GENOGRAMS AS THREAT APPEALS: USING THE EXTENDED PARALLEL PROCESS MODEL WITH FAMILIAL CARDIOVASCULAR DISEASE

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To the Dean of the Graduate School:

I am submitting herewith a dissertation written by Betty Carlson Bowles entitled "Genograms as Threat Appeals: Using the Extended Parallel Process Model with Familial Cardiovascular Disease." I have examined this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy with a major in Health Studies.

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ABSTRACT

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GENOGRAMS AS THREAT APPEALS: USING THE EXTENDED PARALLEL PROCESS MODEL WITH FAMILIAL CARDIOVASCULAR DISEASE

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This study explored the use of genograms as threat appeals in motivating intent to perform health promotion and disease prevention behaviors in participants with a history of cardiovascular disease (CVD) recruited by convenience and snowball sampling. The Extended Parallel Process Model (EPPM) contends that efficacy must exceed threat for protection motivation to dominate resulting in message acceptance and danger control responses. If threat exceeds efficacy, defensive motivation will dominate, resulting in fear control responses and message rejection. Surveys modified from the Risk Behavior Diagnosis Scale, theoretically grounded in the EPPM, assessed threat (perceived severity of and susceptibility to CVD) and efficacy (perceived response efficacy and self-efficacy related to recommended preventive behaviors). These surveys, administered before and after completing a health genogram, determined dominance of threat control or fear control responses, intent to practice health promotion behaviors, and stage of change. There were statistically significant shifts toward fear control for physical activity, tobacco avoidance and total scores, but not for weight maintenance. There were no significant differences in intent to practice health promotion behaviors. Stages of change were advanced for documentation of family health history and for practicing health promotion behaviors. Higher fear scores in those within the fear control spectrum validated that construct of the EPPM. This study has implications for health educators in assisting families to assess susceptibility to disease by documenting family health history. Additionally, health educators should assess perceived threat and perceived efficacy in the client, and intervene at the earliest possible stage with strategies that strengthen response- and self-efficacy to ensure that fear appeal messages are accepted and behaviors are changed to promote health and prevent disease.

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CHAPTER I

INTRODUCTION

A Healthstyles 2004 Survey revealed that 96.3% of Americans acknowledge the importance of knowing their family medical history, but only 29.8% have attempted to gather that information (Yoon et al., 2004). Khoury and Mensah (2005), from the Office of Genomics and Disease Prevention, report that family history is the best and most inexpensive genomic tool available. Family history of disease equates with increased risk of disease and is the most consistent risk factor across the lifespan. This clinical tool has the potential to aid diagnoses, prompt targeted testing and evaluation, and guide patient education and health promotion initiatives (Trotter & Martin, 2007).

According to the American Heart Association (2008), approximately 80,700,000 adults, or one in three Americans, have some form of cardiovascular disease (CVD). Decades of research have provided evidence that family history in a first-degree relative has been associated with CVD (Arnett et al., 2007). Because CVD runs in families, a family medical history in the form of a genogram can help predict disease risk and identify recommendations for health promotion and disease prevention behaviors. If high-risk individuals identified by family history adopt recommended prevention strategies, family history has the potential to become a tool for public health and preventive medicine (Yoon et al., 2002).

According to Witte, Meyer, and Martell (2001), most people ignore logical health risk messages and respond only when the message arouses them emotionally. Persuasive messages that gain compliance by arousing fear are called fear or threat appeals. Fear or threat appeals, which grew out of the Health Belief Model (HBM), have focused on health risks and have used persuasive messages that evoke anxiety and fear. These appeals have attempted to persuade people to change their attitudes, intentions, and behaviors in order to improve their health (Witte, 1991).

This study explored the use of health genograms as threat appeals in motivating intent to perform health promotion and disease prevention behaviors in participants with a history of CVD. The Extended Parallel Process Model (EPPM) (Appendix A), which originated from the Health Belief Model (HBM) and expanded the Parallel Process Model, was utilized for this study. The HBM hypothesizes that behavior change to improve health depends on perceived susceptibility, perceived severity, perceived benefits and barriers, cues to action, and self-efficacy. According to the HBM, increased perception of susceptibility to a disease should be associated with an increased likelihood of engaging in behaviors that will prevent, detect, or cure the problem (Janz, Champion & Strecher, 2002).

Research has shown that fear appeals are powerful persuasive devices, but only in certain circumstances (Lapinski & Witte, 1998). According to the EPPM (Witte, 1991), a health threat initiates two appraisals: 1) danger control, a cognitive process; or 2) fear control, an emotional process. Danger control is assessed in terms of perceived severity of the threat and perceived susceptibility to the threat. With low severity or susceptibility, there is no motivation to respond to the threat, and the health message may be ignored. The greater the threat, the more motivated the individual is to begin the next appraisal of self-efficacy and efficacy of the recommended response. If self-efficacy and response-efficacy are high, danger or threat control processes will dominate. Threat control motivation stimulates adaptive attitude, intention or behavior changes to control the threat. However, if self-efficacy and response-efficacy are low, fear control processes dominate over danger control processes, resulting in denial, defensive avoidance, or reactance. If this happens, fear appeal campaigns backfire. Therefore, for fear appeals to be successful, the individual must perceive that the threat is serious, that one is personally susceptible to it, that the recommended response will control the threat, and that one is capable of making the recommended response (Witte, 1991). This study utilized the Risk Behavior Diagnosis (RBD) Scale (Witte, Meyer & Martel, 2001), which is theoretically grounded in the EPPM. Participants were assessed on their perception of severity and susceptibility related to CVD, collectively termed threat, and their perception of response efficacy and self-efficacy related to preventive behaviors.

In addition to the perceived threat and efficacy constructs of the EPPM, this study assessed the participant's stage of change from the Transtheoretical Model (TTM) before and after the genogram was completed. The TTM (Prochaska, Redding, & Evers, 2002) states that behavior change occurs through a series of stages (precontemplation, contemplation, preparation, action, and maintenance). This theory includes consciousness-raising as one of the constructs in advancing readiness to change. This study explored whether completion of the online family health genogram raised the participants' consciousness of their CVD risk and advanced their stage of change or their readiness to adopt health promotion behaviors.

Statement of Purpose

The purpose of this study was to introduce the health genogram as a threat appeal in motivating intent to perform health promotion and disease prevention behaviors in participants with a family history of CVD. The study applied the EPPM (Appendix A) by using the health genogram as a fear or threat appeal. Participants with a history of CVD completed surveys based on the RBD scale before and after completion of a health genogram. The surveys assessed their (a) perceived threat of CVD, (b) perceived efficacy of recommended preventive behaviors, (c) perceived self-efficacy to perform the preventive behaviors, (d) intent to perform those behaviors, and (e) stage of change for family history collection and for health promotion and disease prevention behaviors related to CVD.

Research Questions

The research questions for this study were:

1. Do threat control responses or fear control responses dominate in participants with a family history of CVD, after they complete the specified online health genogram?

2. Does completion of the specified online health genogram influence the participant's intent to practice health promotion behaviors?

3. Does completion of the specified online health genogram advance the participant's readiness to adopt health promotion behaviors?

Null Hypotheses

H_{01} – There is no statistically significant difference in fear control / danger control processes related to weight control, physical activity and smoking avoidance in participants with a family history of CVD.

H_{02} - There is no statistically significant difference in pre- and post-genogram scores on intent to control / maintain weight, engage in regular physical activity, and avoid tobacco in participants with a family history of CVD.

H_{03} – There is no statistically significant difference in pre- and post-genogram scores on stage of change of participants with a family history of CVD.

Delimitation

This study had the following delimitation:

1. Participants for this study were 18 years of age or older reported having at least one first-degree relative with CVD.

Limitations

This study had the following limitations:

1. A non-probability convenience sample was used initially to recruit participants. Thereafter, a network or snowball sampling technique was employed. Thus, no generalizations can be made beyond the scope of this study.

2. Due to convenience sampling, those who agreed to participate may have been motivated to do so based on an interest in family history or suspicion of CVD risk.

Assumptions

This study had the following assumptions:

1. Participants spoke, read, and wrote English at a minimum of eighth-grade level, had access to the internet, and had the computer skills to complete the online health genograms.

2. Participants accurately recalled CVD in first-degree relatives.

3. Participants were honest in descriptions of their family medical history.

Definition of Terms

Cardiovascular disease (CVD) – A cluster of diseases of the circulatory system that includes heart disease, stroke, hypertension, angina pectoris, myocardial infarction, atherosclerosis, aneurysm or other cardiac or vascular diseases (World Health Organization, 2006).

Danger control response / Adaptive behavior change – In EPPM, an adaptive cognitive response to a health threat by controlling it through changes in environment or behavior (Witte, 1991).

Efficacy – The power to produce an effect (Merriam-Webster, 2008); in the EPPM, "the cognitive appraisal of response efficacy and self-efficacy" (Witte, 1991, p. 170).

Familial – tending to occur in more family members than expected by chance alone (Merriam-Webster, 2008).

Fear control response / Maladaptive behavior change – In the EPPM, an emotional response to a threat by denial, defensive avoidance or reactance (Witte, 1991).

First-degree relative – Children, parents and siblings that share one-half of their genes (Shawker, 2004).

Genogram / Pedigree – A diagram detailing the medical history of members of a family as a means of assessing a person's risk of developing disease (Merriam-Webster, 2008; Shawker, 2004).

Response efficacy – In the EPPM, "the perceived efficacy of a recommended protective response" (Witte, 1991, p. 173).

Self-efficacy – In the EPPM, "a person's perceived ability to perform the recommended response" (Witte, 1991, p. 173).

Susceptibility – The state of being predisposed to (Merriam-Webster, 2008); in the EPPM, the probability that the threat will occur to an individual (Witte, 1991).

Threat – Imminent danger or harm (Merriam-Webster, 2008); in the EPPM, "the cognitive appraisal of susceptibility to threat and severity of threat" (Witte, 1991, p. 173).

Threat / fear appeal – Persuasive health communication that describes unfavorable consequences of rejecting the appeal recommendations (Witte, 1991).

Importance of the Study

The WHO global strategy for prevention and control of non-communicable diseases, including CVD, calls for reduction of exposure to preventable common risk factors, especially tobacco, obesity and inactivity (WHO, 1999). Healthy People 2010 (USDHHS, n.d.) set goals to reduce the number of deaths from CVD as well as increase the proportion of the population who practice health promotion and disease prevention behaviors. The CDC Office of Public Health Genomics (OPHG) has a goal "to assess the value of family history in risk assessment and disease prevention" (CDC, OPHG, 2009, p.1). The U.S. Surgeon General's Family History Public Health Initiative calls attention to the importance of learning about family health histories and provides an online tool for documenting those histories (USDHHS, 2006). These global and national campaigns recognize the importance of prevention in reducing disease burden, and recognize family history as a significant risk factor for CVD. With prestigious national campaigns promoting the documentation of family health history, it behooves health educators to explore this mechanism to identify high risk individuals as well as health communication theories that communicate risk. This study explored the health genogram as a threat appeal with the potential to motivate intent to adopt health promotion and disease prevention behaviors.

CHAPTER II

REVIEW OF THE LITERATURE

Persuasive health risk messages that gain compliance by arousing fear are called fear or threat appeals, often called scare tactics. This study explored the use of health genograms as threat appeals in motivating intent to perform health promotion and disease prevention behaviors in participants with a history of cardiovascular disease. A literature search yielded previous research and publications in the areas of family history, cardiovascular disease (CVD) and its risk factors, fear appeals, the Extended Parallel Process Model and its related theories, and the Transtheoretical Model.

The family history literature addressed the recent resurgence of interest in family history within the growing field of genomics, as well as its long-recognized importance in health promotion and chronic disease prevention. This review included the clinical utilization of family health history as well as its increasing use by individuals and families. Finally, family history was discussed in terms of the resources that are available to facilitate its investigation and documentation.

Another major area of the literature search was cardiovascular disease, including the pathophysiology of CVD, and its prevalence and disease burden. The literature also addressed family history as a non-modifiable risk factor for CVD, as well as tobacco use, obesity and inactivity as significant modifiable risk factors. This study explored genograms as fear or threat appeals and utilized the Extended Parallel Process Model with its major constructs of perceived threat (severity and susceptibility) and perceived efficacy (response efficacy and self-efficacy). This literature review addressed the history of fear and threat appeals and the evolution of the Extended Parallel Process Model (EPPM) through the Health Belief Model and the Parallel Process Model. The use and analysis of the EPPM in prior research was summarized from the existing literature. In addition to the EPPM, the literature review addressed the Stages of Change component of the Transtheoretical Model which was used to assess stage advancement in the current study.

Family Health History

Importance of Family Health History

The identification of inherited causes of diseases by the Human Genome Project and the establishment of national clinical practice guidelines have brought the importance of family health history into focus (Wattendorf & Hadley, 2005). With the expanding knowledge of genetics, family history plays a vital role in risk assessment, disease prevention and health promotion (Arnett et al., 2007; Audrain-McGovern, Hughes, & Patterson, 2003; Burke, 2005; Guttmacher, Collins, & Carmona, 2004; O'Donnell, 2004; USDHHS, 2009). Although tremendous progress has been made in primary prevention of CVD over the last two decades, substantial advancements may yet be achieved by studying "the family as its own unit of inference and as a specific target for disease prevention" (Kardia, Modell, & Peyser, 2003, p. 143).

The family medical history can be one of the most powerful clinical tools for health risk identification and intervention (Olsen, Dudley-Brown & McMullen, 2004). It is the simplest applied "genomic tool" available in medical practice (Khoury, Davis, Gwinn, Lindegren, & Yoon, 2005, p. 801). Family history is an initial method for risk assessment for many preventable, chronic conditions (Rich et al., 2004; Scheuner, Wang, Raffel, Larabell, & Rotter, 1997), and a cost-effective method for identifying and intervening with high-risk populations (Bennett, 2004; Johnson et al., 2005). If high-risk individuals identified by family history adopt recommended prevention strategies, family history has the potential to become a tool for public health and preventive medicine (Arnett et al., 2007; Scheuner, 2003; Trotter & Martin, 2007; Yoon et al., 2002).

"A family systems perspective views families as inextricably interconnected" through their common history and their implied future, with interactions that are interdependent, reciprocal, patterned and repetitive (McGoldrick, Gerson, & Shellenberger, 1999, p.7). Family history reflects a complex interaction of genetic, environmental, cultural and behavioral risk factors shared within families (Butterworth, 2007; CDC, NOPHG, 2007; Dolan & Moore, 2007; Johnson, 2009; Johnson et al., 2005; Khoury et al., 2005; McGoldrick et al., 1999; Scheuner, 2003; Watt, McConnachie, Upton, Emslie, & Hunt, 2000; Yoon et al., 2004; Yoon, Scheuner, Jorgensen, & Khoury, 2009; Yusef et al., 2004). A family-centered approach to prevention of disease recognizes these complex causes (Pearson et al., 2002) and interactions (Higgins, 2000). McCusker et al. (2004) contend that in order to produce lasting behavior change to prevent disease, successful family history-based interventions need to involve entire families.

Family history promises to remain a valuable tool for public health and preventive medicine (CDC, NOPHG, 2007; Johnson et al., 2005; Trotter et al., 2007). Family health history is important across the life span as risks change throughout life (Dolan et al., 2007). Familial health risks change as families grow and family members age. Because of this, the family medical history should be treated as a living document, expanding and maturing as family members move through developmental life stages (Olsen et al., 2004). When updated regularly, the family history becomes a tool for increasing awareness of risk as people age (Valdez, Greenlund, Khoury, & Yoon, 2007).

Families affect the health of their members by transmitting genetic predisposition to both wellness and illness (Crouch, 2007). According to Milunsky (2001), each person has an ethical responsibility to communicate vital family health information to relatives, who can then make an informed choice about whether or not to act on that information. Discussions about family health history can be a unifying force in extended families (Green, 2007). Family discussions can enhance prevention efforts by shedding light on modifiable family behaviors that have adverse health outcomes (Wattendorf et al., 2005). Hicken and Tucker (2002) found that family history-based risk information was just as strong a motivator to follow recommended health behaviors as genetically-based risk information. Awareness of family health history can lead to periodic screening, early diagnosis, and lifestyle changes that can determine whether a genetic tendency toward a disease will become a reality (Daus, 1999; Shawker, 2004).

Family histories have the potential to make population health promotion messages more immediately personal and thus encourage and support healthier habits in unaffected family members (Hunt, Gwinn, & Adams, 2003; Yoon et al., 2002). When health messages are personally relevant, the messages are more extensively or systematically processed (Etchegary & Perrier, 2006) and remembered (Berry, Michas, & Bersellini, 2003). Natter and Berry (2005) had study participants use graphical displays to actively illustrate the extent of their health risks and demonstrated improved understanding, decision-making, and problem solving through that process. Accordingly, active participation in the construction of genograms could potentially lead to better understanding of familial health risks, greater motivation to modify risky behaviors, and improve health outcomes.

While knowledge of familial susceptibility to disease may motivate behavior change to reduce risk, it also has the potential to decrease motivation. According to Marteau and Lerman (2001), risk information could increase motivation by strengthening the belief that the current behavior, in combination with the genetic susceptibility, increases the individual's risk, or by strengthening the belief in the effectiveness of the recommended behavior change. Conversely, family risk information could decrease motivation by weakening the beliefs that behavior change can reduce risk, or that the individual is capable of changing behavior. In spite of the recognized clinical value of family history, a thorough exploration of family history is the exception rather than the rule in primary health care.

Clinical Utilization of Family Health History

According to Acting Surgeon General Steven Galson, family history has always been important, but has been underutilized (USDHHS, 2009). Suther and Goodson (2003) identify lack of detailed family histories as one of physicians' perceived barriers to provision of genetic services. Jason Mitchell of the American Academy of Family

Practice's Center for Health Information Technology contends that family history is the most accessible genetic information available, but is difficult for busy physicians to gather (Porter, 2009). Acheson, Wiesner, Zyzanski, Goodwin, and Stange (2000) found that family history was discussed at 51% of primary care physician's initial visits, for an average of 2.5 minutes, and at only 22% of follow-up visits. Genograms were found on only 11% of patient records. The traditional practice of dictating medical notes is a significant barrier to including a graphic depiction of family history in the medical record (Olsen et al., 2004). Various clinical specialties have suggested that physicians perform poorly in collecting and interpreting family health history for risk assessment and intervention (Acheson et al., 2000; Frezzo, Rubinstein, Dunham, & Ormond, 2003; Gramling, Nash, Siren, Eaton, & Culpepper, 2004; Ruo et al., 2001; Scheuner, Yoon, & Khoury, 2004).

Several studies have assessed the accuracy of clinical family histories. Sweet, Bradley, and Westman (2002) compared the family history in medical records with the family history entered by patients directly into a touch screen family history computer program. There were significant discrepancies and omissions in the medical record leading to incorrect risk assessment and referral. Frezzo et al. (2003) compared the medical documentation and risk assessment between a questionnaire, a pedigree, and a chart review and found that 20% of patients at high risk did not have the risk documented in the medical record. Mitchell et al. (2004) demonstrated the inaccuracy of a family health history interview in identifying high-risk individuals.

Many barriers were identified for failure to meet this medicolegal standard of care. Suggested barriers include time constraints and failure to realize the importance of family history in accurate risk assessment and intervention. Yarnall, Pollack et al. (2003) found that because of time constraints in primary care physicians' practices, the patient's medical needs take precedence over preventive care and counseling. Additional barriers suggested by Rich et al. (2004) include reimbursement policies that limit time for history taking, and perceived limited history-taking knowledge and skills of the practitioner. Gramling et al. (2004) contend that family physicians lack confidence in their ability to screen for family history.

Valdez et al. (2007) suggest several legal and ethical issues related to the use of family history as a screening tool in clinical practice. Legal issues involve informed consent, ownership of the family history data, the obligation to disclose that data, and the requirements for reporting. The consequences of labeling individuals as high risk must be considered in terms of medical insurability. While the individual should be encouraged to inform family members of increased risk, Wattendorf et al. (2005) contend that the physician's duty to inform other family members is less clear, with disclosure weighed carefully between respect for privacy and any duty to warn. Since the genogram may contain information previously unknown to relatives, Bennett (2004) suggests that patient confidentiality must be carefully weighed against clinical and genetic relevance when choosing what information to record in a genogram.

While expanding knowledge of human genetics presents tremendous potential for improvements in clinical practice, it also poses clinical challenges and the potential for risk of harm. This harm includes the cost of needless testing, unnecessary anxiety, and possible inappropriate or hazardous therapy. According to Rich et al. (2004) genetic testing can have serious effects on family relationships, lifestyle choices, and decisions about reproduction. They believe that with advances in human genetics, radical new resources and strategies for the application of family health history will be essential to aid primary care physicians. Standardized methods of recording will also be essential for the full potential of family history in clinical practice to be realized (Qureshi et al., 2005). A potential strategy to enhance the family history utilization in clinical practice is self-documentation of family health history.

Self-Documentation of Family Health History

The problems inherent in clinical documentation of family health history suggest the need for self-administered instruments for family health history collection. Weir (2005) suggests that physicians ask patients to complete a family health history questionnaire before their medical appointment to give them time to collect the information, and to save the physician's consultation time. According to Valdez et al. (2007), family history of CVD is self-reported fairly accurately because its seriousness causes concern within families, and because there is little stigma attached to the information. In the Family Heart Study, Bensen et al. (1999) validated family histories and demonstrated that self-documentation of family history of CVD can be effectively accomplished with an accuracy of 87%, 85% and 81% for spouse, parent, and sibling respectively. Other researchers assessed how accurately sons and daughters reported the cause of their parents or siblings deaths, age of death, and whether there were conditions or illnesses that ran in their families (Watt et al., 2000). Respondents correctly identified CVD as the cause of death 89% of the time. However, only 23% of sons and 34% of daughters perceived a history of CVD as a familial condition or risk.

Murabito et al. (2004), using offspring participants of the multi-generational Framingham Heart Study, compared the accuracy of CVD risk factors and events reported by offspring with validated outcomes from medical records and clinical assessments. They concluded that positive reports of parental hypertension and hypercholesterolemia had positive predictive values, but reports of early-onset heart attack and stroke, and death from heart disease had relatively low positive predictive values. They concluded that if offspring were more aware of family health history, they could more accurately estimate their own risk to motivate healthy lifestyle changes. Ivanovich et al. (2002) found a higher reporting accuracy of health history among first-degree relatives than among second- and third-degree relatives, as well as a greater likelihood that close relatives would share health information than distant relatives. These findings underscore the importance of involving families in the collection and ongoing maintenance of accurate family health histories. To that end families need to be aware of the resources that are available to assist them with the investigation and documentation of their family health history.

Family Health History Resources

Numerous resources have become available during the last decade to assist individuals and families to formulate their own family health history. Investigation and documentation of family history can be difficult. Families may be large, geographically dispersed, or estranged. Health histories may not be known, or affected family members or key family informants may be deceased. Dealing with family groups also presents legal, ethical, or social dilemmas. In spite of these barriers, greater emphasis on family health history is needed to reduce the burden of CVD (Higgins, 2000).

Wattendorf et al. (2005), representing the National Human Genome Research Institute of the National Institutes of Health, reported that documenting family health history ranges from asking a few questions to diagramming complex pedigrees or genograms. They contend that the three-generation pedigree is the most effective method for recording family health history, and that visualizing a pictorial family history such as a genogram may clarify risks not previously appreciated. Crouch (2007) advocates the use of family history in the genogram format as a versatile patient education tool.

Yoon et al. (2009) suggest that family history tools should have key design principles. These principles include self-administration, ease of use, and adaptability for integration into electronic medical records or public health surveillance systems. Medical pedigrees that can be completed online at home allow families to consult relatives and access family records during the data collection process. Electronic records are especially beneficial because they can be stored, retrieved, updated, and linked to educational websites for disease prevention and health promotion information (Yoon, Scheuner, & Khoury, 2003). Tools for generating a medical pedigree are available from reputable online sources. Several national organizations and governmental agencies have endorsed and developed family history tools. These tools help to identify high risk individuals, and enhance health promotion and disease prevention strategies.

In recognition of the importance of family history for the prevention of disease and promotion of health, the National Office of Public Health Genomics (NOPHG) at the CDC began the CDC's Family History Public Health Initiative in 2002 (CDC, NOPHG, 2007). This initiative, in collaboration with the National Human Genome Research Institute at the National Institutes of Health, the Agency for Healthcare Research and Quality, and the Health Resources and Services Administration, focuses attention on the importance of family history. As part of this initiative, a web-based tool to formulate "My Family Health Portrait" was launched in 2004. This tool guides users through the process of recording pertinent health history, produces graphic reports for presentation to family members and health care providers, and allows individuals to save the reports for future revision. This initiative designates Thanksgiving Day as National Family History Day to encourage families to discuss familial health problems and the reduction of modifiable risk factors (USDHHS, 2006b). In January of 2009, an updated and improved version of My Family Health Portrait was launched making it easier to record and share family health information. This version is built on health information technology standards which will make it usable in electronic health records (USDHHS, 2009).

Family Healthware is a new interactive online family history screening tool developed by the CDC to assess six diseases including heart disease and stroke. It is being evaluated by three academic centers to determine if tailored prevention messages based on family risk assessment will motivate high risk families to make lifestyle changes including earlier and more frequent screening, chemoprevention, and prophylactic procedures and surgeries (Yoon et al., 2009).

Acknowledging the importance of accurate family medical history, the American Medical Association (AMA) developed online tools to aid the individual in recording family history to improve communication between the physician and the patient and promote a sense of partnership in healthcare management. In addition, publications and medical record forms assist the health care provider with essential family history information for identifying disease risk and developing a plan for prevention and/or treatment (AMA, 2009; O'Connor, 2004).

The National Human Genome Research Institute provides links to numerous family medical history tools and resources including various pedigree drawing software programs. Family history resources for health providers are available from the American Society of Human Genetics (n.d.). The Clinical Genetics Society (2001) provides detailed instructions on its website for construction of a genogram or pedigree. The National Society of Genetic Counselors (2009) and the Genetic Alliance (2009) also provide instructions for collecting and recording family history information. The March of Dimes (2009) website includes information for clinicians entitled *Genetics and Your Practice* that details the importance of obtaining family history information as well as step-by-step instructions for constructing a pedigree.

A collaborative project by the American Folklife Center, the American Society of Human Genetics, the Genetic Alliance, and the Institute for Cultural Partnerships provides an online guide for constructing a family health history. This valuable tool includes suggestions for family conversations and sample questions for interviews of family members (USDHHS, n.d.). The Mayo Clinic (2008) provides instructions online for compiling a medical family tree or pedigree. These instructions include what information to gather, how to gather the information, how to compile the information into a family tree, and what to do with the completed family medical history.

Various risk assessment programs are also available online. The American Heart Association (AHA) has a *Heart Attack Risk Calculator* that assesses risk factors and calculates a 10-Year risk of heart attack (AHA, 2009a.). *Your Disease Risk* assesses heart disease risk and formulates a personalized plan for reducing that risk (Siteman Cancer Center, 2007). The Mayo Clinic (2008) has a brief *Heart Disease Risk Calculator* that assesses the risk of having a heart attack or dying of heart disease within the next 10 years.

Individuals and families need to be encouraged to avail themselves of the many resources available for investigation and documentation of their family health history. Guttmacher et al. (2004), representing the National Human Genome Research Institute of the National Institutes of Health, state that "Almost every patient has access to a free, well-proven, personalized genomic tool that can serve as a cornerstone for individualized disease prevention. This valuable tool is the family history" (p. 2333).

Cardiovascular Disease

Prevalence and Burden of Cardiovascular Disease

Heart disease and stroke are the most prevalent and most costly diseases, yet they are among the most preventable. The leading causes of death are heart disease and stroke, accounting for a third of all deaths both in the United States (CDC, 2009) and worldwide (Mackay & Mensah, 2004). More than 80 million Americans currently live with some form of CVD with an estimated 935,000 heart attacks and 795,000 strokes occurring each year. CVD is also the leading cause of disability in the U.S. workforce (Centers for Disease Control and Prevention [CDC], 2009). On an annual basis, the AHA, in collaboration with the CDC National Center for Health Statistics (NCHS), the National Heart, Lung, and Blood Institute (NHLBI), the National Institute of Neurological Disorders and Stroke, and other government agencies, compiles and publishes the most current statistics on heart disease and stroke. According to the *Heart Disease and Stroke Statistics 2009 Update* (2009b), the 2006 prevalence of CVD was 80 million or 33% of the population. Hypertension accounts for 73.6 million, coronary heart disease 16.8 million, heart failure 5.7 million, and stroke 6.5 million (Lloyd-Jones et al., 2009). According to the 2007 prevalence estimates, the death rate for blacks exceeded that for whites by 46% for stroke and 32% for heart disease (NCHS, 2007). The overall CVD death rate for 2006 was 262.9 per 100,000. Based on this rate, 2,400 people died of CVD every day in the United States, which translates to one death every 37 seconds (AHA, 2008; Lloyd-Jones et al., 2009). For the year 2005, 32% of cardiovascular deaths occurred in persons younger than 75 years of age which is below the average life expectancy of 77.8 years of age (AHA, 2009; Lloyd-Jones et al., 2009). The 2006 data indicates that 829,072 or 34.2% of all deaths in the United States were attributed to CVD. This is one in every 2.9 deaths (AHA, 2008; Lloyd-Jones et al., 2009). It is estimated that in 2009 an American will have a coronary event every 25 seconds and these events will result in one death per minute (Lloyd-Jones et al., 2009). The global burden of heart disease is projected to rise from around 47 million disability-adjusted life years (DALYs) in 1990 to 82 million DALYs in 2020 (Mackay et al., 2004). The total direct and indirect cost of CVD and stroke in the United States for 2009 is estimated at $475.3 billion (CDC, 2009; Lloyd-Jones et al., 2009).

Globally, 15 million people suffer a stroke annually. Of these, 5 million die and another 5 million are permanently disabled (Mackay et al., 2004). About 795,000 Americans experience a stroke annually, averaging about one stroke every 40 seconds. The 2006 prevalence is 6.5 million or 2.9% of the population. In the U.S. someone dies of a stroke every three to four minutes. The 2006 data indicate that one out of every 18 deaths in the United States can be attributed to stroke (Lloyd-Jones et al., 2009). Even though the incidence of stroke is declining in many developed countries, the absolute number of strokes continues to rise because of the aging population. The global burden of stroke is projected to increase from around 38 million DALYs in 1990 to 61 million DALYs in 2020. In 2004, stroke cost $33 billion globally in direct medical care and therapy costs and $20.6 billion in lost productivity for a total of $53.6 billion (Mackay et al., 2004).

Between 1950 and 2004 the overall age-adjusted death rate from all causes declined by 45%. For that same period the age-adjusted death rate for heart disease declined 63%, and for stroke 72% (NCHS, 2007). Over the last decade the death rate from CVD has declined 26.4% and the death rate from stroke declined by 29.7% (Lloyd-Jones et al., 2009). In spite of these declines, CVD remains a severe threat. Heart disease remains the leading cause of death for all ages, and stroke remains the third leading cause of death (behind malignant neoplasms). Mortality from heart disease and stroke are associated with risk factors such as hypertension, smoking, obesity and physical inactivity. The decline in CVD mortality is related to better control of these risk factors, early detection, and improved treatment (Mackay et al., 2004; NCHS, 2007). In particular, there has been a decrease in smoking among adults and lower levels of blood pressure and blood cholesterol (Mackay et al., 2004). From 1990 to 2004 a long-term downward trend continued with a 33% decline in mortality from cardiovascular disease (NCHS, 2007).

Gaps remain in the death rates between genders and races. The figures for 2005 indicate an overall age-adjusted death rate from heart disease to be 211.1 per 100,000 population. Those rates are 260.9 and 172.3 for males and females respectively. Blacks have the highest rate with 329.8 and 228.3 for males and females respectively. This compares to 258.0 and 168.2 for white males and females. The death rates for stroke also differ among genders and races. The overall age-adjusted death rate from stroke is 46.6 per 100,000 population. Those rates are 46.9 for males and 45.6 for females. The highest rates are for black males and females at 70.5 and 60.7 respectively, compared to white males and females at 44.7 and 44.0 respectively. The death rates for the black population exceeded those for the white population by 32% for heart disease and 46% for stroke. The lowest death rates for heart disease are among Asian or Pacific Islander males and females with rates of 141.1 and 91.9 respectively (NCHS, 2007). In order to better understand the prevalence and disease burden for CVD, the risk factors that contribute to CVD must be considered.

Risk Factors for Cardiovascular Disease

According to the World Health Organization (WHO), the most common non-communicable diseases are cardiovascular disease, cancer, chronic obstructive pulmonary disease, and diabetes. These diseases have common risk factors that are preventable and related to lifestyle including tobacco use, unhealthy diet, and physical inactivity (WHO, 1999). The WHO Global Report: *Preventing Chronic Disease: A Vital Investment* (WHO, 2005) states that these risk factors are the leading cause of death and disease burden in all countries regardless of economic status. Presence of these factors can help estimate the risk of CVD (U.S. Preventive Services Task Force, 2004).

The WHO in collaboration with the CDC (Mackay et al., 2004) recognize over 300 risk factors associated with cardiovascular disease. Some risk factors such as advancing age, heredity/family history, gender, or ethnicity/race are non-modifiable. However, at least a third of all CVD is attributable to five risk factors - tobacco use, alcohol use, hypertension, hypercholesterolemia, and obesity. These major risk factors are considered modifiable because they can be prevented, treated, and controlled (Mackay et al., 2004). Mokdad, Marks, Stroup, and Gerberding (2004) identified that 40% of deaths in the United States could be attributed to tobacco, poor nutrition, physical inactivity, and alcohol consumption. They predict that poor diet and inactivity may soon overtake smoking as the leading modifiable cause of death.

Interventions for risk factors have been shown to reduce the occurrence of CVD. However, Futterman, and Lemberg (1998) contend that 50% of all patients with CVD do not have any of the conventional risk factors. This claim has led to considerable interest in non-traditional risk factors such as inflammation or infection (Becker, Boer & van der Wal, 2001; McKechnie & Rubenfire, 2002). To refute the 50% claim, Khot et al. (2003) studied the prevalence of risk factors and determined that only 15% to 20% of patients with CVD lacked at least one of the conventional risk factors, including family history, smoking and obesity. They concluded that although research on nontraditional risk factors and genetic causes of CVD is important, significant emphasis still needs to go to lifestyle behaviors causing the conventional risk factors.

Greenland et al. (2003) examined three long-term prospective cohort studies and determined that CVD risk factor exposures were very common among those who developed fatal and non-fatal CVD. In all three studies prior exposure to at least one major risk factor ranged from 87% to100% in fatal CHD. According to Libby and Theroux (2005), aggressive management of modifiable risk factors reduces cardiovascular events and should accompany other therapies for acute CVD. King, Mainous and Geesey (2007) found that men who adopted healthy lifestyles in middle-age experienced a prompt benefit of lower rates of CVD and mortality.

In 14 years of follow-up in the Nurse's Health Study, low risk women who did not smoke, were not overweight, exercised at least half an hour per day, and had moderate alcohol consumption comprised only 3% of the population, but had 83% lower 14-year risk of coronary events compared to the high-risk categories (Stampfer, Hu, Manson, Rimm, & Willett, 2000). Yusuf et al. (2004) determined that lifestyle risk factors, including smoking, obesity, and physical inactivity, account for most of the risk of myocardial infarction in both sexes, at all ages, and in all regions of the world. These risk factors are particularly striking in the young suggesting that lifestyle changes have the potential to prevent most premature cases of myocardial infarction.

Reeves and Raffety (2005), using data from the 2000 Behavioral Risk Factor Surveillance System, combined four healthy lifestyle characteristics (HLCs): nonsmoking, healthy weight, fruit and vegetable consumption, and regular physical exercise. The sum of the four HLCs determined a healthy lifestyle index. An index of four was defined as a single healthy lifestyle indicator. The prevalence of the individual HLCs were: nonsmoking, 76.0%, healthy weight, 40.1%; fruit and vegetable consumption, 23.3%; and regular physical activity, 22.2%. The prevalence of the healthy lifestyle indicator (all four HLCs) was only 3.0%. According to the Institute for Clinical Systems Improvement (2008), these healthy lifestyle indicators are associated with a decade of increased life expectancy, and those who adopt them in midlife have total mortality rates that are 40% lower than those who do not adopt them.

People pay attention to visible risk factors such as smoking and obesity in explaining CVD, but these fail to explain early deaths in low risk individuals and long survival in high risk individuals. Because these violations of coronary candidacy undermine people's belief in the worth of behavioral modifications to reduce risk, McConnachie, Hunt, Emslie, Hart, and Watt (2001) studied these "unwarranted survivals" and "anomalous deaths". They found that men with low visible risk who died prematurely from CVD had poorer risk profiles on less visible risk factors, and those with high visible risk who survived had more favorable profiles on other risk factors. They concluded that health promotion messages would be more credible if they discussed these anomalies and the limits of prediction.

The WHO Global Strategy for Prevention and Control of Non-communicable Diseases proposes a reduction in exposure to risk factors and a focus on controlling the risk factors at the family level because of their social and cultural entrenchment within the family. This global strategy acknowledges that even modest reductions in risk factors can have a substantial benefit to public health (WHO, 1999). Jomini et al. (2002) found the prevalence of modifiable risk factors to be twice as high in those with familial premature coronary artery disease compared to the general population or those with sporadic disease. Grundy et al. (2004) and Bertuzzi, Negri, Tavani, and La Vecchia (2003) recommend therapeutic lifestyle changes for any person who has lifestyle-related risk factors for CVD. Tobacco cessation, regular and adequate physical activity, and maintenance of normal BMI are all associated with lower risk of CVD events (Fung, 2008). Family history, a non-modifiable risk factor for CVD, and modifiable risks factors of tobacco use, obesity, and inactivity are addressed individually in this literature review.

Family history as a modifiable risk factor for CVD. Family medical history is a risk factor for almost all diseases of public health significance, including most chronic diseases such as CVD (Khoury & Mensah, 2005). Even with progress in determining the genetic basis of disease since the human genome was sequenced, family history remains the best method for identifying individuals with genetic susceptibility to CVD (Kullo & Ding, 2007; Scheuner, 2003). Family history is a major predisposing factor (Kaikkonen, Kortelainen, Linna, & Huikuri, 2006; McCusker et al., 2004; Scheuner, Whitworth, McGruder, Yoon, & Khoury, 2006b; Silberberg, Wlodarczyk, Fryer, Robertson, & Hensley, 1998; Thompson, 2002; Valentine et al., 2004; Yarnell et al., 2003; Yoon et al., 2009; Zureik et al., 2006) and independent predictor of CVD (Hunt et al., 2003; Kardia et al., 2003; Lloyd-Jones et al., 2004; Nasir et al., 2004; Vaidya et al., 2007; Valdez et al., 2007; Parikh et al., 2007; Yusuf et al., 2004).

Scheuner (2004) contends that the familial aggregation of CVD is explained by familial aggregation of risk factors. Most CVD occurs in families with a history of CVD. Williams et al. (2001) analyzed 122,155 family pedigrees submitted in the Utah Health Family Tree Study. The 14% with positive family histories for CVD represented 72% of the early CVD cases. The 11% who had positive family histories for stroke represented 86% of early strokes. The researchers concluded that family history is a predictor of CVD, and pedigrees are useful tools for population-based disease prevention programs in the identification of high-risk families for targeted screening and intervention.

Decades of research provides evidence that family history in a first-degree relative is associated with CVD (Arnett et al., 2007). Family history is an important risk factor for CVD with a two- to three-fold risk in first degree relatives of individuals with CVD (Mackay et al., 2004; Scheuner, 2004). The Malmo Preventive Project in Sweden found that a maternal CVD death before the age of 75 years confers a substantial risk of CVD morbidity and mortality in sons that cannot be explained by other risk factors. The association was weaker for paternal CVD, and not significant for daughters (Nilsson, Nilsson, & Berglund, 2004). Leander, Hallqvist, Reuterwall, Ahlbom, and de Faire (2001) demonstrated that family history of coronary heart disease is a strong risk factor for myocardial infarction (MI), and that its effect is synergistic with other cardiovascular risk factors as well. Sesso et al. (2001) found that early history of parental MI conferred a greater risk of CVD than did MI at older ages. They further found that maternal history of MI at any age was significant. Nasir et al. (2004) reported a highly significant association between a family history of premature coronary heart disease (CHD) in a first degree relative and the presence and extent of atherosclerosis in asymptomatic individuals. A multi-ethnic study supported this finding and recommended that family history be included in current methods of global risk assessment and practice guidelines (Nasir et al., 2007).

Using data from the HealthStyles 2003 survey, researchers assigned stratified risks based on the number of family members with CVD, degree of kinship, and age of onset. Those with stronger familial risk for CVD had five times the early-onset CVD when compared with those in the weak risk group (Scheuner et al., 2006b). Zureik et al. (2006) compared carotid-femoral pulse-wave velocity and carotid ultrasound examination to paternal longevity and demonstrated that paternal age at death would predict in the offspring the degree of atherosclerosis. Kim et al. (2004) demonstrated that family history of stroke in first-degree relatives was significantly associated with an increase in stroke after adjustment for risk factors such as BMI, physical activity and smoking.

Several studies indicate that a history of sibling CVD confers a greater risk than parental CVD. Possible explanations for the increased risks are shared genetic makeup and common environmental exposures (Murabito et al., 2005). Nasir et al. (2004) demonstrated that a sibling history of premature CHD was more strongly associated with subclinical atherosclerosis than a parental history. Twin studies using the Swedish Twin Registry identified sibling death from CVD as a risk factor for CVD in the surviving twin. Marenberg, Risch, Berkman, Florderus, and deFaire (1994) did a 26-year twin follow-up and found that at younger ages, death from CVD is influenced by genetic factors, but this effect decreases at older ages. Zdravdovic et al. (2002) did the 36-year follow-up on twins and demonstrated a genetic influence in CVD mortality throughout the entire life span. Friedlander et al. (2001) reported that a family history of myocardial infarction is positively associated with the risk of early myocardial infarction and suggested that CVD in a sibling might be a higher risk factor than CVD in a parent. Using data from the Framingham Offspring Study, Murabito et al. (2005) found that having a sibling with CVD was a risk factor in middle-aged adults above that accounted for by parental CVD and other risk factors. These findings provide evidence that sibling CVD is a predictor of familial vulnerability to the disease.

Because CVD runs in families, a family medical history can help predict disease risk and identify recommendations for health promotion and disease prevention behaviors (Arnett et al., 2007; Williams et al., 2001). The Utah Department of Health, University of Utah, and Baylor College of Medicine followed 151,188 families who constructed family health pedigrees through a school program over a 20 year span. High-risk families were identified for follow-up interventions. At the 10 year evaluation, high risk families had increased health promotion behaviors, showing that family history is a cost effective way to identify high-risk families and to motivate them to utilize prevention strategies (Johnson et al., 2005). However, in two five-year follow-ups of the Coronary Artery Risk

Development in Young Adults (CARDIA) study, the occurrence of a heart attack or stroke in an immediate family member did not lead to self-initiated, sustained change in modifiable risk factors (Kip, McCreath, Roseman, Hulley, & Schreiner, 2002).

The collection and interpretation of family health history is the best way to identify those with a genetic susceptibility to CVD (Dudley-Brown, 2004; Scheuner, 2004). Scheuner, Yoon, and Khoury (2004) found that those at highest risk for common chronic disease might have a Mendelian disorder that can be recognized by analyzing patterns of the disease on a genogram or pedigree. In these cases an accurate family history can lead to risk assessment, genetic counseling and education, and recommendations for screening and prevention in high risk families. Fischer et al. (2005) suggested that, because of the high heritability of particularly severe manifestations of coronary artery disease that screening of asymptomatic relatives may be an important implication for clinical practice. Wiegman et al. (2003) analyzed lipid profiles of children with a positive family history of CVD and concluded that children with positive family history would receive significant benefit from early lifestyle modification as well as pharmacological intervention to prevent CVD.

The literature review also yielded results of prior research on the awareness of family history and reactions to it. Scheuner, Whitworth, McGruder, Yoon, and Khoury (2006a) studied the awareness of family history of CVD. Knowledge of disease was more accurate for first than second-degree relatives. There was significantly greater knowledge of disease status of mothers than fathers, and of maternal first degree-relatives compared to paternal relatives. Women and men had similar awareness of familial health status.

Several studies have addressed reactions to knowledge of family history. Hunt, Davison, Emslie, and Ford (2000) conducted a longitudinal study of how people deal with thoughts, beliefs, and information about their own inherited predispositions to CVD. Perception of family history of CVD was positively related to the number of relatives with CVD, to self-ascribed candidacy or susceptibility, to greater level of importance of family illnesses, and to the importance of lifestyle in prevention of CVD. In a follow-up study, researchers (Hunt, Emslie, & Watt, 2001) investigated factors that affected whether people perceive a family history of CVD. They found that perceptions depended on family health knowledge, and the number, closeness and age of affected relatives. Walter, Emery, Braithwaite, and Marteau (2004) reported that people with a family history of CVD develop a personal sense of vulnerability or "coronary candidacy" (p. 592) that includes physical characteristics, personality, and behaviors in addition to inheritance. Walter and Emery (2005) describe a personal sense of vulnerability to familial disease that extends beyond the counting of affected relatives to the emotional impact of witnessing a family member's illness,, and to likenesses and emotional closeness with the affected family member. Walter and Emery (2006) reported that people who perceived their risk as inherited felt more vulnerable and in less control, while those who perceived their risk as lifestyle-related and felt capable of modifying behaviors, also felt more capable of controlling their risk. Likewise Marteau and Weinman (2006) reported that an inherited predisposition to heart disease reduced the expectation that behavioral means of coping would be effective. McCusker et al. (2004), using data from the 2001 Healthstyles survey, found that almost half reported a family history of CVD, but that there were no significant associations between lifestyle changes and family history-based CVD risk.

Hall, Saukko, Evans, Qureshi, and Humphries (2007) examined how clinicians and patients understand and communicate family history in the context of CVD risk assessment. They found that patients value and are satisfied with family history assessment when the clinician focuses on modifiable risks. Patients had concerns and uncertainties if the clinician did not explore the patient's explanation of their family history, did not discuss the relationship between family history and other risk factors, or did not explain the family history in relation to overall risk. Yoon et al. (2002) believe that family history of CVD could be useful in teaching families that genetic susceptibility is not health destiny, since modifiable risk factors interact with familial risk to contribute to CVD. Modifiable risk factors, including tobacco use, obesity, and inactivity, are explored in the literature review.

Tobacco as a modifiable risk factor for CVD. Cigarette smoking and exposure to tobacco smoke are associated with chronic diseases and death, economic losses to society, and a substantial burden on the healthcare system. Worldwide, more than five million people die annually from the use of tobacco (CDC, 2008). In the United States, tobacco use and exposure to tobacco smoke resulted in 443,595 premature deaths (269,655 males and 173,940 females), approximately 5.1 million years of potential life lost, and $96.8 billion in lost productivity each year (Adhikari et al., 2008). The National Health Interview Survey for 2006 indicated that 21% of all U.S. adults were current smokers and an additional 21% were former smokers (Pleis & Lethbridge-Cejku, 2007).

In 2007, 22% of men and 17.5% of women in the U.S. were smokers. American Indian or Alaska Natives were more likely (32.4%) to be current smokers than African Americans (23.0%), whites (21.9%), Hispanics (15.2%), and Asians (10.4%) (CDC, 2008; Lloyd-Jones, 2009).

Tobacco use is a major cause of CVD in women. For women under 50 years of age, the majority of heart disease is attributable to smoking. Women who smoke have an increased risk of ischemic and hemorrhagic stroke that is reversible after 5–15 years of smoking avoidance. Women who use oral contraceptives compound the risk of CVD. Women who stop smoking greatly reduce their risk of dying prematurely. The risk decreases to that of those who never smoked with 10-15 years of smoking cessation. Tobacco use also increased the risk of peripheral vascular atherosclerosis in women (CDC, 2001).

Tobacco use is determined to a great extent by behaviors learned in childhood. Worldwide, 14% of 13 to 15-year-olds smoke cigarettes (Mackay et al., 2004).
In the U.S., 23% of high school students are cigarette smokers, with 1,300 persons under the age of 18 years becoming regular smokers each day (CDC, 2008). The Youth Risk Behavior Surveillance survey for 2007 indicated that in ninth through twelfth grade students, 21.3% of boys and 18.7% of girls reported current tobacco use. The prevalence was higher among whites (23.2%) than among blacks (11.6%) and Hispanics (16.7%). Nationwide, 13.6% of students had smoked cigars and 7.9% had used smokeless tobacco (Eaton et al., 2008).

Multiple risk assessment tools including the National High Blood Pressure Education Program coordinated by the National Heart, Lung, and Blood Institute and the National Institutes of Health assess cigarette smoking as a major CVD risk factor (USDHHS, 2003). Your Disease Risk (Siteman Cancer Center, 2007) is an online risk assessment tool that considers tobacco use a modifiable risk factor for CVD.

Khot et al. (2003) determined that smoking decreased the age at the time of a CHD event by nearly a decade in all risk factor combinations. Hawe, Talmud, Miller, and Humphries (2003) found that the risk of CVD was three times higher in smokers with a family history of CVD compared to non-smokers with a family history of CVD, which is greater than the risk posed by smoking or family history alone.

Use of tobacco increases the risk of CVD, especially among those who smoke heavily or started smoking at an early age. Passive smoking presents an additional risk (Mackay et al., 2004). Smoking promotes heart disease and stroke by damaging the lining of blood vessels, increasing cholesterol plaques in the artery walls, reducing blood vessel distensibility and compliance, and leading to hardening of the arterial walls. Smoking accelerates the heart rate, raises blood pressure, raises LDL-cholesterol, lowers HDL-cholesterol, and causes spasm of the coronary artery. Smoking causes an increase in fibrinogen levels and platelet aggregation, promoting clot formation (Goldstein et al., 2001; Mackay et al., 2004; Siteman Cancer Center, 2007).

Worldwide, a fifth of all CVD is attributed to tobacco use. In those under the age of 65, two-fifths of deaths from stroke are linked to smoking (Mackay et al., 2004). Tobacco use is the leading preventable cause of death in the United States causing an estimated 438,000 deaths, or one of every five deaths, annually. Tobacco use results in a 2- to 3-fold increased risk of dying from cardiovascular disease (CDC, 2008). Cigarette smokers have double the risk of stroke and more than ten times the risk of developing peripheral vascular disease. Smoking causes 3.3 million years of potential life lost for men and 2.2 million years for women. Smokers die an average of 14 years earlier than non-smokers (CDC, 2008; Lloyd-Jones, 2009). Jomini et al. (2002) found that cigarette smoking was the most prevalent risk factor for premature coronary artery disease. Yusuf et al. (2004) reported that smoking accounted for 36% of the population attributable risk of myocardial infarction worldwide.

Women who smoke have a higher risk of CVD than men who smoke. Women who smoke double their risk of CVD by smoking only three to five cigarettes a day compared to men who smoke six to nine cigarettes a day (Mackay et al., 2004). Yusuf et al. (2004) found that smoking showed a graded relation with the odds of myocardial infarction, with smoking of even five cigarettes per day increasing risk. This implies that there is no safe level of smoking, and that if smoking cessation is not possible, the risk could be significantly reduced by smoking reduction. The risk of stroke decreases to that of non-smokers after 5 years of cessation (Goldstein et al., 2001).

Approximately 126 million children and nonsmoking adults are exposed to second hand smoke (Lloyd-Jones, 2009). Exposure to secondhand smoke has immediate adverse effects on the cardiovascular system, increasing the risk of CVD by 25-30% (Pechacek & Babb, 2004; USDHHS, 2006a). Each year, 62,000 people die in the United States from CVD attributed to passive smoking. In adults, passive smoking clogs and weakens arteries causing heart attack, angina and stroke. In children, passive smoking reduces the oxygen carrying capacity of the blood, damages arteries, causes early-onset atherosclerosis, and sudden infant death syndrome (Mackay et al., 2004). The Pueblo Heart Study (Bartecchi et al., 2006) reported a 27% decrease in hospitalizations for myocardial infarction 18 months after a municipal smoke-free ordinance was enforced. Alsever et al. (2009) extended the analysis for an additional 18 months and reported a 41% reduction in hospitalizations from the pre-implementation period.

Globally, health care costs associated with smoking-related illnesses result in a global net loss of $200 billion per year (Mackay et al., 2004). In the United States, direct medical costs ($96 billion) and lost productivity costs ($97 billion) associated with smoking combine for a total of $193 billion per year (CDC, 2008; Lloyd-Jones, 2009). Healthcare costs associated with passive smoking average $10 billion annually in the United States (CDC, 2008).

Tobacco smoking is strongly associated with peripheral vascular disease (PVD). Researchers reported that the prevalence of smoking was twice as high in the siblings of those with premature PVD who demonstrated asymptomatic PVD on ultrasound (Valentine, Verstraete, Clagget, & Cohen, 2000). Valentine et al. (2004) later demonstrated that smoking and family history combine to increase the risk of PVD in young adults. In light of the strong associations between smoking and CVD, the AHA Guidelines for Primary Prevention of Cardiovascular Disease and Stroke (Pearson et al., 2002) recommends complete cessation of smoking and avoidance of exposure to environmental tobacco smoke.

Madlenski, Esplen, Gallinger, McLaughlin, and Goel (2003) have demonstrated that risk information may motivate participation in preventive screening and intervention programs. McBride et al. (2002) explored risk perceptions and rates of smoking cessation when genetic susceptibility feedback was added to standard smoking cessation interventions. They reported that feedback of increased genetic susceptibility was not associated with significantly increased smoking cessation or risk perceptions. However, they also reported that feedback of not being susceptible did not undermine success at smoking cessation.

Sanderson and Wardle (2005) studied smokers' reactions to genetic testing for smoking-related diseases and reported that 65% of participants were motivated to quit smoking upon receiving a positive test result of genetic susceptibility to disease. Sanderson and Michie (2007) examined the potential impact of genetic testing for heart disease susceptibility on psychological predictors of smoking cessation. They reported that smokers with a high genetic risk of heart disease had greater perceived risk and greater motivation to quit smoking than smokers in other test groups. They suggested that genetic testing for heart disease risk may enhance smoking cessation interventions. In addition to tobacco use, another modifiable risk factor that must be considered in the impact of cardiovascular disease is obesity.

Obesity as a modifiable risk factor for CVD. Overweight and obesity are known risk factors for coronary heart disease, hypertension, hypercholesterolemia, stroke and many other chronic diseases (Weight-control Information Network [WIN], 2007). The risk of CVD increases for those with a higher Body Mass Index (BMI) and is especially acute for people who carry extra body fat around the waist. Waist-to-hip ratio of more than 0.90 in men or 0.85 in women is associated with nearly a threefold increased risk of CVD (Goldstein et al., 2001; NHLBI, 1998). Abdominal obesity is an independent risk factor for stroke in all races and ethnic groups, and is a stronger risk factor than BMI (Suk et al., 2003). Weight loss reduces the risk for CVD by lowering blood pressure, reducing serum triglycerides, increasing high-density lipoprotein, decreasing total serum cholesterol and low-density lipoprotein, and decreasing blood glucose levels (NHLBI, 1998).

The WHO (2005) estimates that 2.6 million people die annually as a result of being overweight or obese. Globally, for the year 2005 it was estimated that over 1 billion people were overweight and 300 million were obese. If current trends continue it is estimated that over 1.5 billion people will be overweight by 2015 (WHO, 2005). In the United States, the 2005 prevalence of overweight (BMI $\geq$ 25.0) was 142 million or 66.0% of the population, and the prevalence of obesity (BMI $\geq$ 30.0) was 57.3 million or 31.4% (AHA, 2008). These figures are up from 44.8 % and 13.3% respectively since 1960 (WIN, 2007). The National Center for Health Statistics (2009) also shows an increase from 0.9% to 6.2% in the extremely obese category (BMI $\geq$ 40.0). The prevalence of obesity varies by sex, race and ethnicity. The prevalence of overweight in men has increased significantly while the prevalence in women is leveling off (Ogden et al., 2006). In 2004, 34% of women and 30% of men were obese. While the prevalence of obesity among men was similar by race and ethnicity, the prevalence among women differed with half of blacks and one-third of whites being obese (NCHS, 2007).

Obese individuals have a 10% to 50% increased risk of death from all causes compared to healthy weight individuals with most of the increased risk due to CVD. The direct cost of overweight and obesity in this country is $61 billion and the indirect cost is $56 billion for a total of $117 billion annually. The cost of lost productivity is $3.9 billion (WIN, 2007). When National Health and Nutrition Examination Survey (NHANES) mortality data was combined with data on BMI, Flegal, Graubard, Williamson, and Gail (2007) determined that 13% of total CVD mortality was associated with obesity (BMI $\geq$ 30). Although there has been an increase in obesity over the past quarter century from 13% to 31%, Gregg et al. (2005) noted a corresponding reduction in hypercholesterolemia, hypertension, and smoking among obese persons by 21, 18 and 12 percentage points respectively compared with obese individuals three to four decades ago. While these risk factors are lowering, obesity is still associated with a higher prevalence of important CVD risk factors. The AHA Guidelines for Primary Prevention of Cardiovascular Disease and Stroke (Pearson et al., 2002) recommends achievement and maintenance of desirable weight and waist circumference through weight management programs of caloric restriction and caloric expenditure. Suk et al. (2003) contends that prevention of obesity and weight reduction needs greater emphasis in stroke prevention programs.

Overweight has serious consequences in young people. The prevalence of overweight and obesity in children and adolescents is increasing (Ogden et al., 2006). Worldwide, approximately 18 million children under five years of age are overweight (Mackay et al., 2004). Nationwide, 13.1% of students are overweight (Eaton et al., 2008).

The prevalence of overweight among preschoolers is 14%, doubling that of a decade ago (NCHS, 2007). The prevalence of overweight for school-age children is 17.5%, and for adolescents is 17% (AHA, 2008; WIN, 2007). The highest prevalence is among African American female school-age children (24.8%) and adolescents (23.8%) and Mexican American male school children (24.8%) (AHA, 2008). The NHANES of the NCHS showed the prevalence of overweight in school age children increased from 4.0% in 1971-1974 to 17.0% in 2003-2006, and in adolescents from 6.1% to 17.6%. Even in children 6 to 23 months of age, high weight-for-age increased from 7.2% to 11.5% during that time period (Lloyd-Jones et al., 2009). These trends are having adverse effects on the health of children and youth that persist into adulthood.

Not only is obesity increasing in children, but CVD is now manifesting in childhood. These children will become seriously ill at younger ages with complex, expensive co-morbidities, and die at younger ages utilizing a disproportionate share of medical resources (Lavisso-Mourey, 2007). They will have lower quality of life than healthy children, and low quality of life similar to that of children diagnosed with cancer (Scheimmer, Burwinkle, & Varni, 2003). One of the contributors to overweight and obesity is inactivity. However, physical inactivity is an important independent modifiable risk factor for cardiovascular disease.

Physical inactivity as a modifiable risk factor for CVD. Regular physical activity is fundamental to maintaining or improving physical and mental health in general, and in reducing the risk of CVD in particular (Hennel & Lemire, 2002). It is an important determinant of energy expenditure, energy balance and weight control. Physical activity reduces blood pressure, improves the level of high density lipoprotein cholesterol, improves blood glucose control, lowers plasma fibrinogen and platelet activity, and decreases blood viscosity (Goldstein et al., 2001; Sundquist, Qvist, Sundquist & Johansson, 2004; WHO, 2004). Physical inactivity increases the risk of obesity, heart disease and stroke, and is an important modifiable risk factor in the rising burden of chronic disease around the world (Guthold, Ono, Strong, Chatterji, & Morabia, 2008; Loyd-Jones, 2009; WHO, 2004; Yancy et al., 2007).

The WHO (2005) estimates that 1.9 million people die annually as the result of inactivity. In spite of health promotion recommendations, only about 40% of the population engages in regular leisure-time physical activity (AHA, 2008) or vigorous activity lasting more than ten minutes. Inactivity is higher in females (12%) than males (8.4%) and increases with age from 5.5% at 18 years to 24% at 65+ years. Blacks adults are more likely to report inactivity (16.7%) than are white adults (10.7%). The lack of vigorous leisure-time activity is inversely associated with educational attainment (Lloyd-Jones, 2009). Globally, more than 60% of the population is not sufficiently active. This physical inactivity causes about 1.9 million deaths and 20% of CVD (Mackay et al., 2004). The National Health Interview Survey for 2006 indicated that 62% of all U.S. adults never participated in any type of vigorous leisure-time physical activity (Pleis et al., 2007).

Physical exercise is linked to longevity, independent of genetic factors. The risk of CVD can be reduced by 30% with only 150 minutes of moderate physical activity or 60 minutes of vigorous physical activity each week. Compared with less active people, moderately active and highly active individuals reduce their risk of stroke by 20% and 27% respectively. In spite of the documented evidence of the benefit of physical activity, over a quarter million people in the United States die annually from inactivity (Mackay et al., 2004). A comparison of sedentary and highly active individuals showed that their relative risk for CVD death was 60% higher (RR=1.6) (Thompson, 2002). Physical inactivity is responsible for 12.2% of the global burden of myocardial infarction after accounting for other risk factors such as tobacco, diabetes, hypertension, hypercholesterolemia and obesity (Yusuf et al, 2004). In the U.S., the direct costs of physical inactivity accounted for an estimated $24 billion in health care costs in 1995 (Mackay et al., 2004).

In the United States, physical activity decreases precipitously around the age of 10 years, especially among girls (Mackay et al., 2004). As reported in the Youth Risk Behavior Surveillance survey for 2007, one-fourth of all students spent more than three hours per day using computers outside of school or watching television. Less than one third of students attend daily physical education classes. In children aged 9 to 13 years of age, 61.5% participate in no organized physical activity after school and 22.6% engage in no free-time physical activity. By 16 years of age, 31% of white girls and 56% of black girls report no leisure-time physical activity. Smoking, pregnancy and increased BMI are associated with the decline in physical activity among girls (Eaton, 2008). Treatment recommendations for children include more than 1 hour of active play per day and limitation of screen time to less than 2 hours per day (Karvey, 2006).

Even at an older age, physical activity can significantly reduce the risk of CVD as well as that of other diseases such as diabetes, colon cancer, breast cancer and depression (Mackay et al., 2004). Leisure time activity decreases all-cause mortality among men and women over 65 years of age. Even occasional activity increases longevity in the elderly according to Sundquist et al. (2004). They found that even once weekly physical activity decreased all-cause mortality, and especially CVD mortality. Hillsdon, Brunner, Guralnik, and Marmot (2005) contend that participation in a physically active lifestyle during middle age is critical to maintaining high physical functioning at older ages. This mid-life activity extends the years of independent living and leads to improved quality of life in older age.

Tavani et al. (2004) assigned adult lifestyle risk scores to risk factors for ischemic heart disease in participants with a family history of heart disease and compared them to those without family history. They concluded that the risk for heart disease could be substantially reduced by selected lifestyle interventions. The Nurses' Health Study, in which over 84,000 women were followed for 14 years, demonstrated that women who did not smoke, maintained a healthful diet, exercised for a half hour a day, and consumed alcohol moderately, experienced 80% fewer cardiac events than the rest of the population (Stampfer et al., 2000).

While it is individual choice and personal or familial responsibility to practice health promoting behaviors, individual motivation to be physically active is increasingly difficult in a society characterized by step- and labor-saving devices, and aggressive marketing of sedentary entertainment and transportation. Given this increase in inactivity and sedentary behaviors, physical activity promotion becomes a critical function of public health practice (Yancy et al., 2007). Moderate activity such as brisk walking for 30-60 minutes on most days is associated with significant reductions in CVD incidence and mortality (Bauman, 2004; Brown, Burton, & Rowan, 2007; Goldstein et al., 2001; Haennel & Lemire, 2002). The AHA Guidelines for Primary Prevention of Cardiovascular Disease and Stroke (Pearson et al., 2002) recommends at least 30 minutes of moderate intensity physical activity on most, and preferably all, days of the week augmented by flexibility training and increase in daily lifestyle activities. In addition, the WHO (2004) recommends muscle strengthening and balance training to improve balance and function in older adults. While family history, tobacco use, obesity and inactivity are all individual risk factors for CVD, the risks are compounded when there are multiple risk factors.

Multiple risk factors for CVD. Greenland, Smith, and Grundy (2001) estimated that the 35% of the U.S. adult population who have no identifiable CVD risk factors, would be considered low risk for coronary events both short- and long-term, and can be reassured about their risk status without further assessment or screening. Conversely, multiple risk factors significantly compound the risk for CVD. Family history is synergistic with smoking in predisposing to CVD (Leander et al., 2001). Hawe et al. (2003) found that the risk for those with a positive family history who were also current smokers was over three times (RR=3.01) that of non-smokers without a family history, which is greater than the risk posed by either smoking or family history alone. In a 30-year follow-up of the Chicago Heart Association Detection Project in Industry, participants with two or more risk factors had six times the CVD mortality rate as those in the low-risk category of no risk factors (Daviglus et al., 2004). Both smoking and obesity are independent predictors of mortality from CVD, but the combination poses an especially high risk (Koster et al., 2008). Obese smokers live 14 fewer years than nonsmokers of normal weight (Mackay et al., 2004). Freedman et al. (2006) found that combining obesity with current smoking poses especially high risks for CVD mortality in people under 65 years of age, with 6- to 11-fold risks compared to those of normal weight who had never smoked. Fear of weight gain upon smoking cessation can be a barrier for obese smokers. The National Heart, Lung, and Blood Institute (1998) clinical guidelines emphasize the importance of abstinence from smoking with the treatment of weight gain through dietary therapy, physical activity and behavioral therapy. Thomas et al. (2002) studied adults who were attempting to change both dietary and physical activity habits, and found that physician advice was a strong predictor of attempts to change both lifestyle habits. This finding supports the positive impact of health professional advice.

Pronk, Peek, and Goldstein (2004) identify an increasingly urgent need for a collaborative approach by multiple stakeholders in addressing multiple behavioral risk factors and their interventions. Based on evidence in the literature for the efficacy of smoking reduction, weight loss and physical activity, the Addressing Multiple Behavioral Risk Factors in Primary Care Project suggests a unifying conceptual framework for describing, delivering, and evaluating health behavioral counseling interventions in the primary health care setting (Goldstein, Whitlock, & DePue, 2004). Prochaska et al. (2005) contend that treating multiple health behavior risks on a population basis is one of the most promising approaches to enhancing health and reducing health care costs. They demonstrated that proactive, home-based, and stage-matched programs can produce a relatively high impact on multiple risks for chronic diseases.

Unless the spread of risk factors is controlled, the world faces an epidemic of CVD. According to the Director-General of the WHO, CVD will reach global epidemic proportions unless urgent preventive action is taken (Mackay et al., 2004). This research study explored the use of genograms as threat appeals to motivate health promoting behavior changes. In addition to the literature on CVD and its risk factors, the review of the literature yielded information about fear or threat appeals as well as information about the Extended Parallel Process Model and its related theories.

Theoretical Foundations

Fear / Threat Appeals

Most persuasive health risk messages are fear appeals. According to Witte, Meyer, and Martell (2001), most people ignore logical health risk messages, and respond only when the message arouses them emotionally. Persuasive health risk messages that gain compliance by arousing fear are called fear or threat appeals, often also called scare tactics. Fear appeals contain two components – a threat and a recommended response. The threat tells the negative consequences that can occur and the recommended response tells what can be done to avoid the threat (Witte et al., 2001). Threat appeals focus on health risks and use persuasive messages that evoke anxiety and fear. These appeals attempt to persuade people to change their attitudes, intentions, and behaviors in order to improve their health (Witte, 1991). Fear appeals offer feasible recommendations that are presented as effective in averting the threat. The central constructs of fear appeals are fear, threat and efficacy. Witte (1993) contends that the more intense and personalized the message, the greater the perceptions of threat and fear. She emphasizes the importance of separating the impact of threat from the impact of fear, because failure to do so makes it impossible to differentiate the unique and independent effects of each on persuasive outcomes of fear appeals. According to Ruiter, Kok, Verplanken, and van Eersel (2003), attempts to promote healthy behaviors by arousing fear profit from approaches that stress the negative consequences of not performing the recommendations than those that stress the positive consequences of performing the recommendations.

Research has shown that fear appeals are powerful persuasive devices, but only in certain circumstances (Lapinski & Witte, 1998). The intended outcome is message acceptance, and attitude, intention and behavior change. However, possible alternate outcomes are defensive avoidance, resistance, denial or minimization of the threat, and reactance (Witte, 1992a).

According to Green and Witte (2006), the first fear appeal study in 1953 showed a negative relationship between the fear appeal and the recommended health behavior, resulting in two generations of health educators avoiding fear appeals. However, subsequent fear appeal literature suggests that fear appeals are reliable and consistent in producing behavior change. Four meta-analyses using different statistical methods each concluded that the stronger the fear appeal, the greater the change in attitude, intention, and behavior (Green et al., 2006). The meta-analysis by Sutton (1982) reported significant positive effects for strength of fear appeal on intentions and behaviors. Two additional meta-analyses by Boster and Mongeau (1984) and Mongeau (1998) examined the influence of a fear appeal on perceived fear, attitudes and behaviors, and found moderate associations between reported fear and strength of fear appeal, and modest but reliable relationships between the strength of a fear appeal and attitude change. The most recent meta-analysis by Witte and Allen (2000) of 100 experimental studies using fear appeals supported the persuasive effects of fear appeals when they are accompanied by high-efficacy messages. These investigators contend that strong fear appeals result in high levels of perceived severity and perceived susceptibility and are thus more persuasive than weaker fear appeals. These fear appeals increase motivation to make adaptive danger control behavior changes when accompanied with high-efficacy messages. As long as individuals believe they are capable of making the recommended response that is effective in averting the threat, the greater the fear or threat, the greater the behavioral change. When accompanied by low-efficacy messages, strong fear appeals produce defensive responses (Green et al., 2006; Witte et al., 2000). Threat or fear appeals evolved from the Health Belief Model (HBM), which is one of the most commonly used conceptual models of health behavior change.

Health Belief Model

The Health Belief Model (HBM) is viewed as the grandmother of most modern health education theories. It was developed by a group of social psychologists in the early 1950s as a framework for promoting prevention behaviors. The HBM proposes that preventive health behavior is influenced by five factors: perceived barriers to making the recommended response, perceived benefits to making the response, perceived susceptibility to the health threat, perceived severity of the threat, and cues to action (Witte, 2006). According to the HBM, people take action if they believe that they are susceptible to a disease condition, that it has potentially serious consequences, that the recommended action would reduce the severity of the condition or their susceptibility to it, and that the barriers to taking action are outweighed by the benefits. Within the HBM, cues to action are triggers that activate a person's readiness to change health-related behaviors, and self-efficacy is a person's confidence in his ability to change those behaviors (Janz, Champion, & Strecher, 2002). The fear appeal may be thought of as the "cue to action" in the HBM. According to Witte (2006), fear appeals are experimental variants or explanatory versions of the HBM. The most recent fear appeal theory is the Extended Parallel Process Model (EPPM) that is an expansion of Leventhal's danger control / fear control Parallel Process Model.

Parallel Process Model

Hovland, Janis and Kelly's (1953) work with the fear-as-acquired drive model purported that fear arousal was needed to elicit a motivational drive state, but that too much fear would result in defensive avoidance. Based on this model, Leventhal (1971) developed the Parallel Process Model. This model focused on cognitive processes rather than emotional processes, arguing that protective adaptive behavior resulted from attempts to control a threat (cognition), not attempts to control the fear (emotion) aroused by the threat. According to this model, individuals who think about the threat and develop strategies to avert it were engaging in danger or threat control processes, while those who focus on the fear and ways to control it experience fear control processes. While this model offers the useful distinction between the cognitive and emotional reactions, it lacks precision and fails to specify what factors would elicit each response and when one process would dominate the other (Witte, 1992a). To address these deficiencies, Witte (1991) proposed the Extended Parallel Process Model (EPPM), which expands on Leventhal's Parallel Process Model.

Extended Parallel Process Model

Expanding on the Parallel Process Model, the EPPM specifies what leads to danger control versus fear control, explains when one process should dominate over the other, and details the expected outcomes of each process. According to the EPPM, a health threat initiates two cognitive appraisals – an appraisal of the threat and an appraisal of the efficacy of the recommended response. The first appraisal of the threat involves perceived severity of the threat and perceived personal susceptibility to the threat. If the threat is deemed irrelevant or trivial, there is no motivation to process the health message further and the message is ignored. In contrast, if the appraisal determines vulnerability to the threat (susceptibility), or that it could lead to serious harm (severity), the result is fear and motivation to act. The greater the threat, the more motivated the individual is to begin the next appraisal of efficacy. If the individual believes that the recommended response is effective in averting the threat (response efficacy), and if he believes he is capable of performing the recommended response (self-efficacy), he will take action to protect himself from the danger (protection motivation). This danger control motivation stimulates adaptive attitude, intention, or behavior changes to control the threat, usually by accepting the message of the fear appeal. Thus, when perceived threat (severity and susceptibility) and perceived efficacy (response-efficacy and self-efficacy) are high, danger control processes dominate and result in adaptive outcomes (Roberto, 2004; Witte, 1998; Witte et al., 2001; Witte, 2006).

When perceived threat is high but perceived efficacy is low, fear control processes dominate and result in maladaptive responses. These individuals respond to and cope with the fear, not the threat (Witte, 1992a; Witte, 2006). If this happens, fear appeal campaigns backfire. At some critical point, if perceptions of threat exceed perceptions of efficacy, the individual will shift into fear control processes (Witte et al., 2001; Witte, 2006). If the individual realizes he cannot prevent a threat because he believes the response is ineffective or believes he is incapable of performing the recommended response, fear control processes will begin to dominate over danger control processes. When this happens the individual will respond to and cope with the fear, not the danger (defensive motivation) through defensive avoidance, denial or reactance. Defensive avoidance occurs when the individual blocks further thoughts or feelings about the threat, or ignores or distorts the message. Denial occurs when the individual refuses to believe the severity of the threat or his susceptibility to it. Reactance occurs when the individual believes he is being manipulated and rejects the message or becomes angry about the entire issue (Roberto, 2004; Witte, 1998; Witte et al., 2001; Witte, 2006).

Therefore, for fear appeals to be successful, the individual must perceive the threat is serious, that he is personally susceptible to it, that the recommended response will control the threat, and that he is capable of making the recommended response (Witte, 1992a; Witte, 2006). Green et al. (2006) state that "the essence of the model is that perceived threat (causing fear arousal) motivates action, and perceived efficacy (causing hope) determines the nature of that action" (p. 249). The EPPM has potential implications for health educators in promoting efficacy as well as threat in order to motivate individuals to adopt health promotion behaviors (Witte, 1991). This model has been extensively applied in a variety of research methods, for a variety of research topics, and with a variety of research participants.

The EPPM has been tested with a variety of research methods including experiments (Gore & Bracken, 2005; McMahan, Witte, & Meyer, 1998; Roberto, Meyer, Johnson, & Atkin, 2000; Witte, 1992b; Witte, 1994a; Witte, Berkowitz, Cameron, & McKeon, 1998; Witte & Morrison, 1995a; Witte & Morrison, 1995b; Witte, Sampson, Liu, & Morrison, 1995), surveys (Murray-Johnson et al., 2001; Witte et al., 1993; Witte, 1994b; Witte, Cameron, McKeon, & Berkowitz, 1996), focus groups (Witte, 1997; Witte, Cameron, & Nzyuko, 1995; Witte et al., 1996; Murray-Johnson et al., 2004) and interviews (Smith, Ferrara, & Witte, 2007). Teen pregnancy prevention (Witte, 1997), tractor safety (Witte et al., 1993), HIV/AIDS prevention (Murray-Johnson et al., 2001; Witte, 1992b; Witte, 1994a; Witte, 1994b; Witte, Cameron et al., 1995; Witte & Morrison, 1995a; Witte & Morrison, 1995b; Witte, Girma & Girgre, 2002-2003), adoption of AIDS orphans (Smith et al., 2007), genital warts (Witte, Berkowitz, Cameron, et al., 1998), meningitis (Gore et al., 2005), noise-induced hearing loss (Murray-Johnson et al., 2004), gun safety (Roberto et al., 2000), electromagnetic fields (McMahan, Witte, & Meyer, 1998), and radon awareness (Witte, Berkowitz, McKeon, et al., 1998) are some of the topics in EPPM studies. EPPM studies have focused on many different populations including juvenile delinquents (Witte & Morrison, 1995b), junior high school students (Murray-Johnson et al., 2001; Witte, Sampson, et al., 1995), high school students (Witte, 1997; Witte & Morrison, 1995b), college students (Gore et al., 2005; McMahan et al., 1998; Witte, 1992b; Witte, 1994a; Witte, 1994b; Witte & Morrison, 1995a; Witte, Berkowitz, Cameron, et al., 1998), Ethiopian urban youth (Witte et al., 2002-2003), Kenyan prostitutes (Witte, Cameron, et al, 1995), Namibian households (Smith et al., 2007), African-American smokers (Witte et al., 1996), farmers (Witte et al., 1993), gun owners (Roberto et al., 2000), coal miners (Murray-Johnson et al., 2004) and truck drivers (Witte, Cameron, et al., 1995). One experimental study involved communicating CVD risk levels (McKay, Berkowitz, Blumberg, & Goldberg, 2004). With these diverse methods, topics and populations, threat appeals appear to interact with efficacy in a way that high threat and high efficacy messages promote danger control processes leading to message acceptance. Conversely, high threat and low efficacy messages promote fear control processes leading to message rejection and various psychological defenses (Witte & Allen, 2000). While the primary theoretical background for this study is the EPPM, and the Risk Behavior Diagnosis Scale assessed its constructs in study participants, the survey also assessed the participants' readiness to change.

Stages of Change

In addition to assessing the perceived threat and efficacy constructs of the EPPM, this study assessed the participants' stage of change with regard to their collection of family history and their behaviors to prevent CVD before and after completing the genogram. These Stages of Change derive from the Transtheoretical Model (TTM). The TTM (Prochaska, Redding, & Evers, 2002) states that behavior change occurs through a series of stages along a continuum of no action to consistent action - precontemplation, contemplation, preparation, action, and maintenance. In the stage of precontemplation the individual does not intend to change his behavior. He may not realize he is engaging in risky behavior or he may deny that the behavior puts him at risk of harm. In the contemplation stage the individual begins to think about the risky behavior and the need to change it. The next stage, in which the individual begins to change, is the preparation stage. It is in this stage that the commitment to change is made and actions are taken to prepare for the behavior change. In the action stage the individual performs the recommended change consistently. The final stage is maintenance, during which time the new behavior is continued and steps are taken to avoid relapsing into the formerly risky behaviors (Lapinski et al., 1998).

The TTM is useful to researchers and health educators in analyzing and segmenting a population or audience based on their readiness to change their behaviors. Health educators can then strategically tailor health communication messages to move individuals through the stages. These tailored messages differ across the stages. Messages that move people from precontemplation to contemplation focus on increasing awareness and knowledge of the health threat and recommended response. Motivational messages move people from contemplation to preparation and from preparation to action. Messages that focus on response efficacy or self-efficacy move people from action to maintenance (Witte, Meyer & Martell, 2001).

The TTM includes consciousness-raising as one of the Processes of Change in advancing readiness to change. Another construct that fits well with the Extended Parallel Process Model is that of self-efficacy which balances confidence with temptation. Confidence in the individual's ability to engage in the recommended health-promoting behavior is weighed against the temptation to engage in the unhealthy behavior in various challenging situations (Prochaska et al., 2002). This study explored whether completion of the online family health genogram raises the participants' consciousness of their CVD risk and advances their stage of change or their readiness to adopt health promotion behaviors. This review of the literature provides background for the current study that will introduce genograms as threat appeals in motivating intent to perform health promotion and disease prevention behaviors in participants with a family history of CVD.

CHAPTER III

METHODOLOGY

Population and Sample

Volunteers with a family history of cardiovascular disease were initially recruited from the general population to participate in the study by taking a survey, completing an online genogram, and taking a second survey. To recruit participants, a flyer (see Appendix B) was posted in numerous locations, distributed at various community organizations or gatherings, sent by mail or e-mail attachment, and used as a script for verbal announcements. Thereafter a network or snowball sampling technique was used where initial participants were asked to recommend others who might be interested in participating in the study. In using the Risk Behavior Diagnosis Scale (RBD) to test the relationship between threat and attitudes or intentions to change behaviors, at least 150 participants are needed (Witte, Meyer, & Martell, 2001).

The initial participants were recruited from the university population including faculty, staff and students. To recruit volunteers from non-academic settings, presentations were made at meetings of a senior organization sponsored by the local medical center, the regional genealogical society, the hospital auxiliary, and the altar society at a local church. All initial participants were asked to refer additional volunteers. These network referrals contacted the researcher by phone or e-mail and indicated their interest in participating. One participant who was particularly interested in familial cardiovascular disease referred a dozen participants by recruiting relatives at a family gathering. Several retired Air Force nurses expressed interest after an initial recruit included contact information in a newsletter. A total of 214 participants signed consent forms with 171 (79.9%) returning both surveys and another 43 (20.1%) returning only the first survey and therefore not completing the study.

Demographic data were requested on the second survey. Results of participant responses to demographic questions are illustrated in Table 1. Over three-fourths of the participants were female (80.5%) which reflects the recruiting efforts in female dominated organizations. Over one-third (34.3%) were between 18 and 30 years of age, with fairly even distribution between 31 and 75 years of age, and only 3 (1.2%) participants were older than 76 years of age. Most (86.1%) of the participants had over 12 years of education, which can be explained by the initial recruitment within the university community. The majority (82.8%) of the participants indicated the ethnicity of most of their ancestors to be White. All other ethnic categories were represented and were within 2 percentage points of the ethnic make-up of Texas (U.S. Census Bureau, 2009) which suggests that the sample was representative of the population. About two-thirds of the participants were employed either full time (43.8%) or part-time (21.9%). The remaining one-third was unemployed (18.9%) or retired (15.4%). Over half (58.4%) ranked their job as moderately active, while over one-fourth (28.9%) considered their job inactive. The largest category (54.3%) had annual family incomes over $76,000 while the smallest category (8.0%) had incomes less than $25,000.

Table 1

Participant Demographic Responses

Category	Response	Frequency	%
Gender	Female	124	80.5%
	Male	30	19.5%
	Total N	154	90.0%
Age	18-30 years	58	34.3%
	31-45 years	38	22.5%
	46-60 years	34	20.1%
	61-75 years	36	21.3%
	76+ years	3	1.8%
	Total N	169	98.8%
Education	<8 years	10	6.0%
	8-12 years	13	7.8%
	>12 years	143	86.1%
	Total N	166	97.1%
Ethnicity	American Indian / Alaskan Native	4	2.4%
	Asian	2	1.2%
	Black / African American	18	10.7%
	Hispanic / Latino	3	1.8%
	Native Hawaiian / Pacific Islander	1	0.6%
	White	140	82.8%
	Other	1	0.6%

Table 1 cont.

Participant Demographic Responses

Category	Response	Frequency	%
	Total N	169	98.8%
Employment Status	Not employed	32	18.9%
	Employed part time	37	21.9%
	Employed full time	74	43.8%
	Retired	26	15.4%
	Total N	169	98.8%
Work Activity	Desk job (sitting)	48	28.9%
	Moderately active (walking)	97	58.4%
	Strenuous (lifting)	21	12.7%
	Total N	166	97.1%
Annual Income	< $25,000	13	8.0%
	$26,000 - $50,000	36	22.2%
	$51,000 - $75,000	25	15.4%
	>$76,000	88	54.3%
	Total N	162	94.7%

In addition to supplying the demographic information discussed above, participants also described their weight as underweight, normal weight, or overweight, their activity level as inactive, active, or very active, and their tobacco use as never used tobacco, used tobacco in the past, or use tobacco now. The results, detailed in Table 2, show that over one-half of the participants considered themselves normal weight and over one-third overweight. Over three-fourths described themselves as active. Over one-half had never used tobacco while almost one-third had used tobacco in the past. Only 13.7% were current tobacco users.

Table 2

Self-Ascribed Weight, Activity Level, and Tobacco Use Status

Category	Response	Frequency	%
Weight	Underweight	5	3.0
	Normal weight	96	56.8
	Overweight	68	40.2
	Total N	169	100.0
Activity Level	Inactive	27	16.0
	Active	130	76.9
	Very Active	12	7.1
	Total N	169	100.0
Tobacco Use	Never use tobacco	93	55.3
	Used tobacco in the past	52	31.0
	Use tobacco now	23	13.7
	Total N	168	100.0

The number of family members included in the participants' genograms ranged from 3 to 75 with a mean of 14.56. The number of family members with CVD ranged from 1 to 22 with a mean of 5.69. Table 3 illustrates that almost one-third of the participants had not thought about gathering their family history prior to the study and another one-third had thought about it but had not yet made plans to do so. Table 4 shows that one-half of the participants had already taken some of the recommended steps to prevent or control CVD, but only one in five consistently follows those recommendations.

Table 3

Initial Stage of Change for Gathering of Family Health History (FHH)

Response	Frequency	%
I have not thought about gathering my FHH.	54	31.6
I have thought about, but not made plans to gather FHH.	55	32.2
I plan to gather in my FHH in the near future.	10	5.8
I have started gathering my FHH.	24	14.0
I have gathered my FHH, and keep it current.	12	7.0
I have gathered my FHH, but not kept it current.	16	9.4

Table 4

Initial Stage of Change for Practicing CVD Preventive Behaviors

Response	Frequency	%
I have not thought about prevention steps.	15	8.8
I have thought about prevention, but have not made plans.	17	9.9
I plan to take recommended prevention steps.	14	8.2
I have taken some recommended prevention steps.	86	50.3
I consistently follow recommended prevention steps.	37	21.6
I have followed recommended steps before, but not now.	2	1.2

Protection of Human Participants

A *Proposal for Activity Using Human Subjects* was approved by the Human Subjects in Research Committee at Midwestern State University (MSU) on November 5, 2008 (see Appendix C). The proposal, with all accompanying documents, and the approval letter from MSU was sent to the Texas Woman's University Institutional Review Board (IRB) along with a memo requesting that the IRB defer approval to MSU. Exempt status was granted by TWU's IRB on December 9, 2008 (see Appendix D).

Data Collection Procedures

The data collection process for this study consisted of four steps. The first step for the participant was to read an American Heart Association brochure included in the mailed packet. The brochure listed risk factors for CVD and recommended health promotion behaviors. The second step was to complete the preliminary survey that included stages of change, the RBD scale, and intent to change behaviors (see Appendix E). The third step was to complete the online genogram at the U.S. Department of Health and Human Services website. Participants received instructions for returning the completed surveys to the investigator. Upon receipt of the completed preliminary survey, the follow-up survey was mailed to the participant. The fourth step was to complete and return the follow-up survey (see Appendix F) that again measured stages of change, RBD scale constructs, and intent to change behaviors. The follow-up survey also elicited demographic data. A crosswalk listing of survey items and their associated theory or construct is illustrated in Appendix G.

Following reliability testing, the initial surveys were sent out on January 1, 2009. The genogram completed by participants was *My Family Health Portrait* at the U.S. Surgeon General's Family History Initiative website (see Appendix H). Initially, several participants reported difficulties with the genogram program. Two participants who were computer experts reported spending up to 15 hours attempting to complete their genograms. In consultation with the USDHHS web support personnel, it was discovered that a new version of the program had been released on January 13, 2009 (less than 2 weeks after the start of the study) and was causing problems for Internet Explorer users. The web support personnel suggested using another browser until the problems could be resolved. A follow-up letter was mailed to all participants explaining the problem, suggesting the alternate browser, and authorizing the option of formulating a manual genogram. Instructions for drawing the genogram from the American Medical Association website (AMA, 2009) were included with the letter and sent in the packet to all subsequent volunteer participants. Another user issue that surfaced during the study was the genograms created in the original version were unable to be opened in the new version. These problems with the *My Family Health Portrait* website could explain why one in five (20.1%) of the original participants did not complete the second phase of the study. Another possible explanation for attrition was the redundancy of the questions which led some initial participants to report they thought they had already completed and returned the survey.

Instrumentation

The RBD scale (Witte, Cameron, McKeon, & Berkowitz, 1996) is a Likert-type survey that allows rapid assessment of beliefs about the health threat and the efficacy of the recommended response. The RBD asks three questions about each of the EPPM constructs (perceived severity, perceived susceptibility, perceived response efficacy, perceived self efficacy). A threat score is determined by adding the scores for the perceived severity and perceived susceptibility. An efficacy score is determined by adding the scores for response efficacy and self-efficacy. Then the threat score is subtracted from the efficacy score to produce the "discriminating value". A positive discriminating value means that efficacy dominates and identifies a threat control response. A negative discriminating value means that threat dominates and identifies a fear control response. Since threat messages must promote perceived severity and perceived susceptibility as well as perceived self- and response-efficacy (Lapinski & Witte, 1998), health messages can be tailored to the response assessed by the RBD scale (Witte, Meyer, & Martell, 2001).

This study of participants with a known family history of CVD used the RBD Scale to assess their perception of severity and susceptibility to the CVD threat. It also assessed their perception of the efficacy of health promotion and disease prevention strategies recommended by the American Heart Association, as well as their own self-efficacy to practice the recommended strategies. In addition to assessing the perceived threat and efficacy constructs of the EPPM, this study also assessed the participant's stage of change from the Transtheoretical Model (TTM) before and after the health genogram was completed.

The Transtheoretical Model (Prochaska, Redding, & Evers, 2002) states that behavior change occurs through a series of stages (precontemplation, contemplation, preparation, action and maintenance). This theory includes the construct of consciousness-raising as one of the processes of change. This study explored whether completion of the online family health genogram raised the participants' consciousness related to their CVD risk and advanced their stage of change or their readiness to adopt health promotion behaviors. In addition, the TTM model was used to assess the participants' experience with collecting and documenting family health history before and after participation in the study.

The Risk Behavior Diagnosis Scale (Witte, Cameron, McKeon, & Berkowitz, 1996) was adapted for use in this study and was tested for validity using a focus group of nursing educators to examine the questions and provide feedback. After modifying the questions based on the focus group input, the tool was reviewed for validity by the EPPM and RBD Scale originator, Kim Witte, PhD. She provided valuable suggestions for modifying the wording of some questions to increase the sensitivity of the perceived severity and perceived response efficacy items. She also suggested omitting the "neutral" option on the Likert scale to force a positive or negative response. After these modifications were made, the survey was then pilot tested by 103 participants and tested for reliability one week later.

Pilot Study

A pilot study (N = 103) of the adapted Risk Behavior Diagnosis (RBD) scale was completed to assess the test-retest reliability of the measure, as well as ascertain if there was significant difference on each item, construct, and factor between the two survey administrations while determining how the items loaded onto underlying factors. The repeated-measures t-tests demonstrated significant differences between the first and second testing for two of the items: the first of the three items involving intent to avoid tobacco ($t = 1.989, p = .049$) and the second of the three items regarding self-efficacy to maintain a healthy weight ($t = -2681, p = .009$). The averages of these items only went from 3.72 (SD – .569) to 3.62 (SD = .732) and 2.95 (SD = .648) to 3.08 (SD = .728), respectively; thus, the practical significance of these differences can be challenged. Still, these items indicate administering the RBC Scale consistently weakens intent to discontinue smoking while strengthening self-efficacy to maintain recommended weight. Upon further analysis, averages of the items regarding intent to avoid tobacco and the items pertaining to self-efficacy to maintain a healthy weight were compared for the first and second testing using repeated-measures t-tests. These comparisons showed the averages of the questions about tobacco were not significantly different ($t = 1.367, p =$

.175), yet the difference between the averages relating to self-efficacy to maintain weight were still significant (t = -2.265, p = .026). The average scores on self-efficacy to maintain weight were 2.95 (SD = .617) for the first survey and 3.034 (SD = .648) for the second survey which still brings into question the practicality of the significance in the difference between these scores. The difference could also be attributed to participants' increased awareness of the importance of weight and making plans to maintain their recommended weight prior to the retest. In looking for significant test-retest differences among the constructs (susceptibility, severity, response-efficacy, self-efficacy, intent, and motivation), a significant difference (t = -2.069, p = .041) was found on the self-efficacy subscale, which may yield a similar explanation to that previously mentioned. The last group of statistics involving comparisons between the test administrations focused on threat score (severity plus susceptibility), efficacy score (response-efficacy plus self-efficacy), and discriminating value (efficacy minus threat). These comparisons found no significant differences on threat (t = 1.291, p = .200), efficacy (t = -0.984, p = .328), and discriminating value (t = -1.427, p = .157).

Data Analysis

The statistical analysis utilized repeated-measures t-tests to look at differences between the test administrations in physical activity, weight, and tobacco use discrimination scores as well as intent to maintain physical activity level and recommended weight, intent not to smoke, stage of change for family health history documentation, and stage of change for CVD prevention. Independent-samples t-tests were also used to assess differences between the fear control group and threat control group for fear level, total family CVD, intent to maintain physical activity level, intent to maintain recommended weight, and intent to not smoke. Also, one-way analyses of variance (ANOVA) were used to compare the discrimination scores and intent among self-identified inactive, active, and very active individuals; underweight, normal weight, and overweight individuals; and past tobacco users, current tobacco users, and non-tobacco users. Additionally, Pearson product-moment correlations were used to look for relationships between the variables and factors in the research.

Summary

The study was approved by the MSU Human Subjects in Research Committee and granted deferral status by the TWU Institutional Review Board. A survey based on the Risk Behavior Diagnosis Scale of the Extended Parallel Process Model, with elements of the Transtheoretical Model, was tested for validity and reliability. Convenience and snowball sampling techniques were used to recruit individuals with a family history of cardiovascular disease. Participants completed initial surveys, recorded family medical history on an online genogram, and completed a follow-up survey. A total of 171 participants completed all phases of the study. The RBD Scale formula was applied to determine if participants were in fear or danger control after completing the family health genogram. That designation was compared with demographic data, the constructs within the EPPM, and the Stages of Change from the TTM.

CHAPTER IV

RESULTS

The surveys completed before and after formulation of the family health genogram yielded a large amount of data. Statistical analyses of the data were done to answer the research questions and address the hypotheses for the study, as well as to look for correlations among the variables.

Research Question 1

The first research question asked "Do threat control responses or fear control responses dominate in participants with a family history of CVD, after they complete the specified online health genogram?" The null hypothesis was that there is no statistically significant difference in fear control / danger control processes related to weight control, physical activity and smoking avoidance in participants with a history of CVD before and after completing the specified online health genogram.

The statistical results related to H_{01} indicated there was a significant change between the two tests based on repeated-measures *t*-tests. Specifically, the discriminating value scores (perceived efficacy minus perceived threat) showed a significant difference for physical activity ($t = 2.34, p = .020$) and tobacco avoidance ($t = 2.64, p = .009$) discriminating value scores, as well as the total discriminating value scores ($t = 2.25, p = 0.026$). Since there was no significant changes in efficacy scores for physical activity ($t = .135, p = .893$), weight management ($t = .623, p = .534$), or tobacco use ($t = .895, p =$

.372), the changes in discriminating value scores were due to increase in threat. The shifts in the means for each of these variables showed that completing the genogram consistently shifted the participants' in the direction of fear control and away from threat control. There was not a significant difference in the weight maintenance discriminating value scores ($t = 1.375$, $p = .171$). These statistical findings enable rejection of H_{01} for physical activity and tobacco use, but acceptance of H_{01} for weight control. Additional statistics relating to physical activity, weight maintenance, and tobacco avoidance discriminating value scores in the second test administration are considered below.

Physical Activity

Using a one-way analysis of variance (ANOVA), the discriminating value scores for physical activity were compared for participants who identified themselves as inactive, active, or very active. This comparison showed a significant difference, $F(2,162) = 14.354$, $p = 0.000$ ($\omega^2 = .14$). Therefore, a Tukey's honestly significant difference (HSD) post-hoc comparison was completed which showed a significant difference from inactive to active (HSD = -.395, $p = .000$), inactive to very active (HSD = -.828, $p = .000$), and active to very active (HSD = -.433, $p = .009$). These differences (illustrated in Figure 1) indicate that as participants' self-reported activity levels increase their discriminating value scores increase, shifting them toward a threat control response. Also, a fear level was determined for each participant by combining self-scores related to feelings of fright, tension, anxiety, discomfort, and nervousness. These scores from the second survey were then compared for the threat control group, with positive discriminating value scores, and the fear control group, with negative discriminating value scores, using an independent-samples *t*-test. This comparison revealed a significant difference ($t = 3.326, p = .001$) between the groups which confirmed that the fear control group consistently scores higher in self-ascribed fear than the threat control group. Additionally, a difference in the number of family members with CVD, which approached significance ($t = 1.546, p = .063$), was found between the fear control group and threat control group indicating that membership in the fear control group correlated with a higher number of family members with CVD.

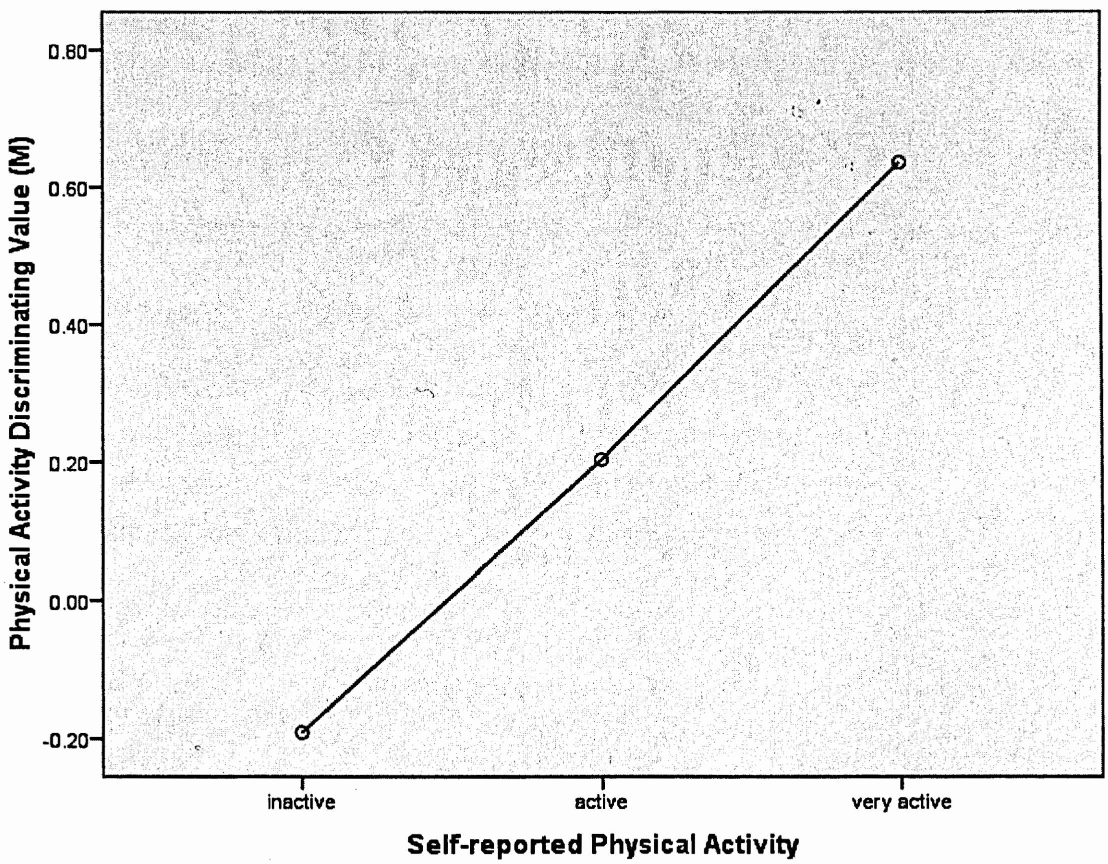


Figure 1. Correlation between physical activity discriminating value scores and self-reported physical activity.

Weight

The discriminating value scores for weight maintenance were also compared using a one-way ANOVA for those who self-identified as underweight, normal weight, and overweight, and a significant difference was found, F (2, 161) = 24.191, p = .000 (ω^2 = .22). The subsequent Tukey's HSD post-hoc comparison revealed a significant difference between those who are overweight and normal weight (HSD = .526, p = .000) and between those who are underweight and overweight (HSD=.577, p = .027). As illustrated in Figure 2, this evidence indicates that those who identify themselves as overweight are more likely to be in the fear control category. Furthermore, using an independent-samples *t*-test, the fear control group and threat control group were found to be significantly different with regard to fear level (t = 1.754, p = .041), confirming that the fear control group had a higher level of self-ascribed fear. Moreover, the total family members with CVD for individuals in these two groups was also significantly different (t = 2.121, p = .018), which illustrates that those in the fear control group had a higher number of family members with CVD. Additionally, using an independent-samples *t*-test to compare those who have been diagnosed with CVD with those who have not been diagnosed with CVD shows a significant difference (t = 2.264, p =.025) in the discriminating value scores for weight management, showing that those with CVD are more likely to have lower discriminating scores and shifts toward fear control behaviors. Furthermore, those who take medication for CVD are also significantly (t - 2011, p = .046) more likely to have lower discriminating value scores for weight management, shifting them toward fear control behaviors.

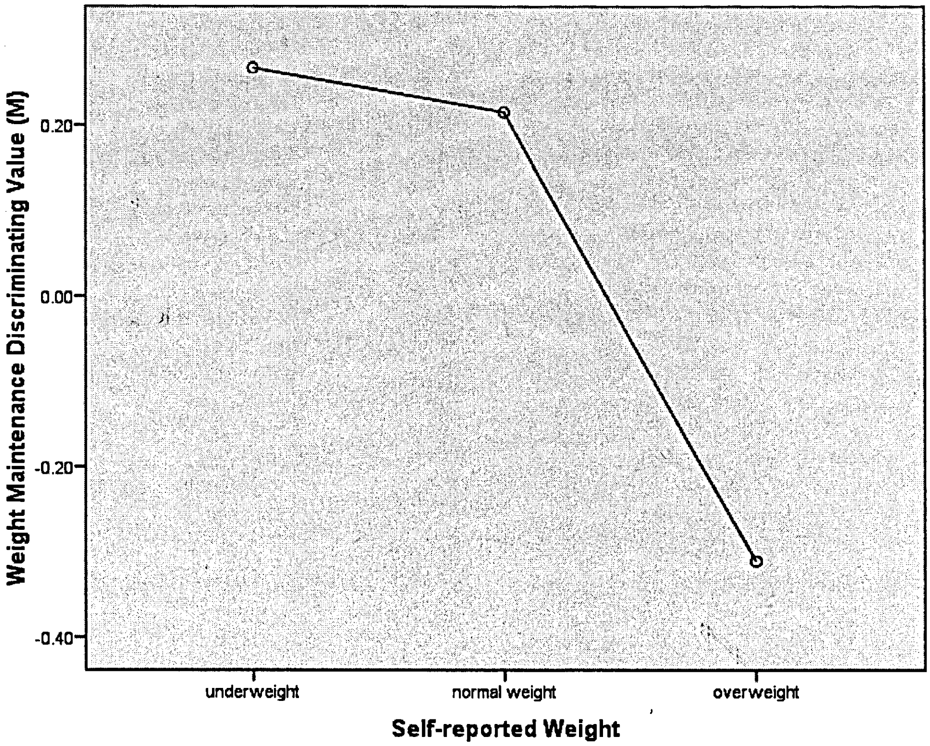


Figure 2. Correlation between weight maintenance discriminating value scores and self-reported weight.

Tobacco

Another one-way ANOVA compared the discriminating value scores for tobacco avoidance with the levels of tobacco use (never used, used in past, and currently use), resulting in a significant difference, F (2, 163) = 31.328, p = .000 (ω^2 = .27). A Tukey's HSD post-hoc comparison showed a significant difference between those who currently use tobacco and those who have never used tobacco (HSD = -.893, p = .000), as well as between those who currently use tobacco and those who have used tobacco in the past (HSD = -.779, p = .000). As seen in Figure 3, these results indicate lower discriminating value scores, and thus greater tendency toward fear control, among those who currently use tobacco or have formerly used tobacco compared to those who have never used tobacco. Again, the fear control group was found to be significantly higher than the threat control group on level of fear ($t = 2.474, p = .007$) and total family members with CVD ($t = 2.167, p = .016$) when compared by independent-samples t-tests.

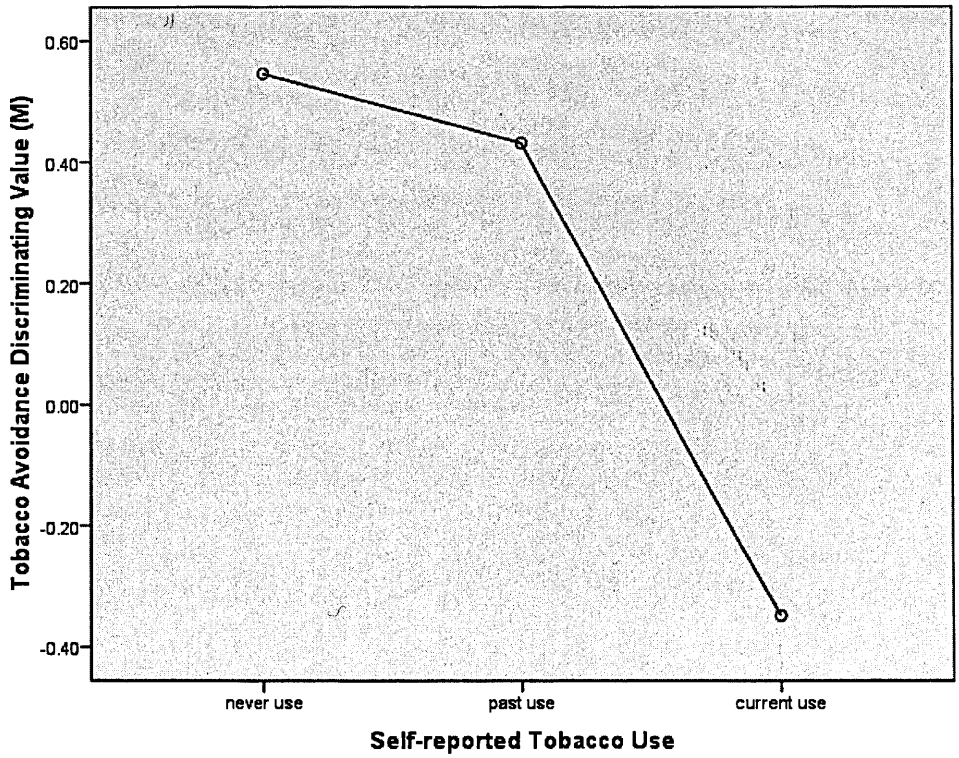


Figure 3. Correlation between tobacco avoidance discriminating value scores and self-reported tobacco use.

Research Question 2

The second research question asked "Does completion of the specified online health genogram influence the participant's intent to practice health promotion behaviors?" The null hypothesis was that there is no statistically significant difference in intent to control / maintain weight, engage in regular physical activity, and avoid tobacco in participants with a history of CVD before and after completing the specified online health genogram. The correlation between the total fear scores and the total discriminating value categories are illustrated in Figure 4.

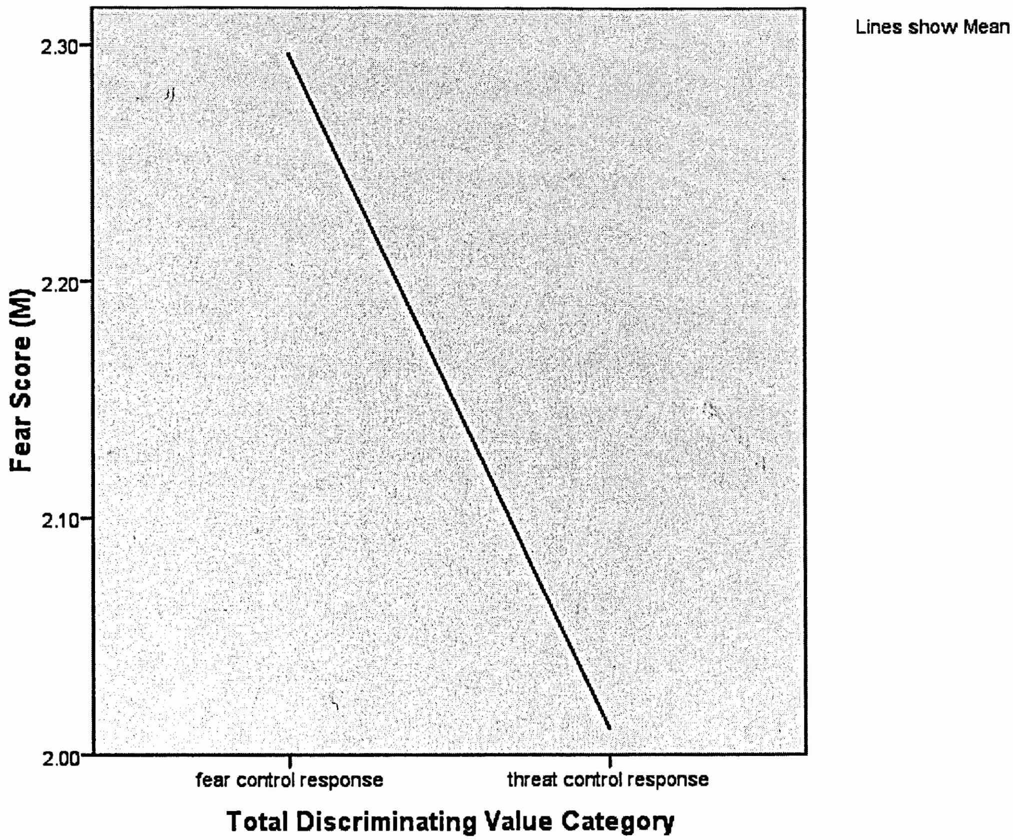


Figure 4. Correlation of fear scores by total discriminating value category (fear control response or threat control response).

The results of the repeated-measures *t*-tests associated with H_{02} showed that there were no significant differences between the test administrations on intent to maintain physical activity level ($t = -.405, p = .686$), intent to maintain recommended weight ($t = .546, p = .586$), or intent to avoid tobacco ($t = .508, p = .612$). These statistical findings allow acceptance of H_{02}. There was a significant difference in intent between those in the fear control group and those in the threat control group when compared using independent-samples t-tests. Specifically, there were significant differences between the groups in intent to maintain physical activity level ($t = -5.742, p = .000$), intent to maintain recommended weight ($t = -4.350, p = .000$), and intent to avoid tobacco ($t = -13.200, p = .000$). These differences, illustrated in Figures 5-7, indicate a higher level of intent for physical activity level, weight maintenance, and smoking avoidance in the threat control group compared to the fear control group.

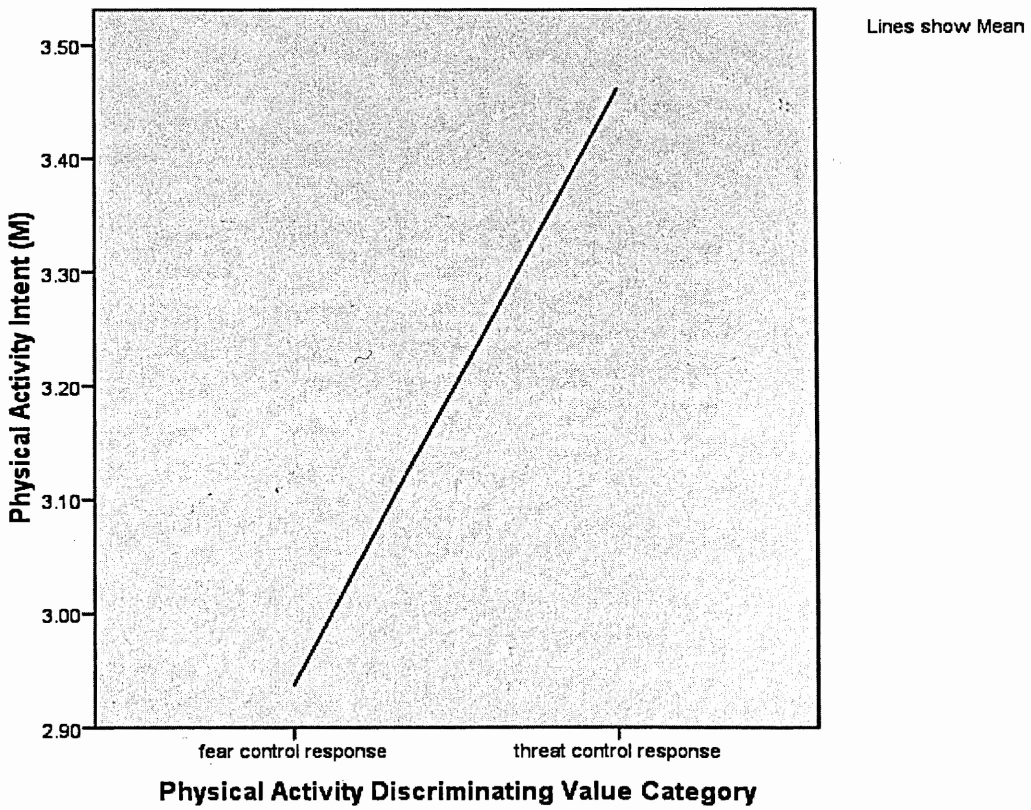


Figure 5. Correlation between physical activity discriminating value category and intent to achieve or maintain recommended physical activity level.

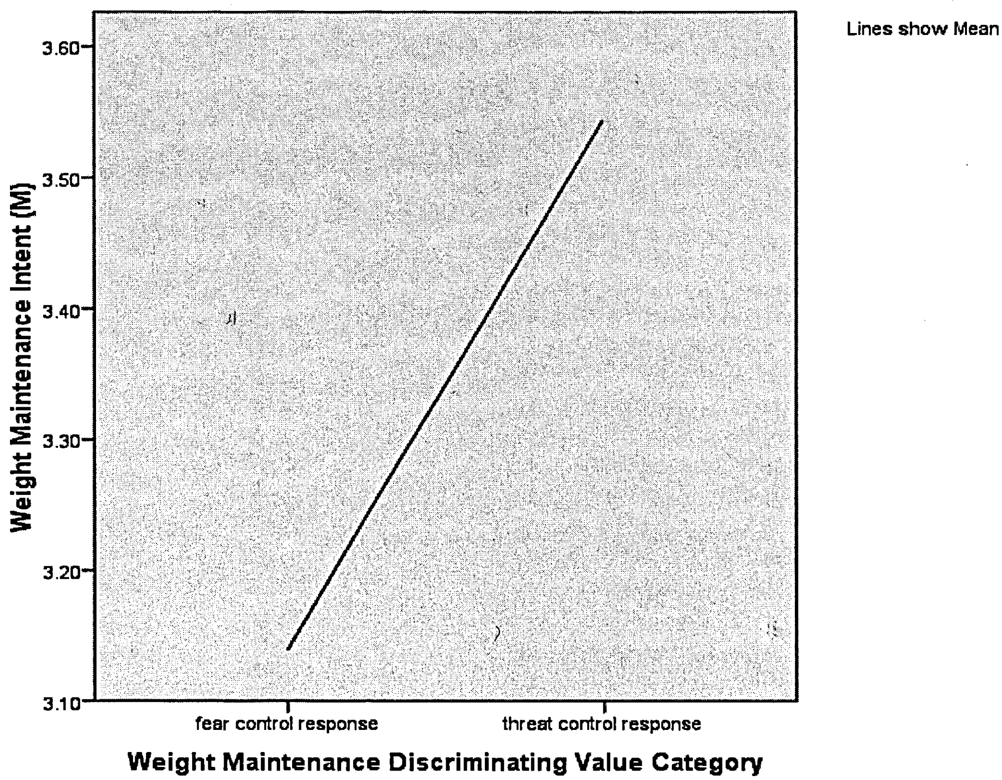


Figure 6. Correlation between weight maintenance discriminating value category and intent to achieve or maintain recommended weight.

Physical Activity

Using a one-way ANOVA, intent to remain physically active was compared among those who identified themselves as inactive, active, and very active, F(2,165) = 18.409, p = 000 (ω^2 = .17). A Tukey's HSD post-hoc comparison showed significant differences in intent between inactive and active (HSD = -.504, p = .000), inactive and very active (HSD = -1.000, p = .000), and active and very active (HSD = -.496, p = .004). As illustrated in Figure 8, these differences demonstrate that as current activity level increases the intent to remain physically active also increases.

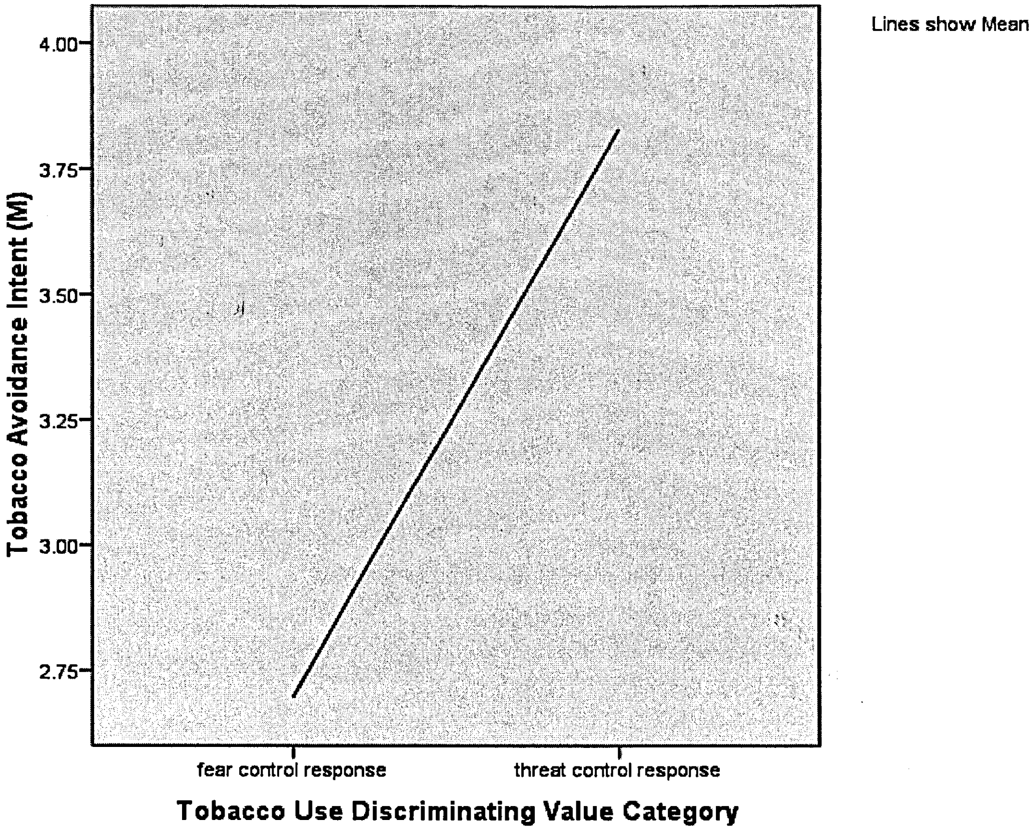


Figure 7. Correlation between tobacco use discriminating value category and intent to avoid tobacco use.

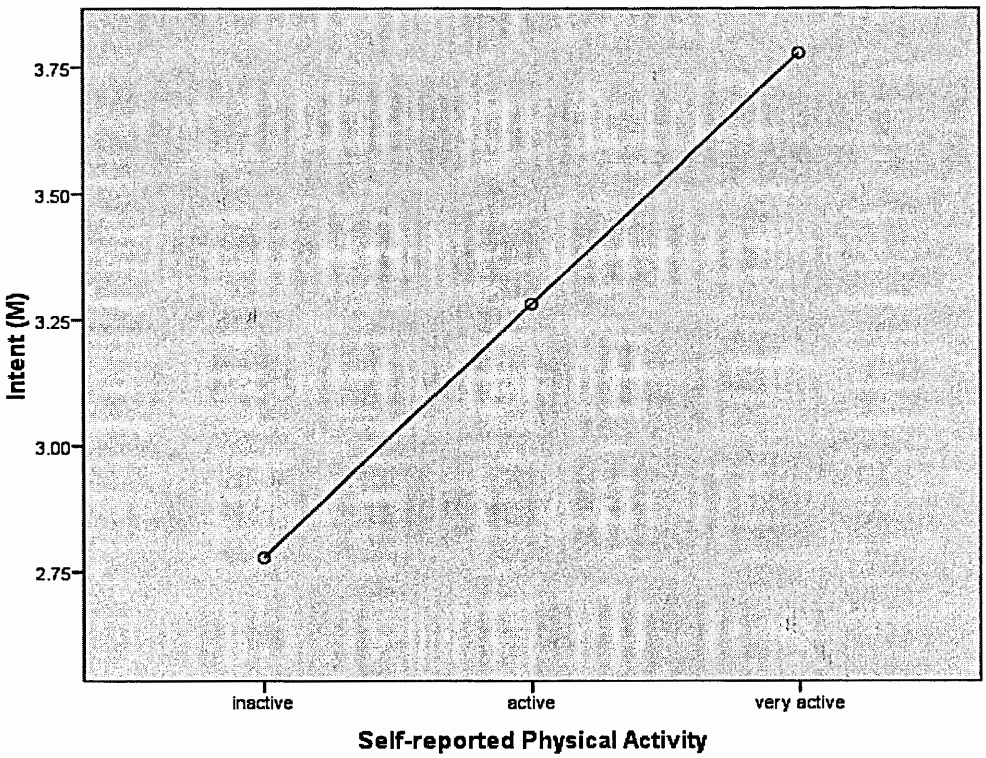


Figure 8. Correlation between intent to achieve and / or maintain recommended physical activity level and self-reported activity status.

Weight

Another one-way ANOVA found significant differences in intent to maintain recommended weight between those who classify themselves as underweight, normal weight, or overweight, F (2, 164) = 5.676, p = .004 (ω^2 = .05). The Tukey's HSD post-hoc comparison revealed a significant difference between those in the normal weight category and those in the overweight category (HSD = .233, p = .007), as shown in Figure 9. Another significant factor relevant to intent is the use of medication for CVD. In comparing those who take medication to those who do not, a significant difference (t =

2.457, $p = .015$) was found in intent to maintain recommended weight, with intent being consistently higher in those who do not take medication.

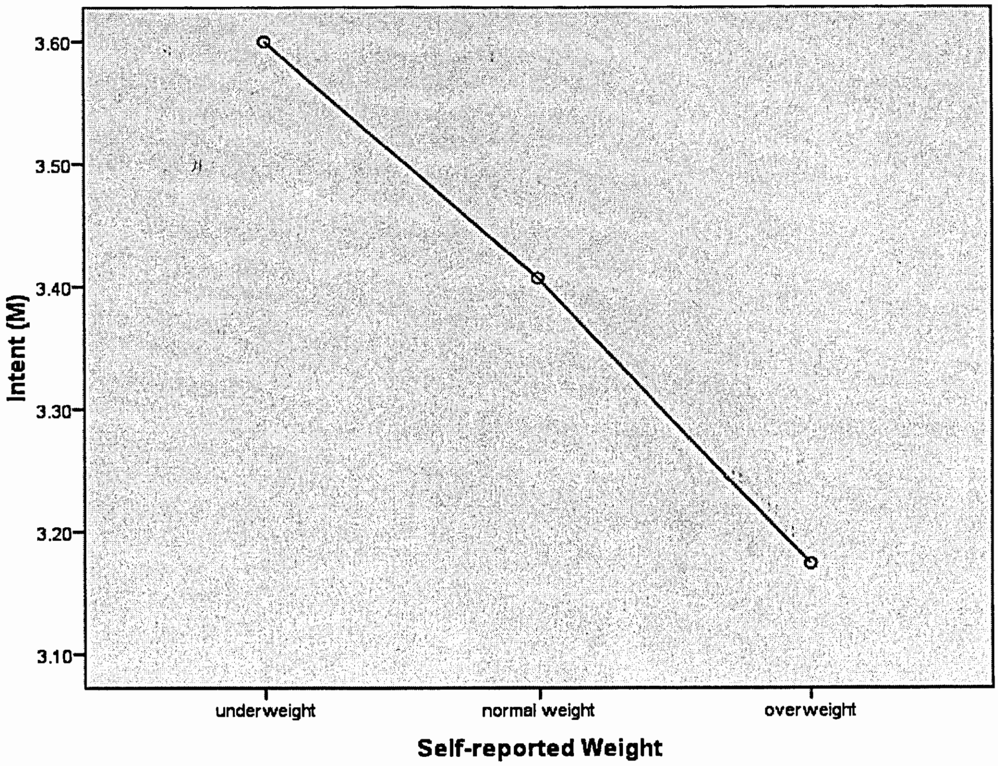


Figure 9. Correlation between intent to achieve and / or maintain recommended weight and self-reported weight.

Tobacco

A one-way ANOVA comparing intent to avoid tobacco among the levels of tobacco use found a significant difference, F (2, 163) = 119.227, $p = .000$ ($\omega^2 = .59$). The Tukey's HSD post-hoc comparison showed significant differences in intent between those who never used tobacco and those who used in the past (HSD = .326, $p = .000$), those who never used tobacco and who currently use (HSD = 1.467, $p = .000$), and those who used tobacco in the past and who currently use (HSD = 1.142, $p = .000$). As illustrated in Figure 10, these differences indicate that those who never used tobacco have greater intent to avoid tobacco than those who have used previously or use currently, and those who have used in the past have more intent to not smoke than those who use now.

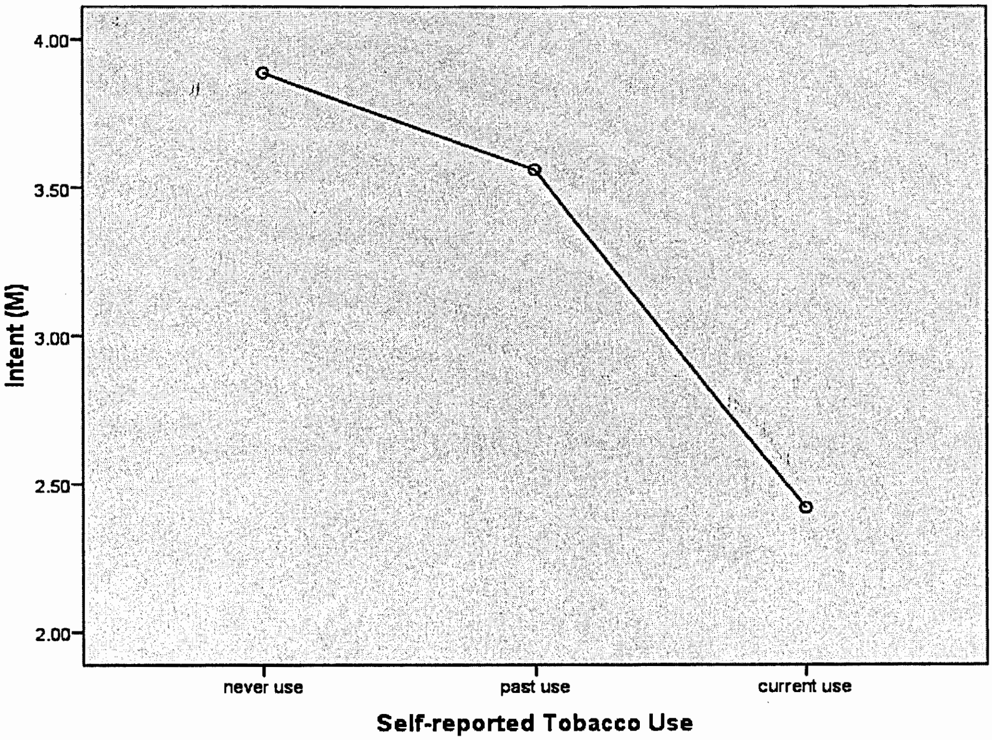


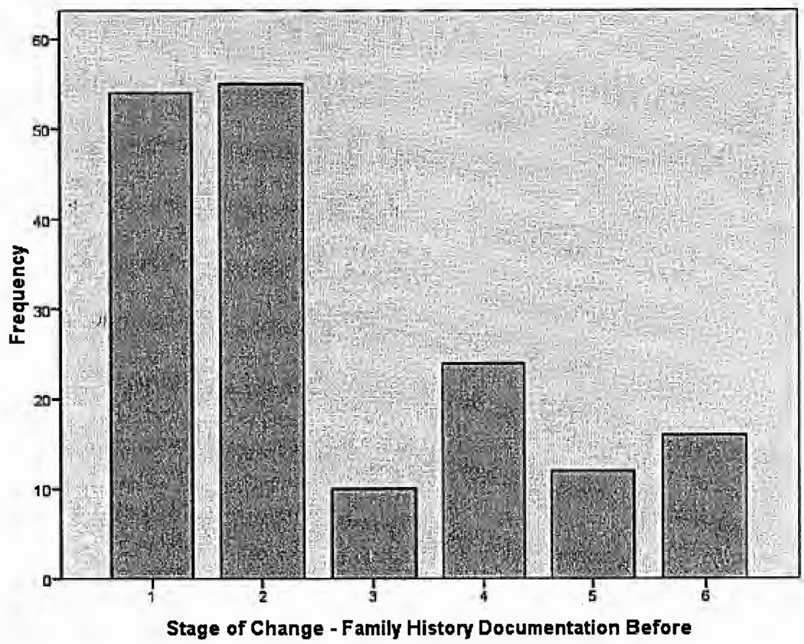
Figure 10. Correlation between intent to avoid tobacco and self-reported tobacco use.

Using the Pearson product-moment correlation, the total number of family members with CVD was significantly correlated to the differences between the two survey administrations in intent to maintain physical activity level ($r = .169, p = .033$) and maintain recommended weight ($r = .213, p = .007$), which shows completing the genogram may increase intent in these areas depending on the prevalence of CVD in the family. Furthermore, perceived susceptibility to CVD was found to be significantly correlated with intent to maintain physical activity level ($r = .194$, $p = .012$) and recommended weight ($r = .330$, $p = .000$); indicating that as the individuals feel more susceptible to CVD there is an increased intent to maintain physical activity level and recommended weight. This correlation was not found for tobacco use.

Research Question 3

The third research question was "Does completion of the specified online health genogram advance the participant's readiness to adopt health promotion behaviors?" The null hypothesis related to this research question was that there would be no statistically significant difference in stage of change in participants with a family history of CVD before and after completing the specified online health genogram. The stages of change were assessed for both CVD preventive behaviors and for documentation of family health history.

The third hypothesis was assessed using repeated-measures *t*-tests to consider the differences in the stages of change between the test administrations. These tests showed there were significant differences in the stage of change for family history documentation ($t = -6.672$, $p = .000$), as illustrated in Figure 11, and in the stage of change for CVD prevention behaviors ($t = -2.191$, $p = .030$). These differences demonstrate that completing the genogram reliably advances the stages of change for both family health history documentation and for CVD preventive behaviors. This indicates that those who have completed the genogram are more aware of their family health history and more likely to engage in preventive behaviors. These statistical findings allowed rejection of H_{03}.



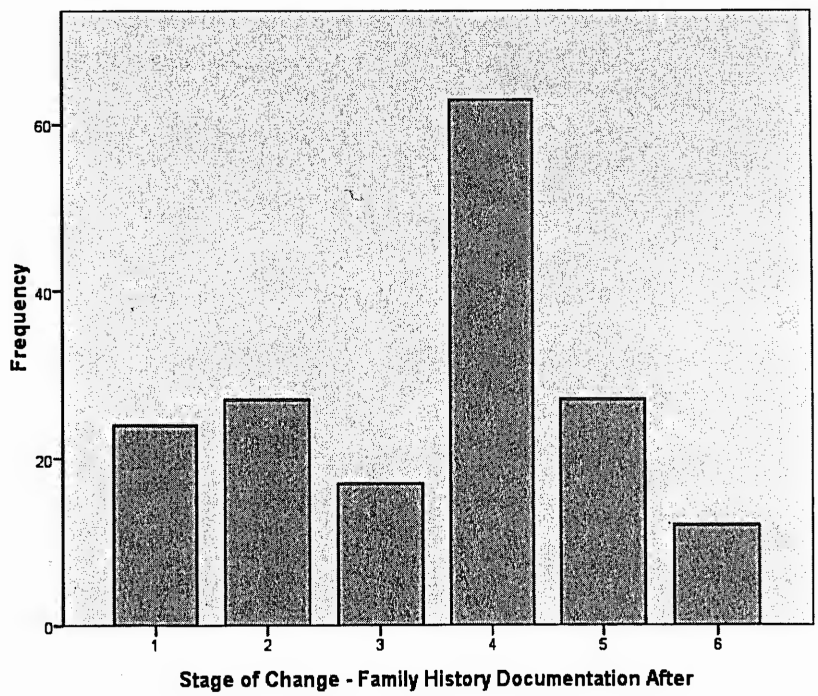


Figure 11. Frequency of participants in various Stages of Change for family history documentation before and after completion of the genogram. The stages are: 1) Precontemplation; 2) Contemplation; 3) Planning; 4) Action; 5) Maintenance; and 6) Relapse.

An independent-samples t-test compared the discriminating value scores for tobacco use with the stage of change for CVD prevention among those in the threat control group and fear control group and showed a significant difference ($t = -2.142, p = .034$). This difference signifies that those in the tobacco threat control group have a more advanced stage of change for CVD prevention than those in the tobacco fear control group. Using an independent-samples t-test to compare those who take medication for CVD to those who do not take medication also reveals a significant difference ($t = -2.160, p = .032$) in stage of change for CVD prevention, indicating that stage of change for CVD prevention is more advanced among those who are currently taking medication. Pearson product-moment correlations were also used to look at relationships between the variables, and significant correlations were found between stage of change for CVD prevention and intent to remain physically active ($r = .237, p = .001$) as well as stage of change for CVD prevention and intent to avoid tobacco ($r = .214, p = .003$). Additionally, a correlation was identified between motivation for health preventive behaviors and stage of change for CVD prevention ($r = .231, p = .002$) and between tobacco avoidance and stage of change for CVD prevention ($r = -.184, p = .009$). These correlations demonstrate that stage of change for CVD prevention increases with intent to remain physically active, intent to avoid tobacco, and motivation to practice preventive behaviors.

In summary, statistical analysis of the data from the surveys administered before and after the completion of the family history genogram answered the three research questions. For the first question, the analysis indicated that fear control responses dominate for physical activity and tobacco avoidance, but not for weight control. For the second question, the analysis showed that completion of the genogram did not significantly influence the intent to maintain physical activity, maintain recommended weight, or avoid tobacco, but did indicate that intent correlated with discriminating value scores. And for the third research question, the analysis showed advancement in stage of change, or readiness, for both family history documentation and for CVD prevention behaviors after completion of the family health genogram.

CHAPTER V

DISCUSSION

Summary

Family history of disease is recognized as the most consistent risk factor for disease across the life span. The family health genogram is recognized as the best and most inexpensive genomic tool available and has the potential to aid diagnoses, prompt targeted testing and evaluation, and guide health education and health promotion efforts. The purpose of this study was to introduce health genograms as threat appeals in motivating intent to perform health promotion and disease prevention behaviors in participants with a history of CVD. The Risk Behavior Diagnosis (RBD) Scale which is theoretically grounded in the Extended Parallel Process Model (EPPM) assesses threat (perceived severity of the threat and perceived susceptibility to it) and efficacy (self-efficacy and efficacy of the recommended response). If efficacy exceeds threat, danger or threat control processes dominate resulting in adaptive attitude, intention or behavior changes to control the threat. However, if threat exceeds efficacy, fear control processes dominate resulting in denial, defensive avoidance or reactance. For fear appeals to be successful, the individual must perceive that the threat is serious, that he is personally susceptible to it, that the recommended response will control the threat, and that he is capable of making the recommended response (Witte, 1991). Participants were assessed on their perception of severity and susceptibility related to CVD, collectively termed threat, and their perception of response efficacy and self-efficacy related to CVD preventive behaviors.

Convenience and snowball sampling techniques recruited 171 participants who completed initial surveys based on the RBD Scale and elements of the Transtheoretical Model (TTM), recorded family medical history using an online genogram, and completed a follow-up survey. The RBD Scale determined if participants were in fear or danger control after completing the genograms. The results indicated a shift toward fear control responses for physical activity and tobacco avoidance, but not for weight control. Completion of the genogram did not significantly influence the intent to maintain physical activity, maintain recommended weight, or avoid tobacco, but did indicate that intent increased as threat control increased for each recommended behavior. There was advancement in stage of change, or readiness, for both family history documentation and for CVD prevention behaviors after completion of the family health genogram (Table 5).

Table 5

Deposition of Study Hypotheses

Hypothesis	Deposition
H_{01} – There is no statistically significant difference in fear control / danger control processes related to weight control, physical activity and smoking avoidance in participants with a family history of CVD.	Rejected (activity, tobacco) Accepted (weight)
H_{02} - There is no statistically significant difference in pre- and post-genogram scores on intent to control / maintain weight, engage in regular physical activity, and avoid tobacco in participants with a family history of CVD.	Accepted
H_{03} – There is no statistically significant difference in pre- and post-genogram scores on stage of change of participants with a family history of CVD.	Rejected

Conclusions

Completion of a family health genogram can act as an effective threat appeal in raising awareness of the severity of and susceptibility to CVD in individuals with a family history of CVD. Graphic presentation of familial patterns of CVD illustrate the individual's or family's susceptibility to this disease.

Completion of a genogram does not in and of itself influence the intent to practice health promotion or disease prevention behaviors. However, when considered relative to the RBD scale of the EPPM, completion of a genogram can raise awareness of the CVD threat (severity plus susceptibility), which can trigger the assessment of efficacy (response efficacy plus self-efficacy). If and when efficacy exceeds threat there is increased motivation or intent to control the threat. However, if threat exceeds efficacy, fear control dominates without further consideration of the threat.

The RBD scale can potentially be used by health educators and clinicians to determine the dominance of threat or efficacy. This determination would allow for the tailoring of health messages to emphasize threat or efficacy and ensure that protective motivation results rather than defensive motivation.

Increased fear generated by the health genogram requires increased efficacy to maintain threat control responses. Individuals in most need of behavior changes, such as smokers, overweight and inactive people, those with high prevalence of CVD in their families, and those who have been diagnosed with CVD, have the most fear, and are thus in defensive motivation with fear control responses to manage their fear rather than the CVD threat. Fear appeals are not appropriate for these individuals unless they are accompanied by vigorous efficacy-building strategies.

Discussion and Implications

Research Question 1

Do threat control responses or fear control responses dominate in participants with a family history of CVD, after they complete the specified online health genogram? The first finding of this study was a shift toward fear control for physical activity and tobacco avoidance after completion of the genogram. The consistent increase in the participants' threat scores (severity plus susceptibility), without a significant change in efficacy scores (response efficacy plus self-efficacy), decreased discriminating value scores and shifted them toward the fear control response (with threat dominating) rather than threat control responses (with efficacy dominating). This confirms that a family health genogram can act as a threat appeal or "scare tactic". This fear arousal, according to Hovland, Janis and Kelly (1953), is needed to elicit a motivational drive state. The Parallel Process Model (Leventhal, 1971) would predict that this increased fear would lead to maladaptive responses to control the fear rather than control the threat. The Extended Parallel Process Model (Witte, 1992a) would expand this theory to explain that if the threat appraisal determines vulnerability to a serious threat, the result is fear and motivation to act. This response triggers the second appraisal of efficacy. If the individual believes the recommended response will avert the threat, and that he is capable of adopting the recommended behavior, he will take action to protect himself from the threat. Thus, the genogram has the potential to illustrate the severity of CVD and the individual's or family's susceptibility to it, and thus motivate health behaviors to prevent or control it. According to Witte, Meyer, and Martel (2001) fear has a tangential role in threat control in that it causes individuals to upgrade their estimates of a perceived threat. It is only when this perceived threat exceeds perceptions of efficacy that people shift into fear control processes and focus on managing their fear instead of the threat or danger. When using a genogram as a fear appeal, it is important for the health educator to promote response efficacy and self-efficacy to ensure that threat does not exceed efficacy and result in fear control.

Of interest is the finding that there were significant differences in discriminating value scores for physical activity and tobacco avoidance, but not for weight maintenance. A confounder in this study may be that a much higher percentage of participants described themselves as overweight (40.2%), compared to those who were inactive (16.0%) or tobacco users (13.7%).

The survey used in this study was a modification of the Risk Behavior Diagnosis Scale. It incorporated a separate self-assessment of fear from the combined responses to feelings of fright, tension, anxiety, discomfort, and nervousness related to genogram formulation. In all behavioral response categories (physical activity, weight, and tobacco use) the fear score was higher for those who fell within the EPPM fear control spectrum than for those within the threat control spectrum. This finding further validates the fear control construct of the EPPM (Witte, 1998), and its related RBD Scale (Witte, Meyer, & Martell, 2001) which calculates the discriminating value score as the difference between the threat and efficacy scores, with fear control occurring when threat exceeds efficacy.

Not only did those in the fear control spectrum have higher fear scores than those in the threat control spectrum, but they also had a higher number of family members with

CVD included in their genogram. A genogram with a graphic illustration of an increased prevalence of CVD in the family would tend to increase perceived susceptibility to CVD. A confounding factor in this regard may be that those who have more family members with CVD have more experience with the morbidity and mortality of the disease. This would increase perceived severity and susceptibility and thus increase the threat and the fear associated with the disease.

Another interesting finding was that as participants' physical activity decreased, their discriminating value scores decreased, shifting them toward fear control. Likewise, as the participants' perceived weight increased from normal they were more likely to be in fear control. Another significant correlation with weight was that those who have been diagnosed with CVD or take medications to control their CVD are more likely to have lower discriminating value scores and thus be in fear control. This means that the threat of CVD is too high, or efficacy to achieve or maintain recommended weight is too low. As previously mentioned fear responses lead to maladaptive behaviors such as defensive avoidance or reactance. For those individuals in fear control, the focus is on controlling their fear rather than controlling the danger. Important implications for health educators would be to emphasize the efficacy of weight management in controlling CVD, and promote the individual's self-efficacy to perform the behaviors necessary to effectively manage weight. Emphasizing the threat would further increase the fear, compound the fear control, and cause the fear appeal to backfire.

Tobacco use also affected the participants' discriminating value scores. There were significant differences between those who had never used tobacco, those who used tobacco in the past and those who currently use tobacco. Those who had never used or used in the past had higher discriminating value scores, indicating threat control, whereas those who currently smoke had lower discriminating value scores, indicating fear control. The same implications apply to tobacco use as to weight management in that health educators must focus on the efficacy of smoking cessation in health promotion and disease prevention. Smokers must be given the tools to increase their self-efficacy, rather than further emphasizing the threat that could plunge them deeper into fear control.

Research Question 2

Does completion of the specified online health genogram influence the participant's intent to practice health promotion behaviors? Analysis showed no statistically significant differences in intent to practice health promotion related to physical activity, weight maintenance, or tobacco avoidance. However, there were significant differences in intent between those in threat control and in fear control, with a higher level of intent in the threat control group. This is an important finding and helps validate the EPPM constructs of threat control and fear control. In threat control individuals are accepting the fear-arousing message and are exercising cognitive processes by thinking of strategies to avert the threat. In fear control they are exercising emotional fear control processes which lead to rejection of the fear-arousing message (Witte, Meyer, & Martell, 2001).

It was not surprising that the participants who were the most active expressed greatest intent to be active, those who were normal weight expressed the greatest intent to maintain recommended weight, and those who never smoked, or formerly smoked and quit, expressed the greatest intent to avoid tobacco. A confounding factor in this regard is that the sample was skewed toward normal weight (56.8%), active or very active (84%) and non-smoker or former smoker (86.3%), as shown in Table 2. As such, a majority of the participants were already practicing these health promotion and disease prevention behaviors. Upon entry into the study, 50.3% of participants stated in the stages of change assessment that they take some of the recommended health promotion steps and another 21.6% stated that they consistently follow the recommended steps (see Table 6). A recommendation for further research would be to separately analyze those in need of each behavior change, or to limit participation to a single behavior change.

Table 6

Stage of Change for Practicing CVD Preventive Behaviors Before and After Completing the Genogram

Stage of Change	Before %	After %
Precontemplation	8.8	2.4
Contemplation	9.9	10.2
Planning	8.2	13.2
Action	50.3	52.1
Maintenance	21.6	21.0
Relapse	1.2	2.1

Threat control requires efficacy to exceed threat. Increased intent to practice health promoting behaviors for those in the threat control spectrum confirmed that the threat appeal can motivate intent to change behaviors, but underscores the need to promote efficacy to avoid fear control. Table 7 proposes health education strategies to increase efficacy and threat control behaviors when assisting clients with recommended behavior changes. Figure 12 illustrates a flow chart for health educators for utilization of those proposed strategies when using a family health genogram as a threat appeal. This flow chart utilizes the RBD Scale and applies the constructs of the EPPM.

An example application of the flow chart follows. A health educator uses a genogram as a threat appeal and uses the RBD scale to assess threat and efficacy for a client with hypertension. If the client perceives the threat as low because CVD does not run in his family and he knows people with heart disease who lead relatively normal lives, there may be no response. However, if he perceives the risk as high because CVD does run in his family and because he sees CVD as a serious disease, a fear response may be initiated which prompts an assessment of efficacy. A recommended behavior change is to increase physical activity. If he believes being more active will help control CVD, and he thinks he can make time to get more exercise, his perceived efficacy will be high and he will proceed to protection motivation and danger control responses. However, if he doesn't believe the physical activity will be beneficial, or he thinks he cannot fit exercise into his already busy schedule, his perceived efficacy will not be adequate to cope with the perceived threat. The health educator should intervene at the earliest possible stage with strategies to build response efficacy and self-efficacy in order to avert a fear control response and message rejection.

Table 7

Health Education Strategies to Increase Response Efficacy and Self-Efficacy

Response Efficacy	Self-Efficacy
Role model good health habits.	Assess current behavior(s) (e.g., likes and dislikes, meal patterns) and reinforce those that can be incorporated into an action plan.
Present research-based evidence for the effectiveness of the recommended behaviors.	Assess current knowledge (e.g., nutrients, calories) and use that as baseline for education to enhance knowledge.
Provide statistics related to lowered risk of disease, or reduced severity of sequelae, after accomplishing the recommended behavior change(s).	Assess skills (e.g., reading food labels, meal preparation) and use those as a baseline for education to increase skills.
Present case-studies of "success stories."	Assess barriers to behavior change and explore strategies to remove them.
Review the relevance of symptoms to undesired behavior(s).	Encourage medical supervision and pharmacological support when appropriate (e.g., nicotine patches or gum).
Review the improvement of symptoms with progress toward recommended behavior change(s).	Set specific, easily-attainable, short-term goals (e.g., walk one block each day for a week).
Emphasize that genetic family history is not health destiny. By changing the modifiable behavioral risk factors, the non-modifiable family history risk factor may be suppressed.	Discourage over-enthusiastic starts (e.g., starvation diets or over-exercise).
	Enlist support from family and friends (e.g. Family walks or group bike rides.
	Suggest support groups and buddy systems (e.g. Weight Watchers or exercise classes).
	Encourage activity / diet / cigarette logs.
	Reward goal attainment with praise and Encouragement and major milestones with certificates of accomplishment.

Flow Chart for Utilizing Strategies to Build Efficacy

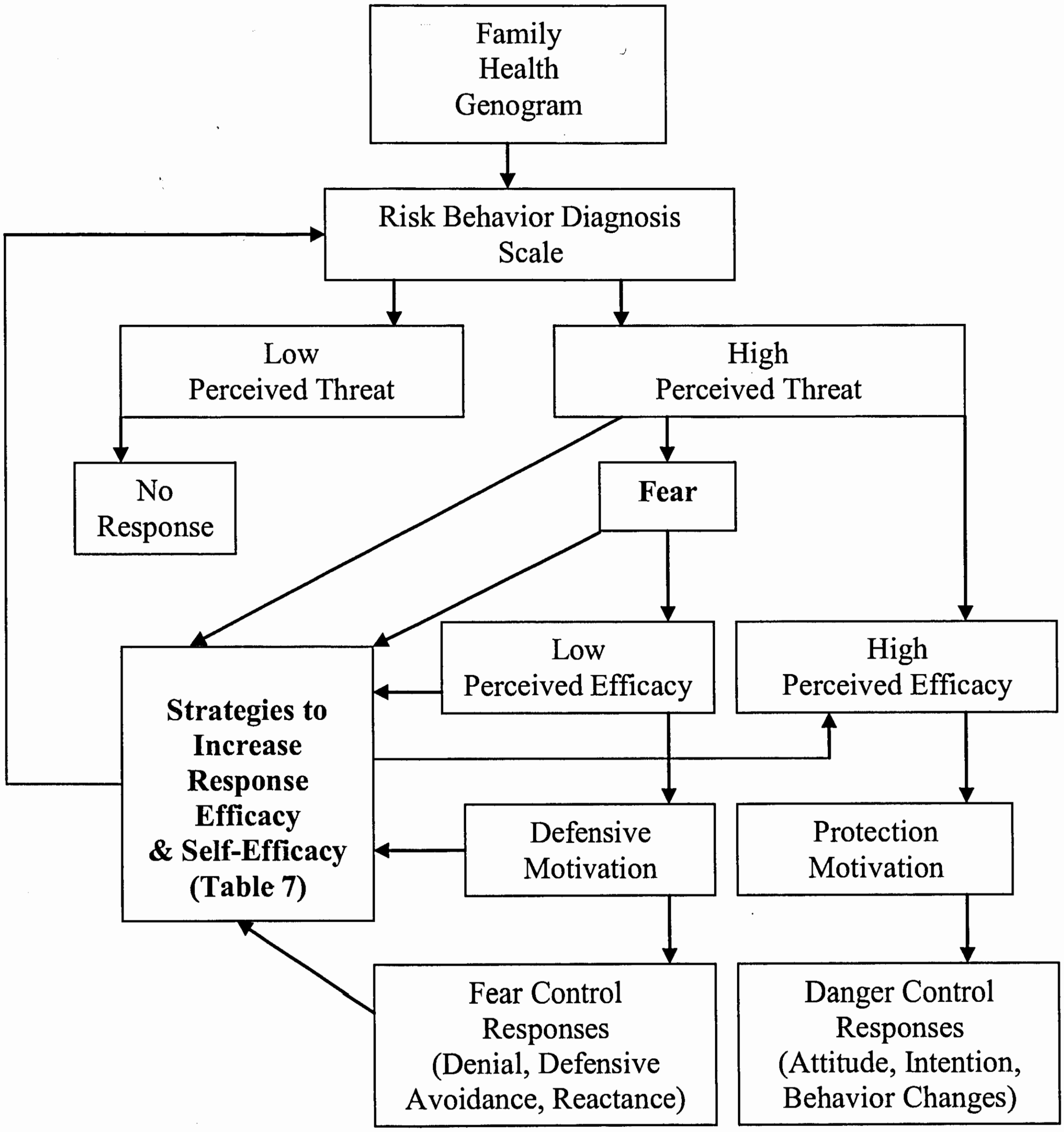


Figure 12. Flow chart for health educators for utilization of strategies to increase response efficacy and self-efficacy (see Table 7) when using a family health genogram as a threat appeal. This decision tree utilizes the Risk Behavior Diagnosis Scale to assess Threat and efficacy and applies to constructs of the Extended Parallel Process Model.

Table 8
Stage of Change for Gathering of Family Health History Before and After Completing the Genogram

Stage of Change	Before %	After %
Precontemplation	31.6	14.1
Contemplation	32.2	15.9
Planning	5.8	10.0
Action	14.0	37.1
Maintenance	7.0	15.9
Relapse	9.4	7.1

Research Question 3

Does completion of the specified online health genogram advance the participant's readiness to adopt health promotion behaviors? The Stages of Change from the Transtheoretical Model were used to assess the participants' experience with documenting family health history as well as their CVD preventive behaviors. The significant difference in stage of change for gathering of family health history after completion of the genogram demonstrated the participants' increased awareness of their family's health history as well as their experience in documenting that history (see Table 8). Those groups who had not thought about gathering their family health history and those who had thought about but not yet planned to gather it each decreased by half, while those groups who had started gathering their family history or gathered family history and kept it current more than doubled after completing the genograms.

There was also a significant advancement in stage of readiness to practice CVD preventive behaviors following completion of the genogram (see Table 5). Those who had not thought about prevention steps decreased considerably after completion of the genogram, while consistent increases were observed in the other categories.

Limitations of the Study

This study had several limitations that resulted from technical difficulties with the online genogram program and with participant misunderstanding of the instructions. The first problem became apparent after the USDHHS launched a new version of *My Family History* just two weeks after the study began. Problems with the new version and the inability to open genograms that had been saved in the previous version caused frustration for many participants due to unnecessary time and delay in completing their genograms. This resulted in attrition as some who completed and returned the first survey chose not to complete and return the second. The follow-up mailing which authorized the use of a manual genogram in lieu of the online genogram introduced variation in the recording of the family medical history into the study.

Another possible cause of the attrition was the redundancy in the questions required by averaging three questions for each construct of the EPPM for each of the behaviors under study. This redundancy led some participants to believe that they had already completed the follow-up survey and thus did not finish the study. This could have been avoided by putting more emphasis on the importance of completing two surveys, even though the surveys would appear redundant.

Failure of participants to read or follow instructions led to certain limitations in the study. While participants were instructed to count only adult relatives in their genograms, it became evident that some had included their very young children. This deviation in protocol precluded the calculation of percentage of adult relatives with CVD, which necessitated using the total number of relatives with CVD in the analysis. Even after completing the genogram, many participants reported no advance in their stage of change regarding collecting family health history. More emphasis should have been placed on identifying any advance in stage that may have occurred from completing the genogram.

Another limitation of the study was discovered when participants who completed the study reported that, prior to participation in the study, they had done extensive work in gathering their family health history. A final limitation of the study was in the measuring of self-reported intent to practice recommended health promotion or disease prevention behaviors rather than actually measuring the behavior changes.

Recommendations for Future Research

Additional research is needed in the area of health education to apply the theories and tools related to fear appeals. Recommendations for future research include:

- Include only those participants for whom a behavior change is recommended.
- Limit participants to those who have not previously collected their family health history.

- Emphasize the importance of completing and returning both surveys.
- Utilize a consistent form of genogram.
- Emphasize the exclusion of children in the count of total persons in the genogram to enable the use of percentages of family members with CVD.
- Explain that the stages of change should reflect the changes that occurred as a result of completing the genogram.
- Employ follow-up questionnaires to assess actual changes in health promotion or disease prevention behaviors rather than only intent to change behaviors.
- Study genograms as threat appeals for other diseases that tend to run in families and for other recommended behavior changes.

Recommendations

Health educators should teach families to document family health history using online genogram tools and to update them on a regular basis. They should utilize the RBD Scale of the EPPM to assess perceived severity and susceptibility as well as perceived response efficacy and self-efficacy for various recommended health behavior changes. Should threat exceed efficacy resulting in fear control, health educators should then employ strategies to increase efficacy in order to ensure that fear appeal messages are accepted and health behaviors are changed to promote health and prevent disease.

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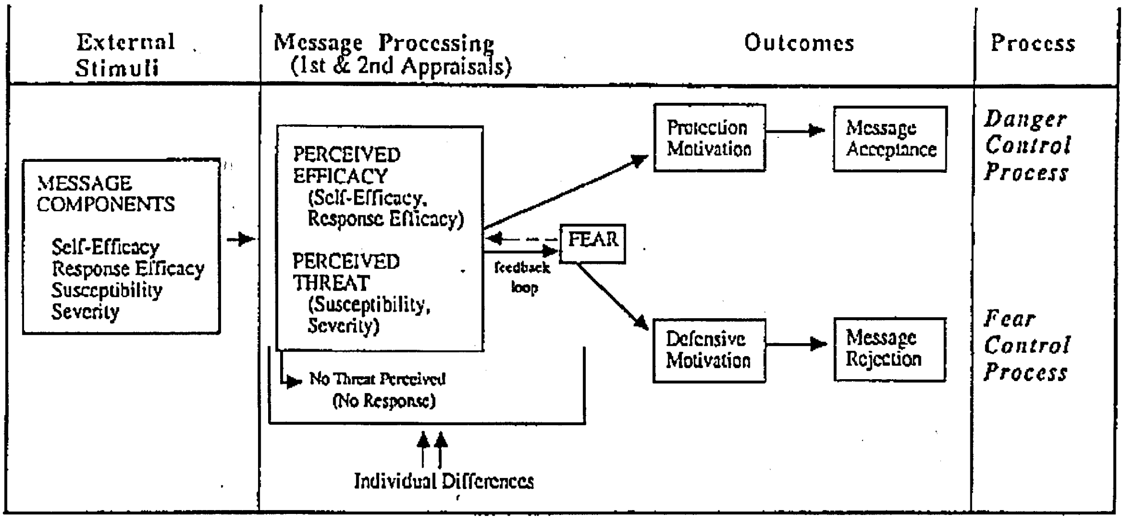
APPENDIX A

The Extended Parallel Process Model

The Extended Parallel Process Model



The Extended Parallel Process Model (EPPM) from *Effective Health Risk Messages: A Step-by-Step Guide*. Used with permission from the author.

APPENDIX B

Recruitment Flyer

Do you know your family health history?

Does anyone in your family have cardiovascular disease*?

I am a doctoral student in the Health Studies program at Texas Woman's University seeking participants for a study involving the online recording of family health history using the *My Family Health Portrait* at the U.S. Surgeon General's Family Public Health Initiative website.

The purpose of the study is to explore the relationship of the history of familial cardiovascular disease* to health promotion and disease prevention behaviors.

Participants must be at least 18 years old, have at least one family member who has had cardiovascular disease*, speak, read and write English, have access to the internet, have computer skills to complete the online medical history.

The potential benefits of participation will be to realize family health risks, and communicate familial risks to health care providers. In addition, the participant can share the family health history with family members who can derive the same benefits. No further incentives will be provided.

Family histories will be completed online and saved with password protection for potential future additions. Except for the total number of family members with cardiovascular disease*, no family health information will be shared with me. Instructions and surveys will be mailed or e-mailed according to the participant's preference. All responses will be kept confidential.

For further information about this study contact:

Betty Carlson Bowles
3410 Taft
Wichita Falls, TX 76308
(940) 397-4048
betty.bowles@mwsu.edu

* Cardiovascular disease is a cluster of diseases including high blood pressure, high cholesterol, atherosclerosis (hardening of the arteries), coronary heart disease, or stroke.

APPENDIX C

Midwestern State University Human Subjects in Research Committee

MIDWESTERN STATE UNIVERSITY WICHITA FALLS, TEXAS 1922

Human Subjects In Research Committee

Institutional Review Board in Compliance with 45 CFR 46

MSU Policy 2.37

MEMORANDUM

TO: Betty Bowles

RE: HSRC Application

DATE: November 5, 2008

Please be advised that your application for research utilizing human subjects has been reviewed and approved by the above named committee. The number assigned this project is:

File Number 08110502

Please include this number in any presentation or publication arising from this research. You may be required to place a copy of this letter within the thesis or other class, department, or college documentation. This approval is valid for one calendar year following granting of approval status. Your may request an extension by submitting a letter requesting such to the HSRC committee chair.

Respectfully,

Gayle Mullen

Chair, Human Subjects in Research Committee

APPENDIX D

Texas Woman's University Institutional Review Board

TWU
TEXAS WOMAN'S UNIVERSITY
DENTON DALLAS HOUSTON

Institutional Review Board
Office of Research and Sponsored Programs
P.O. Box 425619, Denton, TX 76204-5619
940-898-3378 Fax 940-898-3416
e-mail: IRB@twu.edu

November 13, 2008

Ms. Betty Carlson Bowles
Midwestern State University
3410 Taft
Wichita Falls, TX 76308

Dear Ms. Bowles:

Re: *Genograms as Threat Appeals: Using the Extended Parallel Process Model with Familial Cardiovascular Disease*

The above referenced study has been received and reviewed by the Texas Woman's University Institutional Review Board (IRB) and has been determined to be exempt from further review because it has been reviewed and approved by the IRB at Midwestern State University. Participants in the study will be recruited and the study will take place at Midwestern State University in Wichita Falls, Texas.

Another review by the TWU IRB is required if your project changes in any way, and the TWU IRB must be notified immediately regarding any adverse events. If you have any questions, feel free to call the TWU Institutional Review Board at the phone number listed above.

Sincerely,

Dr. David Nichols, Chair
Institutional Review Board – Denton

cc: Dr. Kristin Wiginton, Health Studies
Graduate School

Think SUCCESS Think TWU

APPENDIX E

Family Health Portrait Instructions

and Family Health Portrait Survey #1

Family Health Portrait

Many common diseases can run in families, and a family health history can be a powerful screening tool for prevention and early diagnosis. To assist families in creating a family health history, the U.S. Surgeon General has included a computerized tool on the U.S. Department of Health and Human Services website. This tool makes compiling a family history easy and fun. This study examines your reactions to completing *My Family Health Portrait.*

To be eligible for this study, participants must:

1. Be at least 18 years of age.

2. Have at least one first-degree relative (mother, father, brother, sister, son, daughter) who has or has had some form of cardiovascular disease such as hypertension / high blood pressure, high cholesterol, atherosclerosis / hardening of the arteries, coronary artery disease, angina, myocardial infarction / heart attack, stroke or TIA / transient ischemic attack, or other heart or circulatory problem.

3. Speak, read and write English at a minimum of eighth-grade level.

4. Have access to the internet.

5. Have computer skills to complete the online medical genogram.

To participate in this study of family health portraits please:

1. Complete the Family Health History Survey #1.

   Seal the completed survey in the blank envelope, and return it, and the separate referral form in the enclosed stamped, self-addressed envelope. The sealed blank envelopes will be removed from the mailing envelopes by an assistant before the researcher sees them.

2. Access the U.S. Surgeon General's Family History Initiative at http://www.hhs.gov/familyhistory/

   Click on Access *My Family Health Portrait* web version.

   Follow the instructions for downloading *My Family Health Portrait*. Click on “Download Now”.

Construct your family health portrait including **at least 3 generations of adults**. Save your document so you can edit it as circumstances change or you discover more information.

3. Please use the Referral Form to send me the addresses of other friends or relatives who might participate in this study.

4. Within a week you will receive Survey #2 by mail. Please complete and return it as before. I will send a reminder postcard if I have not received your survey within 2 weeks.

If you have any questions about the study, please contact me at the addresses below.

I certainly appreciate your cooperation and help. Thank you so very much for your participation!

Betty Carlson Bowles, RN, MSN
4018 Kingsbury
Wichita Falls, TX 76308
betty.bowles@mwsu.edu
(940) 397-4048

Family Health Portrait Survey #1

Please complete this Survey AFTER you complete your *Family Health Portrait* online. Please answer all the questions truthfully and completely. Your answers are confidential.

Place an "X" by ONE statement that best represents your thoughts and actions related to family health history BEFORE completing *My Family Health Portrait* (mark only one).

___I have NOT thought about gathering my family health history.

___ I have thought about gathering family health history, but have not yet made plans to do so.

___I plan to gather my family health history in the near future.

___I have started gathering my family health history.

___I have gathered my family health history and made regular additions to keep it current.

___I have gathered my family health history in the past, but have not kept it current.

Place an "X" by ONE statement that best represents your thoughts / actions related to prevention or control of cardiovascular disease BEFORE completing *My Family Health Portrait* (mark only one).

___I have NOT thought about prevention or control of cardiovascular disease.

___I have thought about prevention or control of cardiovascular disease, but have not yet made plans to do so.

___I plan to take recommended steps to prevent or control cardiovascular disease in the near future.

___I have taken some recommended steps to prevent or control cardiovascular disease.

___I consistently follow recommended steps to prevent or control cardiovascular disease.

___I have followed recommended steps to prevent or control cardiovascular disease in the past, but currently do not do so.

Circle the answer that best describes your agreement with each of the following statements.

I am at risk for getting cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

It is possible that I will get cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

I am susceptible to getting cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

Cardiovascular disease is a serious disease.

Strongly disagree Disagree NeutralAgree Strongly agree

Cardiovascular disease is harmful.

Strongly disagree Disagree NeutralAgree Strongly agree

Cardiovascular disease is a severe threat.

Strongly disagree Disagree NeutralAgree Strongly agree

Regular physical activity helps prevent cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

Performing regular exercise reduces the risk cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

The risk of cardiovascular disease is reduced by regular exercise.

Strongly disagree Disagree NeutralAgree Strongly agree

Maintaining normal weight helps prevent cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

Maintaining recommended weight reduces the risk of cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

The risk of cardiovascular disease is reduced by maintaining normal weight.

Strongly disagree Disagree NeutralAgree Strongly agree

Cardiovascular disease can be prevented by avoiding tobacco use.

Strongly disagree Disagree NeutralAgree Strongly agree

The risk of cardiovascular disease is reduced by not using tobacco.

Strongly disagree Disagree NeutralAgree Strongly agree

Not using tobacco reduces the risk of cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

I am able to maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

Maintaining my recommended weight is easy for me.

Strongly disagree Disagree NeutralAgree Strongly agree

I can maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

I am able to engage in regular physical activity.

Strongly disagree Disagree NeutralAgree Strongly agree

It is easy for me to engage in regular physical exercise.

Strongly disagree Disagree NeutralAgree Strongly agree

I can perform physical exercise regularly.

Strongly disagree Disagree NeutralAgree Strongly agree

I am able to avoid using tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

Avoiding the use of tobacco is easy for me.

Strongly disagree Disagree NeutralAgree Strongly agree

I can avoid using tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

I intend to engage in regular physical activity.

Strongly disagree Disagree NeutralAgree Strongly agree

I plan to exercise regularly.

Strongly disagree Disagree NeutralAgree Strongly agree

I will exercise regularly.

Strongly disagree Disagree NeutralAgree Strongly agree

I will take measures to achieve / maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

I plan to achieve or maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

I intend to achieve or maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

I intend to avoid tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

I plan to avoid tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

I will not use tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

Knowing my family health history motivates me to improve my health.

Strongly disagree Disagree NeutralAgree Strongly agree

Because of my family health history I want to adopt healthy behaviors.

Strongly disagree Disagree NeutralAgree Strongly agree

My family health history makes me realize I should change unhealthy behaviors.

Strongly disagree Disagree NeutralAgree Strongly agree

Please seal your completed survey in the blank envelope and return it in the self-addressed stamped envelope that is enclosed.

APPENDIX F

Family Health Portrait Survey #2

Family Health Portrait Survey #2

Please complete this Survey AFTER you complete your *Family Health Portrait* online. Please answer all the questions truthfully and completely. Your answers are confidential.

Which generations did you include in your *Family Health Portrait*?

(Place an "X" by all that apply)

___ Your grandchildren

___ Your children

___ Your generation (yourself, sisters, brothers)

___ Your parents' generation (parents, aunts, uncles)

___ Your grandparents

___Your great-grandparents

How many TOTAL people did you include in your *Family Health Portrait*?

____

Of the people that you included in your *Family Health Portrait*, how many have had some form of cardiovascular disease such as heart disease, stroke, high blood pressure, high cholesterol, or atherosclerosis (hardening of the arteries), etc.

____

Place an "X" by ONE statement that best represents your thoughts and actions related to family health history AFTER completing *My Family Health Portrait* (mark only one).

___ I have NOT thought about gathering my family health history.

___ I have thought about gathering family health history, but have not yet made plans to do so.

___ I plan to gather my family health history in the near future.

___ I have started gathering my family health history.

___ I have gathered my family health history and made regular additions to keep it current.

___ I have gathered my family health history in the past, but have not kept it current.

Place an "X" by ONE statement that best represents your thoughts / actions related to prevention or control of cardiovascular disease AFTER completing *My Family Health Portrait* (mark only one).

___ I have NOT thought about prevention or control of cardiovascular disease.

___ I have thought about prevention or control of cardiovascular disease, but have not yet made plans to do so.

___ I plan to take recommended steps to prevent or control cardiovascular disease in the near future.

___ I have taken some recommended steps to prevent or control cardiovascular disease.

___ I consistently follow recommended steps to prevent or control cardiovascular disease.

___ I have followed recommended steps to prevent or control cardiovascular disease in the past, but currently do not do so.

Circle the answer that best describes your agreement with each of the following statements.

Completing the *Family Health Portrait* made me feel:

Frightened

Strongly disagree Disagree NeutralAgree Strongly agree

Tense

Strongly disagree Disagree NeutralAgree Strongly agree

Anxious

Strongly disagree Disagree NeutralAgree Strongly agree

Uncomfortable

Strongly disagree Disagree NeutralAgree Strongly agree

Nervous

Strongly disagree Disagree NeutralAgree Strongly agree

I am at risk for getting cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

It is possible that I will get cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

I am susceptible to getting cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

Cardiovascular disease is a serious disease.

Strongly disagree Disagree NeutralAgree Strongly agree

Cardiovascular disease is harmful.

Strongly disagree Disagree NeutralAgree Strongly agree

Cardiovascular disease is a severe threat.

Strongly disagree Disagree NeutralAgree Strongly agree

Regular physical activity helps prevent cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

Performing regular exercise reduces the risk cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

The risk of cardiovascular disease is reduced by regular exercise.

Strongly disagree Disagree NeutralAgree Strongly agree

Maintaining normal weight helps prevent cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

Maintaining recommended weight reduces the risk of cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

The risk of cardiovascular disease is reduced by maintaining normal weight.

Strongly disagree Disagree NeutralAgree Strongly agree

Cardiovascular disease can be prevented by avoiding tobacco use.

Strongly disagree Disagree NeutralAgree Strongly agree

The risk of cardiovascular disease is reduced by not using tobacco.

Strongly disagree Disagree NeutralAgree Strongly agree

Not using tobacco reduces the risk of cardiovascular disease.

Strongly disagree Disagree NeutralAgree Strongly agree

I am able to maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

Maintaining my recommended weight is easy for me.

Strongly disagree Disagree NeutralAgree Strongly agree

I can maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

I am able to engage in regular physical activity.

Strongly disagree Disagree NeutralAgree Strongly agree

It is easy for me to engage in regular physical exercise.

Strongly disagree Disagree NeutralAgree Strongly agree

I can perform physical exercise regularly.

Strongly disagree Disagree NeutralAgree Strongly agree

I am able to avoid using tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

Avoiding the use of tobacco is easy for me.

Strongly disagree Disagree NeutralAgree Strongly agree

I can avoid using tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

I intend to engage in regular physical activity.

Strongly disagree Disagree NeutralAgree Strongly agree

I plan to exercise regularly.

Strongly disagree Disagree NeutralAgree Strongly agree

I will exercise regularly.

Strongly disagree Disagree NeutralAgree Strongly agree

I will take measures to achieve / maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

I plan to achieve or maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

I intend to achieve or maintain my recommended weight.

Strongly disagree Disagree NeutralAgree Strongly agree

I intend to avoid tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

I plan to avoid tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

I will not use tobacco products.

Strongly disagree Disagree NeutralAgree Strongly agree

Knowing my family health history motivates me to improve my health.

Strongly disagree Disagree NeutralAgree Strongly agree

Because of my family health history I want to adopt healthy behaviors.

Strongly disagree Disagree NeutralAgree Strongly agree

My family health history makes me realize I should change unhealthy behaviors.

Strongly disagree Disagree NeutralAgree Strongly agree

Tell me about yourself. Circle your responses:

My gender is Male Female

My age is 18-30 31-45 46-60 61-75 76+

My years of education are: Less than 8 8-12 More than 12

My employment status is:

Not employed Employed part time Employed full time Retired

For most of my years of employment I would classify my work as:

Desk job (Sitting) Moderately active (Walking) Strenuous (Lifting)

The ethnicity of most of my ancestors is:

American Indian / Alaskan Native Asian

Black / African American Hispanic / Latino (of any race)

Native Hawaiian / Pacific Islander White Other

My family's annual income is:

Less than \$25,000 \$26,000- \$50,000 \$51,000-\$75,000 More than \$76,000

My physician has said I have / have had:

Heart disease High blood pressure High cholesterol

Heart valve problems Atherosclerosis (hardening of the arteries)

Other heart problems Other circulatory problems

I take medication for my:

Heart High blood pressure High cholesterol Other heart problem

I have had a:

Heart attack Stroke TIA (small stroke) Other heart problems

I have had surgery for:

Coronary Artery Bypass Heart Valve Aneurysm

Pacemaker Other heart problem Other circulatory problem

I believe that I am:

Underweight Normal weight Overweight

I would describe my daily activity level as:

Inactive | Active | Very active

I would describe my tobacco use as:

Never used tobacco | Used tobacco in the past | Use tobacco now

APPENDIX G

Survey Questions and the Associated Theory or Construct

Survey Questions and the Associated Theory or Construct

Question	Theory / Construct to be Measured
Place an "X" by the statement that best represents your thoughts and actions about gathering your family health history after completing *Family Health Portrait.*	Transtheoretical Model /Stages of Change Cardiovascular Disease (CVD)
Completing the Family Health Portrait made me feel: frightened, tense, anxious, uncomfortable, nervous.	EPPM Fear / Threat (post-test only)
I am at risk for getting CVD.	EPPM Threat Susceptibility
It is possible that I will get CVD.	
I am susceptible to getting CVD.	
CVD is a serious disease.	EPPM Threat Severity
CVD is harmful.	
CVD is a severe threat.	
Regular physical activity helps prevent CVD.	EPPM Response Efficacy (Physical Activity)
Performing regular exercise reduces the risk of CVD.	
The risk of CVD is reduced by regular exercise.	
Maintaining normal weight helps prevent CVD.	EPPM Response Efficacy (Weight)
Maintaining recommended weight reduces the risk of CVD.	
The risk of CVD is reduced by maintaining normal weight.	
CVD can be prevented by avoiding tobacco use.	EPPM Response Efficacy (Tobacco)
The risk of CVD is reduced by not using tobacco.	
Not using tobacco reduces the risk of CVD.	

I am able to maintain my recommended weight.	EPPM Self-efficacy (Weight)
Maintaining my recommended weight is easy for me.	
I can maintain my recommended weight.	
I am able to engage in regular physical activity.	EPPM Self-efficacy (Physical Activity)
It is easy for me to engage in regular physical activity.	
I can perform physical activity regularly.	
I am able to avoid using tobacco products.	EPPM Self-efficacy (Tobacco)
Avoiding the use of tobacco is easy for me.	
I can avoid using tobacco products.	
I intend to engage in regular physical activity.	EPPM Outcomes Message acceptance or rejection Intent (Exercise)
I plan to exercise regularly.	
I will exercise regularly.	
I will take measures to achieve or maintain my recommended weight.	EPPM Outcomes Message acceptance or rejection Intent (Weight)
I plan to achieve or maintain my recommended weight.	
I intend to achieve or maintain my recommended weight.	
I intend to avoid tobacco products.	EPPM Outcomes Message acceptance or rejection Intent (Tobacco)
I plan to avoid tobacco products.	
I will not use tobacco products.	
Knowing my family health history motivates me to improve my health.	EPPM Outcomes Message acceptance or rejection Motivation
Being aware of my family health history makes me want to adopt health promotion behaviors.	
My family history makes me realize I should avoid unhealthy behaviors.	

APPENDIX H

Sample Family Health Portrait Diagram Report From *My Family Health Portrait,*

a Tool From the Surgeon General.

Sample Family Health Portrait Diagram Report From *My Family Health Portrait,*

a Tool from the Surgeon General.

Chart Legend			
Male family member	Female family member	Family members with a history of disease	Deceased family members
HA = Heart Attack	Alz = Dementia/Alzheimer's	BoC = Bone Cancer	HD = Heart Disease
HYP = Hypertension	OST = Osteoporosis	Emp = Emphysema	SBA = Stroke/Brain Attack
HCH = High Cholesterol