 2005). Between these times, there has also been an overall shift in the number of people employed in other high activity jobs, to those associated with less physical activity with about twice as
many persons employed in low activity jobs and it is estimated that daily occupation-energy expenditure has decreased by more than 100 calories per day over the past 50 years (Church et al., 2011). Physical activity is also limited by travel time and increased reliance on motor vehicles. The typical person travels 40 mile per day in a personal vehicle and 18.3% of households own three or more vehicles (Brownson et al., 2005).

In schools, the percentage of students attending daily physical education class declined between 1999 and 2001 from 41.6% to 32.2%, suggesting the average amount physical activity during school time is probably decreasing. Inactivity among the youth is also shown to increase by grade level from 24.3% in ninth grade to 38.9% in twelfth grade (Brownson et al., 2005). Lower physical activity levels appear to be compounded by an increase in sedentary activities including watching television, playing video games, and using the computer (Brownson et al., 2005). Studies indicate that American children spend an average of three hours per day using television, computers, or other electronic devices and up to a total of nine hours per day doing sedentary activities (Stamatakis et al., 2013; Tremblay et al., 2011). Although young people report a strong desire to be physically active, many are constrained by external factors including school policy, parental rules in relation to safety, and physical environmental factors (Dollman, Norton & Norton, 2005).

Objective studies indicate that sedentary lifestyle is prominent among adults as well, with the typical adult spending 50-60% of their days in sedentary activities (Barnes et al., 2012; Wilmot et al., 2012). Higher levels of sedentary activity in adults are associated with a 112% increase in RR of diabetes, 147% increase in risk for
cardiovascular disease, 90% increase in risk for cardiovascular mortality, and 49% increase in all-cause mortality risk (Wilmot et al., 2012). Low levels of physical activity result in low daily energy requirements and can lead to obesity if food intake is not limited appropriately. In the United States, low levels of physical activity are often compounded by high caloric intake, creating a high propensity toward obesity.

**Stress.** In a national survey conducted by the American Psychological Association, 50% of people reported feeling more stressed than they did five years ago and 43% reported using food to cope with stress (American Psychosocial Association, 2007). Other non-human animal studies have demonstrated relationships between stress and increased intake of highly palatable foods, such as those high in sugar and fat (Dallman, Pecoraro, & la Fleur, 2005; Groesz et al., 2012; Levine & Morley, 1981; Wilson et al., 2008). Experiencing a drive to eat without a true need for energy is common and may be related to the reactive cortisol response associated with facing more daily stressors (Newman, O’Conner & Conner, 2007). The central control stations of stress response are located in the hypothalamus and brain stem and involve activation of the hypothalamus–pituitary adrenal (HPA) axis and sympathetic-adrenomedullary (SAM) system. Stress produces a response involving repeated activation of the HPA axis by corticotropin releasing hormone (CRP), which is followed by an elevation in cortisol and depends on intensity, duration, and type of stressor (Adam & Epel, 2007). This leads to an activation of adipose tissue lipoprotein lipase and then accumulation of adipose tissue in the abdominal region (Torres & Nowson, 2007). It is currently unclear whether excess glucocorticoid exposure drives obesity or whether obesity itself is responsible for
higher cortisol levels. While these mechanisms are only starting to be investigated in humans, it appears that obesity may be exacerbated by chronic stress and related unsuccessful attempts at food restriction (Adam & Epel, 2007; American Psychological Association, 2007; O'Connor, Jones, Conner, McMillan, & Ferguson, 2008).

**Genetic factors.** Research has shown that obesity also has hormonal and neural factors that are determined genetically. Short and long term signals are involved in satiety and feeding activity and small changes in the expression of these genes can lead to abnormal weight regulation (Mahan & Escott-Stump, 2008). The number and size of fat cells, the distribution of body fat, and the resting metabolic rate (RMR) are also linked to genetics (Mahan & Escott-Stump, 2008). There are numerous gene variants that may contribute a small genetic burden (Shuldiner and Sabra, 2001). Some of these include the ob gene, the GAD2 gene, the FTO gene, and the B3-adrenoreceptor gene.

The OB gene is involved in the production of Leptin, a hormone that promotes satiety, and mutations in the OB gene in mice results in obesity (Mahan & Escott-Stump, 2008). The B-3 andrenoreceptor gene is found in adipose tissue and helps to regulate fat oxidation and control RMR in humans. The FTO gene is linked to a predisposition to the development of diabetes by its effects on body mass (Frayling et al., 2007).

**Gut microbiota.** The human intestine normally hosts about $10^{14}$ organisms of 1000 different species, which are composed by symbiotic innocuous bacteria and potential pathogens, called pathobionts (Valsecchi, Tagliacarne, & Castellazi, 2016). Several studies have indicated that gut microbiota may be related to several conditions...
including obesity, diabetes, fatty liver disease, atherosclerosis, allergies, gastrointestinal diseases, autoimmune diseases, and cancer (Valsecchi, Tagliacarne & Castellazi, 2016). Several studies have indicated that an imbalance in microbiota may lead to the development of obesity in several ways (De wit et al., 2012; Delzeene, Neyrinck, Backhed & Cani, 2011; Tremaroli & Bäckhed, 2012; Valsecchi, Tagliacarne & Castellazi, 2016). First, a suppression of fasting-induced adipose factor may be induced by gut microbiota, leading to enhanced LPL activity and increased storage of fatty acids in adipocytes (Delzeene et al., 2011; Valsecchi et al., 2016). Second, imbalance of microbiota homeostasis leads to increased intestinal permeability, an inflammatory response, and insulin resistance, all commonly associated with obesity (Delzeene et al., 2011; Valsecchi et al., 2016). Third, gut microflora may contribute to obesity by controlling lipid and glucose metabolism through bile acid pools and modulation of farnesoid X receptor (FXR) and TGR5 signaling (Tremaroli & Bäckhed, 2012). Fourth, alterations to the composition of gut microflora may influence metabolic processes through release of hormones, including those that control satiety (Tremaroli & Bäckhed, 2012; Valsecchi et al., 2016).

**Consequences of obesity.** Obesity and its associated comorbidities have significant negative implications for individuals and populations affected and being obese increases an individual’s risk for many health conditions because it affects virtually every organ system in the body (Youdim, 2016). Obesity significantly increases a person’s risk of developing metabolic syndrome, which is a combination of conditions including elevated blood glucose, increased blood pressure, body fat around the waist, and high
cholesterol or triglycerides (Flagel, Graubard, Williamson & Gail, 2008). Major comorbidities of obesity including type 2 diabetes, cardiovascular disease (hypertension and myocardial infarction), certain cancers, fatty liver disease, respiratory conditions, gallstones, osteoarthritis, sleep apnea, GERD, degenerative joint disease, anemia, depression, anxiety, and reproductive problems (Flagel et al., 2008; Samanic et al., 2004). Youdim, 2016). These conditions are more prevalent and tend to worsen as the degree of obesity increases (American Obesity Association, 2012).

Long-term research from the Nurse’s Health Studies (NHS) has shown that being overweight or obese greatly increase a woman’s risk for type 2 diabetes, cardiovascular disease, certain types of cancer, and premature death. In fact, across the initial eight years of NHS, women with a high/normal BMI (23-23.9) were 3.6 or more times more likely to develop diabetes and weight gain after the age of 18 was also a strong risk factor. Similarly in the NHS, gaining 10 or more kilograms from age 18, versus maintaining weight within 3 kilograms, equated to more than a 60% higher risk of cardiovascular disease. With respect to cancer, increases in BMI over the life course significantly elevated the risk of postmenopausal breast cancer, large adenomas in the colon, endometrial cancer, and cancers of the kidney and pancreas (Hruby et al., 2016).

While some researchers have suggested that excess weight is protective against mortality, this “obesity paradox” may be a result of preclinical conditions that lead to weight loss before death. When these methodological issues are appropriately accounted for, obesity is directly linked with morality. In fact, BMI in the overweight or obese range is associated with premature death among generally healthy individuals at baseline
Similarly, moderately high BMI, increased adiposity, and low physical activity during adolescence are associated with premature death in young to middle aged women (van Dam et al., 2006). Individuals with morbid obesity have death rates that are 50-60% higher than individuals with less severe obesity (American Obesity Association, 2016).

Besides an increased risk for chronic disease and death, obesity also has emotional dimensions and can result in psychological distress. It is estimated that about 10% of the population suffers from some form of depression (Stunkard, Faith & Allison, 2003) and there is evidence of a relationship between severity of obesity and depression, with more obese individuals suffering from major depression more often (Luppino et al., 2010; Sunkard, Faith & Allison, 2003). NHS studies have examined weight status and changes in relation to quality of life, using the Short Form 36 Health Survey, which assess 8 domains of physical and emotional health. Findings indicate that weight gain in women was associated with decreased physical function and vitality, increased pain levels, and diminished chances of successful aging (Fine et al. 1999; Sun, Townsend, Okereke, Franco, Hu & Grodstein, 2009).

**Prevention and treatment of obesity.** The main prevention or treatment method for obesity includes changes in lifestyle, including dietary interventions, increased physical activity, and behavioral modification (Mahan & Escott-Stump, 2008). Unfortunately, lifestyle changes do not always produce substantial sustainable weight loss in individuals with severe obesity, which may be a result of consuming more calories than intended, overestimating of physical activity levels, or the decreased
metabolic rate associated with restricted-energy diets (Goodpaster et al., 2010; Mahan & Escott-Stump, 2008). Those who are unsuccessful with lifestyle changes alone may need to try special diets such as very low calorie or very low carbohydrate diets under medical doctors’ supervision for rapid weight loss (Mahan & Escott-Stump, 2008). Reasons for weight regain usually include a regression to old eating behaviors, which resulted in obesity in the first place. Long-term weight loss success requires life-long modification of diet and lifestyle habits, with a combination of diet and exercise showing most promising long-term results (Wu, Gao, Chen & van Dam, 2009). In addition, weight loss medications often are prescribed by doctors to promote weight loss by decreasing appetite or absorption of fats and/or by increasing energy expenditure (Rodgers et al., 2012).

Other individuals may choose to undergo a surgical intervention, such as a Roux-en-Y Gastric Bypass, which are shown to be effective in terms of weight loss, life extension, and reduction of comorbidities (Kral & Näslund 2007; Wickremesekera, Miller, Naotunne, Knowles & Stubbs, 2005). After weight loss surgery, patients typically have a 1-year time span where appetite and the biological drive to eat are reduced. During this time, it is critical for patients to make changes to their eating behaviors and establish eating habits that will allow them to maintain a healthy weight over an extended time. After about one year, many of the internal cravings for high calorie foods return and patients need to apply newly learned behaviors. Each case should be assessed on an individual basis to find the most effective method for long-term
success. It is known that losing as little as 5 to 10% of excess body weight can help reduce the risk or severity of comorbidities associated with obesity (Youdim, 2016).

**Type 2 Diabetes Mellitus**

Type 2 diabetes mellitus (T2DM) is a metabolic disorder of multiple etiologies characterized by chronically elevated blood glucose levels. T2DM is diagnosed through tests that measure the amount of glucose in the blood. Some individuals with diabetes go without symptoms and are unaware of their condition until they are screened and tested appropriately. It is estimated that 8.1 million people (27.7% of people with diabetes) are undiagnosed (National Institute of Diabetes and Digestive and Kidney Diseases, 2016; Centers for Disease Control and Prevention, 2014). Serum blood glucose, hemoglobin A1c (HgA1C) and oral glucose tolerance tests can be used to diagnose and determine severity of T2DM. (Kishore, 2016; National Institute of Diabetes and Digestive and Kidney Diseases, 2016). Typically, diabetes is diagnosed when fasting blood glucose levels are above 126 mg/dL (normal range: 70-100) on at least two occasions or when random blood glucose levels are above 200 mg/dL with symptoms of polyuria and polydipsia (Kaput & Dawson, 2007; American Diabetes Association, 2015).

Under normal conditions, polymers of glycogen are stored in the liver to help maintain blood glucose levels throughout the day. As glucose enters the bloodstream, insulin is released from the pancreas and binds to receptors of cells throughout the body. Insulin, a hormone produced by the beta cells of the pancreas in response to hyperglycemia, plays a central role in regulating blood glucose. In health individuals, insulin lowers blood glucose immediately by enhancing glucose transport into muscle
and fat cells via Glut 4 transporters (Murray et al., 2012; Janson & Tischler, 2012).

Insulin resistance arises when the cell receptors do not recognize and respond to insulin in the appropriate way, leaving elevated levels of glucose and insulin in the bloodstream. When blood glucose levels rise above 160 to 180 mg/dL, glucose spills into the urine and the kidneys must excrete additional water to dilute excess glucose (Kishore, 2016; Janson & Tischler, 2012).

Symptoms of T2DM include increased thirst, urination, and hunger. Other symptoms of T2DM include blurred vision, drowsiness, nausea, tingling or pain in extremities, and decreased endurance during exercise (Kishore, 2016; American Diabetes Association, 2016). Chronic hyperglycemia damages blood vessels through an inflammatory process involving advanced glycation end products (AGEs), which are produced when blood glucose levels are consistently elevated, promoting increased glycation. Glycation of collagen and other proteins can lead to accumulation of plasma proteins, such as LDL, in the walls of the blood vessels; causing them to narrow and restricting blood flow to parts of the body (Murray et al., 2012). Multiple organ systems can be affected including the brain, eyes, heart, kidneys, skin, and nerves (American Diabetes Association, 2016; Mahan & Escott-Stump, 2008). Some problems that manifest in the skin include diabetic dermopathy, necrobiosis, lipoidica diabeticorum, diabetic blisters, exuptive xanthomatosis, and other bacterial or fungal skin infections. Acanthosis Nigricans, a darkening of the skin on the back of the neck or near folded areas, can occur and is a sign of insulin resistance, secondary to T2DM. Individuals with diabetes are 40% more likely to suffer from glaucoma, and 60% more likely to suffer
from cataracts, than individuals without diabetes (American Diabetes Association, 2016; Mahan & Escott-Stemp, 2008). Glaucoma is a condition caused by excess buildup of pressure in the eye, which can eventually lead to blindness. Cataracts are a clouding of the lens of the eye that occurs with aging and may be influenced by other environmental factors (Mahan & Escott-Stump, 2008; National Eye Institute, 2016;).

Diabetic neuropathy is another serious side effect of prolonged elevated blood glucose levels and about 50% of people with diabetes suffer from some form of nerve damage (American Diabetes Association, 2016). Diabetic neuropathy can be peripheral, causing damage to the extremities, or autonomic, effecting multiple organs in the body including the bladder, kidneys, gastrointestinal system, reproductive system, and cardiovascular system. Paralysis of the bladder, diarrhea, constipation, gastroparesis, and erectile dysfunction can all result from autonomic diabetic neuropathy (American Diabetes Association, 2016; Janson & Tischler, 2012). Damage to the small blood vessels in the kidney can result in kidney disease and ultimately kidney failure (Mahan & Escott-Stump, 2008; National Diabetes Federation, 2016). Similarly, damage to blood vessels that supply the heart can result in cardiovascular disease (CVD), the most common cause of death in those with diabetes (National Diabetes Federation, 2016; American Diabetes Association, 2016).

**Global and national prevalence.** Worldwide prevalence of diabetes was estimated to be about 2.8% in 2000 and 4.4% in 2030 with the total number of people with diabetes rising from 171 million to 366 million (Wild, Roglic, Green, Sicree, & King, 2004). Between 2010 and 2030, an estimated 69% increase in the number of adults
with diabetes in developing countries and 20% increase in developed countries is expected (Shaw, Sicree & Zimmet, 2009). The latest global estimate from the International Diabetes Federation is that there were 415 million people with diabetes in 2015 and that by 2040 there will be 642 million.

The pattern of diabetes varies considerably according to a country’s economic status with the majority of individuals affected exceeding the age of 60 years in developed countries, and falling between the ages of 40 and 60 in countries that are developing. (Shaw et al., 2009). Even if the prevalence of obesity remains stable until 2030, the rate of obesity is expected to increase due to an aging population and increased urbanization in developing countries (Wild et al., 2004).

Analyses of nationally representative data from 1980 through 2012 suggest that the incidence and prevalence of diabetes has doubled between 1980 and 2008 and plateauing between 2008 and 2012 (Geiss et al., 2014). In 2012, 29.1 million Americans, or 9.3% of the population had diabetes making it the country’s 7th leading cause of death (Centers for Disease Control and Prevention, 2014; ADA, 2015). Other studies indicate that prevalence may have been as high as 14% among U.S. adults, depending on criteria used (Menke, Casagrande, Geiss & Cowie, 2015; ADA, 2013). An estimated 36% of cases of diabetes went undiagnosed in the US between 2011-2012, (Menke et al., 2015) and deaths from diabetes may be underreported both worldwide and in the United States (Jones, 2015; Dwyer-Lindgren, van Lenthe, Flaxman & Mokdad, 2016).

**Contributing factors.** Genetic factors play a significant role in the development of type-2 diabetes, putting certain ethnicities including African Americans, Asian
Americans, American Indians, and Hispanics who live in the United States at an increased risk (Kishore, 2016; Mahan & Escott-Stump, 2008). The complexity of type 2 diabetes can be attributed to a contribution of genetic factors, environment, and interactions between the two (Kaput & Dawson, 2007). In fact, genetic studies have identified over 50 genes that participate in biochemical, regulatory, or signal transduction pathways that are involved in producing the phenotypes associated with T2DM (Kaput & Dawson, 2007).

Other risk factors for the development of T2DM include obesity, increased age, high blood pressure, history of gestational diabetes, and poor nutrition during pregnancy. In fact, 80-90% of people with diabetes are overweight or obese (Kishore, 2016). Poor dietary and lifestyle choices including over-consumption of calories and physical inactivity are significant contributing factors to the development of obesity and metabolic syndrome, both significant players in the development of T2DM (Mahan & Escott-Stump, 2008; Schulze et al., 2004).

**Prevention and treatment of type 2 diabetes.** The best way to prevent T2DM is to achieve and maintain a healthy weight (Mahan & Escott-Stump, 2008). Like obesity, many of the risk factors associated with T2DM are modifiable, making it reversible through weight loss and glucose control (Gregg et al., 2012). Partial remission of T2DM is defined as HbA1C of 5.7%-6.5% and complete remission is defined as <5.7%(Buse et al., 2009). Studies have shown that even small amounts of weight loss (5% of body weight) can change blood glucose levels toward normal significantly,
improve HbA1C, and/or reverse T2DM all together (Ades, 2015; Franz, Boucher, Rutton-Ramos, & VanWormer, 2015; Mahan & Escott-Stump, 2008).

Dietary and lifestyle interventions are necessary to improve insulin sensitivity and decrease the negative effects of T2DM (Ades, 2015). Diet management is one of the keys to normalizing blood glucose levels and individuals with diabetes benefit greatly from learning about how carbohydrates affect blood glucose. Exercise, in appropriate amounts, also helps individuals with diabetes to improve insulin sensitivity and control body weight. Other common treatments include oral medications and/or insulin injections for those who cannot manage blood glucose levels through diet and exercise alone (Kishore; 2016). It is essential that individuals with T2DM monitor their blood glucose levels throughout the day in order to properly treat high or low levels and prevent further complications (ADA, 2016; Mahan & Escott-Stump, 2008).

**Consequences of type 2 diabetes.** Diabetes is associated with multiple negative psychological and social consequences including decreased life expectancy, decreased quality of life, increased health-care costs, and increased risk of CVD. T2DM is estimated to shorten life span by 3.3-18.7 years (Leung, Pollack, Colditz & Chang, 2015) and contributes a large economic burden at personal, national, and global levels. In fact, diabetes is projected to increase lifetime healthcare expenditures by $8,946 to $159,380 depending on age, race, sex, and BMI classification (Leung et al., 2015).

The total annual global health expenditure for diabetes in 2010 was estimated to fall between $376.0 billion and $672.2 billion, accounting for about 12% of the world’s total health care expenditure (American Diabetes Association, 2013). Moreover,
projections for annual global health care expenditure for 2030 are 30-34% higher and fall between $490.1 billion and $893.0 billion (Zang et al., 2010). In the United States, diabetes accounts for an estimated $322 billion per year and one out of every five dollar is spent caring for people with diabetes (ADA, 2016).

Because of diabetes, it is estimated that each day it is estimated that 200 Americans undergo an amputation, 136 enter end-stage kidney failure, and 1,795 develop severe retinopathy that could lead to blindness (ADA, 2016). Diabetes also significantly increases risk factors for cardiovascular disease, hypertension, dyslipidemia, heart attack, and stroke. In 2003-2006, cardiovascular disease death rates were 1.7 times higher in individuals with diabetes. Similarly, in 2010, heart attack rates were 1.8 times higher and stroke rates were 1.5 times higher in individuals with diabetes (ADA, 2016; CDC, 2014).

**Cardiovascular Disease**

Cardiovascular disease (CVD), also known as heart disease, refers to any disease of the heart and blood vessels (American Heart Association, 2016). The main types of CVD include coronary heart disease, heart failure, heart attack, and stroke. CVD is a complex and multifactorial disease, which is influenced by both genetic and environmental factors (Mahan & Escott-Stump, 2008). A build up of plaque in the arteries, or atherosclerosis, is the main cause of heart attack and stroke (Warnica, 2016). Narrowing and hardening of the arteries are characteristics of atherosclerosis, which limit blood flow through the body (Mahan & Escott-Stump, 2008). While the exact mechanism of the formation of plaque is still being investigated, current research suggests that the initial step involves oxidation of LDL by the enzyme lipoprotein-
associated phospholipase A2. Oxidized LDL particles are subsequently taken up by macrophages, which then invade endothelial cells in the walls of arteries, further damaging blood vessel walls. This process initiates an immune response involving protein vascular cell adhesion molecule 1 along with macrophages and platelets. Accumulation of oxidized LDL along with monocytes results in atheroma, or degeneration, of the arterial wall. (Janson, & Tischler, 2012).

The rupturing of a plaque wall allows for the combination of blood and thrombic factors, creating a thrombus or embolus. A thrombus can completely block blood flow in an artery and an embolus can travel and block a smaller artery elsewhere in the body (Janson & Tischler, 2012). If blood flow, and therefore oxygen and nutrient delivery, to the heart become blocked, heart attack or myocardial infarction (MI) occurs. Similarly, if blood flow to the brain is blocked, ischemic stroke occurs. A hemorrhagic stroke occurs when a blood vessel within the brain ruptures. Heart failure, or congestive heart failure, occurs when the heart cannot pump blood efficiently enough to meet the body’s needs (American Heart Association, 2016).

Global and national prevalence. CVD is the leading cause of death in the United States and worldwide and an estimated 81,000,000 Americans adults (more than 1 in 3) have one or more types of CVD (Lloyd-Hones et al., 2010). According to the 2016 Heart Disease and Stroke Statistics Update, CVD accounts for more than 17.3 million global deaths per year (American Heart Association, 2016). Stroke, the second leading cause of death in 2013, accounted for 11.8% of deaths worldwide and is the leading preventable cause of disability in the United States (American Heart Association, 2016).
In 2014, about 356,500 Americans experienced a sudden heart attack outside of the hospital and each year about 209,000 people have a cardiac arrest while in the hospital (American Heart Association, 2016). With the aging population, the prevalence of CVD is expected to increase significantly in the next 2 decades. By 2030, these increases translate into an additional 27 million people with hypertension, 8 million with CHD, four million with stroke, and three million with heart failure in 2030 relative to 2010 (Heldenreich et al., 2011). By 2030, about 40.5% of the population is projected to have some form of CVD. The absolute number of deaths due to cardiovascular disease has increased substantially since 1990, however, high-income countries have been able to reduce mortality by up to 42% during this span of time. Low-income countries have seen no significant changes in mortality rate over time (Barquera, et al., 2015; Fryar, Chen, & Li, 2012). Between health expenditures and lost productivity, it is estimated that direct and indirect costs of cardiovascular disease total more than $316.6 billion per year (American Heart Association, 2016).

**Contributing factors.** There are multiple environmental and genetic factors that contribute to the development of CVD. While these disease conditions may not be completely preventable, healthy lifestyle choices are shown to slow progression and modify the course of disease (Heidenreich et al., 2011).

**Modifiable risk factors.** There is substantial evidence on the relationship between diet and CVD risk (Engler, 2009). A diet high in saturated fat and/or trans fat has been shown to contribute to CVD (Mahan & Escott-Stump, 2008) and substitution with polyunsaturated and monounsaturated fatty acids may be beneficial (Murray et al.,
The American Heart Association recommends that dietary intake of trans fats be reduced or eliminated with saturated fats limited to 5-6% of total calories (American Heart Association, 2016). Trans fats are plant oils that have been hydrogenated through an industrial process, making them behave more like a saturated fat and can lead to elevated LDL cholesterol levels (American Heart Association, 2016). Saturated fatty acids cause formation of smaller VLDL particles, which carry more cholesterol and are used by the body’s tissues at a slower rate. Polyunsaturated and Monounsaturated fatty acids behave differently by up-regulating LDL receptors, causing an increase in the catabolic rate of LDL. Since LDL is the main atherogenic lipoprotein, its breakdown results in less deposition of cholesterol in the arteries and less opportunity for plaque build up. Diets high in sucrose and fructose also contribute to elevated blood lipids, particularly triacylglycerols because their metabolism bypasses the regulatory step of glycolysis, catalyzed by phosphofructokinase (Murray et al., 2012).

Other modifiable risk factors include hypertension, high cholesterol, obesity, physical inactivity, diabetes, tobacco use, and harmful use of alcohol (World Heart Federation, 2016). Hypertension is the single biggest risk factor for stroke and plays a significant role in heart attacks. Similarly, abnormally high lipid levels can also raise risk for CVD. The relationship between obesity and cardiovascular disease is clear and many large prospective studies, such as the Farmingham Heart Study, the Manitoba Study, and the Harvard School of Public Health Nurses Study have documented obesity as an independent predictor of CVD (Hubert, Feinleib, McNamara & Castelli, 1983; Rabkin, Mathewson, & Hsu, 1977; Wilson, D’Agostino, Sullivan, Parise & Kannel, 2002).
While the progression of atherosclerosis is related to the aging process, inflammatory conditions such as obesity and diabetes may exacerbate development (Bastien et al., 2014). There is now considerable evidence that regional excess abdominal adipose tissue, irrespective of BMI, is associated with metabolic abnormalities such as insulin resistance, increased triglycerides and apolipoprotein B, and decreased HDL cholesterol levels, all potential risks for CVD (Bastien et al., 2014; Després, 2012). Obesity also induces structural abnormalities of the cardiovascular system aiming at increasing cardiac output and decreasing peripheral resistance in order to maintain whole system homeostasis. Over time, an increased cardiac burden can lead to left ventricular hypertrophy (Ku, Lin, Wang, Chang, & Lee, 1994; Bastien et al., 2014).

**Genetics and DNA damage.** CVD and its associated risk factors are multifactorial with many genes interacting with each other and with the environment. Genetic linkage studies and candidate gene association studies have resulted in compelling evidence for the genetic basis of myocardial infraction and atherosclerosis (Engler, 2009; Arnett et al., 2007). It is also known that mediators of inflammation, disturbed lipid metabolism, and oxidative stress are major players in cardiovascular disease (Merched, & Chan, 2013).

Over time, free radical damage is shown to promote atherosclerosis through chemical modification of the proteins or lipids in LDL cholesterol, leading to a form of LDL cholesterol that is not recognized by the Liver (Murray et al., 2012). This modified LDL cholesterol can be taken up by macrophages and subsequently infiltrate beneath blood vessel endothelium. This leads to development of atherosclerotic plaques (Murray
et al., 2012). There are multiple sources of free radicals in the body including ionizing radiation, hydroxyl radicals formed from transition metal ions, and nitric oxide or peroxynitrite, which is the product of a reaction between nitric oxide and superoxide (Murray et al., 2012).

Genetic polymorphisms related to CVD include but are not limited to apolipoprotein E (APOE), fatty acid desaturase (FADS), 5-lipoxygenase (5-LO), peroxisome proliferator-activated receptors (PPARs), apolipoprotein A1 (APOA1), apolipoprotein A2 (APOA2), apolipoprotein A5 (APOEA5), and methylenetetrahydrofolate reductase (MTHFR) (Nuno & Heuberger, 2014). Variation within these genes and others is most often due to single nucleotide polymorphisms (SNPs), which results in a single DNA building block (nucleotide) is replaced by a different nucleotide (Nuno & Heuberger, 2014). Recent research has been conducted in order to determine an interaction between diet and these polymorphisms that may play a direct role in CVD (Andreasen et al., 2008; Nuno & Heuberger, 2014).

**Prevention and treatment of cardiovascular disease.** Treatment of CVD varies depending on the type. Overall, goals of treatment aim to relieve symptoms and reduce the risk factors of a life-threatening emergency. Different methods are used to slow, stop, or reverse the build-up of plaque in the blood vessels. Similarly, sometimes a widening or bypassing of clogged arteries is necessary to prevent or lower the risk of a blood clot.

**Lifestyle changes.** Lifestyle changes are the first measure for prevention and treatment of CVD. Emerging evidence suggests that CVD prevention should occur earlier in life (Heidenreich et al., 2011). Research indicates individuals who reach
middle-age with optimal risk factors only have 6-8% chance of developing CVD in their lifetime (Ovbiagele et al., 2013). Similarly, in the Coronary Artery Risk Development in Young Adults study, risk factors in those under the age of 30 were predictive of subclinical atherosclerosis at a 15-year follow up (Berry et al., 2009). Individuals should maintain a healthy weight, eat a healthful diet, quit smoking, and be physically active. Data collected from NHANES from 1999-2006 suggests that 100% of those with CVD met three or fewer of the five components of a healthy diet (Nuno & Heuberger, 2014). These five healthy diet components include (1) consuming at least 4.5 cups of fruits/vegetables per day, (2) eating fish at least twice per week, (3) eating at least 3 servings of fiber-rich whole grains per day, (4) keeping sodium intake below 1500 mg/day, and (5) keeping sugar sweetened beverage intake below 36 oz per week. Research also shows that extreme stress or anger can trigger a heart attack, making stress management another important preventative measure (U.S. Department of Health and Human Services, 2016).

**Medications.** When lifestyle changes are not enough, oral medication can be prescribed in order to reduce the heart’s workload and relieve symptoms. Some medications are designed to decrease the chance of a heart attack, while others aim at lowering cholesterol levels, blood pressure, or other CVD risks (American Heart Association, 2016). A group of medications known as statins have proved to be effective at lowering blood lipid levels and preventing heart disease. These medications inhibit HMG-CoA reductase and up-regulate LDL receptor activity (Murray et al., 2012). Examples of Statins include atorvastatin, simvastatin, fluvastatin, and pravastatin.
Another medication, Ezetimibe reduces dietary absorption of cholesterol from the intestines via the Neimann-Pick-C 1 protein (Murray et al., 2016). Another class of drugs called fibrates aim at lowering plasma lipid levels by decreasing secretion of VLDL from the liver (Murray et al., 2012).

**Procedures and surgery.** There are multiple procedures and surgical interventions that can be used to treat CVD. A percutaneous coronary intervention (PCI), commonly known as angioplasty, is a non-surgical procedure that opens clogged or narrowed arteries using a thin flexible tube with a balloon or other device on the end. This tool is pushed through a blood vessel in order to compress plaque against the artery wall. Stents can be placed during PCI in order to hold arteries open and keep blood flowing (National Heart, Blood, and Lung Institute, 2016).

A coronary artery bypass grafting (CABG) is a type of surgical procedure that involves removal of arteries or veins form another part of the body and using these to bypass narrowed or blocked arteries. CABG is used to improve blood flow and prevent further complications, such as chest pain or a heart attack (National Heart, Blood, and Lung Institute, 2016). Cardiac rehabilitation is a type of medically supervised exercise training, education, counseling, that improves an individual’s knowledge of their condition as well as strengthens the heart muscle to improve stamina and physical abilities (U.S. Department of Health and Human Services, 2016; National Heart, Blood and Lung Institute, 2016).
Cancer

Cancer is a term that describes an abnormal growth of cells that interfere with normal cell function by expanding and invading adjacent tissues (National Cancer Institute, 2016). Cancer cells are characterized by three key properties: (1) they multiply rapidly with limited control, (2) they lack contact inhibition in vitro, that is they do not stop growing upon reaching other cells, and (3) they invade local tissues and metastasize to other systems of the body (Muray et al., 2012). As cancerous cells grow and multiply, they form a mass of cancerous tissue called a tumor, which can interrupt normal organ system function (National Cancer Institute, 2016). There are different types of cancer including leukemia, lymphomas, carcinomas, and sarcomas (Chabner & Chabner-Thompson, 2016). Leukemia is cancer that manifests in the blood and blood forming tissues, such as the bone marrow. Lymphomas expand in lymph nodes, affecting normal function of the immune system and producing large masses in the armpit, groin, abdomen or chest. Carcinomas are cancers that invade the lining of the organs or glands of the body. Sarcomas are cancers of mesodermal cells, which form muscles blood vessels, bone, and connective tissues (Chabner & Chabner-Thompson, 2016).

Global and national prevalence. Cancer is a major public health problem in the United States and worldwide. It is the second leading cause of death in the United States and is suspected to surpass heart disease as the leading cause of death over the next few years (Siegel, Miller, & Jemal, 2015). Between 1999 and 2013, there were more than 1.5 million cases of invasive cancer with 584,000 deaths from cancer per year in the United States. This included 14,700 cases among children under the age of 20 years old (US
Cancer Statistic Working Group, 2016). While there have been significant improvements in cancer survival over the past three decades in both blacks and whites, an increase in survival rate for lung and pancreatic cancer has been much slower. The five-year survival rate for lung cancer was 18% and for pancreatic cancer was 7%, partly due to more than 50% of these cases being diagnosed at a distant stage (Siegel et al., 2015). Over the past two decades there has been a slow decline in Cancer deaths, which can be attributed to fewer Americans smoking and advances in cancer prevention (Siegel et al., 2015).

**Contributing factors.** While it is never certain why some individuals develop Cancer and others do not, research has shown that risk factors can increase likelihood. Some of the risk factors associated with Cancer include exposure to radiant energy or carcinogenic chemicals, viruses, hereditary predisposition. Modifiable risk factors that increase the likelihood of developing cancer include tobacco use, decreased physical activity, overweight or obesity, poor diet, alcohol intake, unsafe sexual practices, and over-exposure to sunlight (American Cancer Society, 2016; Greenwald, Clifford, & Milner, 2001; Murray et al., 2012)

**Radiant energy.** Ultraviolet rays, x-rays, and gamma rays are shown to damage DNA through various mechanisms, making them mutagenic and carcinogenic. DNA damages caused by radiant energy include formation of pyrimidine dimers, formation of apurinic or apyrimidinic sites, formation of single-or-double strand breaks, and/or cross-linking of DNA strands (Murray et al., 2012). Exposure to ultraviolet radiation, mainly from sunlight, increases the risk of developing skin cancer, especially in individuals with
low melanin content in their skin (Murray et al., 2012). Radiant energy is more
dangerous in individuals who lack DNA repair mechanisms, such as those with
xeroderma pigmentosa and ataxia telangiectasia (Murray et al., 2012).

**Carcinogenic chemicals.** Many chemicals are carcinogenic and it is estimated
that 80% of human cancer cases are caused by environmental factors involving chemicals
(Murray et al). It is believed that most chemicals interact covalently with DNA, forming
adducts, which may be repaired or mis-repaired, resulting in mutation. Chemical
carcinogenesis involves two stages, initiation and promotion. During initiation, exposure
to a carcinogenic chemical causes irreversible DNA damage. During promotion, the cell
begins to grow and proliferate (Murray et al., 2012). Some examples of carcinogenic
chemicals include arsenic, asbestos, chromium, coal, engine exhaust, ethanol, mineral
oils, nickel compounds, plutonium, radon, tobacco, and others (American Cancer Society,
2016).

**Viruses.** Viruses may cause approximately 15% of human cancer cases. Some
viruses that are associated with human cancers include Epstein-Barr, Hepatitis B,
Hepatitis C, Human herpesvirus type 1, human papilloma virus, and human T-cell
leukemia virus type 1 (Murray et al., 2012). In viruses, like these, genetic material from
the genome is incorporated into the host cell. In RNA viruses, reverse transcription of
viral RNA to viral DNA occurs before incorporation into the host genome. Once a virus
has made its way into host DNA, deregulation of the cell cycle, inhibition of apoptosis,
and disruption of cell signaling pathways, and down-regulation of tumor suppressor
genes P53 and RB can occur (Murray et al., 2012).
**Hereditary and genetic basis.** It is estimated that about 5% of cancers may include a hereditary predisposition. The discovery of about 350 genes involved in cancer have been identified and it is estimated that as many as 2000 may exist. (Murray et al., 2012). Genetic polymorphisms have been discovered and research is underway to identify mutations in genes that cause and speed the spread of cancer (Murray et al., 2012). Large-scale analysis of genetic sequences has started to lead to new therapeutic approaches and genetic screening of families may allow for early interventions (Davis & Milner, 2004; Murray et al., 2012).

**Diet and lifestyle.** Similar to other chronic conditions, dietary and lifestyle factors can play a role in cancer development. Overweight and obesity are associated with increased risk for certain cancers (World Health Organization, 2009) and scientific evidence suggests that one third of all cancer deaths in the United States may be attributed to poor lifestyle behaviors (Mahan & Escott-Stump, 2008). Another one third of cancer deaths may be attributed to smoking and tobacco use (Mokdad et al., 2004;), with 71% of lung cancer being attributed to smoking (World Health Organization, 2009). Alcohol use is predicted to be responsible for 30% of deaths due to esophageal cancer and liver cancer (World Health Organization, 2009). Excessive energy intake, leading to excess body weight and obesity appears to increase cancer risk (Semanic et al., 2004) and re-occurrence (Kroenke, Chen, Rosner & Holmes, 2005). Physical activity helps individuals control their body weight. Excess adiposity is associated with increased levels of circulating estrogens, androgens, insulin, and insulin-like growth factors, which are all involved in increased cell and tumor growth (Mahan & Escott-Stump, 2008).
Processed and red meats have recently been labeled as “probably carcinogenic to humans,” and some studies indicate that excessive consumption may contribute to the development of cancer (Boada, Henríquez-Hernández, & Luzardo, 2016). Processed meat refers to meat that has been transformed through salting, curing, fermenting, smoking, or other flavor enhancing processes to improve flavor or extend shelf life. Multiple studies have indicated a relationship between consumption of processed meats and colorectal cancer and stomach cancer and consumption of red meats with pancreatic and prostate cancer (Working, 2015).

**Treatment of Cancer.** Treating cancer is complex, involving a team of healthcare professionals working together to make decisions based on multiple factors (American Cancer Society, 2016). Considerations must be made about the likelihood to cure the disease, ability to prolong life when a cure is impossible, effect of treatment on symptoms, side effects of treatment, and the patient’s personal wishes (Chabner, & Chabner, 2016). The main goal for treatment of cancer is to achieve the longest survival time with the highest quality of life possible. Individuals undergoing Cancer treatment must understand the risks involved with treatment (National Cancer Institute, 2016).

When a cancer diagnosis is first made, the initial objective is to remove the cancer, through single treatment or a combination of surgery, radiation therapy, and chemotherapy. Chemotherapy is the use of potent drugs to kill rapidly producing cancer cells in the body. Chemotherapy is known for its negative side effect due to the damaging nature of these drugs. Radiation therapy is the use of high doses of radiation exposure to kill cancer cells and prevent them from growing and reproducing. Radiation
typically also affects normal cells in the same proximity of cancer cells but the normal cells are able to repair themselves while cancer cells cannot. Surgical procedures can be used to prevent, diagnose, stage, and treat cancer. Lastly, palliative surgery can also be used to relieve discomfort by fixing a problem that is causing discomfort or disability.

Another type of cancer therapy includes a class of drugs that target polymerization and depolymerization of tubulin into microtubule polymers. Tubulin is a globular protein that forms microtubule structures inside cells. Microtubules make-up the structural cytoskeleton of the cell and also allow for intracellular transport within the cell. Medications such as colchicine, nocodazole, vincristine, and colcemid all inhibit polymerization by disrupting the tubulin monomer. Other drugs, such as a family of taxane drugs, break down existing microtubules. Without microtubule function, cell mitosis is inhibited. These medications seem to inhibit mitosis in cancer cells to a greater degree than normal cells, making them desirable for cancer therapy. Unfortunately, they also come with additional negative side effects, limiting their use in some patients (Janson & Tischler, 2012).

Stem cells, which have a unique ability to produce unaltered daughter cells and to create special cell types, may play a significant role in cancer (Clevers, 2011; Murray et al., 2012). Scientists are still investigating how stem cells contribute to cancer cell proliferation and invasion of other tissues. It is believed that cancer stem cells are more resilient to current cancer therapies and that targeting an underlying pool of cancer stem cells may be more effective at preventing reoccurrence of tumor growth (Boston Biomedical, 2016; Clarke, 2005; Murray et al., 2012) Current research suggests that
specifically killing cancer stem cells could improve clinical outcomes for patients with cancer because this would eliminate the potential for self-renewal that drives tumor formation (Boston Biomedical, 2016; Hu et al., 2012; Li et al., 2015).

**American Dietary Recommendations For Prevention of Chronic Disease**

The 2015-2020 dietary guidelines for Americans are designed to help people in the United States make healthier choices and improve their eating habits in order to maintain a healthy weight, prevent chronic disease, and increase longevity. The guidelines are made up of five overarching guidelines along with key recommendations including specific nutritional targets and daily limits. The U.S. Department of Health and Human Services and the U.S. Agriculture Association create these guidelines based upon current scientific evidence and recommendations from a Federal Advisory committee made up of well-respected researchers in the field of nutrition, health, and medicine (USDHHS & USDA, 2015).

**Core Concepts of Healthy Eating**

The 2015-2020 Dietary Guidelines for Americans recommend that individuals follow a dietary pattern that can be maintained across the life-span and that provides an appropriate amount of calories to reach and maintain a healthy weight and to prevent chronic disease. To meet nutritional needs, it is recommended to choose a variety of nutrient dense foods within recommended amounts, limiting calories from saturated fats, trans fats, and added sugars and reducing sodium intake. Key recommendations include less than 10% of calories per day from added sugars and saturated fats and less than 2,300 mg of sodium per day. Alcoholic beverages should be limited to one per day for
women and up to two per day for men, both of legal drinking age (USDHHS & USDA, 2015).

**Fruit, Vegetables, and Legumes**

The 2015-2020 dietary guidelines recommend a total of two and one half cups of vegetables per day including dark green, red & orange, beans & peas, and other starchy varieties. Recommendations also include two cups of fruit per day. Butter, salt, and creamy sauces should be limited in vegetable choices to reduce excess calorie, fat and salt intake. Fruits choices should come from whole fruits and 100% fruit juices. Since fruit juice is low in dietary fiber, 50% of fruit intake should come from whole fruit choices. Fruit, vegetables, and legumes provide a wide variety of essential vitamins and minerals. These foods are also good sources of dietary fiber, which helps to increase satiety and promotes gastrointestinal health. Fruits and vegetables are also known for their antioxidant properties, which help prevent free radical and DNA damage (USDHHS & USDA, 2015).

**Grains**

Current guidelines recommend 6oz of grains per day, with one half of total intake coming from whole grain varieties. Whole grains are recommended over refined grains because they are a better source of dietary fiber, vitamins, and minerals. Whole grains are also lower on the glycemic index, meaning they have a less severe effect on blood sugar levels and may lower LDL cholesterol as well (USDHHS & USDA, 2015).
Dairy

The most recent guidelines recommend 3 cups of low-fat dairy per day. This includes fat-free and 1% milk, cheese, yogurt, or fortified soy products. Dairy products are a good source of calcium and Vitamin D along with other essential vitamins and minerals (USDHHS & USDA, 2015).

Protein

Dietary guidelines recommend five and one half ounces of lean protein per day. Good sources include seafood, meat, poultry, eggs, nuts, seeds, and soy products. Protein foods help build and maintain lean body mass and can also promote satiety. They are a good source of iron, which is especially important for young children and pregnant women. Seafood is a good source of omega-3 fatty acids, which are shown to have antioxidant effects and promote heart health. Individuals should be mindful of the mercury content of the fish they consume and women who are pregnant or breast-feeding should not eat certain fish, which are high in methyl mercury (USDHHS & USDA, 2015).

Oils

The guidelines recommend 27 grams, or 5 teaspoons of oil per day. Oils should be liquid at room temperature and come from plan sources. Oils provide essential fatty acids, Vitamin E and promote satiety. Saturated and trans fats should be limited to less than 10% of total calories per day (USDHHS & USDA, 2015).
Limit on Calories for Other Uses

Once food group recommendations are met for the day, the Dietary Guidelines for Americans recommends limiting other calories to up to 270 kcals per day. These calories should be limited because they do not contribute to nutrient recommendations and are typically contain excess calories coming from refined grains, added sugars, and saturated or trans fats. These excess nutrients are not favorable toward achievement of healthy eating patterns and chronic disease prevention (USDHHS & USDA, 2015).

Dietary Recommendations For College Students

Since most college students fall between the ages of 18 and 26, the dietary recommendations for college students are spelled out in the 2015-2020 Dietary Guidelines for Americans, as listed above. The USDA has launched an initiative to get college students involved and on board with healthy eating. The MyPlate On Campus program encourages students to become MyPlate ambassadors who will support and share the MyPlate message with their student body. The USDA has created resources, all available online, for college ambassadors and the educators and staff who support them as they communicate the MyPlate message.

Additional resources for college students, are available from the USDA in order to encourage students to stay active and practice healthful eating behaviors while living on campus (USDA, 2016). These tips focus on exercise, finding a balance between daily physical activity and total energy intake, and making good choices in the dining halls (USDA, 2015). USDA also recommends that students track their dietary intake using, SuperTracker, a free online diet logging application.
Dietary Habits of College Students

During college years, students begin making independent life choices involving study habits, living arrangements, social interactions, physical activity involvement, and food choices (Marquis, 2005; Morrell & Burke, 2007). During the transition to university living, young adults make more eating decisions and it is during this time that many establish life-long dietary preferences (Birch, 1999). Although adolescence was once considered an age of optimal health, young adulthood is gaining important recognition as a time for health promotion and disease prevention (Nelson et al., 2008). Many times, greater lifestyle freedom that accompanies college years results in excessive weight gain (Holm-Denoma, Joiner, Vohs, & Heatherton, 2008; Racett et al., 2005). Multiple factors and unhealthful behaviors may contribute to weight gain during college years including low intake of fruits and vegetables, excessive caloric intake, decreased physical activity levels, and excessive alcohol intake (Anding, Suminski, & Boss, 2001; Clapp, Shillington, & Segars, 2000; Dinger, 1999; Ha, & Caine-Bish, 2009; Racett et al., 2005). Some studies indicate high consumption of sugar sweetened beverages among this population, which may contribute to weight gain (Schulze et al., 2004; West et al., 2006).

The presence of obesity and unhealthy lifestyle patterns in early life are associated with increased chronic disease risk indicating that this may be a critical developmental period for adoption of lasting healthy habits (Field, Cook, & Gillman, 2005). While multiple barriers and factors play into college students’ food selection, including taste preference, social situations, time constraints, convenience, cost, and healthfulness, there is a need for additional research to understand how the unique characteristics of early
adulthood may contribute to establishing long-term healthy behaviors (Davy, Benes & Driskell, 2006; Greaney et al., 2009).

**Perception of College Students Related to Nutrition and Health**

Even though research has shown that eating habits tend to become less healthful during college years and young adulthood (Grace, 1997; Winkleby & Cubbin, 2004), students believe that they consume a healthy variety of low fat foods including adequate amounts of fruits and vegetables (Davy et al., 2006). However, frequent snacking, high consumption of sugar sweetened beverages, high consumption of fast foods (6-8 times per week), and skipping breakfast are all common eating behaviors among this population, undermining the belief that they live a healthy lifestyle (Driskell, Kim, & Goebel, 2005; West et al, 2006; Winkleby & Cubbin, 2004). Many students consume high amounts of discretionary calories coming from fatty, sugary, or salty snacks (Brunt, Rhee & Zhong, 2008; West et al., 2006).

**Health Status of College Students**

Obesity, hypertension, high cholesterol, and sedentary lifestyle are all common conditions seen in young adults (Lofgren, Burke, Morrell & Reilly, 2008) and recent literature has suggested that obesity has escalated most rapidly among individuals between the ages of 18 and 29 and those with some college education (Huang, Shimel, Lee, Delance & Strother, 2007). The most current data collected by the American College Health Association via the National College Health Assessment, conducted in spring, 2016, demonstrates that 36.8 % of American college students are overweight or obese. More specifically, 22.9% are overweight with a BMI between 25-19.9, 8.4% have
a BMI between 30-34.9, 3.3% have a BMI between 35-39.9, and 2.2% have a BMI > 40 (ACHA, 2016). Some studies suggest that higher stress levels in students may be related to unhealthy diet and health behaviors (Pelletier, & Laska, 2016).

Along with overweight and obesity, increasing numbers of college students are presenting with early indicators of chronic disease including metabolic syndrome (Keown, Smith, & Harris, 2009). One study that examined metabolic disturbances in this population found that at least one risk factor for metabolic syndrome in 60% of men and 50% of women (Burke, Reilly, Morrell & Lofgren, 2009). Researchers from the University of Kansas found that out of 163 students, 27% had at least one component of metabolic syndrome, 11.7% had high total cholesterol, 5.5% had elevated LDL cholesterol, 13.5% had low HDL cholesterol, and 6% had pre-diabetes (Huang et al., 2004). Similarly, research conducted on 226 college students in New Jersey revealed that nearly one third had elevated blood cholesterol and half reported having a parent with high blood pressure and/or high blood cholesterol (Spencer, 2002).

Significant dietary differences are found between college students of a healthy weight and those who are overweight or obese (Brunt et al., 2008), with individuals of lower BMI reporting higher intake of vegetables and lower intake of meat products (Bruhnt, Rhee & Zhong, 2008). The typical diet of college aged students seems to be too high in saturated fat (Spencer, 2002), and too low in fruits and vegetables to be protective against risks for developing chronic disease (Chen et al., 2016; DeBate, Topping, & Sargent, 2001; Hiza & Gerrior, 2002; Hjartåker, Knudsen, Tretli & Weiderpass, 2015; Rautiainen, Levitan, Mittleman & Wold, 2015). In addition, it appears that there is little
improvement in dietary habits throughout the college period, (Driskell, Kim & Goebel, 2005; Small, Bailey-Davis, Morgan, & Maggs, 2012), which may lead to establishment of long-term behaviors. Knowing the relationship that exists between poor dietary choices, development of chronic disease, and increased mortality, research is needed to explore the motivational influences for health behavior, which may change for individuals during early adulthood (Nelson et al., 2008).

**Nutrigenomics, Nutrigenetic, and Nutritional Genomics**

Genomics is defined as the approach of describing the mapping, sequence, and analysis of all genes within the genome of a given species (Mutch et al., 2005). Nutrigenomics, nutrigenetics, and nutritional genomics are all scientific studies involving a relationship between genetics and nutrition but their definitions differ considerably. Nutritional genomics involves the genome-wide influences of nutrition and has far reaching potential for prevention of chronic disease. Nutritigenomics is defined as the science of the effect of genes on dietary response. Nutrigenetics is defined as the science of the role of nutrition and bioactive food parts in gene expression (Fenech et al., 2011).

The study of genomic information, along with the use of “omic” technologies allows for the acquisition of new knowledge aimed at gaining a better understanding of how nutrients and genes interact depending on genotype (Fenech et al., 2011). Modern technologies have been used to analyze the complete sequenced human, mouse, and rate genomes (Chinwalla et al., 2002; Gibbs et al., 2004; McPherson et al., 2001; Venter et al., 2001). These technologies and the vast amount of information stemming from their findings have lead to the development of comprehensive analytical and data mining
software, while transforming the way we understand nutrition, health, and disease (Mutch et al., 2005). The ultimate goal of these types of research is to develop personalized nutrition strategies for individuals or populations resulting in optimal health and disease prevention (Fenech et al., 2011).

There are three main considerations that make nutritional genomics useful and important. First, there is immense diversity in inherited genomes between different ethnic groups and individuals, making nutrient bioavailability and metabolism unequal among them (Hinds et al. 2005; Jorde & Woodling, 2004; Kaput, 2008). Second, food availability and choices are unique depending on environmental influences such as economic status, cultural influences, geographical location, and taste perceptions or preferences (Mahan & Escott-Stump, 2008; Moore, Roux, Nettleton & Jacobs, 2008). Third, nutritional status (over/under-nutrition) itself can influence gene expression and stability. For example, malnutrition (under or over) can lead to mutations in gene sequencing, which can cause expression of adverse phenotypes during different stages of life (Afman & Muller, 2006; Fenech et al., 2011).

Dietary reference values and safe upper limits are determined based on data obtained from the general population. For this reason, these recommendations may not be optimal for genetic subgroups whose metabolism of nutrients may differ from that of another. One of the main goals of nutrigenomics is to personalize nutrition recommendations to match unique genomes, allowing for metabolism to occur in the most efficient and effective manner. The fundamental hypothesis of nutrigenomics and nutrigenetics are that: 1) Nutrition may affect health outcomes by directly influencing the
expression of genes in metabolic pathways or by affecting the likelihood of genetic
mutation. 2) The effects of nutrients depend on inherited genetic variants that alter the
uptake and metabolism of nutrients. 3) Better health outcomes may be achieved by
matching appropriate nutritional recommendations to an individual’s inherited and
acquired genome with consideration to life-stage, dietary preferences, and health status
(Fenech et al., 2011).

**Current Use of Genetic Testing for Personalized Nutrition**

Nearly 1,000 genes have been associated with human disease and 97% of these
can result in monogenetic diseases such as celiac disease, phenylketonuria, and
galactosemia. A monogenetic disease is a disease resulting from a single defective gene
on the autosomes and although they are relatively rare, they affect millions of people
worldwide (Mutch et al., 2005; World Health Organization, 2017). Typically, these
conditions are diagnosed at birth or in early adulthood, although Celiac’s Disease can be
diagnosed at any stage of life. Genetic testing and personalized nutrition are currently
being used to diagnose and treat these diseases, which can be classified into three main
categories including dominant, recessive, and X-linked. The nature of monogenic
diseases depends on the specific functions that are typically performed by the affected
gene. Recessive diseases are autosomal diseases where two copies of an abnormal gene
are passed down from both parents. A monogenetic dominant disease differs in that they
involve damage to only one gene copy. X-linked diseases are those that are caused by
mutations on X-chromosome and can be inherited when one copy of the gene is inherited
from a parent who has the disorder (World Health Organization, 2017).
Unlike monogenetic conditions, chronic diseases that are reaching epidemic proportions across the globe often arise from dysfunctional biological networks (polygenetic) and not single mutated genes. For this reason, dietary interventions to prevent chronic diseases are complex, requiring knowledge of how a complex mixture of nutrients (diet) will interact to change biological functions (Ghosh, 2009). However, in order to use genetic blueprints (genotypes) for dietary prevention of disease, the mechanisms driving the connection between diet and the outward expression of our genes (phenotype) must be identified first. An important aim of nutrigenomics research is to study genome-wide influences of diet, specifically focusing on the role of metabolic stress in the creation of metabolic syndrome, inflammation, insulin resistance, and chronic diseases such as obesity, diabetes, heart disease, and some types of cancer (Afman, & Muller, 2006).

**Celiac Disease**

Celiac disease is a chronic inflammatory condition of the small intestine with known heritable characteristics and is characterized by permanent intolerance to gluten/gliadin. Genetic variants in HLA-DQ genes indicate a high level of risk and probability for development of Celiac Disease (Ludvigsson et al., 2014; Romanos et al., 2009). It is also known that Celiac’s Disease runs in families, with twins having a 75% concurrence of disease development (Greco et al., 2002). This condition requires strict avoidance of gluten (Ludvigsson et al., 2014) in order to limit the inflammatory reaction, which directly affects intestinal cell structure and function by altering gene expression (Pavlidis et al., 2015). Studies have indicated that certain dietary components including
long chain omega-3 fatty acids, plant flavonoids, and carotenoids may act through a variety of mechanisms including decreasing inflammatory mediators through cell signaling and genetic expression, therefore reducing the production of damaging oxidants (Calder, 2011; Ferretti, Bacchetti, Masicangelo, & Saturni, 2012). Nutrition therapy for celiac disease includes these food parts due to their role in preserving the intestinal barrier and protecting against toxicity (Pavlidis et al., 2015).

**Phenylketonuria (PKU)**

PKU is an inborn error of metabolism, characterized by the defective phenylalanine hydroxylase (PAH) enzyme and is inherited as an autosomal recessive trait (Mutch et al., 2005). The human phenylalanine hydroxylase gene includes two sections of polymorphic sites and this genetic variation has been reported to decrease the enzyme’s activity (DiLella, Kwok, Ledley, Marvit, & Woo, 1986). Individuals with PKU must avoid foods high in protein and phenylalanine to prevent buildup of excess phenylalanine in the blood, which can lead to serious neurological damage (Sweeney, Roberts, Fletcher, 2011).

**Galactosemia**

Galactosemia is a rare recessive disorder affecting galactose-1-phosphate uridyltransferase enzyme (GALT) (Mutch et al., 2005). Without proper functioning of this enzyme, there is failed conversion of galactose to glucose leading to an accumulation of galactose in the blood. Elevated serum galactose can cause mental retardation if left untreated (Mahan & Escott-Stump, 2008). A galactose-restricted diet is the main treatment for individuals with galactosemia.
**Personalized Nutrition and Chronic Disease Prevention**

Companies such as GenoVive, Nutrigenomix, Habit’s, and Arivale are currently using direct to consumer genetic testing to create personalized nutrition plans for their clients (Arivale, 2017; GenoVive, 2015; Habit, 2016; Nutrigenomix, 2013). Advertising an approach that is centered on the notion that health depends not only on an individual’s diet, but also on the way their body responds to what they eat, these companies help clients develop an approach to nutrition that is based on their unique genetic blueprint. According to Dr. Alan Greene, M.D. Chief Officer at Habit: “The value of personalized nutrition is already a foundational truth based on science: The official dietary recommendations of the National Academy of Sciences, the Institute of Medicine, and the USDA Food and Nutrition Board, which are based on extensive review of available science, recognize that optimal intakes vary by age, gender, and life stage” (Habit, 2016).

Personalized nutrition companies use their client’s blood, biometrics, and/or genetics as distinct data points in relation to other factors such as lifestyle, physical activity, dietary patterns, stress levels, and sleep patterns. Next, the companies determine the best dietary choices for each client’s genotype in order to optimize bodily functions for that individual. Some companies, such as Arivale, also use microbiome tests to explore the diversity of gut bacteria. Decision tree logic, algorithms, and analysis of SNPs are all techniques used to create personalized dietary recommendations (Arivale, 2017; Habit, 2016).

Among Nutrigenomic companies, some of the most commonly tested genes include MTHFR, VDR and FTO. The MTHFR gene codes for the enzyme,
methylene tetrahydrofolate reductase, which catalyzes the conversion of 5,10 methylenetetrahydrofolate to 5-methyltetrahydrofolate, a co-substrate for remethylation of homocysteine to methionine (Janson & Tischler, 2012). This enzyme is also responsible for the conversion of folate from its inactive form to its active form. Individuals with the A allele for the MTHFR gene variant are shown to have lower activity of the MTHFR enzyme, which can lead to increased levels of homocysteine and elevated cardiovascular disease risk (Crider et al., 2011). Nutrigenomic companies have the opportunity to pinpoint individuals who are at increased risk for high homocysteine by testing for this genetic variation.

The VDR gene codes for the vitamin D3 receptor (Haussler et al., 2008) and plays an important role in metabolism of calcium and phosphorus. The VDR gene influences intestinal calcium absorption, bone cell growth, bone density, and may play a role in other chronic conditions such as type 2 diabetes and cardiovascular disease (Mahan & Escott-Stump, 2008). Nutrigenomic companies have the opportunity to identify individuals who are at an increased risk for low vitamin D levels. Companies may recommend supplementation based on which VDR gene variants are found for each individual.

The FTO gene has been linked to appetite regulation and the A-allele for the rs9939609 variant is associated with increased risk for obesity (Frayling et al., 2007; Tanofsky-Kraff et al., 2009). It is believed that individuals with high impact FTO variants may benefit from decreased total dietary fat consumption or modified fat consumption, depending on their current diet and lifestyle (Kilpeläinen et al., 2012). These individuals
are also informed on the high importance of physical activity because the effect of the FTO gene is significantly less in physically active individuals (Andreasen et al., 2008).

After completing genetic tests, behavioral science is utilized in order to provide additional support through one-on-one coaching. Registered dietitians, physicians, and other health care providers employ counseling techniques in effort to keep clients focused, prioritize goals, and motivate sustainable long-term behavior change (Arivale, 2017; Habit, 2016).

While it is becoming increasingly recognized that not all people respond to diet equally, research in the developing field of nutrigenomics is lacking and the Academy of Nutrition and Dietetics (AND) does not recommend nutrigenetic testing as a means of providing personalized nutrition recommendations on the grounds that it does not have enough evidence based research to support its effectiveness (San-Cristobal et al., 2013). The position AND requests that commercialized tests become more transparent to consumers, reporting data about the analytical techniques used and providing the number and names of SNPs. In addition, AND expresses concerns with the application of personalized nutrition care, based on the fact that many health care professionals lack the knowledge to translate genetic results into common language, therefore presenting a risk for misunderstandings and misuse of genetic information among patients (Afman & Muller, 2006; Rosen, Earthman, Marquart, & Reicks, 2006; San-Cristobal et al, 2013). Furthermore, cost for enrollment in these programs can range from $1,800 to $3,500 per year, (Arivale, 2017; GenoVive, 2015). Consumers must pay these fees in full because
they are not covered under current insurance plans, putting nutrigenomic testing out of reach for individuals who cannot afford these expenses.

**Barriers and Considerations Related to Nutrigenomics**

Nutrigenomics is an emerging science with high expectations, but there are major concerns about whether the goal of matching foods to individual genotypes is within reach. Whether or not nutrigenomic foods and personalized diets flourish in the world’s market depends on multiple hurdles being overcome. Some consider the use of nutrigenomics for personalized nutrition to be controversial, and possibly unethical (Pavlidis et al., 2015; San-Cristobal et al., 2013). Others are not convinced that enough research has been done to support the nutrition claims being made by companies who offer genetic testing, and therefore do not trust the validity of these recommendations (Castle & Ries, 2007; Fenech et al., 2011; Korthals & Komduur, 2010). Moreover, many individuals are unaware of the existence of nutrigenomic technology or lack enough knowledge to understand and appreciate its importance in health promotion (Fenech et al., 2011; Horne et al., 2016; Morin, 2009). These barriers need to be understood and addressed in order to promote successful implementation of nutrigenomic technology and adoption of personalized nutrition (AFFECT).

**Knowledge and Awareness of Nutrigenomics**

Studies have indicated that there may be limited awareness and a lack of general knowledge about current nutrigenomic processes (Goddard et al., 2007; Lapham et al., 2000; Morin, 2009) and no studies have tested these factors across college-students. In a study of Canadian consumers and physicians, findings indicate that members of the
public are unfamiliar with the term “nutrigenomics,” however others were able to provide a simple definition. Even though knowledge was limited among these participants, this did not deter many from appreciating the potential value of better information linking nutrition and one’s genetic profile (Morin, 2009). In a population-based survey on direct-to-consumer nutrigenomic testing in Michigan, Oregon, and Utah, awareness of genetic testing was highest in Oregon (24.4%) and lowest in Michigan (7.6%) indicating significant differences in awareness among different states. Nationally, 14% of respondents indicated awareness of nutrigenome tests. It was estimated that only about 1% of the total population had used a genetic test. Other predictors of awareness in this study were higher income and increasing age, except among those 65 years or older (Goddard et al., 2009). In another study, conducted by Synovate Inc, 5,250 consumers responded to the HealthStyles survey and 14% of respondents were aware of direct-to-consumer genetic tests. Education and age less than 55 years were significant independent predictors of awareness (Goddard et al., 2007). In a study examining the genetic knowledge of patients with chronic disease from the Netherlands, half to three fourths of respondents reported having little knowledge about genetics and older respondents reported significantly less knowledge (Morren, Rijken, Baanders & Bensing, 2007). Individuals that were younger and better educated were more likely to be aware of the nutrigenomics tests in all three of these studies (Morren et al., 2007; Goddard et al., 2007; Goddard et al., 2009). Since college students typically are younger and more educated than the overall U.S. population, a survey to determine their awareness, knowledge, and interest in nutrigenomic testing may prove useful.
Knowledge of nutrigenomics is also limited among dietitians and physicians, indicating a need for training in this area. HealthStyles survey data revealed that 44% of physicians are aware of nutrigenomic tests but only 44% have ever had patients ask about such tests, and 74% had never discussed results of a nutrigenomic test with a patient (Goddard et al., 2007). In a national survey of 390 dietitians, a mean knowledge score of 41% demonstrated generally low levels of knowledge in genetics and diet-gene interactions. In dietitians, higher reported confidence was associated with higher knowledge scores (Whelan, McCarthy & Pufulete, 2008). The Human Genome Education Model Project, a collaborative project of Georgetown University Medical Center and the Alliance of Genetic Support Groups communicated similar findings. Surveys were sent to 3,600 health professionals including dietitians, occupational therapists, physical therapists, speech-language-hearing specialists, and social workers. Almost 80% of respondents reported taking no formal courses in genetics and 80% had heard little to nothing about the Human Genome Project (Lapham et al., 2000). Two-thirds of these health care professionals reported an interest in continuing education in genetics. This study along with others present the need for a solid foundation in genetics in order to apply nutritional genomics to the treatment and prevention of chronic disease in the future (Lapham et al., 2000; Rosen et al., 2006).

**Perceived Benefit of Nutrigenomics**

When individuals and populations perceive a benefit to outweigh a risk, they are more likely to engage in an associated behavior (Frewar et al., 2004; AFFECT). In college students, perception of the risk/benefit ratio for nutrigenomic testing has not yet
been researched. Several studies suggest that general support for genetic testing exists among the public but that the perceived benefit may vary depending on the disease under study (Jallinoja et al., 1998; Hietala, Hakonen, Aro, Niemela, Peltonen & Aula, 1995; Decruyenaere, Eyers-Kiebooms, & Van den Berghe, 1993). In a survey conducted among Finns, 94% of respondents approved of genetic testing and 78% agreed that genetic testing should be available to anybody who wishes to have information about his/her disease genes (Jallinoja et al., 1998). A focus group study including 90 Canadian consumers and health care professionals indicated overall support of nutrigenomic testing, with perceived benefits of making a positive impact on behavior, generating wider interest in nutrition, encouraging preventative care, and helping to target interventions, ultimately resulting in health care savings (Morin, 2009). Another survey of physicians (primary care and endocrinologists) and patients was designed to assess general views about type 2 diabetes genetic testing to predict risk, motivate behavior change, and guide medication prescription. This study found that 88% of physicians and 79% of patients had somewhat or very positive views of genetic testing and that 74% of physicians and 83% of patients would recommend or request such a test (Grant, et al., 2009). Multiple studies report positive perceptions toward genetic testing with high expectations that they would improve patient motivation to adopt healthier behaviors (Grant et al., 2009; Morren et al., 2006; Morin, 2009). While there appears to be a high baseline state of enthusiasm for nutrigenomic testing, additional research is needed in this area to determine the effectiveness of these tests and overall satisfaction with personalized nutrition the outcomes.
Perceived Risk of Nutrigenomics

The scientific intersection between bioethics, nutrigenomics, and personalized nutrition is very important (Patrinos & Prainsack, 2014). Many scientists believe more research is needed to provide adequate background evidence before nutrigenomic technologies can be used to implement personalized nutrition (Pavlidis et al., 2015; San-Cristobal et al., 2013). Others feel that nutrigenomics will be overly expensive, seeking monetary gain over health promotion (Ronteltap, van Trijp & Renes, 2009; Morin, 2009). In the Russian population, cost was the most common reason for not undergoing genetic testing (Makeeva, Markova & Puzyrey, 2009). In another study, the percentage of participants who indicated they would be willing to take a genetic test dropped from 48% to 5% after being told it would cost £250 (Cherkas, Harris, Levinson, Spector & Prainsack, 2010). Consumer based studies like these imply that members of the public may be concerned that genetic tests are overly expensive and cost seems to be a critical factor in their use (Fallaize et al., 2013). Enrollment fees for companies such as Arivale and GenoVive range from $1,800 to $3,500 per year (Arivale, 2017; Genogive, 2015). A newer company, Habitat, offers a $299.00 Nutrition Test Kit, which includes a personalized nutrition plan and biology report (Habitat, 2016). Since these expenses are not covered under current insurance plans, many consumers are faced with financial barriers and are unable to participate in Nutrigenomic testing.

Privacy is another major issue that concerns the public in regards to nutrigenomic testing and collection of DNA data (Fallaize, Macready, Butler, Ellis & Lovegrove, 2013; Jallinoja et al., 1998; Morin, 2009;). Misuse of genomic information was a common
concern reported by various studies. Many participants believed the results of genetic tests could lead to discrimination and/or difficulties finding a job, obtaining insurance coverage, or adopting children (Garver & Garver, 1994; Haga et al., 2013; Jallinoja et al., 1998; Morin, 2009; Morren et al., 2006). In one study, about one half of consumers worried their results would affect their ability to take out insurance policies and one third were concerned their results would limit their ability to get a job (Morren et al., 2006). Recent recommendations have been made from the European Society of Human Genetics advising European government to proceed with additional legislation that would ensure employers and insurance companies use genetic information in a responsible and ethical manner (van El & Cornel, 2011).

**Fear of Disease Susceptibility Results**

In addition to privacy and ethical issues, many consumers are also likely to have fears regarding genetic test outcomes. About 14% of Russian respondents and 5.3% of American respondents agreed that the idea of genetic testing was frightening (Fallaize et al., 2006; Haga et al., 2013). In another study, the majority of Dutch participants were not fearful of genetic test results, but 41.5% said they would not have the test done if the disease could not be treated (Morren et al., 2006). In a similar study, 7.5% of Americans reported that they would not want a DNA test if the disease could not be treated (Haga et al., 2013). Many health care professionals and members of the Canadian public worried that results from these tests may be unclear or cause anxiety (Morin, 2009). A longitudinal study of participants who had undergone a commercially available genetic test found no association between the genomic testing and psychological risks and most
participants reported that the test was of high personal utility (Bloss, Wineinger, Darst, Schork & Topol, 2013).

**Ability to Facilitate Behavior Change Based on Personalized Diets**

One large barrier to the success and efficacy of nutrigenomic based testing is the ability of nutrigenomic testing for personalized nutrition to facilitate behavior change (Fallaize et al., 2013). To this date, evidence is limited, and predominantly comes from weight-loss intervention studies (Fallaize et al, 2013), making it critical to investigate these outcomes further. Food4Me, one of the first studies to investigate consumer acceptance and satisfaction with personalized nutrition, found that among European adults, personalized nutrition advice via internet intervention produced larger and more appropriate changes in dietary behavior than a conventional approach (Celis-Morales, Lara, & Mathers, 2015). However, no evidence was found that phenotype or genotype information enhanced personalized nutrition advice more than advice based on current individual dietary intakes alone (Celis-Morales et al., 2016). Findings from a meta-analysis of randomized-controlled-trials (RCTs), evaluating effectiveness of internet-based personalized nutrition interventions, found increased fruit and vegetable consumption compared to non-personalized advice (Morales, Lara & Mathers, 2015). Another study of 2240 participants found no association between genomic testing and behavior changes (fat intake or exercise) after three months or one year (Bloss et al., 2013). In another RCT of Canadian young adults, comparing the effectiveness of four pieces of personalized nutrition advice to conventional advice, it was reported that genotype-based advice resulted in greater reductions in sodium intake among those who
carried the ACE gene compared to controls. The proportion of individuals who kept sodium consumption below the recommendation of 1500 mg/day increased from 19% at baseline to 34% after one year (Nielsen & El-Sohemy, 2014). In another study comparing a DNA-based weight loss diet to a traditional weight loss diet reported greater dietary adherence, longer-term weight loss, and greater improvements in fasting blood glucose levels in participants who received personalized nutrition (Arkadianos et al., 2007). Overall, it appears that disclosing genetic information may have a positive impact on behavior change when compared to conventional approaches but some research evidence is conflicting (Bloss et al., 2013; Fallaise et al., 2013; ). More research is needed in this area to determine whether value is added to nutrition interventions when genotype and phenotype information is considered and reported.

**Current Research and Implications of Nutrigenomics for College Students**

Young adulthood is an important developmental period when long-term lifestyle behaviors may be established. During this time it is also shown that the risk for obesity and other chronic disease increases due to multiple environmental factors including unhealthy dietary patterns and decreased physical activity (Nelson et al., 2008). The study of nutrigenomics and nutrigenetics has promoted an increased understanding of how nutrition influences metabolic pathways, homeostatic control, and how this regulation may be disturbed in early phases of disease development (Afman & Muller, 2006). With these findings, there are high expectations for nutrigenomics in prevention of chronic disease.
As nutrigenomic research evolves rapidly over the coming years, college students will likely be involved in the collaborative effort that may eventually lead to evidence-based dietary intervention strategies for restoring health and fitness and preventing chronic disease across the lifespan (Afman & Muller, 2006). Given the complex nature of nutrigenomic information, College students, who are typically educated young adults, may be more likely than other populations to understand and appreciate the relevance of the practice in optimizing health and delaying onset of disease. Researchers are currently investigating causal relationships between bioactive dietary components and prevention or outcome of disease (Mutch et al., 2005), but long-term intervention trials are time-consuming and will require willingness to participate by the current population and future generations. Increasing awareness, knowledge, and positive outlook toward nutrigenomic science is detrimental to the success of these research endeavors. College students may face personal and ethical dilemmas associated with nutrigenomics, and may need to decide whether perceived benefits outweigh risks before they proceed in the face of uncertainty. They may consider how the advancement of nutrigenomic technology and practice could impact their own children and grandchildren. Growing acceptance and knowledge of nutrigenomics may, therefore, affect the longevity and quality of life of many generations to come.

**Current Nutrigenomic Research**

An understanding of how genetic variation interacts with environment, including diet, is a rapidly evolving area. The human genome consists of about 25,000 genes including 3.3 billion bases or nucleotides of DNA. Single nucleotide polymorphisms
SNPs are the most common form of genetic variation and there are an estimated 12 million SNPs in the human genome sequence (Fenech et al., 2011). Genetic polymorphisms are estimated to occur in at least 1% of the population. Other types of variations include nucleotide repeats, insertions, and deletions. Genetic variation can cause altered gene function and changes to protein structure and function. Proteins coded for by genes have important metabolic functions in the body and take on various roles including enzymes, receptors, transporters, antibodies, hormones, and communicators (Mahan & Escott-Stump, 2008).

To determine genetic basis for disease, whole genome studies and candidate gene association studies are currently being used (Engler, 2009). Whole genome studies encompass two major approaches including linkage analysis of families and whole genome association analysis of unrelated individuals (Franchini, Peyvandi, & Mannucci, 2008). Linkage analysis of families is a method that is helpful in finding mutations in genes that are inherited along a family line. Whole genome association studies are done to identify which of 12 million common SNPs in the entire human genome are associated with a greater risk or incidence of a particular disease. The HapMap project is a public resource, which was designed to determine common patterns of DNA sequence variation (SNPs) for complex diseases, such as cardiovascular disease, cancer, diabetes, cancer, and others (Gibbs et al., 2003). The work of this project has identified common clusters of alleles, or haplotypes, frequently inherited with many other neighboring SNPs. The HapMap project has made whole-genome studies more efficient and affordable by reducing the number of SNPs that need to be analyzed (Engler, 2009). New technologies
and data analysis tools are rapidly advancing in the field of nutrigenomics in order to sort and organize the large amount of data that comes from these genetic maps.

**Prevention of Chronic Disease Using Personalized Nutrition**

Nutrigenomic knowledge has the potential to provide improved methods for prevention of some of the most prevalent and deadly chronic diseases. For example, multiple nutrigenomic and nutrigenetic studies have indicated that complex interactions may explain differences observed in obese phenotypes, which vary within and across populations. Genes play an important role in body weight homeostasis through multiple mechanisms including appetite, physical activity, adipocyte differentiation, insulin signaling, mitochondrial function, lipid turnover, thermogenesis, and energy efficiency (Joffe & Houghton, 2016). In one study, personalized calorie-controlled diets were created using 24 variants in 19 genes involved in metabolism. Weight loss and weight maintenance between 50 individuals on the tailored diets was compared to weight loss and weight maintenance in individuals on generic diets. Results showed that the group receiving the personalized dietary advice performed better during the weight loss period, and were more able to maintain their weight over the following year (Arkadianos et al., 2007; Fenech et al., 2011). Since there is strong evidence of a correlation between development of obesity and other chronic conditions, interventions like these could lead to prevention of other diet-related diseases.
CHAPTER III
METHODOLOGY

Research Design

The purpose of this cross-sectional survey research study was to examine the knowledge and perception of college students toward genetic testing for personalized nutrition. Independent variables included gender, age, current academic standing, college major, awareness of nutrigenomics testing, and current or past enrollment in college level genetics or nutrition courses. Dependent variables were genetics knowledge and perception of college students toward genetic testing for personalized nutrition. The Institutional Review Board at Kent State University approved this study.

Recruitment

A convenience sample of undergraduate and graduate students at a public Mid-Western state university was used for this research. Participants included part and full time undergraduate and graduate students enrolled in the Spring 2017 semester at Kent State University. Student email addresses were obtained from Kent State University’s registrar’s office. The survey was sent out to a total of 37,232 participants in email listserv using Qualtrics (v.1.817s), an online survey software program.

Research Sample

Participants in the study were full or part-time students and included both undergraduate and graduate students enrolled at Kent State University for Spring 2017 semester. Anyone under the age of 18 years old was excluded from the study.
Instrument of Measure

The survey questionnaire included four main sections including 1) general demographics, 2) perception of nutrigenomics, 3) factors that influence participation in nutrigenomic testing, and 4) general genetics knowledge. There survey consisted of a total of 30 questions.

Part I: General Demographics

Part I of the survey included six general demographic questions including age, ethnicity, weight, height, class ranking, and participation in college level nutrition or genetics courses. Participants who did not meet the criteria of being 18 years or older, were unable to complete the survey. Participants who did not agree to the terms of consent were also unable to complete the survey. Descriptive statistics were used to calculate means, standard deviations, frequencies, and percentages for analysis of data collected from Part I.

Part II: Perceptions Toward Nutrigenomic Testing

Part II of the survey assessed the perception or attitudes of participants toward nutrigenomics through a series of 22 questions. These questions required participants to consider possible positive and negative aspects of using genetic testing for personalized nutrition. Perceptions of college students were measured using a five-point Likert-style scale, ranging from (1) ‘strongly disagree’ to (5) ‘strongly agree’ with (3) indicating ‘neither agree nor disagree.’ Items in this part of the questionnaire were either positive/supportive or negative/opposing. Negative questions were reverse scored and an average score was determined for each item in this section. Higher scores in this section
indicated more positive attitudes toward genetic testing for personalized nutrition. More negative scores indicated that participants perceived risks, or negative aspects of nutrigenomic testing, to outweigh benefits (positive aspects).

Part III: Factors Influencing Decision to Participate in Nutrigenomics Testing

Part III of the survey assessed the degree that common factors either encouraged or discouraged the participants tendency to take a genetic test for personalized nutrition. Participants were presented with a list of 12 common factors and were asked to indicate how much influence these had upon their decision to take a nutrigenomic test. Items in this section of the survey were measured using a five-point Likert-type scale, ranging from (1) ‘not at all likely’ to (5) ‘completely likely, with (3) indicating ‘moderately likely.’ A text box was provided for participants to enter any other factors that are not listed in this section.

Part IV: General Genetic Knowledge

This part of the survey assessed general genetic knowledge of college students. Questions in this section of the survey were measured using a true or false quiz. Genetics knowledge was scored based on responses to a 19 question assessment, where participants could choose from 1) True, 2) False, or 3) Don’t Know. Participants were instructed not to “guess” on answers, but rather to choose ‘Don’t Know’ if they did not know the answer, feel comfortable answering, or comprehend the question. Scores for this part of the survey were measured using a continuous scale. The general genetics knowledge score was scored by identification of the correct answer as a score of one. Incorrect answers were scored as zero. “Don’t know” answers were also coded as zero.
Total knowledge score was calculated by the sum of correct answers. The mean score was determined by averaging the percentage of correct answers for each participant.

Procedures

Upon KSU’s Institutional Revenue Board (IRB) approval, email invitation was sent to 37,232 Kent State University students in one listserv from Qualtrics, an online survey instrument. A link to the survey was provided via email along with an attached consent form and an invitation to participate. The email also briefly explained the questionnaire and ensured anonymity. Students were given 13 days to complete the survey, and two reminder emails were sent after day one and day eight. Participation in the survey was voluntary and personal identifiable information remained completely confidential. Participants were informed that they were allowed to discontinue taking the survey at any time. An incentive was used, giving participants a chance to win one out of four $25.00 Amazon Gift Cards by random selection. Students interested in participating in the prize drawling were directed to a separate Qualtrics survey where they entered their email address. This allowed survey data to be kept separate from the student email addresses collected for the prize drawling. Winners of the prize drawling received their $25.00 Amazon Gift Card via email two days after the survey was closed.

Data Analysis

Analysis of results was completed using the Statistical Package for Social Sciences (SPSS) version 24. Analysis of survey results was completed following the thirteen day data collection period. Descriptive statistics (mean, standard deviation, frequency distribution, and percentages) were used to analyze demographic data
including age, gender, class ranking, and history of taking college level nutrition or genetics course. The independent variables were gender, age, college major, current academic standing, awareness of nutrigenomic testing, and current or past enrollment in college level genetics or nutrition courses. The dependent variables included general genetics knowledge, and perceptions toward genetic testing for personalized nutrition care.

Independent sample t-tests were conducted comparing the mean scores of genetics knowledge and nutrigenomic perception among gender groups, and groups who were either familiar with genetic testing for personalized nutrition care or not. Familiarity with genetic testing was determined by asking participants if they had ever read or heard about nutrigenomic tests. Similarly, t-tests were also used to see whether college students who took a nutrition and/or genetics course scored differently on the genetics knowledge and nutrigenomic perception assessments.

A Pearson correlation coefficient was calculated for the relationship between genetics knowledge scores and perception of nutrigenomics scores. ANOVA was used to determine whether there were differences in genetics knowledge and perceptions toward nutrigenomics among groups of different class ranks (freshman, sophomore, junior, and graduate students) and different majors (1) Health and Medicine, 2) Social Sciences 3) Business, Math, Science & Technology, and 4) Arts, Humanities, & Others). Tukey post-hoc was used to determine the location of significant differences among groups. A significance of $P \leq 0.05$ was set for all t-test and ANOVA measurements.
CHAPTER IV
JOURNAL ARTICLE

Introduction

Nutrigenomics aims to determine the influence of common dietary components on the genome, attempting to relate different phenotypes to specific metabolic responses (Mutch et al., 2005). Experts believe that nutrigenomic developments could eventually lead to personalized evidence-based dietary intervention strategies for promoting health and preventing diet-related diseases (Morren et al., 2006). While nutrigenomics has been used for many years in the diagnosis of rare genetic diseases, usually diagnosed at birth, new nutrigenomic developments could reduce the onset and impact of complex non-communicable diseases that currently account for two-thirds of the world’s deaths and are the main cause of poor health, disability, and death in the United States (Afman & Muller, 2006; Bauer et al., 2014).

Public support of nutrigenomic technology, especially in emerging adults, will be necessary for the successful implementation of this technology and the achievement of the highly desirable goal of personalized nutrition care (Pavlidis et al., 2015). However, many Americans are unaware that nutrigenomic technology even exists (Goddard et al., 2007) and other potential consumers are highly concerned with cost, privacy of genetic information, and other ethical issues (Ghosh, 2008; Morren et al., 2006). Furthermore, many health care professionals lack proper training in this area (Lapham et al., 2000), and therefore, are uncomfortable interpreting or discussing results of nutrigenomic tests with their patients (Whelan et al., 2008). Since the study of nutrigenomics requires a solid foundation in nutrition, genetics, and biochemistry, this field is often difficult, even for
highly educated individuals to understand and appreciate (Görman et al., 2013; Lapham et al., 2000).

Since nutrigenomic technology is still in its developing stages, the perception of future consumers toward genetic testing for personalized nutrition could provide valuable information about the future progress of these scientific endeavors, and simultaneously give insight into the challenges that may hinder forward momentum in this field (AFFECT; Goddard et al., 2007). Considering the depth of knowledge necessary to comprehend nutrigenomic processes, perhaps college students, who are more educated (Ryan & Bauman, 2016), and more exposed to new scientific and technological advances (Smith et al., 2011), might find these concepts to be especially appealing, making them a critical group of potential future consumers. College students also make up a highly diverse and influential population, coming from a broad range of cultural and seriocomic backgrounds.

With approximately 17 million college students enrolled in over 6,000 post-secondary institutions, accounting for nearly half (47%) of Americans between the ages of 17-24 years old (Knapp, Kelly-Reid, & Ginder, 2011), such institutions could be well suited for addressing perceptions toward nutrigenomic technology. The college transition period involves adjustment to a new social environment where students learn independence and have exposure to unfamiliar academic ideas and viewpoints (Hurtado, 2005; Thakral et al., 2016). However, no studies have examined how college students perceive the idea of using genetic information to personalized dietary interventions.
Therefore, the primary purpose of this study is to assess the knowledge and perception of college students toward genetic testing for personalized nutrition.

**Methodology**

**Research Design**

The purpose of this quantitative, non-experimental study was to examine the awareness, knowledge and perception of college students towards genetic testing for personalized nutrition care. This study was approved by the Kent State University Institutional Review Board.

Independent variables included gender, age, current academic standing, major, awareness of nutrigenomics testing, and current or past enrollment in college level genetics or nutrition courses. Dependent variables were genetics knowledge and perception of college students toward genetic testing for personalized nutrition care.

**Research Sample**

The participants in the current study were part and full time undergraduate and graduate students enrolled in the Spring 2017 semester at Kent State University, a public Midwestern state university, in Kent, Ohio. Participants had to be at least 18 years old and agree to all study parameters, which were outlined in the consent form, displayed in Appendix A.

**Instrument of Measure**

The four-part survey, included in Appendix B, consisted of 60 total questions. Part I of the survey included general demographic questions such as age, gender, and class ranking. This section of the survey also asked whether or not students had ever
taken a college level nutrition or genetics course. If students did not meet criteria for age (under 18 years), the survey was terminated. Part II of the survey was focused on assessing perceptions or attitudes toward nutrigenomics. These questions were measured using a five-point Likert-style scale, where (1) indicated strong disagreement to the statement, and (5) indicated strong agreement with the statement. Part III of the survey assessed the degree that common factors either encouraged or discouraged the participants decision to take a nutrigenomic test. Participants were presented with a list of factors and were asked to indicate how much influence these had upon their decision to take a genetic test for personalized nutrition. Items in this section of the survey were measured using a five-point Likert-type scale, where (1) indicated that the factor was not at all likely to influence their decision and (5) indicated that the factor was completely likely to influence their decision. Part IV of the survey assessed general genetic knowledge of participants. Questions in this section of the survey were measured using a test where participants could choose from 1) True, 2) False, or 3) Don’t Know.

**Procedures**

Student email addresses were obtained from Kent State University’s registrar’s office. The survey was administered through Qualtrics (v.1.817s), an online survey software program to a total of 37,232 students. Students received an invitation to participate via email along with a brief explanation of the study, and request for participation. The email also included a link connected to the Qualtrics survey, where they were presented with an electronic consent form. Participants were also invited to
participate in a drawing, where they could win one out of four $25.00 Amazon gift cards. The survey was open for 13 days and two reminder emails were sent throughout the survey period.

**Data Analysis**

Analysis of results was completed using the Statistical Package for Social Sciences (SPSS) version 24. Analysis of survey results was completed following the thirteen day data collection period. Descriptive statistics (mean, standard deviation, frequency distribution, and percentages) were used to analyze demographic data, perception scores, degree that factors influenced participation in nutrigenomic testing, and genetics knowledge scores. Independent t-tests were conducted comparing the mean scores of genetics knowledge and nutrigenomic perception depending on gender, familiarity (awareness) with nutrigenomic testing and those who were unfamiliar, and college students who indicated participation in a nutrition and/or genetics course vs those who did not. ANOVA analysis was used to determine whether there were differences in genetics knowledge and perceptions toward nutrigenomics among groups based on class standing and college major. Tukey post-hoc was used to determine the location of significant differences. Correlation analysis was calculated for the relationship between genetics knowledge scores and perception of nutrigenomics scores. A significance of $P \leq 0.05$ was set for all t-test and ANOVA measurements.

**Results**

Out of 37,232 survey invitations, a total of 3,564 students started the survey, equating to a 9.57% response rate. Participants under the age of 18 years old ($n= 15$) or
those who did not agree to the terms of the consent form (n=13) were excluded and unable to complete the survey. The remaining 3,536 participants were able to continue through the remainder of the four part survey. A total of 21 participants clicked the link to begin the survey but did not provide any responses. Six hundred and sixteen incomplete survey responses were also excluded from the data analysis, and 2,899 students completed the survey in full, equating to an 82.0% completion rate. One response was eliminated from the age analysis due to unrealistic data.

Demographics

The demographic data of participants is highlighted in Table 1. Participants ranged from 17 to 72 years old with a mean age of 25.04 ± 8.8 years (n= 2898). More than two thirds of participants were female and the majority of participants were undergraduate students. Approximately 30% of students in this study reported participation in a college level nutrition course and 15% reported participation in a college level genetics course.

Table 1

*General Characteristics of College Students Surveyed about Nutrigenomic Testing*

<table>
<thead>
<tr>
<th>Gender</th>
<th>%</th>
<th>n</th>
</tr>
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<tbody>
<tr>
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<td>Female</td>
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</tr>
<tr>
<td>Transgender</td>
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<td>12</td>
</tr>
<tr>
<td>Other</td>
<td>0.4</td>
<td>12</td>
</tr>
<tr>
<td>Prefer not to answer</td>
<td>0.5</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>2899</td>
</tr>
</tbody>
</table>
Table 1 (continued)

*General Characteristics of College Students Surveyed about Nutrigenomic Testing*

<table>
<thead>
<tr>
<th>Class Rank</th>
<th>%</th>
<th>n</th>
</tr>
</thead>
<tbody>
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<td>18.5</td>
<td>536</td>
</tr>
<tr>
<td>Sophomore</td>
<td>17.6</td>
<td>510</td>
</tr>
<tr>
<td>Junior</td>
<td>16.6</td>
<td>482</td>
</tr>
<tr>
<td>Senior</td>
<td>23.1</td>
<td>669</td>
</tr>
<tr>
<td>Master’s Student</td>
<td>18.1</td>
<td>543</td>
</tr>
<tr>
<td>Doctoral Student</td>
<td>5.5</td>
<td>159</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>2899</td>
</tr>
</tbody>
</table>

^a^ = 1 response eliminated due to unrealistic data

*Abbreviations.* n, number of members in sample.

**Genetics Knowledge**

This section of the survey assessed general genetic knowledge of college students with a series of true or false questions. Table 2 displays results of this knowledge assessment. For this section of the survey, one hundred and thirty-three responses were excluded from scores for the first ten questions due to missing data, leaving 2,766 valid responses for these questions. One hundred and eighty survey responses were excluded from scores in the last nine questions due to missing data., leaving 2,719 valid responses for these questions. On average, participants answered 55.52% (n = 2766) of questions correctly in this section. A Pearson correlation coefficient was calculated for the relationship between participants’ genetics knowledge and their perceptions toward nutrigenomic testing. Based on survey results, a positive correlation was found, \( r (2764) = .14, p \leq 0.001 \), indicating a significant linear relationship between positive perceptions toward nutrigenomic testing and higher genetics knowledge scores.
An independent sample t-test (Table 3) was used to compare the mean knowledge scores between participants who indicated participation in a college level nutrition and/or genetics course and those who did not. There was a significant effect for students who indicated participation in a college level nutrition course, \( p = .002 \), with those who indicated participation receiving higher scores on the genetics knowledge assessment. Students who participated in a nutrition course had a mean score of 10.88. Similarly, students who indicated participation in a college level genetics course also scored significantly higher on the genetics knowledge assessment, \( p \leq 0.001 \), with a mean score of 14.67.

Another independent sample t-test was conducted comparing the mean genetics knowledge scores of participants who indicated some familiarity with nutrigenomic testing and those who indicated no familiarity. Based on results of this test, participants who reported awareness of nutrigenomic testing scored significantly higher on the genetics knowledge assessment than participants who indicated no awareness \( p \leq 0.001 \).

A one-way ANOVA (Table 4) was conducted comparing the genetics knowledge scores of students based on their class standing. A significant difference in genetics knowledge was found among class ranks, \( p = 0.03 \). Tukey post-hoc test revealed no significant difference seen between freshman, sophomores, juniors, and seniors \( p \geq 0.05 \), however, graduate students scored significantly lower on the genetics knowledge test than both juniors \( p =0.038 \) and seniors \( p =0.032 \).
A second one-way ANOVA was also conducted comparing genetics knowledge among majors. Results from this test are displayed in Table 5. Majors were categorized into four main groups including; 1) Health and Medicine, 2) Social Sciences 3) Business, Math, Science & Technology, and 4) Arts, Humanities, & Others. A statistically significant difference was found among majors, \( p \leq 0.001 \). While Tukey post-hoc revealed no significant difference in genetic knowledge scores among ‘Business, Math, Science & Tech’ and ‘Social Sciences’ majors \( p \geq .05 \), ‘Business, Math, Science, and Technology’ majors scored significantly higher on the knowledge assessment than ‘Arts and Others’ majors \( p = .007 \). In addition, ‘Health and Medicine’ major scored significantly higher on the genetics knowledge test than all other majors \( p \leq 0.001 \). On average, health and medicine majors \( n = 674 \) scored the highest on the genetics knowledge assessment, while art and other majors \( n = 455 \) scored the lowest.

Table 2

*Genetic Knowledge Test Scores among College Students*

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean ± SD (n)</th>
<th>% Correct (n)</th>
<th>% Incorrect (n)</th>
<th>% Don’t Know (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>True or False</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A gene is a portion of DNA which codes for a protein, which leads to a trait.</td>
<td>0.84 ± 0.36 (2766)</td>
<td>84.3 (2333)</td>
<td>2.7 (75)</td>
<td>12.9 (358)</td>
</tr>
<tr>
<td>Males inherit two X-chromosomes at birth, one from their mother and one from their father.</td>
<td>0.59 ± 0.49 (2766)</td>
<td>59.5 (1647)</td>
<td>28.9 (800)</td>
<td>11.5 (319)</td>
</tr>
</tbody>
</table>
Table 2 (continued)

*Genetic Knowledge Test Scores among College Students*

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean ± SD</th>
<th>% Correct</th>
<th>% Incorrect</th>
<th>% Neutral</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>True or False</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The human genome project has estimated that humans have between 20,000 and 25,000 genes.</td>
<td>0.30 ± 0.46</td>
<td>30.2</td>
<td>12.5</td>
<td>57.3</td>
</tr>
<tr>
<td>(2766)</td>
<td>(836)</td>
<td>(345)</td>
<td></td>
<td>(1585)</td>
</tr>
<tr>
<td>Genes contain chromosomes.</td>
<td>0.21 ± 0.41</td>
<td>21.4</td>
<td>63.7</td>
<td>15.0</td>
</tr>
<tr>
<td>(2766)</td>
<td>(591)</td>
<td>(1761)</td>
<td></td>
<td>(414)</td>
</tr>
<tr>
<td>A genotype is the genetic make-up of an organism (BB, Pp, ff)</td>
<td>0.67 ± 0.47</td>
<td>67.4</td>
<td>4.5</td>
<td>28.1</td>
</tr>
<tr>
<td>(2766)</td>
<td>(1865)</td>
<td>(125)</td>
<td></td>
<td>(776)</td>
</tr>
<tr>
<td>In humans, each cell normally contains 23 pairs of chromosomes, for a total of 46.</td>
<td>0.83 ± 0.38</td>
<td>82.9</td>
<td>4.6</td>
<td>12.5</td>
</tr>
<tr>
<td>(2766)</td>
<td>(2293)</td>
<td>(127)</td>
<td></td>
<td>(346)</td>
</tr>
<tr>
<td>A phenotype is a physical expression of alleles (brown eyes or blue eyes).</td>
<td>0.76 ± 0.43</td>
<td>76.3</td>
<td>2.7</td>
<td>21.0</td>
</tr>
<tr>
<td>(2766)</td>
<td>(2111)</td>
<td>(74)</td>
<td></td>
<td>(581)</td>
</tr>
<tr>
<td>A mutation occurs when the structure of a gene changes.</td>
<td>0.80 ± 0.40</td>
<td>79.5</td>
<td>5.2</td>
<td>15.3</td>
</tr>
<tr>
<td>(2766)</td>
<td>(2199)</td>
<td>(143)</td>
<td></td>
<td>(424)</td>
</tr>
<tr>
<td>Mutations always lead to negative health outcomes.</td>
<td>0.83 ± 0.38</td>
<td>82.6</td>
<td>5.1</td>
<td>12.3</td>
</tr>
<tr>
<td>(2766)</td>
<td>(2285)</td>
<td>(142)</td>
<td></td>
<td>(339)</td>
</tr>
<tr>
<td>An allele is the different forms of a gene, represented by letters.</td>
<td>0.51 ± 0.50</td>
<td>51.3</td>
<td>8.0</td>
<td>40.7</td>
</tr>
<tr>
<td>(2766)</td>
<td>(1419)</td>
<td>(222)</td>
<td></td>
<td>(1125)</td>
</tr>
<tr>
<td>A dominant trait is the one hidden in the F1 generation.</td>
<td>0.36 ± 0.48</td>
<td>36.4</td>
<td>11.3</td>
<td>52.2</td>
</tr>
<tr>
<td>(2719)</td>
<td>(991)</td>
<td>(308)</td>
<td></td>
<td>(1420)</td>
</tr>
</tbody>
</table>
Table 2 (continued)

*Genetic Knowledge Test Scores among College Students Continued*

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean ± SD (n)</th>
<th>% Correct (n)</th>
<th>% Incorrect (n)</th>
<th>% Neutral (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>True or False</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DNA repair is a collection of processes where a cell identifies and repairs DNA molecules that encode its genome.</td>
<td>0.51 ± 0.50 (2719)</td>
<td>50.9</td>
<td>3.3</td>
<td>45.9</td>
</tr>
<tr>
<td>A point mutation is a type of mutation that causes a single nucleotide base substitution, insertion, or deletion.</td>
<td>0.44 ± 0.50 (2719)</td>
<td>43.9</td>
<td>3.5</td>
<td>52.7</td>
</tr>
<tr>
<td>An example of a genotype that is heterozygous is AA.</td>
<td>0.48 ± 0.50 (2719)</td>
<td>47.7</td>
<td>18.4</td>
<td>34.0</td>
</tr>
<tr>
<td>An example of a genotype that is homozygous is cc.</td>
<td>0.59 ± 0.49 (2719)</td>
<td>59.2</td>
<td>6.3</td>
<td>34.4</td>
</tr>
<tr>
<td>Mutations can create variations in protein “switches” that control protein function.</td>
<td>0.49 ± 0.50 (2719)</td>
<td>49.2</td>
<td>3.3</td>
<td>47.4</td>
</tr>
<tr>
<td>Mutations cannot be reversed through DNA repair.</td>
<td>0.26 ± 0.44 (2719)</td>
<td>26.4</td>
<td>18.1</td>
<td>55.5</td>
</tr>
<tr>
<td>A recessive trait can be carried in a person’s genes without appearing in their phenotype.</td>
<td>0.78 ± 0.42 (2719)</td>
<td>77.9</td>
<td>3.4</td>
<td>18.7</td>
</tr>
</tbody>
</table>

*Note.* General knowledge score was scored by identification of the correct answer as a score of 1. Incorrect answers were scored as a score of 0.

*Abbreviations.* SD, standard deviation; n, number of members in sample.
Table 3
Differences in Genetics Knowledge Scores according to College Genetics and/or Nutrition Course Participation

<table>
<thead>
<tr>
<th>College Course Participation</th>
<th>Mean ± SD</th>
<th>n</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nutrition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>10.88 ± 4.29</td>
<td>809</td>
<td>0.002*</td>
</tr>
<tr>
<td>No</td>
<td>10.28 ± 4.52</td>
<td>1957</td>
<td></td>
</tr>
<tr>
<td>Genetics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>14.67 ± 4.20</td>
<td>411</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td>No</td>
<td>9.72 ± 4.48</td>
<td>2355</td>
<td></td>
</tr>
</tbody>
</table>

Note. General knowledge score was scored by identification of the correct answer as a score of 1. Incorrect answers were scored as a score of 0. Total scores were calculated from the sum of correct responses. Total possible score = 19.0
*Show t-test statistical significance, where statistical significance was set at p ≤ 0.05.
Abbreviations. SD, standard deviation; n, number of members in sample.

Table 4
Differences in Genetics Knowledge Scores according to College Class Rank

<table>
<thead>
<tr>
<th>Class Rank</th>
<th>Mean ± SD</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Freshman</td>
<td>10.09 ± 4.15</td>
<td>500</td>
</tr>
<tr>
<td>Sophomore</td>
<td>10.62 ± 4.20</td>
<td>483</td>
</tr>
<tr>
<td>Junior</td>
<td>10.83 ± 4.33a</td>
<td>457</td>
</tr>
<tr>
<td>Senior</td>
<td>10.77 ± 4.54b</td>
<td>645</td>
</tr>
<tr>
<td>Graduate Student</td>
<td>10.07 ± 4.82ab</td>
<td>681</td>
</tr>
<tr>
<td>Total</td>
<td>10.46 ± 4.46</td>
<td>2766</td>
</tr>
</tbody>
</table>

Note. General knowledge score was scored by identification of the correct answer as a score of 1. Incorrect answers were scored as a score of 0. Total scores were calculated from the sum of correct responses. Total possible score = 19.

a = statistically significant difference between juniors and graduate students, (p =0.038); b = statistically significant difference between seniors and graduate students.
Abbreviations. SD, standard deviation; n, number of members in sample.
### Table 5
**Differences in Genetics Knowledge Scores according to College Major**

<table>
<thead>
<tr>
<th>Class Rank</th>
<th>Mean ± SD</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Health and Medicine</td>
<td>11.57 ± 4.21&lt;sup&gt;a,b,c&lt;/sup&gt;</td>
<td>674</td>
</tr>
<tr>
<td>Business, Math, Science And Technology</td>
<td>10.43 ± 4.94&lt;sup&gt;a,d&lt;/sup&gt;</td>
<td>816</td>
</tr>
<tr>
<td>Social Science</td>
<td>10.05 ± 4.24&lt;sup&gt;b&lt;/sup&gt;</td>
<td>821</td>
</tr>
<tr>
<td>Arts, Humanities &amp; Others</td>
<td>9.60 ± 4.00&lt;sup&gt;c,d&lt;/sup&gt;</td>
<td>455</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>10.46 ± 4.46</td>
<td>2766</td>
</tr>
</tbody>
</table>

*Note.* General knowledge score was scored by identification of the correct answer as a score of 1. Incorrect answers were scored as a score of 0. Total scores were calculated from the sum of correct responses. Total possible score = 19.

<sup>a</sup> = statistically significant difference between health and medicine and business, math, science and technology, (p ≤ 0.001); <sup>b</sup> = statistically significant difference between health and medicine and social sciences, (p ≤ 0.001); <sup>c</sup> = statistically significant difference between health and medicine and arts, humanities, and others (p ≤ 0.001); <sup>d</sup> = statistically significant difference between business, math, science, and technology and arts, humanities, and others, (p = 0.007).

**Abbreviations.** SD, standard deviation; N, number of members in sample.

### Awareness and Perceptions Toward Nutrigenomic Testing

When students were asked if they were familiar with genetic testing for personalized nutrition, only about 25% reported some familiarity with nutrigenomic testing. The remaining 75% of participants indicated they had never heard or read about these tests. Results from assessment of perceptions regarding genetic testing for personalized nutrition are displayed in Table 6. On a modified 5-point Likert style scale, ranging from (1) ‘strongly disagree’ to (5) ‘strongly agree’ with (3) indicating ‘neither agree nor disagree’, participants most strongly agreed with the statement, “Genetic
testing for personalized nutrition will lead to the prevention of some diseases,” with a mean response of $3.93 \pm 0.69$ (n=2899). Meanwhile, participants were least concerned with statements regarding religious beliefs, discrimination, and privacy. Students showed most concern with cost and availability of nutrigenomic tests, and about 44% of participants agreed with the statement, “Nutrigenomic testing would cost too much and would only be available to the rich.”

Independent sample t-tests were conducted comparing mean perception scores based on gender and between participants who indicated some familiarity with nutrigenomic testing and those who indicated no familiarity. There was no significant difference in perception of nutrigenomics between genders ($p \geq 0.05$), however, participants who were familiar with nutrigenomic testing had a significantly more positive perception towards nutrigenomics than participants who indicated no awareness, ($p \leq 0.001$).

Independent sample t-tests (Table 7) were also used to compare the mean perception scores between participants who indicated participation in a college level nutrition and/or genetics course and those who did not. There was a significant effect for students who indicated participation in a college level nutrition course, ($p = .004$), with those who indicated participation receiving higher scores on the genetics knowledge assessment. Students who participated in a nutrition course had a mean score of 3.63. Similarly, students who indicated participation in a college level genetics course also
scored significantly higher on the genetics knowledge assessment, \( (p = 0.003) \), with a mean score of 3.66.

Mean nutrigenomic perception scores according to class rank (freshman, sophomores, junior, senior, graduate student) are highlighted in Table 8. One way ANOVA revealed a significant difference in the perception scores between the five class ranking groups \( (p = 0.001) \) and Tukey test was used to detect where significant differences were found. Juniors \( (p = 0.038) \) and seniors \( (p = 0.005) \) scored significantly higher than freshman, indicating more positive perceptions. Seniors also scored significantly higher than graduate students \( (p = 0.014) \), indicating more positive perceptions.

A one-way ANOVA was conducted comparing perceptions of genetic testing for personalized nutrition among majors and results are reported in Table 9. Majors were categorized into four groups including; 1) Health and Medicine, 2) Social Sciences 3) Business, Math, Science & Technology, and 4) Arts, Humanities, & Others. A statistically significant difference was found among majors, \( F(3, 2895)=8.17, p \leq 0.001 \) and Tukey post-hoc test was used to see where significant differences were found. While there was no significant difference in perception among ‘Health and Medicine’ majors and ‘Business, Math, Science, and Technology’ majors \( (p \geq 0.05) \), ‘Health and Medicine’ majors had a significantly more positive perception than ‘Social Science’ \( (p = .002) \) and ‘Arts and Other’ \( (p = .003) \) majors. Overall ‘Health and Medicine’ majors (n=705) had the most positive perception toward nutrigenomics, while ‘Art and Other’
majors (n=477) had the least positive perception.

Table 6
Perceptions of Nutrigenomic Testing among College Students

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean ± SD</th>
<th>A/SA (n)</th>
<th>NEU (n)</th>
<th>D/SD (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Positives/Benefits</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knowing their own genetics allows individuals to control their lifestyle more easily.</td>
<td>3.85 ± 0.82</td>
<td>75.9 (2199)</td>
<td>17.9 (520)</td>
<td>6.2 (180)</td>
</tr>
<tr>
<td>Genetic testing for personalized nutrition will help people to live longer.</td>
<td>3.78 ± 0.77</td>
<td>69.8 (2023)</td>
<td>25 (724)</td>
<td>5.2 (152)</td>
</tr>
<tr>
<td>All individuals should be offered genetic testing for personalized nutrition by their family doctor.</td>
<td>3.65 ± 0.91</td>
<td>62.1 (1800)</td>
<td>27.0 (784)</td>
<td>10.9 (315)</td>
</tr>
<tr>
<td>Genetic testing for personalized nutrition will make medical cures for diseases more possible.</td>
<td>3.80 ± 0.80</td>
<td>71.6 (2076)</td>
<td>22.2 (643)</td>
<td>6.2 (180)</td>
</tr>
<tr>
<td>Genetic testing for personalized nutrition should be promoted extensively.</td>
<td>3.33 ± 0.83</td>
<td>38.8 (1130)</td>
<td>48.7 (1412)</td>
<td>12.3 (357)</td>
</tr>
<tr>
<td>I believe it is essential to assign more money to nutrigenomic developments.</td>
<td>3.19 ± 0.81</td>
<td>31.1 (901)</td>
<td>53.9 (1562)</td>
<td>15 (436)</td>
</tr>
<tr>
<td>Screening for known genes is the way forward for medicine and nutrition.</td>
<td>3.80 ± 0.75</td>
<td>71.2 (2065)</td>
<td>23.9 (692)</td>
<td>4.9 (142)</td>
</tr>
<tr>
<td>Gene testing for personalized nutrition will lead to the prevention of some diseases.</td>
<td>3.93 ± 0.69</td>
<td>80.1 (2321)</td>
<td>16.9 (489)</td>
<td>3.0 (89)</td>
</tr>
</tbody>
</table>
Table 6 (continued)

*Perceptions of Nutrigenomic Testing among College Students*

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean ± SD&lt;sup&gt;a&lt;/sup&gt;</th>
<th>% A/SA (n)</th>
<th>% NEU (n)</th>
<th>% D/SD (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Positives/Benefits</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In my lifetime, I expect to see significant medical improvements due to the use of genetics in nutrition.</td>
<td>3.83 ± 0.81</td>
<td>70.8 (2053)</td>
<td>23.7 (688)</td>
<td>5.5 (153)</td>
</tr>
<tr>
<td>My genes have influenced my health.</td>
<td>3.86 ± 0.87</td>
<td>71.7 (2078)</td>
<td>21.4 (621)</td>
<td>6.9 (200)</td>
</tr>
<tr>
<td>I would like to know about future diseases through genetic testing.</td>
<td>3.89 ± 0.84</td>
<td>75.8 (2199)</td>
<td>17.8 (515)</td>
<td>6.4 (185)</td>
</tr>
<tr>
<td>Genetic testing for personalized nutrition should be available to everyone.</td>
<td>3.85 ± 0.86</td>
<td>71.6 (2075)</td>
<td>22.0 (638)</td>
<td>6.4 (186)</td>
</tr>
<tr>
<td><strong>Negatives/Risks</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genetic testing for personalized nutrition will result in discrimination.</td>
<td>2.42 ± 0.89</td>
<td>11.5 (332)</td>
<td>30.5 (885)</td>
<td>58 (1682)</td>
</tr>
<tr>
<td>Nutrigenomic testing would cost too much and would only be available to the rich.</td>
<td>3.32 ± 0.95</td>
<td>43.9 (1273)</td>
<td>38.4 (1112)</td>
<td>17.7 (514)</td>
</tr>
<tr>
<td>I am worried that genetic testing may lead to eugenics (the science of improving the human population by controlling breeding to increase desirable characteristics)</td>
<td>2.97 ± 1.10</td>
<td>34.0 (985)</td>
<td>29.8 (863)</td>
<td>36.2 (1051)</td>
</tr>
<tr>
<td>I am scared of finding out the results of genetic testing for personalized nutrition.</td>
<td>2.39 ± 1.09</td>
<td>19.5 (563)</td>
<td>19.5 (564)</td>
<td>61.1 (1772)</td>
</tr>
<tr>
<td>I do not believe that genetic testing for personalized nutrition is backed by sound science.</td>
<td>2.45 ± 0.84</td>
<td>7.6 (220)</td>
<td>40.9 (1186)</td>
<td>51.5 (1493)</td>
</tr>
</tbody>
</table>
Table 6 (continued)

*Perceptions of Nutrigenomic Testing among College Students*

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean ± SD&lt;sup&gt;a&lt;/sup&gt;</th>
<th>% A/SA (n)</th>
<th>% NEU (n)</th>
<th>% D/SD (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Negatives/Risks</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I am concerned that not enough will be done to protect the confidentiality and privacy of my genetic information.</td>
<td>2.78 ± 1.12</td>
<td>27.6 (800)</td>
<td>27.9 (809)</td>
<td>44.5 (1290)</td>
</tr>
<tr>
<td>Genetic testing for personalized nutrition goes against my religious beliefs.</td>
<td>1.70 ± 0.86</td>
<td>3.5 (101)</td>
<td>14.1 (409)</td>
<td>82.4 (2389)</td>
</tr>
<tr>
<td>I am concerned that my genetic information will be made available for research purposes.</td>
<td>2.70 ± 1.08</td>
<td>24.6 (711)</td>
<td>28.1 (814)</td>
<td>47.3 (1374)</td>
</tr>
<tr>
<td>Genetic testing for personalized nutrition is an invasion of my privacy.</td>
<td>2.19 ± 0.93</td>
<td>8.4 (245)</td>
<td>23.1 (669)</td>
<td>68.5 (1985)</td>
</tr>
<tr>
<td>I think there is too much focus on genetics when money could be spent on the world’s starving population.</td>
<td>2.74 ± 1.00</td>
<td>20.3 (587)</td>
<td>39.7 (1152)</td>
<td>40.0 (1160)</td>
</tr>
</tbody>
</table>

<sup>a</sup> = calculated from a 5-point Likert style scale where (1) equals *strongly disagree* and (5) equals *strongly agree.*

*Abbreviations.* A/SA, agree/strongly agree; NEU, neutral; D/SD, disagree/strongly disagree; SD, standard deviation; (n), number of members in sample.
Table 7

*Differences in Perceptions of Nutrigenomic Testing according to College Genetics and/or Nutrition Course Participation*

<table>
<thead>
<tr>
<th>College Course Participation</th>
<th>Mean ± SD</th>
<th>n</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nutrition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3.63 ± 0.44</td>
<td>848</td>
<td>0.004*</td>
</tr>
<tr>
<td>No</td>
<td>3.58 ± 0.46</td>
<td>2051</td>
<td></td>
</tr>
<tr>
<td>Genetics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3.66 ± 0.43</td>
<td>423</td>
<td>0.003*</td>
</tr>
<tr>
<td>No</td>
<td>3.59 ± 0.46</td>
<td>2476</td>
<td></td>
</tr>
</tbody>
</table>

*Note. Mean scores were calculated from a 5-point Likert style scale where (1) equals *strongly disagree* and (5) equals *strongly agree.*

*Show t-test statistical significance, where statistical significance was set at p ≤ 0.05.

*Abbreviations. SD, standard deviation; n, number of members in sample.*

Table 8

*Differences in Perception of Nutrigenomic Testing according to College Class Ranks*

<table>
<thead>
<tr>
<th>Class Rank</th>
<th>Mean ± SD(^a)</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Freshman</td>
<td>3.55 ± 0.44(^{a,b})</td>
<td>536</td>
</tr>
<tr>
<td>Sophomore</td>
<td>3.60 ± 0.48</td>
<td>510</td>
</tr>
<tr>
<td>Junior</td>
<td>3.63 ± 0.44(^a)</td>
<td>482</td>
</tr>
<tr>
<td>Senior</td>
<td>3.64 ± 0.45(^{b,c})</td>
<td>669</td>
</tr>
<tr>
<td>Graduate Student</td>
<td>3.56 ± 0.44(^d)</td>
<td>702</td>
</tr>
</tbody>
</table>

*Note. Statistical significance was set at p ≤ 0.05. Mean scores were calculated from a 5-point Likert style scale where (1) equals *strongly disagree* and (5) equals *strongly agree.*

\(^a\) = statistically significant difference between Freshman and Juniors, (p = 0.038); \(^b\) = statistically significant difference between freshman and seniors, (p = 0.005); \(^c\) = statistically significant difference between seniors and graduate students, (p = 0.014).

*Abbreviations. SD, standard deviation; n, number of members in sample.*
Table 9

| Differences in Perception of Nutrigenomic Testing according to College Class Major |
|-----------------|-----------------|--------|
| Major            | Mean ± SD^a      | n      |
| Health and Medicine | 3.64 ± 0.45^a,b  | 705    |
| Business, Math, Science & Tech | 3.63 ± 0.46^b,e  | 849    |
| Social Sciences  | 3.56 ± 0.44^c,e  | 868    |
| Arts, Humanities, & Others | 3.54 ± 0.45^b,e  | 477    |
| Total            | 3.60 ± 0.45      | 2899   |

Note. Statistical significance was set at \( p \leq 0.05 \). Mean scores were calculated from a 5-point Likert style scale where (1) equals strongly disagree and (5) equals strongly agree.

^a = statistically significant difference between health and medicine and social sciences, \( p = 0.002 \); ^b = statistically significant difference between health and medicine and arts, humanities and others \( p \leq 0.001 \); ^c = statistically significant difference between business, math, science, and technology and social sciences, \( p = 0.002 \); ^d = statistically significant difference between business, math, science and technology and arts, humanities, and others \( p = 0.003 \).

Abbreviations. SD, standard deviation; n, number of members in sample.

Factors Influencing Decision to Participate in Nutrigenomic Testing

This section of the survey addressed common factors affecting participation in genetic testing for personalized nutrition. Results from this assessment are displayed in Table 9. On a modified 5-point Likert style scale, ranging from (1) ‘not at all likely’ to (5) ‘completely likely,’ with (3) indicating ‘moderately likely,’ participants were most likely to be encouraged to participate in genetic testing because of family history of a particular disease. The most common reason they would not participate, as indicated by survey responses, was lack of money to pay for testing or possible treatments.

Participants scored lowest against participation on factors regarding beliefs, privacy concerns, or thinking it’s useless.
Independent sample t-tests were conducted (Table 10) comparing the likelihood of factors to influence participation in nutrigenomic testing between males and females. There was a significant difference between genders on all factors \((p \leq 0.05)\), except “I think it’s useless,” \((p = 0.345)\). Overall, female respondents indicated that they were significantly more encouraged to participate than male respondents by factors including; ‘Anxiety about health,’ ‘Doctor’s recommendation,’ ‘Availability of more detailed information,’ ‘Curiosity,’ ‘Family or friend’s advice,’ and ‘Family history of a particular disease’. While both males and females indicated that they were more influenced toward undergoing testing than not, males reported being more discouraged by factors including, ‘I think it’s useless,’ ‘It goes against my beliefs,’ and ‘It’s in invasion of my privacy,’ than female counterparts. Females were more discouraged the factor, ‘Lack of money to pay for testing or possible treatments,’ than male counterparts.

Among free answer responses, many students indicated that they might not want to take a nutrigenomic test due to limited understanding, interest, and evidence of accurate results. Other common barriers included worry about inappropriate use of genetic information, especially by insurance companies. Common reasons for wanting to undergo a nutrigenomic test were improved health, fitness, and quality of life.
Table 10
Factors that Influence Decision on Participation in Nutrigenomic Testing

<table>
<thead>
<tr>
<th>Item</th>
<th>Gender</th>
<th>Mean ± SD</th>
<th>N</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Encouragers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety about health</td>
<td>Male</td>
<td>2.48 ± 1.19</td>
<td>731</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>2.79 ± 1.19</td>
<td>2048</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>2.71 ± 1.20</td>
<td>2816</td>
<td></td>
</tr>
<tr>
<td>Doctor’s Recommendation</td>
<td>Male</td>
<td>3.16 ± 1.11</td>
<td>732</td>
<td>0.008*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>3.28 ± 1.07</td>
<td>2048</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>3.25 ± 1.08</td>
<td>2817</td>
<td></td>
</tr>
<tr>
<td>Availability of more detailed information</td>
<td>Male</td>
<td>3.41 ± 1.06</td>
<td>732</td>
<td>0.004*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>3.54 ± 1.03</td>
<td>2046</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>3.50 ± 1.04</td>
<td>2815</td>
<td></td>
</tr>
<tr>
<td>Curiosity</td>
<td>Male</td>
<td>3.44 ± 1.19</td>
<td>732</td>
<td>0.036*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>3.54 ± 1.12</td>
<td>2048</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>3.52 ± 1.14</td>
<td>2817</td>
<td></td>
</tr>
<tr>
<td>Family or friend’s advice</td>
<td>Male</td>
<td>2.68 ± 1.10</td>
<td>731</td>
<td>0.044*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>2.77 ± 1.10</td>
<td>2048</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>2.74 ± 1.10</td>
<td>2818</td>
<td></td>
</tr>
<tr>
<td>Family history of a particular disease</td>
<td>Male</td>
<td>3.63 ± 1.13</td>
<td>732</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>3.92 ± 1.03</td>
<td>2049</td>
<td></td>
</tr>
<tr>
<td><strong>Discouragers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lack of money to pay for testing or possible</td>
<td>Male</td>
<td>3.05 ± 1.28</td>
<td>731</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td>treatments</td>
<td>Female</td>
<td>3.31 ± 1.30</td>
<td>2045</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>3.25 ± 1.30</td>
<td>2813</td>
<td></td>
</tr>
<tr>
<td>Lack of time</td>
<td>Male</td>
<td>2.59 ± 1.17</td>
<td>730</td>
<td>0.016*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>2.71 ± 1.18</td>
<td>2046</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>2.68 ± 1.18</td>
<td>2813</td>
<td></td>
</tr>
<tr>
<td>Fear to discover results</td>
<td>Male</td>
<td>2.08 ± 1.21</td>
<td>731</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>2.28 ± 1.19</td>
<td>2048</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>2.22 ± 1.20</td>
<td>2816</td>
<td></td>
</tr>
<tr>
<td>I think it’s useless</td>
<td>Male</td>
<td>1.67 ± 1.01</td>
<td>730</td>
<td>0.345</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>1.63 ± 0.92</td>
<td>2045</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>1.64 ± 0.94</td>
<td>281</td>
<td></td>
</tr>
</tbody>
</table>
Table 10 (continued)

*Factors that Influence Decision on Participation in Nutrigenomic Testing*

<table>
<thead>
<tr>
<th>Item</th>
<th>Gender</th>
<th>Mean ± SD</th>
<th>N</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Discouragers</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>It goes against my beliefs</td>
<td>Male</td>
<td>1.40 ± 0.89</td>
<td>731</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>1.27 ± 0.69</td>
<td>2046</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>1.31 ± 0.75</td>
<td>2813</td>
<td></td>
</tr>
<tr>
<td>It is an invasion of privacy</td>
<td>Male</td>
<td>1.69 ± 1.03</td>
<td>731</td>
<td>0.012*</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>1.58 ± 0.90</td>
<td>2043</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>1.91 ± 1.34</td>
<td>2811</td>
<td></td>
</tr>
</tbody>
</table>

*Note.* The mean was calculated from data from a 5-point Likert style scale where one equals ‘Not at all likely’ and five equals ‘completely likely’. *Show statistical significance, where statistical significance was set at p ≤ 0.05.

*Abbreviations.* SD, standard deviation; N, number of members in sample.

**Discussion**

The primary purpose of this descriptive study was to examine the knowledge and perception of college students toward genetic testing for personalized nutrition. Study results indicated: 1) there was a general lack of genetics knowledge among college students, therefore Hypothesis 1 was accepted; 2) college students perceive genetic testing for personalized nutrition to have benefits that outweigh risks, therefore Hypothesis 2 was accepted; 3) there was no significant difference between males and females in the perception of genetic testing for personalized nutrition; therefore Hypothesis 3 was not accepted.
Characteristics of Study Population

Demographic data collected from this study is congruent with a Spring 2017 University report, which revealed that: 1) the majority (83.8%) of students are undergraduate 2) seniors make up the largest (27.3%) class level when compared to freshman, sophomores, juniors, and graduate students, and 3) the majority of students are between the ages of twenty-three and twenty-seven (Institutional Research, 2017).

In the current study, 29.3% of participants reported that they had participated in a college level nutrition course and 14.6% reported participation in a college level genetics course. This result mirrors findings from the Human Genome Education Model Project, where 80% of 3,600 health care professionals reported no formal education in genetics in either their graduate or undergraduate programs (Lapham et al., 2000). These findings are indicative of the need to for basic genetics education at the college level. In addition, these results imply that knowledge may be lacking in health care professionals due to limited availability of college genetics courses.

Awareness and Knowledge of Nutrigenomics

Only about one quarter of college students reported that they had heard or read about nutrigenomic testing. Data from the present study is representative of previous, similar research among Canadian consumers, health care professionals, and members of the public, which also revealed limited awareness and a lack of general knowledge about current nutrigenomic processes (Goddard et al., 2007; Lapham et al, 2000; Morin, 2009). Compared to a national survey completed in the United States, where only 14% of respondents indicated awareness of nutrigenomic tests, the present study indicates greater
awareness among college students (24.9%). In the same national survey, education and age less than 55 years old were significant independent predictors of familiarity with nutrigenomics (Goddard et al., 2007). Since college students are typically younger and at a higher academic standing than most Americans, this may explain the increased awareness of college students toward nutrigenomic testing and may also suggest among that they are a well-suited population of potential consumers and/or advocates.

In the present study, college students answered an average of 55% of genetics knowledge questions correctly, indicating a general lack of genetics knowledge among this population. Other survey research conducted in the Netherlands, revealed that about one half to three fourths of patients with chronic diseases reported having little or no knowledge about genetics with older respondents reporting significantly less than younger counterparts (Morren et al., 2007). Based on these findings, it can be speculated that older adults in the United States may have less knowledge regarding nutrigenomics than a younger population, like the one included in the present study. In other survey research of 600 dietitians in the UK, also assessing genetics knowledge, scores were generally poor, averaging 41% correct on a validated multiple choice test (Whelan et al., 2008). Similarly, in a study conducted by the Academy of Nutrition and Dietetics, only about half of dietitian respondents (n= 913), indicated clarity regarding the definition of nutrigenomics (Rosen et al., 2006). The same patterns can be seen among general practitioners, gynecologists, and pediatricians, where scores averaged 40%, 52%, and 62% correct, respectively (Baars, Henneman, & Kate, 2005). These findings support previous findings of significant gaps in genetics knowledge among health care
professionals (Burke, Stone, Bedward, Thomas & Farndon, 2006) and imply that college programs could be an appropriate target for filling these gaps and establishing a foundation of background genetics knowledge.

The present study also revealed that those students who indicated they had participated in a college level genetics course, displayed a higher understanding of basic genetics concepts on the knowledge assessment. These findings are in line with survey research among gynecologists in the United States, where physicians with formal education in genetics scored significantly higher on a series of genetics knowledge questions than those with no training (Wilkins-Haung, Hill, Power, Holzman, & Szchulkin). This discovery is important because other surveys among health-care professionals indicate a lack of confidence in providing genetics services among those with no genetics education, therefore affirming the value of providing genetics education starting at the college level (Grant et al., 2009; Lapham et al 2000; Rosen et al., 2006; Whelan et al., 2008).

Overall, findings from the current study indicate a low level of participation in genetics education at the college level along with an overall lack of genetics knowledge among college students. This may help to explain the deficiency in genetics and nutrigenomics knowledge among trusted health care providers, an issue affirmed by multiple other studies. The relationship between genetics education and genetics knowledge in college students, as found in the present study, in combination with previous reports of low confidence among physicians without genetics education, affirms
the value of genetics education in college programs and beyond, especially for those who will directly apply nutrigenomics in clinical settings.

In the present study participants within higher class ranks (graduate students) scored lower on the genetics knowledge assessment than those in lower class ranks (undergraduate students). While this result was somewhat unexpected, it may be explained by the broad nature of undergraduate programs, which typically include courses from a wide variety of subjects, perhaps giving undergraduate students more current exposure to concepts in genetics. Alternatively, lower scores among graduate students could be explained by their more narrowed focus of study and limited recent exposure, as they pursue more specialized degrees. Another possible explanation for the differences in genetics knowledge among class ranks may be related to the proportion of undergraduate and graduate participants with science and health backgrounds. For example, the average knowledge score for graduate students may have been influenced by a lesser proportion of participants with non-health and medicine related educational backgrounds.

Also notable in the present study, students majoring in health or medical related fields outperformed students with majors in business, math, technology, social sciences, arts, humanities, and other majors. These findings make good sense considering the higher probability of health and medical related studies to include genetic disciplines than those focused in other area. Specifically, at Kent State University, many biology, biomedical, pre-medicine, pre-dentistry, nursing, and other programs require genetics courses, while many other programs lack genetics prerequisites.
Perceptions of Nutrigenomic Testing

To the authors’ knowledge, the present study is the first one to reveal a supportive attitude toward nutrigenomic concepts among college students. Overall, participants indicated stronger agreement with positive statements regarding nutrigenomic testing, while they agreed less with negative statements. Some previous studies have suggested support for genetic testing (Jallinoja et al., 1998; Hakonen et al., 1995; Decruyenaere et al., 1993), but many of these are not specific to the use of genetic testing in nutrition care. Therefore, the present study was designed to gauge attitudes regarding ethical, legal, and social implications and to determine whether college students perceive the benefits of nutrigenomic testing to outweigh the risks commonly associated. In this study, college students most strongly agreed with the statement, “Genetic testing for personalized nutrition will lead to the prevention of some diseases,” and were most concerned that, nutrigenomic testing would cost too much and would only be available to the rich. Overall, participants were most inclined to agree with statements regarding the possibility of nutrigenomic testing to aid in disease prediction and prevention and indicated less agreement with statements about promotion and assignment of more money to nutrigenomic developments. This result mirrors findings from other survey research studies, where both patients and physicians reported that genetic tests would have a significant impact on motivating healthier behavior changes (Grant et al., 2009), and where cost was the most prominent barrier to using genetic testing (Makeeva et al., 2009; Cherkas et al., 2010). The present study and similar past research imply that college students along with other consumers are concerned that genomic testing may be overly
expensive, and excessive cost could be a critical factor affecting decisions to use nutrigenomic technology as a tool for nutrition care (Cherkas et al., 2010; Fallaize et al., 2013).

Privacy issues, religious beliefs, and fear to discover disease susceptibility were all concerns indicated as obstacles to nutrigenomics by previous research studies, (Fallaize et al., 2013; Haga et al., 2013; Morin, 2009; Morren et al., 2006), however, results from the present study reveal that these are not major concerns for college students. In fact, college students most strongly disagreed with the statement, “Genetic testing for personalized nutrition goes against my religious beliefs,” and also demonstrated limited agreement with the statement, “I am concerned that not enough will be done to protect the confidentiality and privacy of my genetic information.” These results may demonstrate an open mindset with greater focus on possible benefits that nutrigenomic testing could provide to nutrition care, again making college students a potential group of supporters and consumers.

In the present study, there was a significant correlation between genetics knowledge and perceptions toward nutrigenomics. This finding is similar to one found among chronic disease patients, where patients who perceived having more knowledge were also more enthusiastic about nutrigenomics (Morren et al., 2006). This relationship between knowledge and positive attitudes implies that, investment of resources in raising consumer awareness may be beneficial as nutrigenomic tests become more available.
Factors Influencing Decision to Participate in Nutrigenomic Testing

Previous research has suggested that many factors influence individuals and populations as they decide to partake in the use of new technologies, and that it is when they perceive potential benefits to outweigh potential risks, that they are more likely to engage in an associated behavior (Frewar et al., 2004; AFFECT). Since nutrigenomics research is limited, the present study is the first to examine which factors might influence the decision of college students to participate in nutrigenomic testing. Results from the present study are in accordance with findings from previous research, which highlighted a range of factors that could either encourage or discourage participation (Cappelli et al., 1999; Jallinoja et al., 1998; Hietala et al., 1993). However, in the present study, college students reported being more strongly encouraged to use nutrigenomic tests than discouraged against their use. In addition, this research found that students were most encouraged by ‘Family history of a particular disease’ and most strongly discouraged by ‘Lack of money to pay for testing or possible treatments.’ This finding agrees with another study, where the percentage of participants who indicated they would be willing to take a genetic test dropped from 48% to 5% after being told it would cost £250 (Cherkas et al., 2010). While college students may show interest, and recognize the value of nutrigenomic advances, cost is a major issue for this population and others. These results collaboratively highlight the need to develop more affordable means of discovering genetic susceptibility to disease so that nutritional genomics can be applied to prevention of chronic disease across all socioeconomic groups.
Limitations

As with any research study, there are limitations that exist within the construct and execution of this study. The first limitation is the validity and reliability of the questionnaire, which utilized questions that were developed by the researcher. Validity and reliability studies on this questionnaire have not been evaluated. The second limitation of this study was that data was self-reported by participants, and therefore, validity of responses cannot be guaranteed. A third limitation of this study is that since participation was voluntary, respondents may have been more interested and/or knowledgeable in genetic related questions than non-respondents. Finally, there were no questions regarding participants’ family history of chronic disease, which has the possibility of being a significant variable in perception and knowledge of chronic disease prevention approaches, such as nutrigenomics. This study, if replicated, could be improved by investigating participants’ family history of chronic disease and its affect on nutrigenomic knowledge and perception. In addition, readability of some survey questions should be addressed to improve clarity. Lastly, validity and reliability studies on all questions would improve the credibility of this research.

Applications

In the current study, less than 25% of college students reported that they had ever heard or read about nutrigenomic tests, which places emphasis on the need to expand awareness about this field. It is possible that more exposure to nutrigenomics at the college level, could improve awareness among this population. In the current study, college students scored an average of 55% on a general genetics knowledge test,
indicating limited knowledge about basic concepts in genetics. In addition, less than 30% of students reported that they had ever taken a college level nutrition course, and only 15% a genetics course. As past research has demonstrated deficiencies in genetics knowledge among health care professionals, a greater emphasis on genetics training in pre-professional college programs is warranted, especially for students in health or medical related fields of study. More education at this level could benefit both patients and providers, as genomic technology will likely continue to permeate nutrition and medical practices in coming years.

In addition, there is a need for educational efforts from alternative sources, such as professional organizations and government agencies, who have a responsibility to provide reliable information for all members of the public. Results from the current study illustrate that more awareness and knowledge about nutrigenomic testing may translate into a more positive outlook about nutrigenomic developments. For this reason, companies, health care professionals, and scientists need to work together to communicate where gaps in knowledge currently exist, so that young adults can make informed decisions and take appropriate precautions as these nutrigenomic tests become increasingly available. With this, educating college students about nutrigenomics could also promote greater interest in the subject, leading to opportunities for larger scale projects made possible through publicly funded research, such as the Human Genome Project.

Another important consideration, highlighted in this, and previous studies, affirms the need for more affordable methods to translate genomic information into personalized
dietary advice. Students reported most concern on multiple survey questions with the high cost of these genetic tests, supporting previous study findings. This should be considered by suppliers who could possibly provide services at a more conservative price as well as researchers who may consider digging deeper into the efficacy and cost effectiveness of applying genetic data in nutrition care. Professionals should realize if the adoption of this new technology adds significant cost to already expensive health care costs, individuals may question whether the cost of a nutrigenomic test would justify its use over more mainstream ‘trial and error’ approaches to nutrition care. A strategy for overcoming this obstacle may be to advocate for reimbursement by insurance companies, which could greatly increase availability of nutrigenomic tests across clinical settings. Successful implementation of personalized nutrition care will depend on the ability of nutrigenomic providers to more cost-effectively test an individual’s genome and create individualized nutrition prescriptions.

As it is expected that nutrigenomic tests will become more mainstream in medical and nutrition care, certain ethical issues will need to be addressed to protect the well-being of the public (Bergmann, Görman, & Mathers, 2008). Given the high cost of nutrigenomic tests, there is a risk for widening disparities in health care, which are already negatively effecting the health of our nation. Limiting availability of nutrigenomic tests among population groups due to cost, will likely increase barriers to accessing quality care and lead to worse health outcomes among these people. In addition, higher health disparities could limit overall improvements in the quality of care across the nation, leading to unnecessary costs. Since many ethnic and low-income
groups already receive poorer quality of care and suffer from worse health outcomes, government agencies need to understand their duty to prioritize the interests of all groups over those of corporations who offer these tests. Since the commercialization of nutrigenomic tests in the U.S. is expanding, a harmonized protocol for the nutrigenomic process as well as validation requirements will need to be established to ensure high quality and accurate results of all tests. To address these issues, scientific initiatives and legislation will need to be developed and implemented.

**Conclusion**

Experts believe that there is great potential for nutrigenomics to lead to evidence-based dietary intervention strategies that will restore health and fitness and reduce the onset and impact of complex diseases that currently account for nearly two-thirds of the world’s deaths (Afman & Muller, 2006; Bauer et al., 2014). However, due to multiple barriers, and especially excessive cost to consumers, public support of “omics” technology is unclear. With millennial college students making up the largest generation is U.S. history, the knowledge and perceptions of this population may be crucial to forward progress in this developing field. This study and previous studies aimed at investigating and communicating the current perceptions of an influential generation about nutrigenomics. Examining these discoveries could help researchers and health care providers to develop strategies, realize future opportunities, and overcome potential challenges, allowing nutrigenomic technology to flourish in the coming years.

To successfully use genetic blue-prints for dietary prevention of disease, researchers and scientists must uncover the mechanisms driving the connection between
diet and the outward expression of our genes, (Afman & Muller, 2006) and then determine how these dietary interventions translate into improved health outcomes. As these goals are achieved and nutrigenomic tests become more available, scientific initiatives and legislation should will need to be developed to address ethical concerns and protect public interest. Additional research is needed to more clearly understand how genetic testing for personalized nutrition is perceived by college students and other populations. Furthermore, researchers need to more accurately interpret the interaction between genes and diet and establish evidence-based practices for using nutrigenomic technology, so that health-care professionals and consumers can find confidence in this scientific field.
APPENDIX A

CONSENT FORM
Appendix A
Consent Form

Knowledge and Perceptions toward Genetic Testing For Personalized Nutrition

Thank you for your interest in taking this online questionnaire. Before taking part in this study, please read the consent form below and click on the "I Agree" button at the bottom of the page if you understand the statements and freely consent to participate in the study.

Consent Form

This study involves a web-based questionnaire designed to understand how college students perceive the use of genetic testing in order to develop personalized nutrition regimens. This study is being conducted by Professor Eun-Jeong Ha, an associate professor of Nutrition & Dietetics, and Julianne Wilkins, a graduate student in Nutrition & Dietetics. The Kent State University Institutional Review Board has approved this study. No deception is involved, and the study involves no more than minimal risk to participants (i.e., the level of risk encountered in daily life).

Participation in the study typically takes around 15 minutes and is strictly anonymous. Participants begin by answering a series of demographic questions. Next, questions will ask about perceptions toward nutrigenomics. Several questions will also examine which influencing factors would make respondents more inclined to participate or not participate in the nutrigenomic testing. Finally, participants will be asked a series of questions to evaluate a genetics knowledge baseline.

All responses are treated as confidential, and in no case will responses from individual participants be identified. Rather, all data will be pooled and published in aggregate form only. Participants should be aware, however, that the experiment is not being run from a "secure" https server of the kind typically used to handle credit card transactions, so there is a small possibility that responses could be viewed by unauthorized third parties (e.g., computer hackers).

No adverse reactions have been reported thus far during the completion of this questionnaire. Participants will be given the option of entering a raffle for four $25 Amazon e-gift cards. Contact information obtained for the gift card raffle will not in any way be linked to responses from the questionnaire. Participation is voluntary. Refusal to take part in the study involves no penalty or loss of benefits to which participants are otherwise entitled, and participants may withdraw from the study at any time without penalty or loss of benefits to which they are otherwise entitled.

If participants have further questions about this study or their rights, or if they wish to lodge a complaint or concern, they may contact the principal investigator, Professor Eun-Jeong Ha, at (330) 672-2701; or the Kent State University Institutional Review Board, at (330) 672-2704.

If you are 18 years of age or older, understand the statements above, and freely consent to participate in the study, click on the "I Agree" button to begin the survey.

I Agree I Do Not Agree
APPENDIX B
SURVEY QUESTIONNAIRE
Appendix B
Survey Questionnaire

Q1 How old are you?

Q2 What is your gender?
○ Male (1)
○ Female (2)
○ Transgender (3)
○ Prefer not to answer (4)
○ Other (5)

Q3 What grade are you in?
○ Freshman (1)
○ Sophomore (2)
○ Junior (3)
○ Senior (4)
○ Master Student (5)
○ Doctorate Student (6)

Q4 Have you ever taken a college level nutrition course?
○ Yes (1)
○ No (0)

Q5 Have you ever taken a college level genetics course?
○ Yes (1)
○ No (0)

Q6 What is your major? ____________________________

Q7 Nutrigenomics is the study of the interaction between nutrition and genes. Some companies are offering genetic tests that are advertised to improve health and prevent disease. (Example companies include Nutrigenomix Inc., Interleukin Genetics, Habit, GenoVive, Arivale, etc.) Have you heard or read about these genetic tests?
○ Yes (1)
○ No (0)
Q8 The following statements are about genetic testing for personalized nutrition (Nutrigenomics). To what extent do you agree or disagree with each statement?

<table>
<thead>
<tr>
<th>Statement</th>
<th>Strongly disagree (1)</th>
<th>Disagree (2)</th>
<th>Neither agree nor disagree (3)</th>
<th>Agree (4)</th>
<th>Strongly agree (5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knowing their own genetics allows individuals to control their lifestyle more easily. (Q8_1)</td>
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<tr>
<td>Genetic testing for personalized nutrition will result in discrimination. (Q8_2)</td>
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<td>Genetic testing for personalized nutrition will help people to live longer. (Q8_3)</td>
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<td>All individuals should be offered genetic testing for personalized nutrition by their family doctor. (Q8_4)</td>
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<td>Nutrigenomic testing would cost too much and would only be available to the rich. (Q8_5)</td>
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<td>I am worried that genetic testing may lead to eugenics (the science of improving the human</td>
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</table>
population by controlling breeding to increase desirable characteristics). (Q8_6)
Genetic testing for personalized nutrition will make medical cures for diseases more possible. (Q8_7)
I am scared of finding out the results of genetic tests for personalized nutrition. (Q8_8)
Genetic testing for personalized nutrition should be promoted extensively. (Q8_9)
I do not believe that genetic testing for personalized nutrition is backed by sound science. (Q8_10)
Genetic testing for personalized nutrition goes against my religious beliefs. (Q8_11)
I believe it is essential to assign more money to nutrigenomic developments.
Q8 Continued. The following statements are about genetic testing for personalized nutrition (Nutrigenomics). To what extent do you agree or disagree with each statement?

<table>
<thead>
<tr>
<th>Statement</th>
<th>Strongly disagree (1)</th>
<th>Disagree (2)</th>
<th>Neither agree nor disagree (3)</th>
<th>Agree (4)</th>
<th>Strongly agree (5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screening for known genes is the way forward for medicine and nutrition. (Q8_13)</td>
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<td>Gene testing for personalized nutrition will lead to prevention of some diseases. (Q8_14)</td>
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<td>In my lifetime, I expect to see significant medical improvements due to use of genetics in nutrition. (Q8_15)</td>
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<td>I am concerned that my genetic information will be made available for research purposes. (Q8_16)</td>
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<td>My genes have influenced my health. (Q8_17)</td>
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<tr>
<td>Genetic testing</td>
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for personalized nutrition is an invasion of my privacy. (Q8_18)
I would like to know about future diseases through genetic testing. (Q8_19)
I think there is too much focus on genetics when money could be spent on the world's starving population. (Q8_20)
Genetic testing for personalized nutrition should be available to everyone. (Q8_21)
I am concerned that not enough will be done to protect the confidentiality and privacy of my genetic information. (Q8_22)

<table>
<thead>
<tr>
<th>Q9 To what extent do the following reasons make you want OR not want to undergo a nutrigenomic test for personalized nutrition?</th>
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<tbody>
<tr>
<td>Not at all likely (1)</td>
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<td>Question</td>
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<tr>
<td>-------------------------------------------------------------------------</td>
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<tr>
<td>Anxiety about health (Q9_1)</td>
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<tr>
<td>Doctor's recommendation (Q9_2)</td>
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<tr>
<td>Availability of more detailed information (Q9_3)</td>
</tr>
<tr>
<td>Curiosity (Q9_4)</td>
</tr>
<tr>
<td>Family or friend's advice (Q9_5)</td>
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<tr>
<td>Family history of particular disease (Q9_6)</td>
</tr>
<tr>
<td>Lack of money to pay for testing or possible treatments (Q9_7)</td>
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<tr>
<td>Lack of time (Q9_8)</td>
</tr>
<tr>
<td>Fear to discover results (Q9_9)</td>
</tr>
<tr>
<td>I think it's useless (Q9_10)</td>
</tr>
<tr>
<td>It goes against my beliefs (Q9_11)</td>
</tr>
<tr>
<td>It is an invasion of privacy (Q9_12)</td>
</tr>
<tr>
<td>Other, please specify (Q9_13)</td>
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</tbody>
</table>

The following questions are designed to assess your knowledge in genetics. Please read each question carefully. If you do not understand or don't feel comfortable answering a question, please choose "Don't know." Please do not guess if you do not know the
answer. Instead, choose "Don't know." All the correct answers for this section will be provided at the end of the survey.

Q10 A gene is a portion of DNA, which codes for protein, which leads to a trait.
- True (1)
- False (0)
- Don't know (3)

Q11 Males inherit two X-chromosomes at birth, one from their mother and one from their father.
- True (0)
- False (1)
- Don't know (3)

Q12 The human genome project has estimated that humans have between 20,000 and 25,000 genes.
- True (1)
- False (0)
- Don't know (3)

Q13 Genes contain chromosomes.
- True (0)
- False (1)
- Don't know (3)

Q14 A genotype is the genetic make-up of an organism (BB, Pp, ff).
- True (1)
- False (0)
- Don't know (3)

Q15 In humans, each cell normally contains 23 pairs of chromosomes, for a total of 46.
- True (1)
- False (0)
- Don't know (3)

Q16 A phenotype is a physical expression of alleles (brown eyes or blue eyes).
- True (1)
- False (0)
- Don't know (3)
Q17 A mutation occurs when the structure of a gene changes.
- True (1)
- False (0)
- Don't know (3)

Q18 Mutations always lead to negative health outcomes.
- True (0)
- False (1)
- Don't know (3)

Q19 An allele is the different forms of a gene, represented by letters.
- True (1)
- False (0)
- Don't know (3)

Q20 A dominant trait is the trait which is hidden in F1 generation.
- True (0)
- False (1)
- Don't know (3)

Q21 Epigenetics is the study of changes in an organism's gene expression without a change in the genetic code.
- True (1)
- False (0)
- Don't know (3)

Q22 DNA repair is a collection of processes where a cell identifies and repairs DNA molecules that encode its genome.
- True (1)
- False (0)
- Don't know (3)

Q23 A point mutation is a type of mutation that causes a single nucleotide base substitution, insertion, or deletion.
- True (1)
- False (0)
- Don't know (3)
Q24 An example of a genotype that is heterozygous is AA.
- True (0)
- False (1)
- Don't know (3)

Q25 An example of a genotype that is homozygous is cc.
- True (1)
- False (0)
- Don't know (3)

Q26 Mutations can create variations in protein "switches" that control protein function.
- True (1)
- False (0)
- Don't know (3)

Q27 Mutations cannot be reversed through DNA repair.
- True (1)
- False (2)
- Don't know (3)

Q28 A recessive trait can be carried in a person's genes without appearing in their phenotype.
- True (1)
- False (0)
- Don't know (3)

Would you like to see the correct answers to the previous genetics questions?
- Yes (1)
- No (2)
REFERENCES
REFERENCES


