Stony Brook University

Psychosocial Aspects of Risk Perceptions for Cardiovascular Disease, Breast Cancer, and Lung Cancer in Younger and Older Women

A Dissertation Presented

by

Jada Gabrielle Hamilton

to

The Graduate School

in Partial Fulfillment of the

Requirements

for the Degree of

Doctor of Philosophy

in

Social/Health Psychology

Stony Brook University

May 2009

Stony Brook University

The Graduate School

Jada Gabrielle Hamilton

We, the dissertation committee for the above candidate for the

Doctor of Philosophy degree, hereby recommend

acceptance of this dissertation.

Marci Lobel, Ph.D.
Dissertation Advisor
Associate Professor, Department of Psychology

Anne Moyer, Ph.D.
Chairperson of Defense
Assistant Professor, Department of Psychology

Richard Heyman, Ph.D.
Research Professor, Department of Psychology

Catherine Messina, Ph.D.
Research Associate Professor, Preventive Medicine, Stony Brook University

This dissertation is accepted by the Graduate School.

Lawrence Martin
Dean of the Graduate School

Abstract of the Dissertation

Psychosocial Aspects of Risk Perceptions for Cardiovascular Disease, Breast Cancer, and Lung Cancer in Younger and Older Women

by

Jada Gabrielle Hamilton

Doctor of Philosophy

in

Social/Health Psychology

Stony Brook University

2009

Health problems of particular relevance to women include cardiovascular disease, the leading cause of death in women, breast cancer, the most prevalent form of cancer in women apart from skin cancer, and lung cancer, the leading cause of cancer deaths in women. Individual women differ in their perception of risk for these diseases. Although perceptions of disease risk have critical public health implications because they are associated with the adoption of protective and screening health behaviors, little is known about psychosocial factors that contribute to disease risk perceptions. However, research on individual traits and social influences suggests that these and related factors may affect perceptions of disease risk, and that such associations differ for women of varying ages.

The present study used structural equation modeling to test theoretically-based hypotheses about women's perceptions of risk for cardiovascular disease, breast cancer, and lung cancer, with four main goals: 1) to compare competing models of risk

perceptions in both younger and older women; 2) to investigate relationships among perceptions of risk, beliefs about disease etiology, and the adoption of healthful behaviors; 3) to identify psychosocial factors (e.g., optimism, social exposure to disease) that contribute to perceptions of disease risk; and 4) to explore the association of traditional gender roles with perceptions of disease risk. Exploratory analyses examining the extent to which objective risk factors moderate associations between psychosocial factors and perceptions of disease risk were also conducted. Survey data from 634 participants, including 454 younger (ages 18-25) and 180 older (ages 40 and above) women, were analyzed. Surveys included well-validated instruments and measures designed for this study.

Results confirm that women's risk perceptions are multifactorial, based upon perceptions of personal risk, as well as perceptions of risk for others and estimates of the prevalence and mortality rates of a disease. Although perceptions of disease risk were unrelated to current levels of preventive behaviors, greater perceptions of risk were associated with screening behaviors in some women. Whereas social influences primarily contributed to younger women's disease risk perceptions, both social influences and individual traits contributed to older women's risk perceptions. Objective risk factors were largely unrelated to women's global perceptions of risk for chronic disease. Older and younger women's beliefs about disease risks differed, as did the influences on these groups of their dispositional traits and interpersonal relationships. Results have important implications for conceptualizing and measuring risk perceptions, and for investigating how perceived risk contributes to health behavior practices.

Table of Contents

List of Tables

List of Figures

List of Appendices

Acknowledgements

In many ways, it seems impossible that this chapter of my life is coming to an end. As I reflect on the past five years, I am overwhelmed with gratitude and respect for all the amazing people that helped me reach this point.

My deepest and most sincere thanks go to my advisor, Marci Lobel. When I first met you while visiting Stony Brook's campus in 2004, I was struck by your unique combination of intelligence, warmth, and approachability. I knew I could learn a lot by working with you, but I never guessed just how much I would take away from this experience. You have given me so many opportunities to grow as a researcher and a teacher. Whenever I needed support, you provided it; whenever I needed a push, you gave it. I have such admiration for all that you manage to do, and so much appreciation for the outstanding example with which you have provided me. I have been truly lucky to have you as a friend and a mentor, and I look forward to continuing our relationship as colleagues in the future.

My appreciation goes to the members of my dissertation committee. My chair, Anne Moyer, has been a wonderful teacher and resource over the years. Working with you has truly been a joy. Thank you also to Rick Heyman and Catherine Messina, who provided many thoughtful comments and questions regarding my dissertation research. Your expertise and insight have helped me think about my work in new and exciting ways.

In completing this research, I had the opportunity to interact with a number of talented undergraduate research assistants. Their hard work, dedication, and enthusiasm helped to make this project possible. Thank you to Katherine Ambrosino, Svetlana

Bershadsky, Valerie Cuda, Amy Drislane, Sabrina Dukach, Michelle Felice, Jessica Hayes, Azadeh Issapour, Andrew Marchese, Amanda McKenna, Farah Mohamed, Stephen Mondia, Jennifer Morrissey, Fanny Ng, Helen Randazzo, Vicky Tan, and Karen Wallace.

I am also grateful for the generous financial support that funded this research, which was provided by an Individual Development Award from United University Professors and the Department of Psychology at Stony Brook University.

It has been a privilege to know the many fantastic women of the Lobel Lab. I have enjoyed working with Susan Darlow and Lilly Grey, and I have appreciated the help and advice I received from Jennifer Graham, Christine Veloso, and Cheryl Woods-Giscombé. Special thanks also go to Dolores Cannella; you have always been so patient with my countless questions! I have been lucky to have you as someone to look up to over the years, and as a dear friend, too.

I am eternally grateful for the wonderful friendships I have developed as a graduate student. My friends have helped me to celebrate the good times, and commiserated with me during the difficult ones. As a new student, I appreciated the wisdom so generously provided by Karen Langsam and Nefta Mitchell. The countless laughs, drinks, and pints of ice cream I shared with Alice Blackshear and Janette Ponticello were the best remedies for the worst of days. I owe John Borghi, Angel Gonzalez, and Michael Greenstein many thanks for letting me deplete their printing accounts week after week, for solving my frequent computer dilemmas, and for the numerous long, meandering conversations on the third floor that I will sincerely miss.

Additionally, thank you to Greg Perryman, Julie Riederer, Jennifer Rodden, and Dylan Selterman for their kind support and friendship, especially during this last year.

I am very appreciative of Aaron Cropper and Maggie Kelly, my friends out there in the "real world." I certainly do not get to see you often enough, but I am so thankful for your patience, understanding, and support. Thank you also to Kyle Smith, who encouraged me to start this journey in the first place. Your unwavering friendship and confidence in my abilities means so much to me.

The love and support of my family has been instrumental in everything I have accomplished. My gratitude goes to my grandparents, Anne and Leonard Snyder. My love of the world of academia has been shaped by your dedication to higher education and the fondness with which you recall your own experiences. I am also appreciative of my sister, Jelisa Hamilton. Your hard work and determination in pursuing your dreams is inspiring. My mother, Susan Hamilton, deserves more recognition than I can even begin to express. You have always been my greatest advocate and cheerleader. Your faith, strength, and compassion are truly phenomenal, and I am honored and proud to be your daughter.

My heartfelt gratitude goes to my partner and friend, Victor Luna. Your passion for research is an inspiration, and your love for me is a blessing. I do not know how I could have made it through the frustrations and uncertainties of the past year without your extraordinary support and patience. I am so very appreciative of the sacrifices you have made, and am so excited to see what the next chapter holds for us both.

Chronic diseases pose serious threats to the well-being of all women. Diseases of major concern include cardiovascular disease, the leading cause of death in women (Rosamond et al., 2008), breast cancer, the most prevalent form of cancer in women apart from skin cancer, and lung cancer, the leading cause of cancer deaths in women (American Cancer Society, 2008). Although risk for these diseases is influenced by individual characteristics including age and family history, risk is also increased by modifiable health behaviors such as physical inactivity, poor diet, and cigarette smoking. One variable consistently associated with behavior change and the adoption of protective health behaviors is perceived risk. However, differences exist among women in their perceptions of disease risk, and perceptions of risk vary across diseases. Little is currently known about psychosocial factors, such as individual traits or social exposure to disease, which may influence perceptions of disease risk. Furthermore, it is possible that objective factors that moderate one's risk for disease, such as age, may interact with psychosocial factors to predict perceptions of risk. The proposed study will address these and related issues.

Cardiovascular Disease

Cardiovascular disease encompasses a number of ailments including hypertension, coronary heart disease, myocardial infarction, angina pectoris, heart failure, and stroke. Common risk factors for cardiovascular disease include age, having a family history of disease, physical inactivity, obesity, smoking, high blood cholesterol, diabetes mellitus, and consuming an unbalanced diet high in saturated fat, salt, and cholesterol and low in fruits, vegetables, and fiber (Lloyd-Jones et al., 2006; Rosamond et al., 2008; Silberberg, Fryer, Wlodarczyk, Robertson, & Dear, 1999). At age 50, a woman's lifetime risk for

developing cardiovascular disease is approximately 39% (Lloyd-Jones et al., 2006). Mortality rates of 245.3 female deaths per 100,000 have been reported for cardiovascular disease; however, rates differ for women of varying ethnicity, with deaths among Black women (333.6 per 100,000) exceeding those among White women (238.0 per 100,000; Rosamond et al., 2008). Cardiovascular disease caused approximately one death per minute among women in 2004, and the number of deaths attributable to cardiovascular disease is greater than that for cancer, chronic lower respiratory disease, Alzheimer's disease, accidents, and diabetes mellitus combined (Rosamond et al., 2008).

Breast Cancer

Breast cancer is the most prevalent form of cancer, apart from skin cancer, among women of all racial and ethnic backgrounds. Risk factors for breast cancer include being female, age, having a family history of breast cancer (and for some, possessing a mutation in the *BRCA1/BRCA2* genes), being overweight or obese at menopause, physical inactivity, use of hormone therapy, and alcohol consumption (American Cancer Society, 2008). A woman's lifetime risk of developing invasive breast cancer is 12% (Ries et al., 2008). Breast cancer is the second-leading cause of cancer deaths among all women (except for those of Hispanic origin, for whom breast cancer is the leading cause of cancer deaths). The mortality rate for breast cancer is 24.4 deaths per 100,000 for all women (U.S. Cancer Statistics Working Group, 2007).

Lung Cancer

Lung cancer is the second most-commonly diagnosed cancer among White and American Indian/Alaska native women, and the third most-commonly diagnosed cancer among Black, Asian/Pacific Islander, and Hispanic women. Cigarette smoking is the

greatest risk factor for the development of lung cancer, although disease risk is also increased by occupational and environmental exposure to secondhand smoke, asbestos, radon, certain metals and organic chemicals, air pollution, and radiation. Age, personal history of tuberculosis, family history of lung cancer, and a genetic susceptibility to lung cancer have also been identified as risk factors for the disease (American Cancer Society, 2008; National Cancer Institute, 2007). Women face a lifetime risk of approximately 6% for developing lung cancer (Ries et al., 2008). Lung cancer has a mortality rate of 40.9 deaths per 100,000 women, and is the leading cause of cancer deaths for all women except those of Hispanic origin, for whom lung cancer is the second-leading cause of cancer deaths (U.S. Cancer Statistics Working Group, 2007).

Disease Awareness, Health Behaviors, and Perceptions of Disease Risk

From a public health perspective, the high levels of morbidity and mortality associated with cardiovascular disease, breast cancer, and lung cancer are reasons for concern. Additionally, these chronic diseases have significant economic consequences, with estimated direct and indirect costs in 2008 of $448.5 billion for cardiovascular disease and $228.1 billion for cancers of all kinds (National Heart, Lung, and Blood Institute, 2007). Despite these staggering figures, women are not fully aware of their risk for chronic disease. For example, only 57% of women surveyed in 2006 by the American Heart Association recognized cardiovascular disease as the leading cause of death, an increase in awareness compared to previous years (Christian, Rosamond, White, & Mosca, 2007). In a study of women's knowledge of cancer mortality, approximately 67% of respondents inaccurately identified breast cancer as the primary cause of cancer deaths; approximately 30% correctly identified lung cancer (Healton et al., 2007).

Similarly, women's awareness of disease risk factors is varied. For instance, one study found that only half of participants identified age as a risk factor for breast cancer (Pohls et al., 2004). Data collected from college women in 23 countries revealed that although more than 90% of respondents knew that cigarette smoking increases the risk for lung cancer, only 55% knew such a relationship exists for cardiovascular disease (Steptoe et al., 2002). Less than 7% of college women in the same sample knew that alcohol consumption, exercise, dietary fat, or obesity influences women's breast cancer risk (Peacey, Steptoe, Davidsdottir, Baban, & Wardle, 2006). Similarly, in a sample of women aged 50 to 80 years, only approximately one-third of respondents knew that being obese at menopause or drinking alcohol increases a woman's risk for breast cancer (Messina, Kabat, & Lane, 2002).

Increasing women's awareness of these diseases is an important public health goal, especially because awareness has been associated with protective health behaviors including increased physical activity, weight loss, and decreased consumption of unhealthy foods (e.g., Mosca et al., 2006). Health behaviors can have a dramatic impact on people's risk for chronic disease. For instance, 20% of women's cancer deaths are attributable to overweight and obesity (Calle, Rodriguez, Walker-Thurmond, & Thun, 2003), and cigarette smokers are 2 to 4 times more likely to develop cardiovascular disease, and 13 times more likely to develop lung cancer, than are female nonsmokers (U.S. Department of Health and Human Services, 2008). Since chronic disease susceptibility is characterized by largely modifiable risk factors such as exercise, obesity, diet, and smoking, understanding variables that influence people's likelihood of adopting protective and preventive health behaviors is crucial. One such variable is *perceived risk*,

also referred to as perceived susceptibility or perceived vulnerability. Perceived risk, defined as one's belief about the likelihood of personal harm, is a key element in a number of theoretical models of health-protective behaviors (Weinstein, 1993; Weinstein & Klein, 1995). For example, perceived risk is a predictor of health behaviors in protection motivation theory (Prentice-Dunn & Rogers, 1986; Rogers, 1975, 1983), the health belief model (Becker & Maiman, 1975; Janz & Becker, 1984; Rosenstock, 1974), and subjective expected utility theory (Edwards, 1954; Ronis, 1992). These theories predict that people who perceive themselves at greater risk for a negative health outcome are more motivated to adopt precautionary health behaviors than those who perceive themselves at lower risk.

Results from a number of empirical studies have supported these theoretical predictions. For example, an examination of the health belief model in the context of heart disease found that women's perceptions of disease susceptibility accounted for nearly 51% of the variance in their heart disease preventive behaviors such as limiting alcohol consumption, quitting smoking, and losing weight (Ali, 2002). In addition, women with higher perceptions of heart disease risk are more likely to have seen a healthcare provider, and to have arranged for a family or household member to see a healthcare provider, than women with lower perceptions of disease risk (Mosca et al., 2006). In the context of perceived breast cancer risk, meta-analyses indicate that higher perceptions of risk are significantly associated with mammography use as well as interest in and utilization of genetic testing for mutations in the *BRCA1/BRCA2* genes (Katapodi, Lee, Facione, & Dodd, 2004; McCaul, Reid, Rathge, & Martinson, 1996). However, these same analyses note that evidence is inconclusive regarding an association between

perceived cancer risk and breast self-examination. In a study of men and women undergoing lung cancer screening, participants with higher perceptions of risk for receiving an abnormal screening result reported being more ready to quit smoking than those with lower levels of perceived risk (K. L. Taylor et al., 2007). Similar results were found in a study of current head and neck and lung cancer patients; those with higher perceptions of smoking-related risks were more likely to have quit smoking after their cancer diagnosis than patients with lower perceptions of risk (Schnoll et al., 2002).

Various methodological approaches have been used to assess perceptions of risk. With one approach, participants are asked to provide numerical estimates of the prevalence or mortality rate for a given disease. They may be asked to provide frequency estimates (e.g., "how many women out of 100 will develop heart disease in their lifetime?") or percentage estimates (e.g., "what percentage of women will die from breast cancer?"). People often have difficulty understanding and using numerical information, and this innumeracy may help explain why participants inaccurately respond to such assessments (for reviews, see Gigerenzer, Gaissmaier, Kurz-Milcke, Schwartz, & Woloshin, 2007; Klein & Stefanek, 2007). Participants may also be asked to estimate their absolute risk for disease (e.g., "how likely are you to develop lung cancer in the next 10 years?"), with responses provided on a 5- or 7-point Likert-type scale. These absolute risk estimates can be compared to objective indicators of disease susceptibility, allowing for the accuracy of participants' perceptions of risk to be examined. In some cases, participants overestimate their susceptibility, as compared to objective measures of risk. For example, one study of risk perceptions and actual disease risk established by the Gail model (Gail et al., 1989), a statistical model used to calculate a woman's risk for breast

cancer based on a variety of objective risk factors, found that 86% of the sample overestimated their actual disease risk (Daly et al., 1996). People may also underestimate their disease risk, and such inaccurate risk perceptions can be associated with undesirable outcomes. For example, smokers with the greatest degree of unrealistic optimism, defined as an underestimation of their personal risk for lung cancer as compared to their objective risk, are most likely to believe myths about the curability and risk factors for lung cancer, and are also least likely to have an interest in quitting smoking (Dillard, McCaul, & Klein, 2006). Such beliefs are likely to contribute to these individuals' continued smoking and sustained risk for disease development.

An additional assessment approach requires participants to estimate their disease risk compared to another person. These comparative risk estimates may be assessed directly through a single item contrasting a participant's risk to that of a target individual (e.g., "compared to the average woman, how likely is it that you will develop heart disease?"), or indirectly, through one item assessing the participant's perceived personal risk and a second item assessing the participant's perceived risk for a target individual (Helweg-Larsen & Shepperd, 2001). This approach capitalizes on one of the cognitive strategies people typically use when making judgments. As explained by social comparison theory (Festinger, 1954), when people are uncertain about how to evaluate themselves on an unfamiliar dimension, they compare themselves to similar others, especially those with related attributes (e.g., gender). When making such comparative risk estimates, people typically demonstrate an optimistic bias and estimate their personal level of risk for a negative outcome as much lower than those of other people (van der Pligt, 1998; Weinstein, 1980, 1987), consistent with research on downward social

comparison (e.g., S. E. Taylor & Lobel, 1989; Wills, 1981). This optimistic bias may help protect people from experiencing negative feelings of anxiety or worry about their risk status, but may also prevent them from adopting protective or preventive behaviors (Perloff, 1983; Weinstein, 1980).

Psychosocial Correlates of Perceptions of Disease Risk

Although perceptions of risk play an important role in motivating people to adopt protective behaviors, little is known about factors that may influence people's perceived susceptibility for disease development (Eibner, Barth, & Bengel, 2006; Gerend, Aiken, West, & Erchull, 2004). Theoretical models have yet to identify psychosocial factors that may contribute to perceptions of risk. Related research suggests, however, that both individual traits and social influences may be associated with perceptions of disease risk. Furthermore, awareness of objective risk factors is also likely to be related to people's perceived disease susceptibility. Each of these likely contributors to disease risk perceptions is discussed in the following sections.

Individual traits (optimism, health locus of control). Stable across time and situations, individual traits influence how people understand and interact with the world. One such trait is dispositional optimism, a generalized expectancy for positive rather than negative outcomes (Scheier & Carver, 1985). People with high levels of optimism tend to believe that good things will happen to them, and that they will be capable of handling life's demands. An optimistic outlook is associated with a number of positive psychological and physical outcomes including reduced likelihood of re-hospitalization after surgery, lower risk of adverse birth outcomes including low infant birthweight, lower levels of emotional distress, and the adoption of adaptive coping strategies (e.g.,

Carver et al., 1993; Hamilton & Lobel, 2008; Lobel, DeVincent, Kaminer, & Meyer, 2000; Scheier et al., 1999). Additionally, an association exists between optimistic beliefs and perceptions of disease risk; people who are more optimistic perceive they are at lower risk for a variety of disease-related outcomes (Eibner et al., 2006; Gerend, Aiken, & West, 2004; Norman & Brain, 2007; Radcliffe & Klein, 2002). Optimists' lower perceptions of disease risk may be related to their expectations of positive future events, or may be attributable to confidence in their abilities to cope with or prevent health threats (Aspinwall & Brunhart, 1996).

Health locus of control is another trait related to people's beliefs about their abilities to control their physical well-being. This generalized expectancy about whether people can control their health status also influences how people respond to disease-related issues (Wallston, Wallston, & DeVellis, 1978). Health locus of control is conceptualized as having three separate dimensions. The first, an internally-oriented dimension, is characterized by the belief that one has the ability to affect one's health. The remaining externally-oriented dimensions are characterized by the belief that either powerful others or chance forces control one's well-being. It would seem likely that people who believe they can control their health would also believe they are less susceptible to disease development. This prediction has been supported by a few studies examining how health locus of control is associated with perceptions of disease risk. Internal locus of control has been associated with lower perceptions of risk for breast cancer and heart disease (Gerend, Aiken, & West, 2004; Rowe, Montgomery, Duberstein, & Bovbjerg, 2005), whereas external locus of control has been associated with greater perceptions of risk for these diseases (Gerend, Aiken, & West, 2004).

Social influences (social exposure to disease, perceptions of stigma by others). The social environment can powerfully influence how people perceive their risk for disease. Other people can indirectly influence perceptions of risk by providing information about symptoms, risk factors, and treatments for chronic illnesses. Additionally, if people in one's social network become ill, they may serve as an easily accessible reminder of the disease's prevalence. Such direct social exposure to disease may lead to increased feelings of vulnerability and heightened perceptions of risk. This process may occur through the use of the availability heuristic, a decision-making strategy in which people estimate the probability of an event based on the number of examples they can easily call to mind (Tversky & Kahneman, 1973, 1974). When attempting to determine their own risk for disease, people may estimate the disease's prevalence based on how many disease-afflicted people they personally know. Having more social exposure to the disease may therefore result in greater perceptions of risk for the self. Evidence has been somewhat mixed regarding this possibility. For example, while the awareness of myocardial infarction in one's social network was not associated with perceptions of risk in men and women in one study (Meischke et al., 2000), having friends afflicted with breast cancer, colon cancer, heart disease, or diabetes was positively associated with perceptions of risk for each disease among women in a different study (Montgomery, Erblich, DiLorenzo, & Bovbjerg, 2003).

The ways that others respond to those afflicted with disease may further influence people's perceptions of risk. In some cases, a specific disease is considered shameful or embarrassing, and people typically hold negative views of those afflicted with the stigmatized condition. Weinstein (1987) proposed that people might be motivated to

believe they are less susceptible to diseases that they perceive as being highly stigmatized by others. Perceiving themselves at decreased risk for such diseases may help protect people from emotional distress. Diseases believed to be highly controllable due to perceived behavioral risk factors, such as HIV infection or lung cancer, are generally those that are most stigmatized (Weiner, Perry, & Magnusson, 1988).

Objective risk factors (family history, age, body mass index, preventive health behaviors). People's susceptibility to disease is directly affected by objective risk factors. Some risk factors, such as family history of disease or age, are beyond personal control. Other factors, such as obesity or engaging in unhealthy behaviors, are far more controllable. Relationships between these objective risk factors and disease development are frequently discussed by the mass media and healthcare providers (e.g., Meischke et al., 2002), contributing to people's understanding that such factors elevate their risk for chronic disease. People seem to be particularly aware of the risk conferred by a family history of disease. People's awareness of a family history of diseases including breast cancer, heart disease, colon cancer, diabetes, and osteoporosis have been consistently and positively associated with higher perceptions of disease risk (DiLorenzo et al., 2006; Erblich, Bovbjerg, Norman, Valdimarsdottir, & Montgomery, 2000; Gerend, Aiken, West et al., 2004; Katapodi et al., 2004; Montgomery et al., 2003; Patel et al., 2007; Vernon, Vogel, Halabi, & Bondy, 1993; Wilcox & Stefanick, 1999). Research findings regarding age have been less consistent, despite the fact that risk for chronic illness increases with age. While some studies have found that older participants perceive greater risks for disease than do younger participants (Meischke et al., 2000; Renner, Knoll, & Schwarzer, 2000), others have found the opposite association between age and perceptions of disease

risk (Gerend, Aiken, West et al., 2004; Harwell et al., 2005; Vernon et al., 1993). Furthermore, there is some evidence that people consider their standing on controllable risk factors when estimating their disease risk. For instance, people categorized as overweight based on their body mass index (BMI) had higher perceptions of heart disease risk than did people of a normal weight (Renner et al., 2000). Similarly, women smokers were more likely to rate their risk for developing lung cancer and heart disease as above average, compared to former smokers or women who had never smoked cigarettes (Moran, Glazier, & Armstrong, 2003).

Study Overview

The present study investigated perceptions of risk for cardiovascular disease, breast cancer, and lung cancer in a sample of younger and older women. Psychosocial correlates of general and disease-specific perceptions of risk were examined, as were relationships between perceptions of risk and self-reported adoption of preventive and screening health behaviors. Younger (ages 18 to 25) and older (ages 40 and above) women completed study questionnaires including measures of perceptions of disease risk, preventive and screening health behaviors, individual traits, social influences, and objective risk factors, as well as sociodemographic information. Structural equation modeling, a powerful multivariate analysis technique, was used to test hypotheses related to four main goals: 1) to construct and test competing models of risk perceptions in both younger and older women; 2) to examine relationships among perceptions of risk, beliefs about disease etiology, and the adoption of healthful behaviors; 3) to identify individual traits, social influences, and objective risk factors that contribute to perceptions of disease

risk; and 4) to explore the association of traditional gender roles with perceptions of disease risk.

Hypotheses and Analyses

1. Constructing a Model of Perceptions of Disease Risk

A. Measurement Model Design

There is considerable variability in the methods used to assess people's perceptions of risk. Perceptions of risk may be assessed through numerical estimates, or through estimates of absolute or comparative risk. It is unclear which of these methods is most closely related to how people actually think about the risk for disease (Weinstein, 1998). However, empirical findings suggest that different risk measures assess similar yet independent aspects of perceived risk. For instance, in an investigation of women's perceptions of risk for breast cancer, heart disease, and osteoporosis, items assessing both absolute and comparative dimensions of risk loaded significantly onto single factors representing perceived susceptibility for each disease (Gerend, Aiken, & West, 2004; Gerend, Aiken, West et al., 2004). Conversely, Rowe et al. (2005) found that different measures of perceptions of disease risk were moderately correlated with one another, yet had unique associations with other psychological variables (e.g., health locus of control). Similarly, Weinstein (1984) concluded that although absolute and comparative risk estimates were equally related to objective risk factors and were moderately intercorrelated, each assessed different aspects of people's risk-related cognitions. One goal of the present study was to examine whether measures of perceived risk for the self, perceived risk for the average woman, perceived prevalence rate, and perceived mortality rate can serve as indicator variables for a latent variable representing perceptions of

disease risk. A multiply-indicated latent variable representing perceptions of disease risk is likely to be statistically more powerful and more reliable than individual measures of disease risk perceptions. Structural equation modeling was used to construct separate measurement models of perceptions of risk for cardiovascular disease, breast cancer, and lung cancer.

Hypothesis 1.1: People's estimates of their personal risk for cardiovascular disease, the average woman's risk for cardiovascular disease, the prevalence rate of cardiovascular disease, and the mortality rate of cardiovascular disease will share common error variance, and thus be reliable indicators of a latent variable representing perceptions of risk for cardiovascular disease (see Figure 1.1).

Hypothesis 1.2: People's estimates of their personal risk for breast cancer, the average woman's risk for breast cancer, the prevalence rate of breast cancer, and the mortality rate of breast cancer will share common error variance, and thus be reliable indicators of a latent variable representing perceptions of risk for breast cancer (see Figure 1.2).

Hypothesis 1.3: People's estimates of their personal risk for lung cancer, the average woman's risk for lung cancer, the prevalence rate of lung cancer, and the mortality rate of lung cancer will share common error variance, and thus be reliable indicators of a latent variable representing perceptions of risk for lung cancer (see Figure 1.3).

Exploratory Analyses: Differences may exist in the extent to which the four indicator variables contribute to the latent variable in each measurement model. Therefore, each model was tested to determine whether $a_x = b_x = c_x = d_x$ (where a_x is the

path between the indicator variable of perceived risk for the self and the latent variable representing perceptions of disease risk, for example) Additionally, it is possible that the association of a specific indicator variable with the latent variable differs between diseases. To examine this possibility, tests were conducted to determine whether $a_1 = a_2 = a_3$, $b_1 = b_2 = b_3$, $c_1 = c_2 = c_3$, and $d_1 = d_2 = d_3$ (where a_1 is the path between the indicator variable of perceived risk for the self and the latent variable representing perceptions of risk for cardiovascular disease, a_2 is the path between the indicator variable of perceived risk for the self and the latent variable representing perceptions of risk for breast cancer, etc.).

B. Comparison of Higher-Order Models of Perceptions of Disease Risk

Following the development of well-fitting measurement models of perceptions of risk for each disease, two higher-order models of disease risk were compared.

Disease-Specific Risk Model: One possibility is that people perceive different levels of risk for different diseases. For instance, a woman may perceive her risk for one disease to be quite high, yet she may simultaneously believe that her risk for a different disease is lower than average. Similarly, she may believe that one disease is relatively rare, whereas another is rather common. Although her perceptions of risk for each disease may be influenced by similar factors (e.g., personal experience with a disease, knowledge or presence of objective risk factors, etc.), her disease-specific beliefs exist independently of one another. With a Disease-Specific Risk Model, people's perceptions of risk for a given disease are expected to be independent of their perceptions of risk for other diseases.

General Disease Risk Model: Alternatively, it is possible that people consider risk through the lens of a more general cognitive framework, leading them to perceive risk as either high or low for all diseases. Underlying people's perceptions of risk for specific diseases may be a general factor of risk sensitivity (Sjoberg, 2000). Differences in risk sensitivity may lead to consistency in people's perceptions of disease risk; those high in risk sensitivity may be greatly concerned by all health threats, and therefore perceive greater risks for all diseases, while those low in risk sensitivity may be less aware of such threats and perceive fewer risks. Another possibility is that consistency in women's perceptions of disease risk is driven by beliefs about personal invulnerability. Most people demonstrate a sense of invulnerability to negative life events, believing they are protected from misfortune or victimization (Perloff, 1983; Perloff & Fetzer, 1986). However, it is possible that people differ on this dimension, with some believing they are invulnerable to most negative events, and others believing they are generally at risk.

Regardless of whether such consistency is due to risk sensitivity or beliefs of invulnerability, both explanations would predict that perceptions of risk for different diseases are related to a single underlying construct. One study has provided empirical support for such a model. A single factor representing general perceived susceptibility was found to underlie older women's perceptions of risk for breast cancer, heart disease, and osteoporosis (Gerend, Aiken, & West, 2004). In the present study, with a General Disease Risk Model, a higher-order factor representing general beliefs about risk would underlie women's perceptions of risk for different diseases.

Hypothesis 1.4: If the Disease-Specific Risk Model is accurate, then the best model fit would be found when the latent variables representing perceptions of risk for

cardiovascular disease, breast cancer, and lung cancer are not correlated with one another (see Figure 1.4).

Hypothesis 1.5: If the General Disease Risk Model is accurate, then the best model fit would be found when the latent variables representing perceptions of risk for cardiovascular disease, breast cancer, and lung cancer all load onto a higher-order latent variable representing general beliefs about risk (see Figure 1.5).

Exploratory Analyses: In the General Disease Risk Model, differences may exist in the extent to which perceptions of risk for each disease are associated with the higher-order latent variable representing general beliefs about risk. Therefore, tests were conducted to determine whether e = g = f. It is also possible that people's perceptions of disease risk change over time, since differences in experience, exposure, and knowledge may influence how people estimate their vulnerability to chronic illness. The possibility of age-related differences in the structure of perceptions of disease risk was explored using the best-fitting model (either the Disease-Specific Risk Model or the General Disease Risk Model). This model was examined separately in younger and older women to determine whether the model fit equally well in both groups.

C. Descriptive Information about Perceptions of Disease Risk

Descriptive statistics assessing levels of perceived personal risk for disease, the perceived average woman's risk for disease, the perceived prevalence rate of a disease, and the perceived mortality rate of a disease were calculated for cardiovascular disease, breast cancer, and lung cancer in younger and older women. Since older women have greater experience with and susceptibility to chronic disease, they may possess a better

understanding of the epidemiology and relative risks of these illnesses than do younger women. These differences are reflected in the following hypotheses:

Hypothesis 1.6: Older women's perceptions of risk for cardiovascular disease will be greater than their perceptions of risk for breast cancer or lung cancer.

Hypothesis 1.7: Younger women's perceptions of risk for breast cancer will be greater than their perceptions of risk for cardiovascular disease or lung cancer.

Hypothesis 1.8: Older women's perceptions of risk for cardiovascular disease will be greater than younger women's perceptions of risk for cardiovascular disease.

Exploratory analyses: There is considerable evidence in support of an optimistic bias in people's risk perceptions (e.g., van der Pligt, 1998; Weinstein, 1980, 1987), whereby people estimate their personal level of risk for a negative outcome as much lower than those of others. To investigate the presence of such a bias, women's estimates of their personal risk for disease were compared to their estimates of the average woman's risk for disease. Another possibility is that ethnicity may be associated with perceptions of disease risk. Differences do exist in the prevalence and mortality rates of specific diseases among women of varying ethnicity (e.g., Rosamond et al., 2008; U.S. Cancer Statistics Working Group, 2007). Descriptive statistics comparing levels of perceived disease risk across women of different ethnicities were calculated.

2. Perceptions of Disease Risk, Health Beliefs, and Healthful Behaviors

As previously discussed, perceptions of risk are associated with the adoption of protective health behaviors in a number of psychological theories (Weinstein, 1993). However, perceptions of risk represent one of a host of factors contributing to people's behavioral intentions; heightened perceptions of risk are likely to be a necessary but not

sufficient contributor to the adoption of protective health behaviors (van der Pligt, 1998). Some of these theories (e.g., the health belief model and protection motivation theory) identify the perceived effectiveness of such behaviors as another important precursor to behavior change. Simply put, unless people believe that a certain behavior will be protective and reduce their chances of harm, they are unlikely to be motivated to perform that behavior. This section of the present study used structural equation modeling to examine how perceptions of disease risk and beliefs about the role of healthful behaviors in disease etiology relate to the performance of preventive and screening health behaviors.

A. Measurement Model Design

To investigate this question, it was necessary to first determine how to best model the dependent variables representing preventive and screening behaviors. Preventive behaviors were assessed with individual items addressing self-reports of exercise, consumption of cigarettes and alcohol, vitamin use, calorie monitoring, and eating a balanced diet. To determine whether these items share significant error variance and therefore may be best modeled as loading onto one latent variable representing preventive behaviors, such a model (see Figure 2.1) was constructed and tested for model fit. Similarly, screening behaviors were assessed with individual items addressing self-reports of breast self-exams, mammograms, and receiving physicals from a doctor. These items were also tested to determine whether they share common error variance and may be best modeled as loading onto a latent variable representing screening behaviors (see Figure 2.2).

Hypothesis 2.1: People's estimates of their frequency of exercising, consuming cigarettes and alcohol, using vitamins, monitoring calories, and eating a balanced diet

will share common error variance, and thus be reliable indicators of a latent variable representing preventive behaviors (see Figure 2.1).

Hypothesis 2.2: People's estimates of their frequency of performing breast self-exams, obtaining mammograms, and receiving physicals from a doctor will share common error variance, and thus be reliable indicators of a latent variable representing screening behaviors (see Figure 2.2).

Exploratory Analyses: Age-related differences are likely to exist in the performance of preventive and screening behaviors, particularly in the use of mammography. Younger women's inexperience with mammography caused this variable to be a poor indicator for screening behaviors in this subsample. Therefore, the fit of latent variables representing preventive and screening behaviors were assessed separately for younger and older women.

B. Preventive Behaviors in the Context of Cardiovascular Disease, Breast Cancer, and Lung Cancer

Separate models exploring the relationship between perceptions of disease risk (using the measurement models constructed in Part 1), beliefs about the role of healthful behaviors in disease etiology, and preventive behaviors were constructed for cardiovascular disease, breast cancer, and lung cancer. Within the context of each disease, the fit for two possible models was compared. The Main Effects Model predicts that 1) perceptions of risk and 2) beliefs about the role of health behaviors in disease etiology are *independently* associated with the performance of preventive behaviors. In contrast, the Interaction Model predicts that health beliefs and perceptions of risk *interact* to affect the performance of preventive behaviors. For example, whereas women with

elevated perceptions of risk for cardiovascular disease who also believe that behaviors can affect their disease risk may perform more healthy behaviors, women with elevated risk perceptions who doubt that behaviors can alter their disease risk may fail to perform preventive behaviors.

Hypothesis 2.3: If the Main Effects Model is accurate, then the best model fit will be found when both the latent variable representing perceived disease risk and the observed variable representing beliefs about the role of health behaviors in disease etiology are positively and independently associated with the variable representing preventive behaviors (see Figures 2.3.1, 2.3.2, and 2.3.3 for models tested for each disease).

Hypothesis 2.4: If the Interaction Model is accurate, then the best model fit will be found when the observed variable representing beliefs about the role of health behaviors in disease etiology moderates the positive association between the latent variable representing perceived disease risk and the variable representing preventive behaviors (see Figures 2.4.1, 2.4.2, and 2.4.3 for conceptual models tested for each disease).

Exploratory Analyses: Since experience, exposure, and knowledge could influence people's health beliefs and their association with behavior, age-related differences in the fit of these proposed models were examined. That is, for each disease both the Main Effects Model and the Interaction Model were examined separately in younger and older women.

C. Screening Behaviors in the Context of Breast Cancer

The screening behaviors assessed in this study are relevant to the detection of breast cancer, therefore, models exploring the relationship between perceptions of disease risk (using the measurement model constructed in Part 1), beliefs about the role of healthful behaviors in disease etiology, and screening behaviors were only examined for this disease. Again, the fit for the Main Effects Model was compared to the fit for the Interaction Model.

Hypothesis 2.5: If the Main Effects Model is accurate, then the best model fit will be found when both the latent variable representing perceived breast cancer risk and the observed variable representing beliefs about the role of health behaviors in breast cancer etiology are positively and independently associated with the variable representing screening behaviors (see Figure 2.5).

Hypothesis 2.6: If the Interaction Model is accurate, then the best model fit will be found when the observed variable representing beliefs about the role of health behaviors in breast cancer etiology moderates the positive association between the latent variable representing perceived breast cancer risk and the variable representing screening behaviors (see conceptual model in Figure 2.6).

Exploratory Analyses: Age-related differences in the fit of these proposed models were examined by testing the Main Effects and Interaction Models separately in younger and older women.

3. Psychosocial Correlates of Perceptions of Disease Risk

Perceptions of disease risk are posited to be critical in motivating behavioral changes that can dramatically improve and protect people's physical health. Yet, little is currently known about individual and psychosocial factors that may contribute to

people's perceptions of disease risk. Related research suggests that individual traits, including dispositional optimism and health locus of control, may influence how people think about their susceptibility to disease. Social influences, such as the extent to which a person is exposed to disease through their relationships, and the degree to which a person believes a specific disease is stigmatized by others, are also likely to contribute to perceptions of disease risk. Finally, the presence of risk factors that objectively increase people's risk for disease, including family history, age, BMI, and preventive health behaviors, are also likely to affect their subjective feelings of vulnerability.

A. Full Model of Psychosocial Correlates of Perceptions of Disease Risk

This section of the present study used structural equation modeling to test a hypothesized model of how individual traits (optimism and health locus of control), social influences (social exposure to disease and perceptions of stigma by others), and objective risk factors (family history of disease, age, BMI, and preventive health behaviors) are associated with perceptions of risk for cardiovascular disease, breast cancer, and lung cancer. Predictions regarding these variables are based on past research findings. In the model (see Figure 3.1), the hypothesized relationships between these predictor variables and the dependent variable of perceptions of disease risk (using the best-fitting model of disease risk created in Part 1), include:

Hypothesis 3.1.1: Dispositional optimism will be inversely associated with perceptions of disease risk, such that women who are more optimistic will have lower perceptions of disease risk.

Hypothesis 3.1.2: Greater internal locus of control will be inversely associated with perceptions of disease risk. Greater beliefs in one's ability to control one's health will be associated with lower perceptions of disease risk.

Hypothesis 3.1.3: Greater chance (external) locus of control will be positively associated with perceptions of disease risk. Having greater beliefs in the power of fate to control one's health will be associated with greater perceptions of disease risk.

Hypothesis 3.1.4: Greater powerful others (external) locus of control will be positively associated with perceptions of disease risk. Having greater beliefs in the ability of powerful others to control one's health will be associated with greater perceptions of disease risk.

Hypothesis 3.1.5: Greater social exposure to disease will be positively associated with perceptions of disease risk. Specifically, personally knowing more women who have been afflicted with a disease will be associated with greater perceptions of disease risk.

Hypothesis 3.1.6: Greater perceptions of stigma by others will be inversely associated with perceptions of disease risk. That is, believing that others hold negative views of those afflicted with a disease will be associated with lower perceptions of disease risk.

Hypothesis 3.1.7: Family history of disease will be positively associated with perceptions of disease risk. Women with a family history of disease are expected to have greater perceptions of disease risk than women without a family history.

Hypothesis 3.1.8: Age will be positively associated with perceptions of disease risk. That is, older women are expected to have greater perceptions of disease risk than younger women.

Hypothesis 3.1.9: Higher BMI will be positively associated with perceptions of disease risk. Women with higher BMI are expected to have greater perceptions of disease risk.

Hypothesis 3.1.10: The more frequent performance of preventive health behaviors will be inversely associated with perceptions of disease risk. To examine this hypothesis, the measurement model of preventive behaviors created in Part 2 was used. Women who perform preventive behaviors more often are expected to have lower perceptions of disease risk.

B. Objective Risk Factors as a Moderating Variable of the Association between Social Influences and Perceptions of Disease Risk

There is an intriguing possibility that the presence of objective risk factors may moderate some of the relationships between social influences and perceptions of disease risk described above. As previously noted, objective risk factors are personal characteristics and behaviors that indisputably increase the risk of disease development. Some risk factors are uncontrollable, whereas others are largely preventable. Although not all women are aware of these risk factors (e.g., Peacey et al., 2006; Pohls et al., 2004; Steptoe et al., 2002), many are likely to understand their health-relevance to some extent. The knowledge that one has a greater disease risk due to objective risk factors can be distressing. For instance, women who have an increased risk for breast cancer due to their family history tend to experience more emotional distress and worry than do women

without a genetic predisposition to the disease (Coyne, Kruus, Racioppo, Calzone, & Armstrong, 2003). Distress about one's increased risk status may be even greater when the risk factors are controllable, as this may lead to feelings of guilt or disappointment.

The presence of objective risk factors may moderate the relationship between social influences and perceptions of disease risk through a process similar to cognitive dissonance (Festinger & Carlsmith, 1959). With cognitive dissonance, discomfort is experienced when a person has two simultaneous but conflicting cognitions (including beliefs, attitudes, values, or awareness of one's behaviors). This discomfort motivates the person to reduce the cognitive inconsistency, which can be remedied by altering one of the offending beliefs. In the context of health risk perceptions, people may recognize that they possess objective risk factors (particularly controllable risk factors) making them more susceptible to a disease, and then feel discomfort due to a conflicting cognition, such as the belief that people stigmatize those with the disease. To remedy this discomfort, people who possess objective risk factors may be less likely to acknowledge that they are at risk. Thus, the presence of objective risk factors may moderate the association between perceived stigma by others and perceptions of disease risk.

Objective risk factors may moderate the relationship between social exposure to disease and perceptions of disease risk in a similar manner. For people with increased disease risk due to objective factors, having greater social exposure to disease could be particularly distressing. To alleviate such distress, people may change their beliefs about what the increased prevalence of disease in their social networks means. Rather than perceive their risk as elevated, these people may be particularly motivated to believe that they are invulnerable to disease. These people may believe that since the disease has

already affected many of those around them, it is unlikely to directly impact them as well. Structural equation modeling was used to examine these hypothesized relationships.

Hypothesis 3.2: The presence of objective risk factors will moderate the relationship between perceptions of stigma by others and perceptions of disease risk (see Figures 3.2.1, 3.2.2, and 3.2.3 for conceptual models). For women with low levels of objective risk factors, perceptions of stigma by others is expected to be inversely associated with perceptions of disease risk. A different relationship (greater, opposite, or no association) between perceptions of stigma by others and perceptions of disease risk may be seen in those with higher levels of objective risk.

Hypothesis 3.3: The presence of objective risk factors will moderate the relationship between social exposure to disease and perceptions of disease risk (see Figures 3.3.1, 3.3.2, and 3.3.3 for conceptual models). While greater social exposure to disease is expected to be associated with greater perceptions of disease risk for those with low levels of objective risk factors, a different relationship (smaller, opposite, or no association) between social exposure to disease and perceptions of disease risk may be seen in those with higher levels of objective risk.

4. Is Identification with Traditional Gender Roles Associated with Perceptions of Disease Risk?

People frequently rely on heuristics to evaluate the risk of a health threat (Katapodi, Facione, Humphreys, & Dodd, 2005). Heuristics allow people to make decisions quickly and efficiently, however these cognitive shortcuts often involve the biased processing of information, which can lead to inaccurate conclusions (Tversky & Kahneman, 1974). One heuristic strategy is the representativeness heuristic, wherein

people estimate the probability that a target belongs to a larger category based on the extent to which the target possesses characteristics that are stereotypical of the category (Kahneman & Tversky, 1973). While attempting to determine the susceptibility of a target person to a disease, people may consider how similar the target is to their image of the stereotypical person afflicted with the disease. This process is illustrated in a study of women's perceptions of risk for breast cancer, heart disease, and osteoporosis (Gerend, Aiken, West et al., 2004). Women were asked to decide how similar they were to the typical woman who develops each of the three diseases. Greater perceptions of similarity to the typical woman afflicted with each disease were significantly correlated with higher perceptions of disease risk.

When imagining the stereotypical person most likely to be afflicted with a chronic disease, one characteristic that people may consider is gender. Cardiovascular disease and breast cancer are examples of gendered diseases; that is, people perceive each disease to be more common in either men or women. Specifically, cardiovascular disease is generally perceived as a "male" disease most likely to affect affluent, successful men whose personalities reflect the Type-A behavioral pattern (Aalto, Heijmans, Weinman, & Aro, 2005; Lockyer & Bury, 2002). Additionally, much of the information regarding the treatment and presentation of cardiovascular disease in women is extrapolated from what is known about men's experiences with the disease (Lockyer & Bury, 2002; Shumaker & Smith, 1995). While it is true that more men than women die from cardiovascular disease each year (341.7 versus 245.3 deaths per 100,000 in 2004), cardiovascular disease is still the leading cause of mortality among women (Rosamond et al., 2008). Nonetheless, many women fail to recognize their risk for cardiovascular disease,

believing instead that they are at greater risk for developing breast cancer (e.g., Christian et al., 2007). Breast cancer is widely, and accurately, perceived as a disease that primarily affects women. Male breast cancer accounts for only 1% of all cases of breast cancer (Giordano, Cohen, Buzdar, Perkins, & Hortobagyi, 2004). Although much of what is known about the management of male breast cancer is generalized from what is understood about women (Contractor, Kaur, Rodrigues, Kulkarni, & Singhal, 2008), men have a number of unique psychological and emotional experiences with the disease (e.g., Williams et al., 2003).

Faced with the task of estimating their own risk for cardiovascular disease or breast cancer, people may use the representativeness heuristic and consider how similar they are to the stereotypical person afflicted with each disease. The extent to which people self-identify as masculine or feminine may influence their perceptions of risk through this process, since gender is a salient characteristic of each disease. This section of the present study used structural equation modeling to examine hypothesized relationships between women's identification with traditional gender roles and their overall perceptions of risk for cardiovascular disease and breast cancer. Exploratory analyses were also conducted to investigate similar associations between traditional gender roles and perceptions of risk for lung cancer. To examine these relationships, participants' responses to the Personal Attributes Questionnaire (PAQ; Spence & Helmreich, 1978), which consists of two subscales measuring the extent to which one self-identifies with traditionally masculine and traditionally feminine gender roles, were associated with the latent variables representing perceptions of risk for cardiovascular

disease, breast cancer, and lung cancer (using the measurement models constructed in Part 1).

Hypothesis 4.1: Since cardiovascular disease is perceived to be a disease of men, identification with traditionally masculine gender roles will be positively associated with perceptions of cardiovascular disease risk (see Figure 4.1 for model).

Hypothesis 4.2: Similarly, since cardiovascular disease is perceived to be a disease of men, identification with traditionally feminine gender roles will be inversely associated with perceptions of cardiovascular disease risk (see Figure 4.2).

Hypothesis 4.3: Since breast cancer is perceived to be a disease of women, identification with traditionally feminine gender roles will be positively associated with perceptions of breast cancer risk (see Figure 4.3).

Hypothesis 4.4: Similarly, since breast cancer is perceived to be a disease of women, identification with traditionally masculine gender roles will be inversely associated with perceptions of breast cancer risk (see Figure 4.4).

Exploratory Analyses: Lung cancer is not regarded as a gendered disease. It is the second most commonly diagnosed cancer in both men and women (85.3 and 54.2 cases per 100,000, respectively), and the leading cause of cancer deaths in men and women (70.3 and 40.9 deaths per 100,000 respectively) (U.S. Cancer Statistics Working Group, 2007). Thus, there were no a priori hypotheses about the relationship between identification with traditional gender roles and perceptions of risk for lung cancer. Relationships between masculinity, femininity, and perceptions of lung cancer risk were examined with models similar to those in Hypotheses 4.1 thorough 4.4 (see Figures 4.5 and 4.6).

Age-related differences in relationships between identification with traditional gender roles and perceptions of disease risk were not expected. The models for cardiovascular disease, breast cancer, and lung cancer were examined separately in both younger and older women to determine if age does moderate these relationships.

Method

Participants and Procedure

Younger Women (Ages 18 to 25)

Participants were recruited from the Department of Psychology undergraduate subject pool at Stony Brook University. Eligible participants were female and between the ages of 18 and 25. Participants completed the study questionnaire in our research laboratory in groups of no more than four participants. Participants were provided with a consent letter (see Appendix A) describing the study's purpose as an investigation of "women's thoughts about a variety of topics including health issues and life events." After completing the 30-minute questionnaire, participants were read a short debriefing script and thanked for their involvement. All participants received course credit in exchange for their involvement in the study. A total of 458 participants were recruited into the study between March 2006 and December 2006.

Older Women (Ages 40 and Above)

Participants were identified with the help of students enrolled in various undergraduate Psychology courses. Students enrolled in these courses were provided with contact sheets containing a brief description of the study and a space for an interested woman over the age of 40 to provide her name, mailing address, and telephone number. For those participants who resided in the state of New York and had a telephone number in the 631 or 516 area codes, a maximum of three attempts were made to contact these participants by telephone. Researchers contacted these participants to confirm their mailing addresses and willingness to participate in the study. Telephone calls were made to reduce nonresponse error and to motivate participants to complete and return the study

questionnaire (Visser, Krosnick, & Lavrakas, 2000). After contacting them by telephone, researchers mailed each participant a packet including a cover letter (see Appendix B), consent letter (see Appendix C), copy of the study questionnaire, and a pre-addressed, postage-paid envelope in which to return the completed questionnaire. Packets were also mailed to recruited participants who could not be contacted by telephone (e.g., those who lived outside of the eligible calling area, those who could not be reached after three attempts, those who did not provide a telephone number). Return of the completed questionnaire by mail indicated the participant's consent. A total of 360 packets were mailed between October 2006 and December 2008, with 205 study questionnaires returned (57% response rate).

Measures

All participants completed questionnaires including both experimenter-designed and previously well-validated measures. Study questionnaires assessed outcome variables including measures of perceived risk for the self, perceived risk for the average woman, perceived prevalence rate, and perceived mortality rate for cardiovascular disease, breast cancer, and lung cancer, as well as preventive and screening health behaviors. Predictor variables were assessed with measures of individual traits, social influences, objective risk factors, beliefs about disease etiology, and sociodemographic characteristics.

Outcome Measures

Perceptions of disease risk for the self. Three items were used to assess the extent to which participants felt they were at risk for cardiovascular disease, breast cancer, and lung cancer. Each participant estimated her "chance of developing [specific disease]"

during her lifetime on a 5-point scale ranging from 0 (no chance) to 4 (very high chance). Items similar in phrasing and response-scale format have been used in a number of studies, including the biennial National Cancer Institute Health Information National Trends Survey (HINTS; Nelson et al., 2004). Perceptions of future disease risk were of primary interest in the present study; therefore, a response-option was also provided which allowed participants to indicate whether they had been previously diagnosed with the disease.

Perceptions of disease risk for the average woman. Three items assessed participants' perceptions of disease risk for the average woman. Each participant estimated "the average woman's chance of developing [specific disease] during her lifetime" on a scale ranging from 0 (no chance) to 4 (very high chance).

Perceived prevalence rate of disease. Participants estimated how many women out of 100 would develop cardiovascular disease, breast cancer, and lung cancer (3 items) during their lifetime.

Perceived mortality rate of disease. Participants estimated how many women out of 100 would die from cardiovascular disease, breast cancer, and lung cancer (3 items) during their lifetime.

Preventive and screening health behaviors. Items based on a measure of prenatal health behaviors were used to assess preventive behaviors (DeLuca & Lobel, 1995). These items assessed how frequently in the past month participants exercised for at least 15 minutes, smoked cigarettes, drank alcohol, used vitamins, monitored their calorie intake, and consumed a balanced diet. Responses were made on a scale ranging from 0 (never) to 4 (very often). Open-ended questions were used to measure screening

behaviors; participants reported how many times in the past year they performed a breast self-exam, had a mammogram, and received a check-up or physical from a doctor. Preventive and screening health behaviors are typically assessed with self-report measures, and such measures demonstrate adequate reliability and validity. Self-reported levels of behaviors including exercise, alcohol consumption, vitamin use, and mammography yield results comparable to data obtained from physiological measures and medical records (Caplan et al., 2003; Del Boca & Darkes, 2003; King, Rimer, Trock, Balshem, & Engstrom, 1990; Satia-Abouta et al., 2003; Timperio, Salmon, & Crawford, 2003).

Measures of Predictor Variables: Individual Traits

Optimism. Dispositional optimism was assessed with the 12-item Life Orientation Test (LOT; Scheier & Carver, 1985). Sample items include "in uncertain times, I usually expect the best" and "I hardly ever expect things to go my way" (reverse-scored). Items are rated on a 5-point scale ranging from "strongly disagree" to "strongly agree" and summed, with higher scores indicating greater levels of optimism. Optimism is stable over time (test-retest reliability = .79), and the LOT has been shown to have good internal consistency (α = .76). In the present study, the internal consistency of this measure was high, α = .83 among younger women and α = .84 among older women.

Health locus of control. The 18-item Multidimensional Health Locus of Control (MHLC) Scale – Form B (Wallston et al., 1978) assesses people's general expectancies about whether they have control over their health status. The MHLC scale consists of three internally- or externally-oriented subscales: Internal (sample item: "if I become sick, I have the power to make myself well again"), Chance (sample item: "when I become ill,

it's a matter of fate"), and Powerful Others (sample item: "other people play a big part in whether I stay healthy or become sick"). Items are rated on a scale ranging from 1 (strongly disagree) to 6 (strongly agree), and then summed to create scores for each of the subscales ranging from 6 to 36. The Internal (α = .71), Chance (α = .69), and Powerful Others (α = .72) subscales each demonstrate good internal consistency, and have test-retest reliabilities ranging from .70 to .80 (Wallston, 2004). However, the internal consistency of the Internal (α = .61 for the younger subsample, α = .57 for the older subsample), Chance (α = .62 for the younger subsample, α = .63 for the older subsample), and Powerful Others (α = .53 for both subsamples) subscales was low in the present study.

Identification with traditional gender roles. The 24-item Personal Attributes Questionnaire (PAQ; Spence & Helmreich, 1978) was used to assess the extent to which participants identify with traditional gender roles of femininity and masculinity. This scale has an 8-item Femininity subscale, composed of expressive traits which are both socially desirable and stereotypically female, and an 8-item Masculinity subscale, composed of instrumental traits which are socially-desirable and stereotypically male. Each item includes a pair of opposing trait descriptions (e.g., "not at all understanding of others" and "very understanding of others"), which are evaluated on a 5-point scale. Scores on each scale are summed, and can range from 0 to 32. Among samples of women of varying ages, the PAQ subscales demonstrate good internal consistency; Femininity α = .71 to .77 and Masculinity α = .73 to .79 (Helmreich, Spence, & Wilhelm, 1981). In the present study, the internal consistency of the Femininity subscale was α = .70 for younger women and α = .77 for older women, and of the Masculinity subscale was α = .72 for younger women and α = .71 for older women.

Measures of Predictor Variables: Social Influences

Social exposure to disease. To assess the presence of chronic disease in each participant's social network, participants were asked to "list all of the women you personally know who have developed [specific disease]." Participants identified each woman in general terms (e.g., initials or relationship such as mother or friend) to maintain anonymity. The number of women listed for each disease was summed to create scores representing the extent of social exposure to cardiovascular disease, breast cancer, and lung cancer.

Perceptions of stigma by others. To assess whether participants believed that other people stigmatize those with chronic diseases, participants indicated on 3 items the extent to which they perceived that "people have negative views or attitudes toward women" with cardiovascular disease, breast cancer, and lung cancer on a scale ranging from 0 (people have no negative views) to 3 (people have many negative views).

Measures of Predictor Variables: Objective Risk Factors

Family history of disease. Participants responded to three items assessing whether they had a family history of cardiovascular disease, breast cancer, and lung cancer (yes/no format).

Age. Participants reported how old they were on their last birthday. Participants were categorized as "younger women" if their ages were between 18 and 25 years, and as "older women" if their ages were 40 years or older. All of the participants categorized as "younger women" were recruited from the Department of Psychology undergraduate subject pool at Stony Brook University, and all of the participants categorized as "older women" completed mailed questionnaires.

Body mass index. Body mass index (BMI) is calculated from a person's height and weight, with the resulting score used to categorize people's body composition as underweight, normal, overweight, and obese. Participants reported their height and weight, and their responses were converted to inches and pounds, respectively. These values were then used to calculate each woman's BMI using a program provided by the National Heart, Lung, and Blood Institute (available online at: http://www.nhlbisupport.com/bmi/bmicalc.htm).

Additional Study Variables

Beliefs about disease etiology. Beliefs about the role of health behaviors in disease etiology were assessed with 3 items asking to what extent participants thought that "general health behaviors such as diet, exercise, and smoking" contribute to women's chances of developing cardiovascular disease, breast cancer, and lung cancer. Response options ranged from 0 (no contribution) to 4 (very high contribution).

Sociodemographic characteristics. Participants provided information regarding their race or ethnicity (African-American or Black, Asian or Pacific Islander, Latino or Hispanic, Native American, White or European American, or multiethnic), the total annual income of everyone living in their home (ranging from less than $10,000 to more than $70,000), and their highest level of education (ranging from some high school to Ph.D. or M.D. degree).

Results

Preliminary Analyses

Data Preparation

Descriptive statistics including means, standard deviations, and frequencies were calculated for all study variables. Each variable was examined for missing values. In instances where missing data were minimal and appeared to be random, missing values were replaced using mean imputation. When individual items for scale measures (e.g., LOT, PAQ) were missing at random, the participant's mean score on that scale or subscale was substituted for the missing value. A total of 0.06% and 0.41% of the data were missing at random and were therefore replaced for younger and older participants, respectively. Data were also screened for violations of assumptions of statistical normality. When violations were found, cases with extreme outliers were removed. Data transformations (e.g., square root transformation, log transformation) were conducted on nonnormal variables as needed. Several unique problems with normality were found in items assessing the frequency of screening behaviors. In the subsample of younger women, 2 participants' responses on the measure of breast self-exams were extreme outliers; these cases were removed. To reduce the impact of 5 additional outliers on this variable, these scores were recoded as one unit greater than the highest score in the normal distribution. A similar procedure was utilized for this variable in the subsample of older women, and on the measure of frequency of physicals or check-ups from a doctor in both subsamples.

Since the current study is primarily concerned with perceptions of future disease risk, data from any participant who had been previously diagnosed with cardiovascular

disease, breast cancer, or lung cancer were removed from the dataset. A total of 15 cases were removed from the subsample of older women due to previous disease diagnoses; data from an additional 7 participants were removed due to missing responses on multiple measures of disease risk perceptions. Due to minor modifications to the study questionnaire during the course of data collection, 78 younger participants and 1 older participant did not provide data for the PAQ. In an effort to maximize use of the available data, such participants were not removed from the dataset. Instead, these participants' data were restricted to certain analyses, resulting in small variations in the sample size for different study questions. Disregarding differences due to the PAQ measure, complete data for 454 younger women and 180 older women were available for analyses. Bivariate correlations between study variables were also calculated. Variable means, standard deviations, bivariate correlations, and additional details about data transformations are provided in Tables 1 (younger participants) and 2 (older participants). Demographic information for study participants is provided in Table 3.

Preparation for and Review of Structural Equation Modeling

The majority of analyses were conducted with structural equation modeling, with all analyses performed with the AMOS 17.0 computer software (Arbuckle, 2008) using maximum likelihood estimation. With structural equation modeling, it is possible to examine multiple regression equations simultaneously. Hypothesized path models including both observed (measured) and unobserved (latent) variables can be tested statistically to determine whether the proposed model is a good fit to the sample data (Byrne, 2001). As noted, structural equation modeling allows for the examination of observed variables, which are variables that can be directly measured, as well as latent

variables, which cannot be measured directly. Latent variables are often theoretical constructs that can be defined, or indicated, by a set of measured values. As an initial step, the structure and goodness of fit of latent variables must be confirmed with the construction of a measurement model (the objectives of Hypotheses 1.1, 1.2, 1.3, 2.1, and 2.2). Once the latent variables in a hypothesized model have been constructed, the observed variables and all hypothesized paths can be added to the model.

The fit of a model to the sample data is determined by examining goodness of fit indices. The present study used goodness of fit indices including the Chi-square test, the CFI (Comparative Fit Index), and the RMSEA (Root Mean Square Error of Approximation). With the Chi-square test, a nonsignificant χ^2 statistic indicates that the data do not significantly depart from the model. However, the Chi-square test overestimates model fit with large sample sizes. For this reason, the CFI and RMSEA are also frequently examined as fit indices. The CFI takes sample size into account, and is derived from the comparison of the hypothesized model with an independence model (i.e., a highly restricted model in which all correlations among the variables are zero). CFI values range from 0 to 1, with values greater than .90 indicating good model fit. The RMSEA takes the degrees of freedom in the hypothesized model into account, and is therefore sensitive to the complexity of the model. RMSEA values range from 0 to 1, with values less than .10 indicating good model fit. Data for the 90% confidence intervals around the RMSEA value are also available, providing information about the precision of this statistic. A narrow confidence interval with the lower limit including 0 and the upper limit below .10 indicates good precision (Byrne, 2001).

Several assumptions must be met for the appropriate application of structural equation modeling, particularly concerning sample size and multivariate normality. A minimum of 5 cases per estimated parameter is suggested as an adequate sample size for structural equation modeling (Bentler & Chou, 1987). This assumption was met for all analyses in the current study; however, results of analyses involving fewer than 100 participants may still be underpowered and should be interpreted cautiously. Violations of the assumptions of normality and the presence of multivariate outliers can affect model fit and lead to inaccurate parameter estimates. Mardia's coefficient of multivariate kurtosis and its critical ratio can reveal violations of the assumptions of normality, with a critical ratio value greater than 2.00 indicating that the data are nonnormal. In cases where the data were found to be multivariate nonnormal, data from specific nonnormal variables were transformed. Large Mahalanobis *d*-squared values were also used to identify individual cases that were multivariate outliers. When removing a limited number of multivariate outliers did not result in normally-distributed data, bootstrapping with maximum likelihood estimation was utilized. Bootstrapping is recommended as a technique for testing models in situations where basic assumptions are not met; specifically if the sample size is moderate (but not excessively small), or the data are nonnormal (Byrne, 2001; Yung & Bentler, 1994). When data are multivariate nonnormal, maximum likelihood estimation can result in biased model estimates. Bootstrapping, which involves the comparison of parameter estimates over repeated samples that have been drawn with replacement from the original data sample, can be used to test whether these maximum likelihood estimates are biased to a meaningful extent (Byrne, 2001).

Once the initial fit indices for a given model were calculated, additional statistics were examined to determine if the model was misspecified or if the fit could be improved. The standardized residuals and modification indices of the model were examined to determine if there were conceptually appropriate changes that could be made to the model that would improve its fit to the data. Such changes can involve the correlation of residuals (error terms) thereby indicating significant associations between variables in the model, the addition of a path, or the removal of a nonsignificant path. Specifically, standardized residual values greater than 2.58 and large modification index values can indicate areas of potential change within the model. Appropriate modifications were considered and added to the tested models as needed.

Hypothesis Testing

1. Constructing a Model of Perceptions of Disease Risk

A. Measurement Model Design

Perceptions of risk for cardiovascular disease. As noted in Hypothesis 1.1, people's estimates of their personal risk for cardiovascular disease, the average woman's risk for cardiovascular disease, the prevalence rate of cardiovascular disease, and the mortality rate of cardiovascular disease were expected to be reliable indicators of a latent variable representing perceptions of risk for cardiovascular disease. Bivariate correlations (see Tables 1 and 2) confirmed that these items were significantly correlated with one another in both younger and older participants. In both samples, an examination of the descriptive statistics revealed that the standard deviations of the items assessing the prevalence and mortality rates of cardiovascular disease were substantially larger than those of the items assessing personal disease risk and the average woman's disease risk.

Responses to items assessing disease prevalence and mortality were standardized by dividing all scores by a constant to produce similar variances for all 4 indicator items (e.g., for younger participants, all estimates of the prevalence rate of cardiovascular disease were divided by 36).

In the subsample of younger women, the hypothesized measurement model shown in Figure 1.1 was a poor fit to the data ($\chi^2(2) = 28.58$, $p < .001$; CFI = .96; RMSEA = .17, 90% confidence limits for the RMSEA = .12 to .23). However, the data were multivariate nonnormal (Mardia's coefficient = 12.68). After the removal of 30 cases found to be multivariate outliers (6.6% of the subsample data), and addition of a path correlating residuals of items assessing perceptions of personal risk for cardiovascular disease and the average woman's risk for cardiovascular disease, the measurement model (see Figure 1.1a) was found to be a good fit to the data ($\chi^2(1) = 2.13$, $p = .15$; CFI = 1.00; RMSEA = .05, 90% confidence limits for the RMSEA = .00 to .15).[1] Exploratory analyses examined whether there were differences in the extent to which each indictor variable contributed to the latent variable of perceptions of risk for cardiovascular disease. That is, a series of Chi-square difference tests were conducted to determine whether path $a_1 = b_1 = c_1 = d_1$ (see Figure 1.1). A set of constrained models (for example, where all paths were constrained to be equal, where only path a_1 was constrained to be equal to path b_1, etc.) were tested, and each was compared to an unconstrained model in which each of the 4 paths were allowed to freely vary. The Chi-square value and degrees of freedom for the unconstrained model was then subtracted from the Chi-square value and degrees of freedom for each constrained model. The

[1] Although 6.6% of the sample were removed as multivariate outliers, differences between the standardized path coefficients for each indicator variable in the nonnormal ($n = 454$) and normal ($n = 424$) models differed only minimally (each $\beta \leq .05$).

significance of the resulting Chi-square difference value allows for the determination of the appropriateness of the constrained model; a nonsignificant Chi-square value indicates that the constrained model is not a worse fit to the data than the unconstrained model. Constrained models in which paths $a_1 = b_1$ and paths $c_1 = d_1$ provided a fit to the data similar to that of the unconstrained model. Thus, the magnitude of the paths for perceptions of personal risk and the average woman's risk for cardiovascular disease were not significantly different among younger women, nor were the magnitudes of the paths for perceptions of disease prevalence and mortality rates significantly different.[2]

Among older women, the hypothesized measurement model was found to be a good fit to the data ($\chi^2(2) = 2.46$, $p = .29$; CFI = 1.00; RMSEA = .04, 90% confidence limits for the RMSEA = .00 to .16), as shown in Figure 1.1b. Exploratory analyses examining differences in the contribution of each indicator variable to the latent variable revealed that, as for younger women, the magnitude of the paths for perceptions of personal risk and the average woman's risk for cardiovascular disease were not significantly different from one another, nor were the magnitudes of the paths for perceptions of disease prevalence and mortality rates.

The final well-fitting measurement models were also tested to determine whether these models were invariant across groups. That is, each path (a_1, b_1, c_1, and d_1 in Figure 1.1) was tested to see if its value was equal for both younger and older participants. To test for model invariance, each parameter of interest (in this case, the path coefficients for a_1, b_1, c_1, and d_1) was constrained to be equal across the two groups, and the resulting Chi-square value and degrees of freedom were subtracted from the Chi-square value and

[2] The differences in path values revealed by tests of path equivalence and model invariance (discussed in subsequent pages) are best seen in the unstandardized, rather than standardized (β), path coefficients. For this reason, both unstandardized and standardized path coefficients are included in figures for this section.

degrees of freedom for an unconstrained model in which these parameters were allowed to freely vary. A significant Chi-square difference value indicates that some constrained parameter is not equal across groups. Each parameter of interest is then systematically constrained to be equal across the two groups, and the calculation of Chi-square difference values reveals whether each parameter is invariant across groups (a nonsignificant Chi-square difference value), or whether the parameter's value differs across groups (a significant Chi-square difference value). Tests of model invariance revealed that all 4 paths of interest were invariant across groups. That is, the path coefficients for the indicator variables did not differ significantly between younger and older women.

Perceptions of risk for breast cancer. Hypothesis 1.2 predicts that people's estimates of their personal risk for breast cancer, the average woman's risk for breast cancer, the prevalence rate of breast cancer, and the mortality rate of breast cancer would all be reliable indicators of a latent variable representing perceptions of risk for breast cancer (see Figure 1.2). These items were significantly correlated with one another in both younger and older participants (see Tables 1 and 2). In both subsamples, the variances of the items assessing the perceived prevalence and mortality rates of breast cancer were found to be substantially larger than those of the items assessing personal risk and the average woman's risk; the former items were standardized to produce comparable variances for all 4 items.

Among younger women, the hypothesized measurement model was a poor fit to the data ($\chi^2(2) = 40.65$, $p < .001$; CFI = .94; RMSEA = .21, 90% confidence limits for the RMSEA = .15 to .26). The data were found to be multivariate nonnormal (Mardia's

coefficient = 4.43), therefore 4 outlier cases (0.9% of the data) were removed from the subsample. Additionally, modification indices supported adding the correlation of the residual for perceptions of personal risk for breast cancer with the residual for perceptions of the average woman's risk for breast cancer. The final model (see Figure 1.2a) was an excellent fit to the data ($\chi^2(1) = 0.07$, $p = .79$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .08). Exploratory analyses examined whether there were differences in the extent to which each indictor variable contributed to the latent variable of perceptions of risk for breast cancer. Contrasts between constrained models and the unconstrained model revealed that the magnitudes of the paths between the latent variable and perceptions of personal disease risk and perceptions of the average woman's disease risk were not significantly different, and the magnitudes of the paths between the latent variable and perceptions of disease prevalence and mortality rates were not significantly different.

In the subsample of older women, the hypothesized measurement model was initially a poor fit to the data ($\chi^2(2) = 41.69$, $p < .001$; CFI = .86; RMSEA = .33, 90% confidence limits for the RMSEA = .25 to .43). The data in this subsample were multivariate nonnormal (Mardia's coefficient = 4.30), thus necessitating the removal of 5 outlier cases (2.8% of the data). Although modification indices suggested adding the correlation of the residual for perceptions of personal disease risk with the residual for perceptions of the average woman's disease risk, such a modification produced a Heywood case (the situation in which an inadmissible solution is reached due to the estimation of a standardized path coefficient as greater than 1.00 and a residual variance as negative) for the path between the latent variable and perceptions of the prevalence

rate of breast cancer. Therefore, a model in which the residual for perceptions of the prevalence rate of breast cancer and the residual for perceptions of the mortality rate of breast cancer were correlated was tested instead (see Figure 1.2b), which produced an admissible solution and a well-fitting model ($\chi^2(1) = 0.17$, $p = .68$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .15). Results of the exploratory analyses indicated that the magnitudes of the paths for perceptions of personal disease risk, the average woman's disease risk, and the prevalence rate of the disease were not significantly different.

Tests of model invariance were also conducted to determine whether paths a_2, b_2, c_2, and d_2 (see Figure 1.2) were invariant across younger and older participants. These tests revealed that each path's value was significantly different across groups. While path a_2 (unstandardized coefficient = 0.15 for younger women, 0.38 for older women) and path b_2 (unstandardized coefficient = 0.24 for younger women, 0.41 for older women) had a weaker association with the latent variable in younger compared to older women, path c_2 (unstandardized coefficient = 0.47 for younger women, 0.25 for older women) and path d_2 (unstandardized coefficient = 0.45 for younger women, 0.20 for older women) had a stronger association with the latent variable in younger compared to older women.

Perceptions of risk for lung cancer. In line with Hypothesis 1.3, people's perceptions of personal risk for lung cancer, the average woman's risk for lung cancer, the prevalence rate of lung cancer, and the mortality rate of lung cancer were expected to share common error variance and thus be reliable indicators of a latent variable representing perceptions of risk for lung cancer (see Figure 1.3). Bivariate correlations confirmed that these items were significantly associated with one another in both younger

and older women (see Tables 1 and 2). Again, in both subsamples the items assessing the prevalence and mortality rates of disease were standardized because the variances were substantially larger than those of the items assessing personal disease risk and the average woman's disease risk.

Among younger women, the hypothesized measurement model was initially a poor fit to the data ($\chi^2(2) = 34.84$, $p < .001$; CFI = .96; RMSEA = .19, 90% confidence limits for the RMSEA = .14 to .25). However, the data were nonnormal (Mardia's coefficient = 7.21). After the removal of 5 outlier cases (1.1% of the data) and addition of the correlation of the residual for perceptions of personal risk for lung cancer with the residual for perceptions of the average woman's risk for lung cancer, the model (see Figure 1.3a) was a good fit to the data ($\chi^2(1) = 0.73$, $p = .39$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .12). Exploratory analyses examining differences in the contribution of each indicator variable to the latent variable revealed that the magnitude of the paths for perceptions of personal disease risk and the average woman's disease risk were not significantly different from one another.

In the subsample of older women, the hypothesized measurement model was a poor fit to the data ($\chi^2(2) = 28.23$, $p < .001$; CFI = .93; RMSEA = .27, 90% confidence limits for the RMSEA = .19 to .36). Modification indices suggested the addition of the correlation of the residual for perceptions of personal risk for lung cancer with the residual for perceptions of the average woman's risk for lung cancer. With this change, the model (see Figure 1.3b) was a good fit to the data ($\chi^2(1) = 1.08$, $p = .30$; CFI = 1.00; RMSEA = .02, 90% confidence limits for the RMSEA = .00 to .20). Exploratory analyses contrasting the unconstrained model with various constrained models indicated

that the paths between the latent variable and perceptions of personal risk for lung cancer and the average woman's risk for lung cancer were not significantly different.

Tests of model invariance were conducted to determine whether paths a_3, b_3, c_3, and d_3 (see Figure 1.3) were invariant across younger and older participants. However, attempts to constrain the model for these tests produced Heywood cases, thereby undermining the validity of these results. Although it appears that only path c_3 differed significantly between the subsamples (unstandardized coefficient = 0.52 for younger women, 1.00 for older women), this finding should be interpreted cautiously.

As a final step, the equivalence across diseases of the paths from a given indicator variable to each latent variable representing perceptions of disease risk was evaluated. That is, tests were conducted separately in younger and older participants to determine whether path $a_1 = a_2 = a_3$, $b_1 = b_2 = b_3$, $c_1 = c_2 = c_3$, and $d_1 = d_2 = d_3$. These tests involved the comparison of constrained and unconstrained models to produce Chi-square difference values in a manner similar to that described above. Among younger participants, models in which path $a_1 = a_2 = a_3$, $b_1 = b_2 = b_3$, and $c_1 = c_2 = c_3$ fit as well as the model in which these paths were allowed to freely vary. Thus, there is similarity in the extent to which perceptions of personal disease risk, the average woman's disease risk, and the prevalence rate of a disease are associated with the latent variables representing perceptions of risk for cardiovascular disease, breast cancer, and lung cancer. Among older participants, all of the constrained models produced a worse fit than the unconstrained model. Therefore, significant differences exist in the extent to which perceptions of personal disease risk, the average woman's disease risk, the prevalence

rate of a disease, and the mortality rate of disease are associated with the latent variables representing perceptions of risk for cardiovascular disease, breast cancer, and lung cancer.

B. Comparison of Higher-Order Models of Perceptions of Disease Risk

Hypotheses 1.4 and 1.5 address the comparison of two higher-order models of perceptions of disease risk. In the Disease-Specific Risk Model, people's perceptions of risk for a given disease are expected to be independent of their perceptions of risk for other diseases. Thus, with this model, no associations should exist between the latent variables representing perceptions of risk for cardiovascular disease, breast cancer, and lung cancer. Conversely, in the General Disease Risk Model, people's perceptions of risk for different diseases are expected to be related. A higher-order factor representing general beliefs about risk would underlie the latent variables representing perceptions of disease risk. Each of these models was tested separately in younger and older study participants.

Comparison of models in younger women. In the subsample of younger participants, all cases that were multivariate outliers in the construction of the measurement models for perceptions of risk for cardiovascular disease, breast cancer, and lung cancer were first removed, resulting in a subsample of 419 participants. The Disease-Specific Risk Model (see Figure 1.4) was a poor fit to the data ($\chi^2(51) = 655.43$, $p < .001$; CFI = .78; RMSEA = .17, 90% confidence limits for the RMSEA = .16 to .18). The data were still multivariate nonnormal (Mardia's coefficient = 7.51). After the removal of 45 cases (10.0% of the original subsample of 454 participants), the data were normally distributed (Mardia's coefficient = 2.00). However, the Disease-Specific Risk Model (see Figure 1.4a) remained a poor fit to the data ($\chi^2(51) = 668.63$, $p < .001$; CFI

= .77; RMSEA = .18, 90% confidence limits for the RMSEA = .17 to .19).[3] Since the objective of these analyses was to compare two hypothesized models, modification indices were not examined at this stage.

To test the General Disease Risk Model (see Figure 1.5) in the younger participants, the normally-distributed subsample of 374 participants was also used. This model was a poor fit to the data ($\chi^2(48) = 342.97$, $p < .001$; CFI = .89; RMSEA = .13, 90% confidence limits for the RMSEA = .12 to .14). However, consistent with Hypothesis 1.5, the fit indices revealed that the General Disease Risk Model provided a better fit to the data than did the Disease-Specific Risk Model. Efforts were therefore made to improve the fit of the General Disease Risk Model in the subsample of younger participants. Based on the modification indices, theoretically appropriate paths were added, including: 1) correlations of the residuals for perceptions of the mortality rates for disease, and 2) correlations of the residuals for perceptions of personal risks for disease. This model (see Figure 1.5a) was a good fit to the data ($\chi^2(42) = 141.88$, $p < .001$; CFI = .96; RMSEA = .08, 90% confidence limits for the RMSEA = .07 to .10).

Exploratory analyses examined whether there were differences in the extent to which each latent variable representing perceptions of risk for cardiovascular disease, breast cancer, and lung cancer contributed to the latent variable representing general beliefs about risk in younger women. That is, a series of tests were conducted to determine whether path e = f = g (see Figure 1.5). A model in which path f was constrained to be equal to path g fit as well as an unconstrained model. Thus, there is evidence that the latent variables representing perceptions of risk for breast cancer and

[3] Although 10.0% of the original sample were removed as multivariate outliers, the standardized path coefficients for each indicator variable in the nonnormal ($n = 419$) and normal ($n = 374$) models differed only minimally (each $\beta \leq .08$).

perceptions of risk for lung cancer have similarly strong associations with the latent variable representing general beliefs about risk in younger participants. Younger women may interpret the risks for breast cancer and lung cancer in similar ways.

Comparison of models in older women. In the subsample of older participants, all cases that were multivariate outliers in the construction of the measurement models for perceptions of risk for cardiovascular disease, breast cancer, and lung cancer were first removed, resulting in a subsample of 175 participants. The Disease-Specific Model was a poor fit to the data ($\chi^2(52) = 291.11$, $p < .001$; CFI = .77; RMSEA = .16, 90% confidence limits for the RMSEA = .15 to .18). The data were found to be multivariate nonnormal (Mardia's coefficient = 3.94). After the removal of 5 cases (5.6% of the original subsample of 180 participants), the data were normally distributed (Mardia's coefficient = 1.88). The model (see Figure 1.4b) remained a poor fit to the data ($\chi^2(52) = 294.99$, $p < .001$; CFI = .76; RMSEA = .17, 90% confidence limits for the RMSEA = .15 to .19).[4]

In the subsample of older participants, the normally-distributed subsample of 170 participants was used to test the General Disease Risk Model (see Figure 1.5). This model was also a poor fit to the data ($\chi^2(49) = 174.75$, $p < .001$; CFI = .88; RMSEA = .12, 90% confidence limits for the RMSEA = .10 to .14). However, consistent with Hypothesis 1.5, the fit indices revealed that the General Disease Risk Model provided a better fit to the data than did the Disease-Specific Risk Model. Efforts were therefore made to improve the fit of the General Disease Risk Model in this subsample. Based on the significance of model paths and the modification indices, theoretically appropriate

[4] Although 5.6% of the original sample were removed as multivariate outliers, standardized path coefficients for each indicator variable in the nonnormal ($n = 175$) and normal ($n = 170$) models differed only minimally (each $\beta \leq .06$).

changes were made, including: 1) removing a nonsignificant correlation between the residual for perceptions of the prevalence rate of breast cancer and the residual for the mortality rate of breast cancer; 2) correlating the residual for perceptions of personal risk for breast cancer with the residual for perceptions of the average woman's risk for breast cancer; 3) correlating the residuals for perceptions of the mortality rates for disease with one another; and 4) correlating some residuals for perceptions of personal risk for disease with one another. These changes produced a model (see Figure 1.5b) that was a good fit to the data ($\chi^2(44) = 78.14$, $p = .001$; CFI = .97; RMSEA = .07, 90% confidence limits for the RMSEA = .04 to .09).

Exploratory analyses examined whether there were differences in the extent to which each latent variable representing perceptions of risk for cardiovascular disease, breast cancer, and lung cancer contributed to the latent variable representing general beliefs about risk. These tests (see Figure 1.5) revealed that the three paths were not equivalent in older participants. Variation exists in older women's perceptions of susceptibility to cardiovascular disease, breast cancer, and lung cancer.

Testing the invariance of the General Disease Risk Model across younger and older women. Parameters of interest in the General Disease Risk Model (specifically, paths e, f, and g in Figure 1.5) were tested to see if their values were equivalent for both younger and older participants. Tests of model invariance revealed that the magnitude of the path between the latent variable representing perceptions of risk for cardiovascular disease and the latent variable representing general beliefs about risk (path e) did not differ significantly in younger and older participants. Similarly, the path between the latent variable representing perceptions of risk for breast cancer and the latent variable

representing general beliefs about risk (path f) did not differ significantly between groups. However, the path between the latent variable representing perceptions of risk for lung cancer and the latent variable representing general beliefs about risk (path g) did differ between younger (unstandardized coefficient = 0.36) and older (unstandardized coefficient = 0.79) participants. Thus, general perceptions of risk are more influential in older women's perceptions of risk for lung cancer than in younger women's.

C. Descriptive Information about Perceptions of Disease Risk

Trends in participants' responses to the 4 risk-related items (perceptions of disease risk for the self, perceptions of disease risk for the average woman, perceived prevalence rate of disease, and perceived mortality rate of disease) were examined. Contrasts focused on differences in responses based on disease (cardiovascular disease, breast cancer, or lung cancer), subsample (younger or older women), and item target (self or average woman). Exploratory contrasts also examined whether race or ethnicity may be associated with perceptions of disease risk. Due to the composition of the subsamples, the relationship between ethnicity and perceptions of risk could only be examined among younger women. For contrasts based on disease, subsample, and race or ethnicity, post-hoc analyses were corrected using Tukey's HSD test.

Risk perceptions by disease. The following results are summarized in Table 4. Consistent with Hypothesis 1.7, significant differences existed in younger women's perceptions of disease risk for the self, $F(2, 906) = 30.46$, $p < .001$. Specifically, younger women perceived they were at greatest risk for breast cancer, followed by cardiovascular disease, and finally lung cancer. A similar pattern emerged for younger women's perceptions of disease risk for the average woman, $F(2, 906) = 69.66$, $p < .001$. Whereas

younger women perceived the average woman to be at equal risk for cardiovascular disease and breast cancer, they perceived her to be at significantly less risk for lung cancer. Younger women also estimated that the greatest number of women would develop breast cancer, followed by cardiovascular disease, and then lung cancer, $F(2, 906) = 54.22$, $p < .001$. Younger women estimated that an equal number of women would die from cardiovascular disease and breast cancer, yet significantly fewer women would die from lung cancer, $F(2, 906) = 5.94$, $p = .003$.

Results for older women's perceptions of disease risk for the self were consistent with Hypothesis 1.6; older women perceived they were at greatest risk for cardiovascular disease, followed by breast cancer, and then lung cancer, $F(2,358) = 14.60$, $p < .001$. An identical pattern emerged in their perceptions of cardiovascular disease, breast cancer, and lung cancer risk for the average woman, $F(2, 358) = 73.79$, $p < .001$. Similarly, older women estimated that the greatest number of women would develop and die from cardiovascular disease, followed by breast cancer, and finally lung cancer, $F(2, 358) = 113.34$, $p < .001$ and $F(2, 358) = 59.80$, $p < .001$, respectively.

Risk perceptions by subsample. As shown in Table 5, significant differences existed between a number of younger and older women's perceptions of disease risk. Consistent with Hypothesis 1.8, older women had significantly greater perceptions of cardiovascular disease risk for the self and for the average woman, as well as higher estimates of the prevalence and mortality rates of cardiovascular disease, than did younger women (all ps $\leq .001$). Whereas older and younger women did not differ in their perceived risk for the self, perceived risk for the average woman, or perceived mortality rate for breast cancer, younger women did estimate that a greater number of women

would develop breast cancer than did older women ($p = .05$). Finally, younger and older women did not differ in their perceptions of lung cancer risk for the self or in their estimates of the mortality rate of lung cancer. However, younger women perceived the average woman to be at greater risk for lung cancer than did older women ($p = .03$), and younger women estimated that a greater number of women would develop lung cancer than did older women ($p < .001$).

Risk perceptions by target. Analyses were also conducted to determine whether there was an optimistic bias in people's perceptions of disease risk for the self compared to their perceptions of risk for the average woman (see Table 6). Younger women consistently demonstrated an optimistic bias in their perceptions of disease risk, perceiving their personal risk to be lower than the average woman for cardiovascular disease ($p < .001$, $\eta^2 = .26$), breast cancer ($p < .001$, $\eta^2 = .17$), and lung cancer ($p < .001$, $\eta^2 = .16$). This effect in the context of lung cancer became even stronger when the frequency of cigarette smoking, a well-known risk factor for the disease, was entered as a covariate ($\eta^2 = .30$). Older women demonstrated a similar optimistic bias in their risk perceptions, perceiving their personal risk to be lower than the average woman for cardiovascular disease ($p < .001$, $\eta^2 = .15$), breast cancer ($p < .001$, $\eta^2 = .19$), and lung cancer ($p = .004$, $\eta^2 = .05$). Again, this effect in the context of lung cancer became stronger when the frequency of cigarette smoking was entered as a covariate ($\eta^2 = .11$). It is noteworthy that while both younger and older women displayed an optimistic bias of similar magnitude in the context of breast cancer ($\eta^2 = .17$ and .19, respectively), younger women displayed a greater optimistic bias than older women in their perceptions of

cardiovascular disease risk (η^2 = .26 versus .15) and lung cancer risk (controlled for cigarette smoking; η^2 = .30 versus .11).

Risk perceptions by race or ethnicity. Because differences exist in the prevalence and mortality rates of specific diseases among women of varying ethnicity (e.g., Rosamond et al., 2008; U.S. Cancer Statistics Working Group, 2007), levels of perceived disease risk were compared across younger women of different racial and ethnic backgrounds. Contrasts were conducted to determine whether participants' responses to the risk items varied by their ethnicity, with post-hoc analyses differentiating between women who were African-American or Black only, Asian or Pacific Islander only, Latino or Hispanic only, White or European American only, or multiethnic.[5] Women's perceptions of their personal risk, the average woman's risk, the prevalence rate, and the mortality rate of cardiovascular disease did not differ based on their ethnicity (all *p*s ≥ .06). Similarly, women's perceptions of their personal risk, the prevalence rate, and the mortality rate of breast cancer did not differ based on their ethnicity (all *p*s ≥ .07). Although the contrast for differences in women's perceptions of the average woman's risk for breast cancer based on ethnicity was significant, $F(4, 448) = 2.35$, $p = .05$, the corrected post-hoc comparisons did not reveal any significant differences between groups. In the context of lung cancer, women's perceptions of their personal disease risk did differ based on their ethnicity, $F(4, 448) = 4.48$, $p = .001$. White or European American women ($M = 1.77$) perceived they were at greater personal risk for lung cancer than did African-American or Black women ($M = 1.21$; $p = .001$). White or European American women ($M = 1.77$) also perceived they were at greater personal risk for lung cancer than

[5] Only one participant reported being Native American, therefore this case was removed from this set of analyses.

did Asian or Pacific Islander women (M = 1.49; p = .05). However, when frequency of cigarette smoking was entered as a covariate, this difference disappeared, $F(4, 447)$ = 0.71, p = .59. There were no differences based on ethnicity in women's perceptions of the average woman's risk, the prevalence rate, or the mortality rate of lung cancer (all ps ≥ .12).

2. Perceptions of Disease Risk, Health Beliefs, and Healthful Behaviors

A. Measurement Model Design

Preventive behaviors. As noted in Hypothesis 2.1, people's estimates of their frequency of exercising, consuming cigarettes and alcohol, using vitamins, monitoring calories, and eating a balanced diet were tested as possible indicators of a latent variable representing preventive behaviors. Although there were some nonsignificant correlations between these variables in both younger and older participants (see Tables 1 and 2), the hypothesized measurement model (see Figure 2.1) was tested in each subsample.

Among younger participants (n = 419 due to the removal of all cases that were found to be multivariate outliers in the construction of the measurement models for perceptions of risk for cardiovascular disease, breast cancer, and lung cancer), the hypothesized measurement model was initially a poor fit to the data ($\chi^2(9) = 86.66$, p < .001; CFI = .75; RMSEA = .14, 90% confidence limits for the RMSEA = .12 to .17). Attempts to improve the model's fit revealed that cigarette and alcohol consumption were poor indicators. Contrary to expectations, each was weakly associated with the latent variable. The low incidence of cigarette smoking in this subsample (approximately 75% of participants never smoked), as well as the social role of alcohol use among college women, may explain these unexpected associations with preventive behaviors.

To address these issues, two separate measurement models of preventive behaviors were tested in this subsample. The first (see Figure 2.1a) excluded both alcohol and cigarette consumption as indicator variables, and was intended to be used in analyses involving perceptions of risk for cardiovascular disease and breast cancer. This model was a very good fit to the data ($\chi^2(2) = 2.61$, $p = .27$; CFI = 1.00; RMSEA = .03, 90% confidence limits for the RMSEA = .00 to .11), but the data were nonnormally distributed (Mardia's coefficient = -3.04). After the removal of 30 outliers (14.3% of the original subsample of 454 participants), the data were normally distributed and the model continued to be a good fit to the data ($\chi^2(2) = 4.18$, $p = .12$; CFI = .99; RMSEA = .05, 90% confidence limits for the RMSEA = .00 to .13).[6] The second measurement model constructed in younger women excluded alcohol consumption while retaining cigarette use, and was intended for use in analyses involving perceptions of risk for lung cancer. Retaining cigarette use as an indicator of preventive behaviors in these analyses seemed warranted due to the strong and widely-publicized association between cigarette smoking and lung cancer. To address the low incidence of cigarette use in this subsample, a dichotomized version of the variable, coded as 0 (never) or 1 (almost never, sometimes, fairly often, and very often), was used in the model. Although this model (see Figure 2.1b) was a good fit to the data ($\chi^2(5) = 7.40$, $p = .19$; CFI = .99; RMSEA = .03, 90% confidence limits for the RMSEA = .00 to .08), the data were nonnormally distributed (Mardia's coefficient = -3.47). The data were normally distributed after the removal of 33 outlier cases (15.0% of the original subsample of 454 participants), and the model

[6] Although 14.3% of the original sample were removed as multivariate outliers, standardized path coefficients for each indicator variable in the nonnormal ($n = 419$) and normal ($n = 389$) models were inconsequentially different (each $\beta \leq .03$).

remained a good fit to the data ($\chi^2(5) = 6.41$, $p = .27$; CFI = .99; RMSEA = .03, 90% confidence limits for the RMSEA = .00 to .08).[7]

Testing the hypothesized measurement model of preventive behaviors (Figure 2.1) in older participants ($n = 175$ due to the removal of all cases that were found to be multivariate outliers in the construction of the measurement models for perceptions of disease risk) revealed that the model was a poor fit to the data ($\chi^2(9) = 26.41$, $p = .002$; CFI = .72; RMSEA = .11, 90% confidence limits for the RMSEA = .06 to .15). Alcohol consumption was not significantly associated with the latent variable ($\beta = .18$, $p = .07$); widely-publicized reports on the health benefits of limited alcohol consumption (e.g., Meister, Whelan, & Kava, 2000; Stockley, 1998) may explain the unexpected relationship between this variable and preventive behaviors. Removing this variable resulted in a measurement model (see Figure 2.1c) that was an excellent fit to the data ($\chi^2(5) = 3.73$, $p = .59$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .09).

Screening behaviors. Attempts to fit a measurement model as described in Hypothesis 2.2, in which frequency of performing breast self-exams, obtaining mammograms, and receiving physicals from a doctor were indicators of a latent variable representing screening behaviors were unsuccessful in both younger and older participants. Therefore, a summary variable representing screening behaviors was created for each group. For younger participants, estimates of their frequency of performing breast self-exams and receiving physicals from a doctor were transformed into *z*-scores, and a constant was then added to each score to ensure that all values were

[7] Although 15.0% of the original sample were removed as multivariate outliers, standardized path coefficients for each indicator variable in the nonnormal ($n = 419$) and normal ($n = 386$) models were nearly identical (each $\beta \leq .01$).

positive. These scores were then summed and averaged (M = 2.00, SD = 0.74). The resulting variable was slightly positively skewed and highly kurtotic, necessitating a logarithmic transformation to achieve a normal distribution (M = 0.28, SD = 0.14). For older participants, estimates of their frequency of performing breast self-exams, obtaining mammograms, and receiving physicals from a doctor were transformed into z-scores, and a constant was then added to each score to ensure that all values were positive. These scores were also summed and averaged (M = 2.00, SD = 0.61).

B. Preventive Behaviors in the Context of Cardiovascular Disease, Breast Cancer, and Lung Cancer

To explore the relationship between risk perceptions, beliefs about the role of healthful behaviors in disease etiology, and preventive behaviors, a Main Effects Model was compared to an Interaction Model. To test the Main Effects Model (see Figures 2.3.1, 2.3.2, and 2.3.3), the latent variable representing perceptions of disease risk (constructed in Part 1) and an observed variable representing beliefs about the role of health behaviors in disease etiology each independently predicted the latent variable representing preventive behaviors. With the Interaction Model, the latent variable representing perceptions of disease risk and the observed variable representing beliefs about health behaviors were expected to interact to predict the latent variable representing preventive behaviors (see conceptual models in Figures 2.4.1, 2.4.2, and 2.4.3). To test such an interaction with structural equation modeling, the sample must be separated into different groups based on the moderator variable of interest. A median split was conducted to separate those with higher scores from those with lower scores on the items assessing beliefs about the role of health behaviors in disease etiology. Then, a model in

which the latent variable representing perceptions of disease risk predicted the latent variable representing preventive health behaviors was tested separately in the two groups. A difference in the fit of this model between the groups high and low in beliefs about the role of health behaviors in disease etiology reveals an interaction. For each chronic disease, the Main Effects Model and Interaction Model were tested separately in younger and older participants.

Cardiovascular disease in younger women. In a multivariate-normally distributed subsample of 418 participants, the Main Effects Model (see Figure 2.3.1a) was a poor fit to the data ($\chi^2(35) = 290.72$, $p < .001$; CFI = .87; RMSEA = .13, 90% confidence limits for the RMSEA = .12 to .15). There was also no evidence in support of the Interaction Model. The model (see Figure 2.4.1a) was a poor fit to the data in a normally-distributed subsample of 251 women with weaker beliefs in the role of health behaviors in the etiology of cardiovascular disease ($\chi^2(27) = 162.28$, $p < .001$; CFI = .88; RMSEA = .14, 90% confidence limits for the RMSEA = .12 to .16), and in a normally-distributed subsample of 162 women with stronger beliefs in the role of health behaviors ($\chi^2(27) = 143.87$, $p < .001$; CFI = .85; RMSEA = .16, 90% confidence limits for the RMSEA = .14 to .19). In all instances, the critical path between the latent variable representing perceptions of risk for cardiovascular disease and the latent variable representing preventive behaviors was not significant.

Breast cancer in younger women. In a normally-distributed subsample of 419 women, the Main Effects Model (see Figure 2.3.2a) was a poor fit to the data ($\chi^2(35) = 258.84$, $p < .001$; CFI = .87; RMSEA = .12, 90% confidence limits for the RMSEA = .11 to .14). The Interaction Model was also not supported. This model (see Figure 2.4.2a)

was a poor fit to the data in a normally-distributed subsample of 244 women with weaker beliefs in the role of health behaviors in the etiology of breast cancer ($\chi^2(27) = 176.35$, $p < .001$; CFI = .86; RMSEA = .15, 90% confidence limits for the RMSEA = .13 to .17), and in a normally-distributed subsample of 175 women with stronger beliefs in the role of health behaviors ($\chi^2(27) = 114.59$, $p < .001$; CFI = .87; RMSEA = .14, 90% confidence limits for the RMSEA = .11 to .16). Again, the critical path between the latent variable representing perceptions of risk for breast cancer and the latent variable representing preventive behaviors was not significant in any of the models.

Lung cancer in younger women. In a subsample of 414 women, the Main Effects Model (see Figure 2.3.3a) was a poor fit to the data ($\chi^2(44) = 987.65$, $p < .001$; CFI = .65; RMSEA = .23, 90% confidence limits for the RMSEA = .22 to .24).[8] Additionally, the Interaction Model was not confirmed. The model (see Figure 2.4.3a) was a poor fit to the data in a normally-distributed subsample of 135 younger women with weaker beliefs in the role of health behaviors ($\chi^2(35) = 348.77$, $p < .001$; CFI = .66; RMSEA = .26, 90% confidence limits for the RMSEA = .23 to .28). This model was also a poor fit to the data in a normally-distributed subsample of 283 women with stronger beliefs in the role of health behaviors in the etiology of lung cancer ($\chi^2(35) = 634.51$, $p < .001$; CFI = .65; RMSEA = .25, 90% confidence limits for the RMSEA = .23 to .26). In all three instances, the path between the latent variable representing perceptions of risk for lung cancer and the latent variable representing preventive behaviors was nonsignificant.

Cardiovascular disease in older women. It was not possible to use the latent variable representing preventive behaviors in any tests of the Main Effects Model and

[8] In this analysis, attempts to create a multivariate normal dataset were unsuccessful. Once the sample size reached 414 cases, Mardia's coefficient was 2.05. At this point, the removal of additional cases resulted in an increase, rather than the typical decrease, in the value for Mardia's coefficient.

Interaction Model in the subsample of older participants because the sample moment matrix was not positive definite. The most common reason for such an error with model-fitting stems from multicollinearity; however, none of the variables included in these models were highly correlated. Difficulty in fitting this model may have also been related to the small sample sizes in some analyses, particularly for tests of the Interaction Model. Since a solution could not be reached when the models of interest included the latent variable representing preventive behaviors, a summary variable was created. Estimates of participants' frequency of exercising, consuming cigarettes (reverse-scored), using vitamins, monitoring calories, and eating a balanced diet were summed and averaged to create an observed variable representing preventive behaviors ($M = 2.59$, $SD = 0.71$).

The Main Effects Model (see Figure 2.3.1b) was a poor fit to the data in a normally-distributed subsample of 175 older women ($\chi^2(9) = 33.85$, $p < .001$; CFI = .88; RMSEA = .13, 90% confidence limits for the RMSEA = .08 to .17). Additionally, the path between the latent variable representing perceptions of risk for cardiovascular disease and the observed variable representing preventive behaviors was not significant. Although the Interaction Model (see Figure 2.4.1b) was an adequate fit to the data in a normally-distributed subsample of 110 women with weaker beliefs in the role of health behaviors in the etiology of cardiovascular disease ($\chi^2(5) = 8.23$, $p = .14$; CFI = .97; RMSEA = .08, 90% confidence limits for the RMSEA = .00 to .17), the critical path between perceptions of risk for cardiovascular disease and preventive behaviors was nonsignificant. This model was also a poor fit the data in a normally-distributed subsample of 65 women with stronger beliefs in the role of health behaviors ($\chi^2(5) =$

13.96, p = .02; CFI = .86; RMSEA = .17, 90% confidence limits for the RMSEA = .07 to .27). Again, the path between perceptions of disease risk and preventive behaviors was nonsignificant.

Breast cancer in older women. In a normally-distributed subsample of 175 women, the Main Effects Model (see Figure 2.3.2b) was a good fit to the data ($\chi^2(8)$ = 10.81, p = .21; CFI = .99; RMSEA = .05, 90% confidence limits of the RMSEA = .00 to .11), yet the critical path between the latent variable representing perceptions of risk for breast cancer and the observed variable representing preventive behaviors was not significant. In tests of the Interaction Model, the model (see Figure 2.4.2b) was a good fit to the data in both a normally-distributed subsample of 94 women with weaker health beliefs ($\chi^2(4)$ = 10.32, p = .04; CFI = .96; RMSEA = .13, 90% confidence limits for the RMSEA = .03 to .23), and a normally-distributed subsample of 81 women with stronger health beliefs ($\chi^2(4)$ = .82, p = .94; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .04). However, the path of interest between perceptions of risk for breast cancer and preventive behaviors was nonsignificant.

Lung cancer in older women. Difficulties arose in fitting the Main Effects Model and Interaction Model in the context of lung cancer in the subsample of older women. In each case, the model's solution was inadmissible because it resulted in a Heywood case. Results of these analyses should be interpreted cautiously. Although the Main Effects Model (see Figure 2.3.3b) was an adequate fit to the data in a normally-distributed subsample of 175 women ($\chi^2(8)$ = 22.68, p = .004; CFI = .96; RMSEA = .10, 90% confidence limits for the RMSEA = .05 to .15), the path between the latent variable representing perceptions of risk for lung cancer and the observed variable representing

preventive behaviors was nonsignificant. The Interaction Model was also not supported. In a normally-distributed subsample of 98 women with weaker beliefs in the role of health behaviors in the etiology of lung cancer, the model (see Figure 2.4.3b) was an adequate fit to the data ($\chi^2(4) = 9.00$, $p = .06$; CFI = .97; RMSEA = .11, 90% confidence limits for the RMSEA = .00 to .21), but the path of primary interest was not significant. The same situation occurred in the normally-distributed subsample of 77 women with stronger health beliefs, with the model being an adequate fit to the data ($\chi^2(4) = 7.36$, $p = .12$; CFI = .98; RMSEA = .11, 90% confidence limits for the RMSEA = .00 to .22), but the path of interest failing to reach significance.[9]

In conclusion, neither Hypothesis 2.3 nor Hypothesis 2.4 was supported; both the Main Effects Model and the Interaction Model provided a poor fit to the data. Little evidence was found for a relationship between beliefs about the role of health behaviors in disease etiology and the performance of preventive health behaviors. Furthermore, in both younger and older women, perceptions of risk for cardiovascular disease, breast cancer, and lung cancer were not significantly associated with the performance of preventive health behaviors.

C. Screening Behaviors in the Context of Breast Cancer

The relationship between perceptions of risk for breast cancer, beliefs about the role of healthful behaviors in the etiology of breast cancer, and screening behaviors was explored by comparing the fit of the Main Effects Model (see Figure 2.5) to the fit of the Interaction Model (see Figure 2.6). Model fit was examined separately in younger and older women, using the measurement model of perceptions of risk for breast cancer and the observed summary variable representing screening behaviors.

[9] For this single test, the model did not produce a Heywood case and the solution was therefore admissible.

Breast cancer in younger women. The Main Effects Model was not supported in the subsample of younger women. The Main Effects Model (see Figure 2.5a) did provide a good fit to the data in a multivariate normally-distributed subsample of 418 women ($\chi^2(8) = 15.11$, $p = .06$; CFI = .99; RMSEA = .05, 90% confidence limits for the RMSEA = .00 to .08). However, the path between the latent variable representing perceptions of risk for breast cancer and the observed variable representing screening behaviors was marginally significant ($\beta = .09$, $p = .07$), and the path between the observed variables representing health beliefs and screening behaviors was nonsignificant ($\beta = -.05$, $p = .30$).

Consistent with Hypothesis 2.6, the Interaction Model was supported in younger women. In a normally-distributed subsample of 241 women with weaker beliefs in the role of health behaviors in the etiology of breast cancer, the Interaction Model (see Figure 2.6a) was a good fit to the data ($\chi^2(4) = 3.03$, $p = .55$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .09). The path between perceptions of risk for breast cancer and screening behaviors was nonsignificant for this group ($\beta = .001$, $p = .99$). Interestingly, the path between the indicator variable of perceptions of disease risk for the self and the latent variable representing perceptions of risk for breast cancer was also nonsignificant in this model ($\beta = .10$, $p = .14$). When examined in a normally-distributed subsample of 175 younger women with stronger health beliefs, this model was also a good fit to the data ($\chi^2(4) = 9.38$, $p = .05$; CFI = .98; RMSEA = .09, 90% confidence limits for the RMSEA = .00 to .16), and most critically, the path between the latent variable representing perceptions of risk for breast cancer and the observed variable representing screening behaviors was significant ($\beta = .21$, $p = .006$). Thus, beliefs about the role of health behaviors in breast cancer etiology do moderate the association between

perceptions of risk for breast cancer and the performance of screening behaviors including breast self-exams and visits to a physician. For younger women with weaker beliefs in the role of health behaviors, perceptions of disease risk were not associated with screening behaviors. Yet, for younger women with stronger beliefs in the role of health behaviors in the development of breast cancer, greater risk perceptions were associated with more frequent screening behaviors.

Breast cancer in older women. The Main Effects Model (see Figure 2.5b) was a good fit to the data in a normally-distributed subsample of 175 older women ($\chi^2(8) = 9.66$, $p = .29$; CFI = .99; RMSEA = .04, 90% confidence limits for the RMSEA = .00 to .10), but the path between perceptions of risk for breast cancer and screening behaviors was nonsignificant. The Interaction Model was also not supported in this subsample. In a normally-distributed subsample of 92 women with weaker beliefs in the role of health behaviors in the etiology of breast cancer, the model (see Figure 2.6b) was an adequate fit to the data ($\chi^2(4) = 5.66$, $p = .23$; CFI = .99; RMSEA = .07, 90% confidence limits for the RMSEA = .00 to .18). The path of interest between risk perceptions and screening behaviors was not significant. Similarly, in a normally-distributed subsample of 81 women with stronger beliefs in the role of health behaviors, the model was a good fit to the data ($\chi^2(4) = 0.58$, $p = .97$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .00). Again, the path between perceptions of risk for breast cancer and screening behaviors was nonsignificant. Thus, neither Hypothesis 2.5 nor Hypothesis 2.6 was supported among older women, as perceptions of breast cancer risk were not associated with screening behaviors.

3. Psychosocial Correlates of Perceptions of Disease Risk

A. Full Model of Psychosocial Correlates of Perceptions of Disease Risk

Hypothesized associations of individual traits (optimism and health locus of control), social influences (social exposure to disease and perceptions of stigma by others), and objective risk factors (family history of disease, age, BMI, and preventive health behaviors) with perceptions of risk for cardiovascular disease, breast cancer, and lung cancer were examined (see Figure 3.1). The General Disease Risk Model was the best fit to the data in both younger and older participants (see Part 1); therefore, this higher-order model was used in all analyses. Since the criterion variable in the analyses was general beliefs about risk rather than the measurement models for each disease, different versions of some of the predictor variables were incorporated into the model. First, a summary variable representing total social exposure to disease was created by summing responses to the three items assessing level of social exposure to cardiovascular disease, breast, cancer, and lung cancer. Second, a summary variable representing family history was created by summing responses to the three items assessing family history of disease. Third, a latent variable representing perceptions of stigma, indicated by each of the items assessing perceptions of stigma by others for those with cardiovascular disease, breast cancer, and lung cancer, was created.

The full model of psychosocial correlates of perceptions of disease risk was tested in samples consisting of younger and older women together (n = 634), only younger women (n = 454), and only older women (n = 180). Preliminary tests of the hypothesized model in the full sample and in each subsample revealed it to be a poor fit to the data, the data were multivariate nonnormal, and many of the hypothesized paths were not

significant. Because of the potential overlap in the psychosocial predictors and the complexity of the hypothesized model, an exploratory model-building approach was adopted. Potential predictors were added to the model in a systematic fashion, beginning first with variables that were significantly associated with general beliefs about risk in the preliminary tests of the full model, followed by the other hypothesized predictor variables. As each variable was added to the model, standardized residuals and modification indices were carefully examined to determine whether conceptually-appropriate paths could be added to improve model fit. Whenever predictor variables with significant associations to the risk variables were identified, the sample was trimmed at that point to meet the assumption of multivariate normality.

Full model in younger and older women together. The preliminary test of the hypothesized model (see Figure 3.1) indicated that the model was a poor fit to the data ($\chi^2(332) = 1447.77$, $p < .001$; CFI = .76; RMSEA = .08, 90% confidence limits for the RMSEA = .08 to .09). Additionally, only age, optimism, internal health locus of control, and family history were significantly associated with general beliefs about disease risk. Through the exploratory model-building procedure described above, a model that was a good fit to the data was created in a normally-distributed subsample of 480 younger and older women ($\chi^2(134) = 405.71$, $p < .001$; CFI = .92; RMSEA = .07, 90% confidence limits for the RMSEA = .06 to .07). However, because an excessive number of cases were removed to achieve a multivariate normal distribution (24.3%) this model was tested in the original sample of 634 participants using the bootstrapping with maximum likelihood estimation technique. This final model (see Figure 3.1a) was a good fit to the data ($\chi^2(102) = 365.61$, $p < .001$; CFI = .94; RMSEA = .06, 90% confidence limits for the

RMSEA = .06 to .07). Furthermore, bootstrapping produced standard errors that were similar and bias estimates that were low, indicating that despite the nonnormal data (Mardia's coefficient = 17.10), model parameter estimates were not biased.

As shown in Figure 3.1a, optimism was the only variable uniquely associated with general beliefs about risk. As hypothesized, optimism was inversely associated with women's beliefs about risk. Greater social exposure to cardiovascular disease was positively associated with women's perceptions of risk for cardiovascular disease, and was also uniquely associated with women's perceptions of their personal risk for this disease. Another aspect of social influence, people's perceptions of stigma by others, was associated with disease risk perceptions. However, contrary to predictions, greater perceptions of stigma for people afflicted with cardiovascular disease and lung cancer were associated with greater risk perceptions for each disease. respectively, and not with general risk perceptions. Finally, the objective risk factor of age was associated with perceptions of risk.[10] Those who were older perceived greater risks for cardiovascular disease, but also perceived fewer risks for breast cancer.

Full model in younger women. For younger women, age was excluded from the hypothesized model due to the limited range of this variable in the subsample. The hypothesized model was a poor fit to the data ($\chi^2(309) = 781.17$, $p < .001$; CFI = .87; RMSEA = .06, 90% confidence limits for the RMSEA = .06 to .07). Optimism was the only significant correlate of general beliefs about disease risk in this model. Through the exploratory model-building procedure, a model that was a good fit to the data was created in a normally-distributed subsample of 343 younger women ($\chi^2(137) = 304.82$, $p < .001$;

[10] In this analysis, similar results were obtained when subsample (coded as younger women = 1, older women = 2) was included as a variable in the model instead of age (which was square root transformed to achieve normality).

CFI = .94; RMSEA = .06, 90% confidence limits for the RMSEA = .05 to .07). An excessive number of cases (24.4%) were removed because the data were multivariate nonnormal, thus this model was tested in the original subsample of 454 younger participants using the bootstrapping with maximum likelihood estimation technique. This final model (see Figure 3.1b) was a good fit to the data ($\chi^2(89) = 229.14$, $p < .001$; CFI = .95; RMSEA = .06, 90% confidence limits for the RMSEA = .05 to .07). Bootstrapping produced standard errors that were similar and bias estimates that were low, indicating that the nonnormal data (Mardia's coefficient = 14.50) did not lead to biased parameter estimates.

In this model (see Figure 3.1b), none of the psychosocial variables predicted the underlying factor of general beliefs about risk. Social influences, including social exposure to disease and perceptions of stigma, were associated with specific disease risk perceptions. Social exposure to cardiovascular disease was positively associated with women's perceptions of risk for cardiovascular disease, and was also uniquely associated with women's perceptions of their personal risk for this disease. Women who believed that other people stigmatized those afflicted with cardiovascular disease and lung cancer also had greater perceptions of risk for each disease, respectively. Objective risk factors influenced perceptions of risk as well; women with a family history of breast cancer had greater overall perceptions of risk for breast cancer, and perceived their personal risk for breast cancer to be greater, than did women without a family history.

Full model in older women. Measures of age and education were both incorporated into the hypothesized model for older women. The preliminary test of this model indicated that the hypothesized model was a poor fit to the data ($\chi^2(359) = 593.40$,

$p < .001$; CFI = .83; RMSEA = .06, 90% confidence limits for the RMSEA = .05 to .07). Optimism, total social exposure to disease, family history of disease, and education were significantly associated with general beliefs about risk. By using these variables as a starting point for the exploratory model-building procedure, a model that was a good fit to the data was created in a normally-distributed subsample of 170 older women ($\chi^2(92) = 143.85$, $p < .001$; CFI = .95; RMSEA = .06, 90% confidence limits for the RMSEA = .04 to .08). The bootstrapping technique was also used to test this model in the full subsample of 180 participants, because 5.6% of the subsample was removed to achieve normality, and the sample size was small for a model of this complexity. Bootstrapping produced a final model (see Figure 3.1c) that was a good fit to the data ($\chi^2(92) = 157.57$, $p < .001$; CFI = .94; RMSEA = .06, 90% confidence limits for the RMSEA = .05 to .08). However, bootstrapping revealed that there may be some bias in the parameter estimates, particularly in the estimation of parameters associated with the latent variable representing perceptions of risk for cardiovascular disease and its indicators. It should be noted that the path between the latent variable and the indicator of perceptions of personal risk for cardiovascular disease was not significant in this model.

As shown in Figure 3.1c, the individual trait of optimism was inversely associated with general beliefs about risk. Similarly, level of education was inversely associated with the latent variable representing general beliefs about risk. Therefore, women who were more optimistic and who received more education perceived lower risks for chronic diseases, compared to those who were less optimistic and had lower levels of education. The remaining correlates were associated with women's perceptions of cardiovascular disease risk. Women with greater beliefs in the ability of powerful others to control their

health perceived less risk for cardiovascular disease. Additionally, women with greater social exposure to cardiovascular disease perceived greater risk for the disease. Greater social exposure was also uniquely associated with increased perceptions of personal risk for cardiovascular disease. However, in light of the results from the bootstrapping analysis, these findings must be interpreted cautiously.

B. Objective Risk Factors as a Moderating Variable of the Association between Social Influences and Perceptions of Disease Risk

Hypotheses 3.2 and 3.3 address the possibility that objective risk factors may moderate a relationship between social influences and disease risk perceptions. To test these hypotheses, participants were first separated into different groups based on their levels of objective risk. Then, models in which the observed variable representing perceptions of stigma by others predicted the latent variable representing perceptions of risk for cardiovascular disease, breast cancer, and lung cancer were tested separately in those at lower levels of objective risk and those at higher levels of objective risk (see Figures 3.2.1, 3.2.2, and 3.2.3 for conceptual models). Models in which the observed variable representing social exposure to disease predicted the latent variable representing perceptions of disease risk were also tested in the two groups (see Figures 3.3.1, 3.3.2, and 3.3.3 for conceptual models). A difference in model fit between the two groups would indicate that objective risk moderated the association between social influences and perceptions of disease risk. These hypotheses were tested separately in younger and older participants.

Creating an observed variable representing objective risk. A variable representing total objective risk was calculated in younger and older participants. In

younger participants, this variable included family history of disease, BMI, and preventive health behaviors; in older participants, this variable also included age. Family history of each disease was coded as 0 (no) and 1 (yes). BMI was recoded into three levels based on national guidelines, with participants who were underweight or at normal weight (BMI values ≤ 24.9) coded as 0, participants who were overweight (BMI values of 25 to 29.9) coded as 1, and participants who were obese (BMI values ≥ 30) coded as 2. For health behaviors including exercising, using vitamins, monitoring calories, and eating a balanced diet, responses were recoded as 0 (often or very often), 1 (sometimes), and 2 (never or almost never). Frequency of cigarette smoking was dichotomized into 0 (never) and 1 (almost never, sometimes, often, and very often). Finally, for older women, age was recoded into 0 (ages ≤ 50), and 1 (ages > 50). The age of 50 was selected to reflect an increase in objective risk because this is the approximate age at which women reach menopause, which is associated with an elevated susceptibility to chronic disease (Cheung, Chaudhry, Kapral, Jackevicius, & Robinson, 2004). The recoded values for each of these variables were then summed (M = 5.41, SD = 2.23 in younger participants; M = 5.51, SD = 2.43 in older participants). A median split (median = 6.00 in both subsamples) was conducted to separate those at lower levels of objective risk (n = 305 for younger participants, n = 113 for older participants) from those at higher levels of objective risk (n = 149 for younger participants, n = 67 for older participants).

Perceptions of stigma by others and perceptions of disease risk. Hypothesis 3.2 was not supported among younger women in the context of cardiovascular disease. Perceptions of stigma by others were not associated with perceptions of risk for cardiovascular disease in those at low or high levels of objective risk (see Figure 3.2.1a).

Objective risk did moderate the relationship between perceptions of stigma and perceptions of risk for breast cancer, but the direction of the relationship was contrary to predictions. Although the hypothesized model (see Figure 3.2.2a) was a good fit to the data in a subsample of women at lower levels of objective risk ($\chi^2(4) = 3.96$, $p = .41$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .09), perceived stigma was unrelated to perceptions of risk for breast cancer ($\beta = .02$, $p = .75$). Among women at higher levels of objective risk, the model was an adequate fit to the data ($\chi^2(4) = 10.38$, $p = .03$; CFI = .97; RMSEA = .10, 90% confidence limits for the RMSEA = .03 to .18), and perceived stigma was positively associated with perceptions of risk for breast cancer ($\beta = .25$, $p = .004$). Therefore, women who believed other people stigmatized those afflicted with breast cancer also perceived greater risk for breast cancer; however, this was only true for women with higher levels of objective risk.

Similar results were found in the context of lung cancer. The hypothesized model (see Figure 3.2.3a) was an adequate fit to the data in younger women at lower levels of objective risk ($\chi^2(4) = 16.63$, $p = .002$; CFI = .98; RMSEA = .10, 90% confidence limits for the RMSEA = .06 to .16), but perceived stigma was unrelated to perceptions of disease risk ($\beta = .03$, $p = .59$). For women at higher levels of objective risk, the model was a good fit to the data ($\chi^2(4) = 2.91$, $p = .57$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .11) and their perceptions of stigma were positively associated with their risk perceptions for lung cancer ($\beta = .20$, $p = .02$). However, in this model women's perceptions of personal risk for lung cancer were not significantly associated with the latent variable representing perceptions of disease risk ($\beta = .04$, $p = .68$). Women with an increased susceptibility to disease who also believe that

others stigmatize those afflicted with lung cancer may be motivated to distinguish between their own risk for lung cancer and more general aspects of disease risk, such as prevalence or mortality rates.

Among older women, there was no evidence in support of Hypothesis 3.2. For those at both lower and higher levels of objective risk, perceptions of stigma by others were unrelated to perceptions of risk for cardiovascular disease (see Figure 3.2.1b), breast cancer (see Figure 3.2.2b), and lung cancer (see Figure 3.2.3b), despite the fact that the hypothesized model was an adequate fit to the data in each analysis.[11]

Social exposure to disease and perceptions of disease risk. Hypothesis 3.3 was not supported in younger women. In the context of cardiovascular disease, bootstrapping analyses revealed that the hypothesized model was a poor fit to the data in those at both lower and higher levels of objective risk (see Figure 3.3.1a). The path between the observed variable representing social exposure and the latent variable representing perceptions of risk for cardiovascular disease was significant for those at lower objective risk ($\beta = .22$, $p < .001$) and for those at higher objective risk ($\beta = .21$, $p = .01$). However, the Chi-square difference test revealed that these path coefficients were not significantly different from one another, confirming that the strength of the path was not moderated by objective risk. In the context of breast cancer (see Figure 3.3.2a) and lung cancer (see

[11] Tests of Hypothesis 3.2 in women with higher levels of objective risk in the context of cardiovascular disease and breast cancer (as shown in Figures 3.2.1b and 3.2.2b, respectively), produced Heywood cases. Steps were taken to modify these models and produce an admissible solution; in neither case did this result in a significant path between the observed variable representing perceptions of stigma and the latent variable representing perceptions of disease risk.

Figure 3.3.3a), social exposure was unrelated to perceptions of disease risk for women at lower and higher levels of objective risk.[12]

In the subsample of older women, objective risk did not reliably moderate the relationship between social exposure and perceptions of risk for cardiovascular disease. Amount of social exposure was significantly associated with perceptions of risk for cardiovascular disease in those with fewer objective risk factors ($\beta = .29$, $p = .003$), and was not associated in those at higher levels of objective risk ($\beta = .25$, $p = .14$). However, the model (see Figure 3.3.1b) was a poor fit to the data in women at low objective risk, hampering the interpretation of this finding. Hypothesis 3.3 was also not supported in the context of breast cancer (see Figure 3.3.2b) or lung cancer (see Figure 3.3.3b), as social exposure was not associated with perceptions of disease risk.[13] In conclusion, although social exposure to disease was related to perceptions of risk in some instances, objective risk did not moderate this relationship in younger or older women.

4. Is Identification with Traditional Gender Roles Associated with Perceptions of Disease Risk?

To test whether the extent to which people identify themselves as masculine or feminine may influence their perceptions of disease risk, hypothesized models were analyzed in which participants' responses to the PAQ subscales of traditionally masculine and traditionally feminine gender roles were associated with the latent variables

[12] The test of Hypothesis 3.3 in younger women with higher levels of objective risk in the context of breast cancer (as shown in Figure 3.3.2a), initially produced a Heywood case. Modifying the model to produce an admissible solution did not result in a significant path between the observed variable representing social exposure and the latent variable representing perceptions of risk for breast cancer.

[13] The test of Hypothesis 3.3 in older women with lower levels of objective risk in the context of lung cancer (shown in Figure 3.3.3b) initially produced a Heywood case. Modifying the model to produce an admissible solution did not result in a significant path between social exposure and perceptions of risk for lung cancer.

representing perceptions of risk for cardiovascular disease (a "male" disease; see Figures 4.1 and 4.2) and breast cancer (a "female" disease; see Figures 4.3 and 4.4). Exploratory analyses examined whether identification with traditional gender roles was associated with perceptions of risk for lung cancer (see Figures 4.5 and 4.6). Analyses were conducted separately in younger and older participants.

Gender and perceived risk in younger women. The hypothesized models (see Figures 4.1a and Figure 4.2a) were an adequate fit to the data, but identification with traditional gender roles was unrelated to perceptions of risk for cardiovascular disease. Similarly, identification with traditional gender roles was unrelated to perceptions of risk for breast cancer (see Figures 4.3a and 4.4a). Exploratory analyses indicated that identification with traditionally masculine and traditionally feminine gender roles were both associated with perceptions of risk for lung cancer. The model (see Figure 4.5a) was a good fit to the data ($\chi^2(4) = 9.30$, $p = .05$; CFI = .99; RMSEA = .06, 90% confidence limits for the RMSEA = .00 to .11), with greater identification with traditionally masculine gender roles significantly associated with greater perceptions of risk for lung cancer ($\beta = .10$, $p = .05$). The same pattern (see Figure 4.6a) was found for identification with traditionally feminine gender roles; the model provided a good fit to the data ($\chi^2(4) = 3.07$, $p = .55$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .07), and greater identification with traditionally feminine gender roles was significantly associated with greater perceptions of risk for lung cancer ($\beta = .11$, $p = .04$)

Gender and perceived risk in older women. Among older women, identification with traditionally masculine and traditionally feminine gender roles was unrelated to

perceptions of risk for cardiovascular disease (see Figures 4.1b and 4.2b). Hypotheses regarding identification with traditional gender roles and perceptions of risk for breast cancer were also not supported (see Figures 4.3b and 4.4b). Finally, exploratory analyses revealed that identification with traditionally masculine and traditionally feminine gender roles was not associated with older women's perceptions of risk for lung cancer (see Figures 4.5b and 4.6b).

Discussion

Summary and Discussion of Findings

The present study used structural equation modeling to test theoretically-based hypotheses about younger and older women's perceptions of risk for chronic illnesses including cardiovascular disease, breast cancer, and lung cancer. Analyses explored measurement aspects of disease risk perceptions, the relationship between risk perceptions and protective health behaviors, and psychosocial factors associated with risk perceptions.

As hypothesized, women's perceptions of disease risk are multifactorial. Among both younger and older women, individual items assessing women's perceived risk for the self, perceived risk for the average woman, perceived prevalence rate, and perceived mortality rate of a disease were reliable indicators of latent variables representing global perceptions of risk for cardiovascular disease, breast cancer, and lung cancer. The strength of association between each of these indicators and the latent variables was also examined. Frequently, measures of perceived risk for the self and perceived risk for the average woman did not differ in their association with the latent variables. Measures of perceived prevalence rate and perceived mortality rate also did not differ in their association with women's global disease risk perceptions. These findings may be due to similarities in how the individual items were assessed. Whereas perceived risk for the self and for the average woman were measured with Likert-type scales, perceived prevalence and mortality rate were measured with numerical estimates. These four items reflect various methodological approaches, including absolute, comparative, and numerical estimates, which have been used to evaluate risk perceptions. Results from the

present study corroborate that these items all assess a common aspect of people's risk-related cognitions.

This study compared two hypothesized higher-order models of disease risk perceptions. In the Disease-Specific Risk Model, people's risk perceptions for different diseases were expected to be independent of one another. In the General Disease Risk Model, people's perceptions of risk for different diseases were expected to be related to a common underlying factor. Results were consistent with the General Disease Risk Model; people's global perceptions of risk for cardiovascular disease, breast cancer, and lung cancer were closely related to one another. These results corroborate the findings of a similar study investigating older women's perceptions of risk for chronic illnesses (Gerend, Aiken, & West, 2004). In that study, a single factor representing general perceived susceptibility was found to underlie women's perceptions of risk for breast cancer, heart disease, and osteoporosis. Results of the present study suggest that women's risk perceptions for different diseases share a common underlying factor, thereby supporting the idea that people consider their risk through the lens of a more general cognitive framework. This underlying factor may be health-specific, representing generalized beliefs about health risks. Or, this factor may represent broader, non-specific beliefs about personal vulnerability.

It was hypothesized that older and younger women would differ in their perceptions of disease risk. Consistent with predictions, results indicate that older women may have more complex and varied perceptions of risk than do younger women. Among older women, significant differences existed in the extent to which each of the items assessing perceptions of personal risk, the average woman's risk, prevalence rate,

and mortality rate was associated with the latent variables representing perceptions of risk for cardiovascular disease, breast cancer, and lung cancer. Yet, among younger women, most of these items did not differ in their associations with the latent variables representing perceptions of risk for the various diseases. Older women are likely to have more direct experiences with chronic illnesses, and also face greater actual susceptibility to such threats. These factors may contribute to older women's more elaborate perceptions of disease risk demonstrated in this study. Because disease-related information has a greater relevance to their personal well-being, it was also predicted that older women would have a more accurate understanding of the relative risks of chronic illnesses. Consistent with epidemiological data, older women perceived the greatest risk to be for cardiovascular disease. Younger women, however, felt a greater vulnerability for breast cancer than for cardiovascular disease or lung cancer. Furthermore, whereas all women demonstrated an optimistic bias in their perceived susceptibility to disease, estimating their own risk as significantly lower than the average woman's risk, older women demonstrated a smaller optimistic bias than younger women in their risk perceptions for cardiovascular disease and lung cancer. Unlike older women who have more disease-related knowledge and experiences, younger women may find it easier to distance themselves from the threat of chronic illness.

An additional goal of the present study was to examine how perceptions of disease risk and beliefs about the role of healthful behaviors in disease etiology relate to the performance of preventive and screening health behaviors. A Main Effects Model in which perceptions of risk and health beliefs are independently associated with behavior was compared to an Interaction Model in which health beliefs moderate the association

between perceptions of risk and behavior. Although risk perceptions are associated with the adoption of protective health behaviors in a number of psychological theories (Weinstein, 1993; Weinstein & Klein, 1995), the present study found little evidence of this relationship. Contrary to predictions, neither the Main Effects Model nor the Interaction Model was supported in analyses of the relationship between risk perceptions, beliefs about the role of health behaviors in disease etiology, and the performance of preventive behaviors such as exercising and eating a balanced diet. Younger women's beliefs about the role of health behaviors in disease etiology were associated with preventive behaviors; yet, in both younger and older women, perceptions of risk for cardiovascular disease, breast cancer, and lung cancer were not significantly associated with the performance of preventive behaviors. There was evidence, however, that the performance of screening behaviors was associated with perceptions of risk for breast cancer in younger women, and that this relationship was moderated by health beliefs. Specifically, for younger women with weaker beliefs in the role of health behaviors in breast cancer etiology, perceptions of disease risk were unrelated to screening behaviors. But, for younger women with stronger beliefs in the role of health behaviors, greater risk perceptions were associated with more frequent screening behaviors including breast self-exams and visits to a physician. Elevated perceptions of disease risk may only contribute to the adoption of screening behaviors in people who believe such actions can truly reduce their vulnerability to disease.

The failure to find additional evidence of a link between risk perceptions and behavior may be due to the measurement of protective behaviors. Measures of preventive and screening behaviors that are more comprehensive than the single-item

assessments of recent health behaviors used in this study may yield different results. Additionally, this study examined the relationship between behavior and global perceptions of disease risk (i.e., the latent variables representing perceived risk for cardiovascular disease, breast cancer, and lung cancer); perhaps very specific components of people's risk-related cognitions are more important in providing motivation to adopt precautionary health behaviors. It is also possible that the relationship between perceptions of disease risk and protective health behaviors is dynamic. Greater perceptions of disease risk may result in the practice of health behaviors, but over time, the performance of health behaviors may reduce perceptions of disease risk. A cross-sectional examination of these factors, such as the present study, would not detect such a process.

Hypotheses regarding the role of individual traits, social influences, and objective risk factors in influencing perceptions of disease risk were also investigated in the full sample of women, as well as in the subsamples of younger and older women. In the full sample, optimism was inversely associated with women's general beliefs about risk; those who were least optimistic perceived the greatest susceptibility to chronic illnesses. Those with more social exposure to cardiovascular disease had greater global perceptions of risk for cardiovascular disease, and also perceived their personal risk for this disease to be greater, than did women with less social exposure. Women with greater perceptions of stigma for people afflicted with cardiovascular disease and lung cancer perceived greater risk for each disease, respectively. Additionally, women who were older perceived greater risks for cardiovascular disease, and perceived fewer risks for breast cancer.

A slightly different pattern emerged when the role of psychosocial factors in perceptions of disease risk was examined separately in younger women. None of the hypothesized variables was associated with the underlying factor of general beliefs about risk. As predicted, social exposure to disease was positively associated with perceptions of risk for cardiovascular disease, and with younger women's perceptions of personal cardiovascular disease risk. Greater perceptions of stigma for those afflicted with cardiovascular disease and lung cancer were related to greater global perceptions of risk for each disease, respectively. Also, although objective risk did not moderate this relationship in the context of cardiovascular disease, the relationship between perceptions of stigma and perceptions of risk for breast cancer and lung cancer did differ based on women's levels of objective risk. Among younger women with elevated objective disease risk, perceptions of stigma were positively related to perceptions of risk for breast cancer. These variables were not associated among younger women with decreased objective risk. The same pattern emerged for perceptions of stigma for lung cancer and perceptions of lung cancer risk. Although these variables were positively correlated in women with higher objective risk, women's estimates of their personal risk for lung cancer were not significantly associated with the latent variable representing global perceptions of risk for lung cancer. Finally, younger women with a family history of breast cancer had greater global perceptions of risk for breast cancer, and also perceived greater personal risk for breast cancer.

Among older women, those who were more optimistic and who received more education perceived lower risks for chronic illnesses. Also, women with greater beliefs in the ability of powerful others to control their health perceived less risk for

cardiovascular disease. Greater social exposure to cardiovascular disease was associated with greater perceptions of risk for this disease, as well as greater perceptions of personal cardiovascular disease risk. However, bootstrapping analyses of this model indicate that these results, particularly those regarding correlates of risk perceptions for cardiovascular disease, may not be reliable.

These results illustrate that different patterns of psychosocial correlates of perceptions of disease risk emerged in younger and older women. Social influences primarily contributed to younger women's disease risk perceptions. As hypothesized and consistent with the availability heuristic (Tversky & Kahneman, 1973, 1974), those who knew more women afflicted with cardiovascular disease perceived greater risks for this illness than did women with less social exposure. It was also hypothesized that people would be motivated to believe they are less susceptible to diseases that they perceive as highly stigmatized by others. By perceiving themselves at decreased risk for such diseases, people may avoid emotional distress. However, results indicate that perceptions of stigma by others were positively associated with younger women's perceptions of disease risk. Additionally, perceptions of stigma for breast cancer and lung cancer were positively associated with perceptions of risk for each respective disease in women with greater objective vulnerability to disease. Perhaps such beliefs about stigma are simply indicative of a greater awareness of chronic disease. Younger women who perceive greater stigma for those afflicted with a disease may be more attuned to the threat of that disease. This may be especially true for women with elevated objective risk, as they are likely to be particularly concerned by such health issues. Although it was predicted that women would perceive less overall disease risk as a way to cope with the threat of

stigmatization, it is also possible that women with elevated objective risk instead change their perceptions of personal disease risk. As noted earlier, younger women with the greatest vulnerability to disease had perceptions of personal risk that were not significantly associated with the latent variable. Because these women are more vulnerable and also believe that lung cancer is stigmatized, they may be motivated to differentiate beliefs about their own risk from other risk-related beliefs. Making this distinction may help women cope with distress resulting from their heightened risk and perceptions of stigma.

Among older women, both social influences and individual traits, including optimism, education, and health locus of control, contributed to perceptions of disease risk. Older women who were more optimistic perceived fewer risks for chronic illnesses. Similarly, higher levels of education were associated with lower general beliefs about risk. Education may confer greater confidence and knowledge about how to cope with health threats, leading to lower perceptions of risk. Additionally, education is associated with the adoption of protective health behaviors (Gorin & Heck, 2005; Huisman, Kunst, & Mackenbach, 2005; Jones, Greaves, & Iliffe, 1992), which in turn lowers the risk for chronic disease. Contrary to expectations, older women with greater beliefs in the ability of powerful others to control their health perceived less risk for cardiovascular disease. These women may believe that powerful others, such as doctors and other healthcare providers, can successfully manage and mitigate the risks associated with heart disease. This is consistent with shifts in the dynamic of doctor-patient relationships over recent decades that have led older women to be more trusting of medical authority than younger women (Coulter, 1999; Freedman, 2002).

Contrary to predictions, objective risk factors were largely unrelated to women's perceptions of risk for chronic disease. Despite a well-established relationship between family history and elevated disease risk perceptions (e.g., Katapodi et al., 2004; Montgomery et al., 2003; Wilcox & Stefanick, 1999), family history was only associated with perceptions of risk for breast cancer in younger women. However, the influence of family history on disease risk perceptions may have been obscured by the influence of social exposure to disease. That is, the measure of social exposure used in this study did not assess how disease-afflicted women in the social network were related to the participant, and thus may have been confounded with family history. Indeed, scores on these two measures were significantly correlated in both younger and older women. Age was positively associated with perceptions of cardiovascular disease risk and inversely associated with perceptions of breast cancer risk in the full sample of younger and older women. Yet, age was unrelated to disease risk perceptions when data from the subsample of older women, ranging in age from 40 to 84 years, were examined separately. This suggests that most women in this subsample may have felt at risk because of their age, regardless of whether they were closer to the younger or older end of this distribution. Other well-established risk factors for chronic illness, including BMI and the performance of healthful behaviors, were not significantly associated with perceptions of disease risk in younger or older women. In this study, psychosocial variables, such as optimism, health locus of control, social exposure to disease, and perceptions of stigma by others, appear to have a more powerful influence than objective risk factors on women's perceptions of disease risk. These findings are consistent with previous studies in which objective risk factors identified by the Gail model (Gail et al.,

1989), a statistical model used to calculate a woman's risk for breast cancer, were weakly associated with women's perceptions of breast cancer risk (Audrain-McGovern, Hughes, & Patterson, 2003; Daly et al., 1996). Women appear to base their risk estimates on factors other than those considered important by healthcare providers.

A greater number of factors, including both individual traits and social influences, contributed to older women's perceptions of risk compared to younger women in this study. These additional factors, particularly greater optimism and external health locus of control, may help older women manage their heightened risk for chronic diseases of aging and any associated distress. To cope with this threat, older women may think about their disease risk differently than younger women. For instance, by putting their faith in powerful others, older women may protect themselves from uncomfortable feelings of risk. Whether this process is effortful and motivated, or is nonconscious and automatic, is unclear.

Analyses also examined whether the extent to which people identified themselves as traditionally masculine or traditionally feminine influences their disease risk perceptions. Hypotheses were guided by the representativeness heuristic, which states that people estimate the probability that a target belongs to a larger category based on the extent to which the target possesses characteristics that are stereotypical of the category (Kahneman & Tversky, 1973). It was predicted that when attempting to estimate the risk for cardiovascular disease, a masculine disease, and breast cancer, a feminine disease, people would use the representativeness heuristic and consider how similar they are to the stereotypical person afflicted with each disease. However, predictions were unsupported in younger and older women. Exploratory analyses revealed that, in

younger women, greater identification with traditionally masculine gender roles was associated with greater perceptions of risk for lung cancer. Greater identification with traditionally feminine gender roles was also associated with greater perceptions of risk for lung cancer in younger women. It is not clear why this pattern of results emerged. The only significant correlation to exist among the variables included in these models was between perceived prevalence rate of lung cancer and scores on the Femininity subscale of the PAQ. Thus, it is possible that these findings are spurious.

Implications

Although it has been suggested that individual studies should use multiple types of risk measures (e.g., Weinstein, 1998), the extent to which such measures are conceptually interchangeable or produce equivalent estimates has rarely been tested. Results of this study confirm that various approaches used to assess perceptions of disease risk, including absolute, comparative, and numerical risk estimates, do assess a common aspect of people's risk-related cognitions. These were reliable indicators of latent variables representing women's perceptions of risk for chronic illnesses. Yet, although these constructs are related to one another, they should not be considered interchangeable. Each was differentially associated with global perceptions of disease risk. Furthermore, these variables were differentially associated with psychosocial factors. Most notably, perception of personal disease risk was uniquely associated with social exposure to disease and family history of disease. Considering these findings, it appears that risk perception is a multidimensional construct. Single-item assessments that rely on absolute, comparative, or numerical estimates of risk may produce inaccurate measures of people's risk perceptions, and do not fully capture the richness of people's

risk-related beliefs. Multiply-indicated latent measures of perceptions of disease risk have the additional benefits of being statistically more powerful and reliable than single-item assessments. Adopting such an approach in the conceptualization and measurement of risk perceptions may provide a clearer understanding of the ways in which people think about their vulnerability to disease, and provide researchers the ability to better examine predictors and consequences of this vulnerability.

Results of this study also indicate that younger and older women differ in their perceptions of disease risk. Younger women believe they are at personal risk for different diseases than do older women. Younger women perceive greater risks for breast cancer than other diseases. However, older women perceive greater risks for cardiovascular disease, which is consistent with epidemiological evidence that cardiovascular disease is the leading cause of death in women. Additionally, younger women's perceptions of disease risk are influenced by different psychosocial factors than older women's risk perceptions. Whereas social influences primarily contribute to younger women's disease risk perceptions, both social influences and individual traits contribute to older women's risk perceptions. These findings suggest that there may be meaningful variations in women's risk-related cognitions with age. Yet, the underlying reasons for these differences are unclear. Older women may have a better awareness of health threats because of their greater maturity and experience with chronic illness. Older women are also at greater objective risk for disease than younger women, which may provide them with a better understanding of disease risks. But, knowledge of their elevated risk is also likely to produce feelings of anxiety. Older women's dispositional characteristics may help them to cope with concerns related to their elevated disease risk.

It is also possible that the differences observed between younger and older women in this study are the result of generational differences. For instance, younger women's greater perceptions of risk for breast cancer compared to other diseases may reflect the fact that advocacy surrounding breast cancer has dominated news reports and popular culture for the majority of their lifetime (Braun, 2003). It is clear that age should be considered as a factor in investigations of perceptions of disease risk, and future studies of risk-related cognitions may benefit by examining younger and older women separately. Longitudinal studies could also discern whether age-related differences in perceptions of disease risk are due to developmental or generational effects.

The present study found little evidence in support of the relationship between perceptions of disease risk and the performance of protective health behaviors, despite a strong theoretical basis for their association. Theories such as protection motivation theory (Prentice-Dunn & Rogers, 1986; Rogers, 1975, 1983), the health belief model (Becker & Maiman, 1975; Janz & Becker, 1984; Rosenstock, 1974), and subjective expected utility theory (Edwards, 1954; Ronis, 1992) identify risk perceptions as an important contributor to health behaviors. These theories focus on outcomes such as the likelihood of behavioral change, or motivation for adopting a behavior in the future. Although it is generally assumed that behavioral intentions are very similar to actual behavior, this may not be accurate (Weinstein, 1993). Risk perceptions may be more closely related to behavioral intentions than to the levels of current preventive behaviors that the present study examined. Risk perceptions also represent one of a host of factors contributing to people's healthful behaviors, and as such, may be a necessary but not sufficient contributor to behavioral change (van der Pligt, 1998). It is possible that

additional factors in health behavior theories, such as perceived costs and perceived benefits, are more powerful predictors of people's current levels of preventive health behaviors. That is, although risk perceptions contribute to people's intentions to adopt healthy behaviors, other factors can prevent actual behavioral changes. Indeed, practical or immediate barriers such as lack of time or access to healthcare facilities, which the present study did not examine, have been identified as strong determinants of precautionary behavior (e.g., Salmon, Owen, Crawford, Bauman, & Sallis, 2003). Women, who typically face unique burdens such as greater caregiving responsibilities and limited income, are particularly likely to be affected by such barriers. It has also been hypothesized that risk perceptions are less powerful predictors of behaviors that have broad benefits non-specific to a particular disease, such as exercising and eating a balanced diet, and are instead more closely associated with disease-specific behaviors such as cancer screening tests or vaccine utilization (Brewer et al., 2007). This explanation may account for the significant association of perceptions of breast cancer risk with screening health behaviors such as breast self-exams in the present study.

Results of this study also illustrate that women's disease-specific beliefs do not exist independently of one another. There was some consistency in how women perceived their risks for cardiovascular disease, breast cancer, and lung cancer, and these perceptions were associated with an underlying construct that was conceptualized as general beliefs about risk. This construct may be health-specific, or it may be a broader factor of risk sensitivity (Sjoberg, 2000) or personal invulnerability (Perloff, 1983; Perloff & Fetzer, 1986). In older women, these general beliefs about risk were influenced by individual traits including optimism and educational attainment. Although

people appear to have a generalized orientation in their beliefs about disease risk, psychosocial factors, including locus of control, social exposure to disease, and perceptions of stigma, still influenced women's perceptions of vulnerability to specific diseases.

It appears that women's subjective beliefs contribute more strongly to their risk-related cognitions than do their objective risks. Most notably, optimistic expectations and greater confidence in the abilities of others are related to lower perceptions of disease risks. These subjective beliefs are consistent with the kinds of positive illusions people typically hold about themselves and the surrounding world (S. E. Taylor & Brown, 1988). Such positive illusions are more common in stressful situations, because these beliefs can help people to cope with distress (e.g., S. E. Taylor, Lichtman, & Wood, 1984; Wood, Taylor, & Lichtman, 1985). The threat of chronic disease may be particularly distressing for people. By maintaining an optimistic outlook, and placing their confidence in doctors and healthcare providers, people may decrease their feelings of vulnerability and distress. Even though such beliefs may be inaccurate, these psychosocial factors may help people to effectively cope with health threats. However, it is also possible that positive beliefs are not always beneficial. Positively-biased perceptions may become maladaptive if they prevent people from taking necessary health precautions such as seeking medical care when needed or performing screening tests (Perloff, 1983; Weinstein, 1980).

Study Limitations

This investigation used many well-validated instruments. However, novel items were also designed based on a review of relevant literature, and the reliability and validity of such items is unknown. For instance, it is unclear what kind of woman

participants may have imagined when asked to estimate the "average woman's" disease risk. Participants may have imagined someone similar to themselves in terms of age, ethnicity, and objective risk factors, or they may have envisioned a dissimilar target. This issue may be particularly relevant to younger women's risk perceptions. This may explain why younger women appeared to be more optimistic in their estimates of disease risk than older women. That is, if younger participants imagined the average woman as someone older than themselves, they may have accurately perceived her disease risk as greater than their own. Similarly, participants may have interpreted the questions designed to assess perceptions of stigma in unanticipated ways. Participants were asked to rate the extent to which "other people hold negative views or attitudes" about women with a disease. Rather than thinking about how stigmatized these women are, participants may have considered other negative views, such as others' beliefs that these women have a poor health prognosis due to their disease.

Most items used in this investigation had sufficient variance to address study hypotheses. However, there were substantial differences in participants' responses to measures of social exposure across diseases. Most notably, participants knew far fewer women with lung cancer than other diseases. This may have diminished the ability to detect a relationship between social exposure to lung cancer and perceptions of disease risk. An additional limitation is that the order of the risk measures was not counterbalanced in the study questionnaires. All participants answered questions about cardiovascular disease first, followed by questions about breast cancer, and then lung cancer. It seems unlikely, however, that the order of question presentation influenced participants' responses. Differences emerged across subsamples and diseases in

women's perceptions of risk, and these patterns were not correlated with the order in which the risk items were presented.

The limited sample size available for some analyses may have led to underpowered tests of study hypotheses. Specifically, tests in older women of the moderating role of health beliefs in the relationship between protective health behaviors and perceptions of disease risk, and tests of the moderating role of objective risk factors in the relationship between social influences and perceptions of disease risk may have been underpowered. These analyses, which included as few as 65 older women, did meet the minimum of 5 cases per estimated parameter suggested for structural equation modeling (Bentler & Chou, 1987). However, analyses with samples involving fewer than 100 participants should be interpreted cautiously. It is also important to note that, although structural equation modeling can be used to test causal relationships, results of this study are correlational. Thus, there is still the possibility of reverse causality or bidirectionality in the analyzed relationships, and associations between study variables could be caused by unmeasured factors (i.e., third variables). However, it is unlikely that some of the confirmed relationships, such as social exposure to disease and family history predicting perceptions of disease risk, would operate in the opposite direction.

The generalizability of study results must also be considered. The subsample of younger women was sufficiently large for all study analyses, and was ethnically and socioeconomically diverse, lending confidence to the generalizability of findings from this group. However, the older women in this study were less diverse; participants were primarily White or European American (86%), and the majority were relatively affluent (approximately 63% had annual household incomes of $70,000 or more). Ethnic and

socioeconomic variables in this sample were confounded with age, and could explain the subsample differences found in this study. However, younger women's perceptions of disease risk did not differ based on ethnicity. Thus, it seems unlikely that ethnicity accounts for the subsample differences. Also, no information regarding differences between responders and nonresponders is available. It is possible that women who chose to participate may differ in important ways from those who decided not to complete the study questionnaire. Finally, this study only examined aspects of women's disease risk perceptions. It is not clear whether these findings would also be valid for men. Conducting a similar study in men using diseases that affect both women and men (e.g., cardiovascular disease and lung cancer), as well as sex-specific diseases (e.g., prostate cancer), would clarify whether women and men differ in their risk perceptions and in the predictors of these beliefs.

Future Directions and Conclusions

These results demonstrate that younger and older women differ in their perceptions of risk for chronic illnesses. Yet, questions remain about why these differences were observed. Older women's more varied and complex understanding of disease risk may be a result of their cognitive and social development. As people advance from young adulthood through later life, they may acquire and integrate knowledge about diseases of aging through formal (e.g., education) and social (e.g., family, friends) sources. Younger women, who have had fewer of these informative experiences, may therefore have a less well-developed understanding of disease risk. Alternatively, it is possible that older and younger women vary in their awareness of disease risk due to differences in their exposure to public health campaigns or media

coverage of celebrity and political figures afflicted with disease. Older women in this study may have a more complex understanding of these health threats simply because they have lived through different public health events than younger women. Longitudinal studies examining changes in women's perceptions of disease risk could inform our understanding of such age-related differences.

Future longitudinal investigations should also incorporate measures of current health behaviors and behavioral intentions, particularly those including preventive and screening behaviors with both broad and disease-specific benefits. Data from such investigations would clarify the theorized association between perceptions of disease risk and precautionary behavior. If heightened risk perceptions can be reliably associated with behavioral intentions and subsequent behavioral change, it may be possible to design and test interventions involving psychosocial correlates of disease risk. For example, interventions could encourage women to actively think about disease-affected women in their social networks. Encouraging women to think about their family history of disease may also be effective in reducing feelings of invulnerability. By increasing women's perceptions of disease risk, it might be possible to increase their motivation for adopting healthy behaviors. However, such interventions would also need to carefully monitor participants' distress levels, and there may be ethical challenges associated with increasing people's perceptions of risk. Increasing optimism may be a way to help women gain confidence in their ability to manage health threats, or help women mitigate distress about their elevated disease risk.

Cardiovascular disease, breast cancer, and lung cancer are leading causes of morbidity and mortality in women, and it is crucial that we learn how to best assess

women's understanding of these health risks. Although the exact association between perceptions of vulnerability and the adoption of healthful behaviors remains unknown, it is likely that women's risk-related beliefs contribute to these choices. Women's perceptions of disease risk may have implications for a variety of other domains, as well. For instance, women with greater perceptions of risk may feel more prepared and less distressed if they do become ill than women who feel invulnerable. Women who believe they are susceptible to a disease may provide better social support to loved ones with diseases, or may have more motivation to encourage their healthy friends, siblings, or children to adopt precautionary behaviors. Greater feelings of vulnerability could also be related to women's willingness to support social initiatives designed to improve public health, such as smoking bans to reduce the risk of lung cancer, or funding for recreational parks to encourage exercise and decrease the threat of obesity and cardiovascular disease. Therefore, understanding women's disease risk perceptions, and the psychosocial factors that can influence these beliefs, remains a vital public health goal.

References

Aalto, A.-M., Heijmans, M., Weinman, J., & Aro, A. R. (2005). Illness perceptions in coronary heart disease: Sociodemographic, illness-related, and psychosocial correlates. *Journal of Psychosomatic Research, 58*, 393-402.

Ali, N. S. (2002). Prediction of coronary heart disease preventive behaviors in women: A test of the Health Belief Model. *Women & Health, 35*, 83-96.

American Cancer Society. (2008). *Cancer Facts & Figures 2008*. Atlanta: American Cancer Society.

Arbuckle, J. L. (2008). AMOS 17.0.1. Spring House, PA: Amos Development Corporation.

Aspinwall, L. G., & Brunhart, S. M. (1996). Distinguishing optimism from denial: Optimistic beliefs predict attention to health threats. *Personality and Social Psychology Bulletin, 22*, 993-1003.

Audrain-McGovern, J., Hughes, C., & Patterson, F. (2003). Effecting behavior change: Awareness of family history. *American Journal of Preventive Medicine, 24*, 183-189.

Becker, M. H., & Maiman, L. A. (1975). Sociobehavioral determinants of compliance with health and medical care recommendations. *Medical Care, 13*, 10-24.

Bentler, P. M., & Chou, C. P. (1987). Practical issues in structural modeling. *Sociological Methods & Research, 16*, 78-117.

Braun, S. (2003). The history of breast cancer advocacy. *Breast Journal, 9 Suppl 2*, S101-103.

Brewer, N. T., Chapman, G. B., Gibbons, F. X., Gerrard, M., McCaul, K. D., & Weinstein, N. D. (2007). Meta-analysis of the relationship between risk perception and health behavior: The example of vaccination. *Health Psychology, 26*, 136-145.

Byrne, B. M. (2001). *Structural Equation Modeling with AMOS*. Mahwah, NJ: Lawrence Erlbaum Associates.

Calle, E. E., Rodriguez, C., Walker-Thurmond, K., & Thun, M. J. (2003). Overweight, obesity, and mortality from cancer in a prospectively studied cohort of US adults. *New England Journal of Medicine, 348*, 1625-1638.

Caplan, L. S., McQueen, D. V., Qualters, J. R., Leff, M., Garrett, C., & Calonge, N. (2003). Validity of women's self-reports of cancer screening test utilization in a managed care population. *Cancer Epidemiology Biomarkers & Prevention, 12*, 1182-1187.

Carver, C. S., Pozo, C., Harris, S. D., Noriega, V., Scheier, M. F., Robinson, D. S., et al. (1993). How coping mediates the effect of optimism on distress: A study of women with early-stage breast-cancer. *Journal of Personality and Social Psychology, 65*, 375-390.

Cheung, A. M., Chaudhry, R., Kapral, M., Jackevicius, C., & Robinson, G. (2004). Perimenopausal and postmenopausal health. *BMC Women's Health, 4 Suppl 1*, S23.

Christian, A. H., Rosamond, W., White, A. R., & Mosca, L. (2007). Nine-year trends and racial and ethnic disparities in women's awareness of heart disease and stroke: An American Heart Association national study. *Journal of Women's Health, 16*, 68-81.

Contractor, K. B., Kaur, K., Rodrigues, G. S., Kulkarni, D. M., & Singhal, H. (2008). Male breast cancer: Is the scenario changing, *World Journal of Surgical Oncology*, *6,* 58.

Coulter, A. (1999). Paternalism or partnership? *British Medical Journal, 319*, 719-720.

Coyne, J. C., Kruus, L., Racioppo, M., Calzone, K. A., & Armstrong, K. (2003). What do ratings of cancer-specific distress mean among women at high risk of breast and ovarian cancer? *American Journal of Medical Genetics Part A, 116*, 222-228.

Daly, M. B., Lerman, C. L., Ross, E., Schwartz, M. D., Sands, C. B., & Masny, A. (1996). Gall model breast cancer risk components are poor predictors of risk perception and screening behavior. *Breast Cancer Research and Treatment, 41*, 59-70.

Del Boca, F. K., & Darkes, J. (2003). The validity of self-reports of alcohol consumption: State of the science and challenges for research. *Addiction, 98*, 1-12.

DeLuca, R. S., & Lobel, M. (1995). Conception, commitment, and health behavior practices in medically high-risk pregnant women. *Women's Health, 1*, 257-271.

Dillard, A. J., McCaul, K. D., & Klein, W. M. P. (2006). Unrealistic optimism in smokers: Implications for smoking myth endorsement and self-protective motivation. *Journal of Health Communication, 11*, 93-102.

DiLorenzo, T. A., Schnur, J., Montgomery, G. H., Erblich, J., Winkel, G., & Bovbjerg, D. H. (2006). A model of disease-specific worry in heritable disease: The influence of family history, perceived risk and worry about other illnesses. *Journal of Behavioral Medicine, 29*, 37-49.

Edwards, W. (1954). The theory of decision making. *Psychological Bulletin, 51*, 380-417.

Eibner, F., Barth, J., & Bengel, J. (2006). Predicting perceived vulnerability for breast cancer among women with an average breast cancer risk. *British Journal of Health Psychology, 11*, 607-621.

Erblich, J., Bovbjerg, D. H., Norman, C., Valdimarsdottir, H. B., & Montgomery, G. H. (2000). It won't happen to me: Lower perception of heart disease risk among women with family histories of breast cancer. *Preventive Medicine, 31*, 714-721.

Festinger, L. (1954). A theory of social comparison processes. *Human Relations, 7*, 117-140.

Festinger, L., & Carlsmith, J. M. (1959). Cognitive consequences of forced compliance. *Journal of Abnormal and Social Psychology, 58*, 203-210.

Freedman, T. G. (2002). 'The doctor knows best' revisited: Physician perspectives. *Psycho-Oncology, 11*, 327-335.

Gail, M. H., Brinton, L. A., Byar, D. P., Corle, D. K., Green, S. B., Schairer, C., et al. (1989). Projecting individualized probabilities of developing breast cancer for white females who are being examined annually. *Journal of the National Cancer Institute, 81*, 1879-1886.

Gerend, M. A., Aiken, L. S., & West, S. G. (2004). Personality factors in older women's perceived susceptibility to diseases of aging. *Journal of Personality, 72*, 243-270.

Gerend, M. A., Aiken, L. S., West, S. G., & Erchull, M. J. (2004). Beyond medical risk: Investigating the psychological factors underlying women's perceptions of susceptibility to breast cancer, heart disease, and osteoporosis. *Health Psychology, 23*, 247-258.

Gigerenzer, G., Gaissmaier, W., Kurz-Milcke, E., Schwartz, L. M., & Woloshin, S. (2007). Helping doctors and patients make sense of health statistics. *Psychological Science in the Public Interest, 8*, 53-96.

Giordano, S. H., Cohen, D. S., Buzdar, A. U., Perkins, G., & Hortobagyi, G. N. (2004). Breast carcinoma in men: A population-based study. *Cancer, 101*, 51-57.

Gorin, S. S., & Heck, J. E. (2005). Cancer screening among Latino subgroups in the United States. *Preventive Medicine, 40*, 515-526.

Hamilton, J. G., & Lobel, M. (2008). Types, patterns, and predictors of coping with stress during pregnancy: Examination of the Revised Prenatal Coping Inventory in a diverse sample. *Journal of Psychosomatic Obstetrics & Gynecology, 29*, 97-104.

Harwell, T. S., Blades, L. L., Oser, C. S., Dietrich, D. W., Okon, N. J., Rodriguez, D. V., et al. (2005). Perceived risk for developing stroke among older adults. *Preventive Medicine, 41*, 791-794.

Healton, C. G., Gritz, E. R., Davis, K. C., Homsi, G., McCausland, K., Haviland, M. L., et al. (2007). Women's knowledge of the leading causes of cancer death. *Nicotine & Tobacco Research, 9*, 761-768.

Helmreich, R. L., Spence, J. T., & Wilhelm, J. A. (1981). A psychometric analysis of the Personal Attributes Questionnaire. *Sex Roles, 7*, 1097-1108.

Helweg-Larsen, M., & Shepperd, J. A. (2001). Do moderators of the optimistic bias affect personal or target risk estimates? A review of the literature. *Personality and Social Psychology Review, 5*, 74-95.

Huisman, M., Kunst, A. E., & Mackenbach, J. P. (2005). Inequalities in the prevalence of smoking in the European Union: Comparing education and income. *Preventive Medicine, 40*, 756-764.

Janz, N. K., & Becker, M. H. (1984). The Health Belief Model: A decade later. *Health Education Quarterly, 11*, 1-47.

Jones, A., Greaves, K., & Iliffe, S. (1992). Health protective behaviour and knowledge of coronary heart disease risk factors in a general practice population. *Medical Science Research, 20*, 71-73.

Kahneman, D., & Tversky, A. (1973). On the psychology of prediction. *Psychological Review, 80*, 237-251.

Katapodi, M. C., Facione, N. C., Humphreys, J. C., & Dodd, M. J. (2005). Perceived breast cancer risk: Heuristic reasoning and search for a dominance structure. *Social Science & Medicine, 60*, 421-432.

Katapodi, M. C., Lee, K. A., Facione, N. C., & Dodd, M. J. (2004). Predictors of perceived breast cancer risk and the relation between perceived risk and breast cancer screening: A meta-analytic review. *Preventive Medicine, 38*, 388-402.

King, E. S., Rimer, B. K., Trock, B., Balshem, A., & Engstrom, P. (1990). How valid are mammography self-reports? *American Journal of Public Health, 80*, 1386-1388.

Klein, W. M. P., & Stefanek, M. E. (2007). Cancer risk elicitation and communication: Lessons from the psychology of risk perception. *CA: A Cancer Journal for Clinicians, 57*, 147-167.

Lloyd-Jones, D. M., Leip, E. P., Larson, M. G., D'Agostino, R. B., Beiser, A., Wilson, P. W. F., et al. (2006). Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. *Circulation, 113*, 791-798.

Lobel, M., DeVincent, C. J., Kaminer, A., & Meyer, B. A. (2000). The impact of prenatal maternal stress and optimistic disposition on birth outcomes in medically high-risk women. *Health Psychology, 19*, 544-553.

Lockyer, L., & Bury, M. (2002). The construction of a modern epidemic: The implications for women of the gendering of coronary heart disease. *Journal of Advanced Nursing, 39*, 432-440.

McCaul, K. D., Reid, P. A., Rathge, R. W., & Martinson, B. (1996). Does concern about breast cancer inhibit or promote breast cancer screening? *Basic & Applied Social Psychology, 18*, 183-194.

Meischke, H., Kuniyuki, A., Yasui, Y., Bowen, D. J., Andersen, R., & Urban, N. (2002). Information women receive about heart attacks and how it affects their knowledge, beliefs, and intentions to act in a cardiac emergency. *Health Care for Women International, 23*, 149-162.

Meischke, H., Sellers, D. E., Robbins, M. L., Goff, D. C., Daya, M. R., Meshack, A., et al. (2000). Factors that influence personal perceptions of the risk of an acute myocardial infarction. *Behavioral Medicine, 26*, 4-13.

Meister, K. A., Whelan, E. M., & Kava, R. (2000). The health effects of moderate alcohol intake in humans: An epidemiologic review. *Critical Reviews in Clinical Laboratory Sciences, 37*, 261-296.

Messina, C. R., Kabat, G. C., & Lane, D. S. (2002). Perceptions of risk factors for breast cancer and attitudes toward mammography among women who are current, ex- and non-smokers. *Women & Health, 36*, 65-82.

Montgomery, G. H., Erblich, J., DiLorenzo, T., & Bovbjerg, D. H. (2003). Family and friends with disease: Their impact on perceived risk. *Preventive Medicine, 37*, 242-249.

Moran, S., Glazier, G., & Armstrong, K. (2003). Women smokers' perceptions of smoking-related health risks. *Journal of Women's Health, 12*, 363-371.

Mosca, L., Mochari, H., Christian, A., Berra, K., Taubert, K., Mills, T., et al. (2006). National study of women's awareness, preventive action, and barriers to cardiovascular health. *Circulation, 113*, 525-534.

National Cancer Institute. (2007). *What You Need to Know About Lung Cancer*. Rockville, MD: U.S. Department of Health and Human Services, National Institutes of Health, and National Cancer Institute.

National Heart, Lung, and Blood Institute. (2007). *NHLBI Factbook, Fiscal Year 2007*. Bethesda, MD: National Heart, Lung, and Blood Institute.

Nelson, D. E., Kreps, G. L., Hesse, B. W., Croyle, R. T., Willis, G., Arora, N. K., et al. (2004). The Health Information National Trends Survey (HINTS): Development, design, and dissemination. *Journal of Health Communication, 9*, 443-460.

Norman, P., & Brain, K. (2007). Does dispositional optimism predict psychological responses to counseling for familial breast cancer? *Journal of Psychosomatic Research, 63*, 247-254.

Patel, M. J., de Lemos, J. A., Philips, B., Murphy, S. A., Vaeth, P. C., McGuire, D. K., et al. (2007). Implications of family history of myocardial infarction in young women. *American Heart Journal, 154*, 454-460.

Peacey, V., Steptoe, A., Davidsdottir, S., Baban, A., & Wardle, J. (2006). Low levels of breast cancer risk awareness in young women: An international survey. *European Journal of Cancer, 42*, 2585-2589.

Perloff, L. S. (1983). Perceptions of vulnerability to victimization. *Journal of Social Issues, 39*, 41-61.

Perloff, L. S., & Fetzer, B. K. (1986). Self-other judgments and perceived vulnerability to victimization. *Journal of Personality and Social Psychology, 50*, 502-510.

Pohls, U. G., Renner, S. P., Fasching, P. A., Lux, M. P., Kreis, H., Ackermann, S., et al. (2004). Awareness of breast cancer incidence and risk factors among healthy women. *European Journal of Cancer Prevention, 13*, 249-256.

Prentice-Dunn, S., & Rogers, R. W. (1986). Protection Motivation Theory and preventive health: Beyond the Health Belief Model. *Health Education Research, 1*, 153-161.

Radcliffe, N. M., & Klein, W. M. P. (2002). Dispositional, unrealistic and comparative optimism: Differential relations with the knowledge and processing of risk information and beliefs about personal risk. *Personality and Social Psychology Bulletin, 28*, 836-846.

Renner, B., Knoll, N., & Schwarzer, R. (2000). Age and body make a difference in optimistic health beliefs and nutrition behaviors. *International Journal of Behavioral Medicine, 7*, 143-159.

Ries, L. A. G., Melbert, D., Krapcho, M., Stinchcomb, D. G., Howlader, N., Horner, M. J., et al. (2008). SEER Cancer Statistics Review, 1975-2005. Bethesda, MD: National Cancer Institute.

Rogers, R. W. (1975). A protection motivation theory of fear appeals and attitude change. *Journal of Psychology: Interdisciplinary and Applied, 91*, 93-114.

Rogers, R. W. (1983). A protection motivation theory of fear appraisals and attitude change: A revised theory of protection motivation. In J. R. Cacioppo & R. E. Petty (Eds.), *Social Psychology: A sourcebook* (pp. 153-176). New York: Guilford Press.

Ronis, D. L. (1992). Conditional health threats: Health beliefs, decisions, and behaviors among adults. *Health Psychology, 11*, 127-134.

Rosamond, W., Flegal, K., Furie, K., Go, A., Greenlund, K., Haase, N., et al. (2008). Heart disease and stroke statistics 2008 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation, 117*, E25-E146.

Rosenstock, I. M. (1974). The health belief model and preventive health behavior. *Health Education Monographs, 2*, 354-386.

Rowe, J. L., Montgomery, G. H., Duberstein, P. R., & Bovbjerg, D. H. (2005). Health locus of control and perceived risk for breast cancer in healthy women. *Behavioral Medicine, 31*, 33-40.

Salmon, J., Owen, N., Crawford, D., Bauman, A., & Sallis, J. F. (2003). Physical activity and sedentary behavior: A population-based study of barriers, enjoyment, and preference. *Health Psychology, 22*, 178-188.

Satia-Abouta, J., Patterson, R. E., King, I. B., Stratton, K. L., Shattuck, A. L., Kristal, A. R., et al. (2003). Reliability and validity of self-report of vitamin and mineral supplement use in the Vitamins and Lifestyle Study. *American Journal of Epidemiology, 157*, 944-954.

Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychology, 4*, 219-247.

Scheier, M. F., Matthews, K. A., Owens, J. F., Schulz, R., Bridges, M. W., Magovern, G. J., et al. (1999). Optimism and rehospitalization after coronary artery bypass graft surgery. *Archives of Internal Medicine, 159*, 829-834.

Schnoll, R. A., Malstrom, M., James, C., Rothman, R. L., Miller, S. M., Ridge, J. A., et al. (2002). Correlates of tobacco use among smokers and recent quitters diagnosed with cancer. *Patient Education and Counseling, 46*, 137-145.

Shumaker, S. A., & Smith, T. R. (1995). Women and coronary heart disease: A psychological perspective. In A. L. Stanton & S. J. Gallant (Eds.), *The Psychology of Women's Health: Progress and Challenges in Research and Applications* (pp. 25-49). Washington, DC: American Psychological Association.

Silberberg, J., Fryer, J., Wlodarczyk, J., Robertson, R., & Dear, K. (1999). Comparison of family history measures used to identify high risk of coronary heart disease. *Genetic Epidemiology, 16*, 344-355.

Sjoberg, L. (2000). Factors in risk perception. *Risk Analysis, 20*, 1-11.

Spence, J. T., & Helmreich, R. L. (1978). *Masculinity and Femininity: Their Psychological Dimensions, Correlates, & Antecedents*. Austin, TX: University of Texas Press.

Steptoe, A., Wardle, J., Cui, W., Baban, A., Glass, K., Tsuda, A., et al. (2002). An international comparison of tobacco smoking, beliefs and risk awareness in university students from 23 countries. *Addiction, 97*, 1561-1571.

Stockley, C. S. (1998). Wine in moderation: How could and should recent in vitro and in vivo data be interpreted? *Drug and Alcohol Review, 17*, 365-376.

Taylor, K. L., Cox, L. S., Zincke, N., Mehta, L., McGuire, C., & Gelmann, E. (2007). Lung cancer screening as a teachable moment for smoking cessation. *Lung Cancer, 56*, 125-134.

Taylor, S. E., & Brown, J. D. (1988). Illusion and well-being: A social psychological perspective on mental health. *Psychological Bulletin, 103*, 193-210.

Taylor, S. E., Lichtman, R. R., & Wood, J. V. (1984). Attributions, beliefs about control, and adjustment to breast cancer. *Journal of Personality and Social Psychology, 46*, 489-502.

Taylor, S. E., & Lobel, M. (1989). Social comparison activity under threat: Downward evaluation and upward contacts. *Psychological Review, 96*, 569-575.

Timperio, A., Salmon, J., & Crawford, D. (2003). Validity and reliability of a physical activity recall instrument among overweight and non-overweight men and women. *Journal of Science and Medicine in Sport, 6*, 477-491.

Tversky, A., & Kahneman, D. (1973). Availability: A heuristic for judging frequency and probability. *Cognitive Psychology, 5*, 207-232.

Tversky, A., & Kahneman, D. (1974). Judgment under uncertainty: Heuristics and biases. *Science, 185*, 1124-1131.

U.S. Cancer Statistics Working Group. (2007). *United States Cancer Statistics: 2004 Incidence and Mortality*. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, and National Cancer Institute.

U.S. Department of Health and Human Services. (2008, January 23). *Fact Sheet: Health Effects of Cigarette Smoking*. Retrieved January 9, 2009, from http://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/health_effects.htm

van der Pligt, J. (1998). Perceived risk and vulnerability as predictors of precautionary behaviour. *British Journal of Health Psychology, 3*, 1-14.

Vernon, S. W., Vogel, V. G., Halabi, S., & Bondy, M. L. (1993). Factors associated with perceived risk of breast cancer among women attending a screening program. *Breast Cancer Research and Treatment, 28*, 137-144.

Visser, P. S., Krosnick, J. A., & Lavrakas, P. J. (2000). Survey research. In H. T. Reis & C. M. Judd (Eds.), *Handbook of Research Methods in Social and Personality Psychology* (pp. 223-252). Cambridge, UK: Cambridge University Press.

Wallston, K. A. (2004). Multidimensional Health Locus of Control Scale. In A. J. Christensen, R. Martin & J. Smyth (Eds.), *Encyclopedia of Health Psychology* (pp. 171-172). New York: Kluwer Academic/Plenum.

Wallston, K. A., Wallston, B. S., & DeVellis, R. (1978). Development of the Multidimensional Health Locus of Control (MHLC) scales. *Health Education Monographs, 6*, 160-170.

Weiner, B., Perry, R. P., & Magnusson, J. (1988). An attributional analysis of reactions to stigmas. *Journal of Personality and Social Psychology, 55*, 738-748.

Weinstein, N. D. (1980). Unrealistic optimism about future life events. *Journal of Personality and Social Psychology, 39*, 806.

Weinstein, N. D. (1984). Why it won't happen to me: Perceptions of risk factors and susceptibility. *Health Psychology, 3*, 431-457.

Weinstein, N. D. (1987). Unrealistic optimism about susceptibility to health problems: Conclusions from a community-wide sample. *Journal of Behavioral Medicine, 10*, 481-500.

Weinstein, N. D. (1993). Testing four competing theories of health-protective behavior. *Health Psychology, 12*, 324-333.

Weinstein, N. D. (1998). Accuracy of smokers' risk perceptions. *Annals of Behavioral Medicine, 20*, 135-140.

Weinstein, N. D., & Klein, W. M. (1995). Resistance of personal risk perceptions to debiasing interventions. *Health Psychology, 14*, 132-140.

Wilcox, S., & Stefanick, M. L. (1999). Knowledge and perceived risk of major diseases in middle-aged and older women. *Health Psychology, 18*, 346-353.

Williams, B. G., Iredale, R., Brain, K., France, E., Barrett-Lee, P., & Gray, J. (2003). Experiences of men with breast cancer: An exploratory focus group study. *British Journal of Cancer, 89*, 1834-1836.

Wills, T. A. (1981). Downward comparison principles in social psychology. *Psychological Bulletin, 90*, 245-271.

Wood, J. V., Taylor, S. E., & Lichtman, R. R. (1985). Social comparison in adjustment to breast cancer. *Journal of Personality and Social Psychology, 49*, 1169-1183.

Yung, Y. F., & Bentler, P. M. (1994). Bootstrap-corrected ADF test statistics in covariance structure analysis. *British Journal of Mathematical & Statistical Psychology, 47*, 63-84.

Table 1

Correlations Among Major Study Variables in Younger Women (n = 454)

	1	2	3	4	5	6	7
1. CVD risk for self	—	.34***	.25***	.26***	.25***	.02	-.03
2. CVD risk for avg. w.		—	.38***	.37***	.21***	.09	.06
3. CVD prevalence			—	.80***	.05	.09*	.54***
4. CVD mortality				—	.10*	.12*	.52***
5. BRCA risk for self					—	.35***	.18***
6. BRCA risk for avg. w.						—	.40***
7. BRCA prevalence							—
8. BRCA mortality							
9. LUCA risk for self							
10. LUCA risk for avg. w.							
11. LUCA prevalence							
12. LUCA mortality [a]							
13. Exercise							
14. Cigarette use							
15. Alcohol use							
16. Vitamin use							
17. Monitor calories							
18. Balanced diet							
19. Breast self-exams							
20. Mammograms							
21. Physicals from doctor							
22. Optimism							
23. MHLC – Internal							
24. MHLC – Chance							
25. MHLC – Others							
26. PAQ – Femininity [b]							
27. PAQ – Masculinity [b]							
28. CVD social exposure [c]							
29. BRCA social exposure [c]							
30. LUCA social exposure [c]							
31. CVD stigma							
32. BRCA stigma							
33. LUCA stigma							
34. CVD family history [d]							
35. BRCA family history [d]							
36. LUCA family history [d]							
37. Age							
38. Body mass index [c]							
39. CVD health beliefs							
40. BRCA health beliefs							
41. LUCA health beliefs.							
M	1.83	2.34	37.33	22.72	1.97	2.35	41.55
SD	.87	.58	18.68	14.91	.85	.58	18.83

Note. Table 1 continues on next page.

Table 1 (continued)

	8	9	10	11	12	13	14
1. CVD risk for self	.00	.30***	.11*	.03	.09	-.00	.18***
2. CVD risk for avg. w.	.08	.18***	.15***	.09	.07	.12*	.12*
3. CVD prevalence	.46***	.02	.13**	.41***	.37***	.02	-.08
4. CVD mortality	.55***	-.01	.10*	.38***	.46***	-.02	-.03
5. BRCA risk for self	.18***	.22***	.08	-.01	.05	.02	.05
6. BRCA risk for avg. w.	.38***	.00	.15***	.14**	.17***	-.07	-.09
7. BRCA prevalence	.80***	.04	.18***	.59***	.54***	-.04	-.08
8. BRCA mortality	—	.04	.15**	.47***	.53***	-.08	-.09
9. LUCA risk for self		—	.33***	.19***	.14**	.06	.52***
10. LUCA risk for avg. w.			—	.49***	.42***	.14**	.13**
11. LUCA prevalence				—	.85***	.03	.01
12. LUCA mortality [a]					—	-.02	.00
13. Exercise						—	.09
14. Cigarette use							—
15. Alcohol use							
16. Vitamin use							
17. Monitor calories							
18. Balanced diet							
19. Breast self-exams							
20. Mammograms							
21. Physicals from doctor							
22. Optimism							
23. MHLC – Internal							
24. MHLC – Chance							
25. MHLC – Others							
26. PAQ – Femininity [b]							
27. PAQ – Masculinity [b]							
28. CVD social exposure [c]							
29. BRCA social exposure [c]							
30. LUCA social exposure [c]							
31. CVD stigma							
32. BRCA stigma							
33. LUCA stigma							
34. CVD family history [d]							
35. BRCA family history [d]							
36. LUCA family history [d]							
37. Age							
38. Body mass index [c]							
39. CVD health beliefs							
40. BRCA health beliefs							
41. LUCA health beliefs							
M	23.58	1.57	1.98	32.48	21.11	2.23	.59
SD	15.65	.96	.57	18.92	16.51	1.23	1.19

Note. Table 1 continues on next page.

Table 1 (continued)

	15	16	17	18	19	20	21
1. CVD risk for self	.07	-.00	.01	-.12*	.04	.03	-.07
2. CVD risk for avg. w.	.03	.11*	.14**	.05	.04	-.07	-.03
3. CVD prevalence	-.05	-.01	.01	-.07	.01	-.04	.03
4. CVD mortality	-.01	-.01	.00	-.02	-.01	-.07	.04
5. BRCA risk for self	.13**	.04	.09	.02	.09	.04	.04
6. BRCA risk for avg. w.	.06	.01	.00	.01	-.02	.02	.05
7. BRCA prevalence	.02	-.02	-.02	-.04	-.05	.04	.13**
8. BRCA mortality	.02	-.01	-.02	-.07	-.03	-.02	.14**
9. LUCA risk for self	.24***	-.03	.07	-.04	.01	.02	-.00
10. LUCA risk for avg. w.	.17***	.01	.14**	.07	-.05	.01	.09
11. LUCA prevalence	.07	-.03	.04	-.02	.00	.02	.12**
12. LUCA mortality [a]	.06	-.04	.05	.03	-.03	-.00	.11*
13. Exercise	.15**	.24***	.44***	.38***	.12*	-.01	.03
14. Cigarette use	.43***	-.02	.10*	.06	.02	.02	.00
15. Alcohol use	—	-.04	.14**	.06	.01	.07	.03
16. Vitamin use		—	.20***	.21***	.10*	.02	.08
17. Monitor calories			—	.46***	.05	-.03	.03
18. Balanced diet				—	.03	.00	-.02
19. Breast self-exams					—	.08	.09
20. Mammograms						—	.09*
21. Physicals from doctor							—
22. Optimism							
23. MHLC – Internal							
24. MHLC – Chance							
25. MHLC – Others							
26. PAQ – Femininity [b]							
27. PAQ – Masculinity [b]							
28. CVD social exposure [c]							
29. BRCA social exposure [c]							
30. LUCA social exposure [c]							
31. CVD stigma							
32. BRCA stigma							
33. LUCA stigma							
34. CVD family history [d]							
35. BRCA family history [d]							
36. LUCA family history [d]							
37. Age							
38. Body mass index [c]							
39. CVD health beliefs							
40. BRCA health beliefs							
41. LUCA health beliefs							
M	1.52	1.31	1.42	2.43	1.31	.06	1.48
SD	1.19	1.31	1.32	.99	2.34	.26	.98

Note. Table 1 continues on next page.

Table 1 (continued)

	22	23	24	25	26	27	28
1. CVD risk for self	-.17***	-.01	.13**	.08	-.04	-.14**	.36***
2. CVD risk for avg. w.	-.04	.04	-.06	-.03	.07	.05	.17***
3. CVD prevalence	-.07	.08	-.09	-.02	.05	.00	.21***
4. CVD mortality	-.06	.09	-.03	.01	.04	-.01	.19***
5. BRCA risk for self	-.13**	-.04	.04	-.03	-.05	-.20***	.07
6. BRCA risk for avg. w.	-.06	.00	.02	.00	-.04	-.06	.05
7. BRCA prevalence	-.05	.03	-.03	-.03	.02	.00	.01
8. BRCA mortality	-.07	.01	-.02	-.04	-.03	.01	.01
9. LUCA risk for self	-.13**	.06	.09	.08	.04	-.08	.09*
10. LUCA risk for avg. w.	-.08	.04	.00	.08	.06	.02	.07
11. LUCA prevalence	-.05	.08	.00	.00	.11*	.09	.05
12. LUCA mortality [a]	-.06	.10*	.04	.01	.05	.05	.04
13. Exercise	.11*	.02	.04	-.01	.09	.15**	.05
14. Cigarette use	-.03	.09*	-.01	-.08	-.01	.02	-.05
15. Alcohol use	.03	.02	.02	-.04	.04	.07	-.03
16. Vitamin use	-.02	.04	-.04	.02	-.00	.14**	.03
17. Monitor calories	.10*	.10*	-.04	-.02	.13*	.13*	.05
18. Balanced diet	.16***	.03	-.08	-.09	.10*	.18***	-.07
19. Breast self-exams	-.03	.00	-.04	.03	.07	.07	.04
20. Mammograms	-.02	-.01	.07	.02	.01	.06	.00
21. Physicals from doctor	.01	.02	.02	.07	-.08	.11*	.04
22. Optimism	—	.08	-.12*	-.06	.06	.50***	-.12*
23. MHLC – Internal		—	-.12**	.12*	.11*	.07	-.00
24. MHLC – Chance			—	.27***	-.03	-.22***	-.01
25. MHLC – Others				—	.09	-.22***	.05
26. PAQ – Femininity [b]					—	.06	.01
27. PAQ – Masculinity [b]						—	-.04
28. CVD social exposure [c]							—
29. BRCA social exposure [c]							
30. LUCA social exposure [c]							
31. CVD stigma							
32. BRCA stigma							
33. LUCA stigma							
34. CVD family history [d]							
35. BRCA family history [d]							
36. LUCA family history [d]							
37. Age							
38. Body mass index [c]							
39. CVD health beliefs							
40. BRCA health beliefs							
41. LUCA health beliefs							
M	26.88	25.63	17.18	19.23	24.25	19.16	.52
SD	5.41	3.96	4.38	3.86	3.88	4.57	.97

Note. Table 1 continues on next page.

Table 1 (continued)

	29	30	31	32	33	34	35
1. CVD risk for self	.09	.09	.05	.03	.07	.43***	.06
2. CVD risk for avg. w.	.02	.09	.08	-.05	.06	.15***	.07
3. CVD prevalence	.04	.07	.09*	.07	.03	.15***	.05
4. CVD mortality	.04	.03	.09	.03	.02	.18***	.03
5. BRCA risk for self	.27***	.16***	-.02	-.07	-.03	.06	.43***
6. BRCA risk for avg. w.	.04	.06	-.02	-.03	-.01	.05	.06
7. BRCA prevalence	.10*	.02	-.04	.09*	-.04	-.01	-.11*
8. BRCA mortality	.04	.04	-.06	.09	-.08	-.01	.06
9. LUCA risk for self	.06	.18***	.05	.12**	-.04	.07	.03
10. LUCA risk for avg. w.	-.00	.12*	.06	-.04	.12*	.10*	.04
11. LUCA prevalence	-.01	.06	-.00	.04	.09	.05	.02
12. LUCA mortality [a]	-.05	.07	-.02	.07	.10*	.07	.01
13. Exercise	.18***	.17***	.07	-.03	.06	.04	.07
14. Cigarette use	.03	.06	-.03	-.04	.03	-.01	.02
15. Alcohol use	.16***	.08	-.09*	-.12**	-.04	.11*	.11*
16. Vitamin use	.05	.07	-.02	-.05	.04	.03	.05
17. Monitor calories	.08	.04	.11*	-.06	.11*	.04	.02
18. Balanced diet	.07	.00	.07	-.06	.02	-.06	-.02
19. Breast self-exams	.10*	.07	.06	-.04	-.01	.06	.08
20. Mammograms	-.02	.01	-.02	-.01	-.01	.07	.07
21. Physicals from doctor	.02	.01	-.03	.01	-.00	.04	.04
22. Optimism	.00	-.05	.00	-.04	.01	-.09	-.08
23. MHLC – Internal	-.02	.04	-.01	.12*	.13**	.04	.12**
24. MHLC – Chance	-.06	-.03	.03	.06	.00	.07	-.07
25. MHLC – Others	-.05	.06	.07	.12**	.05	.03	-.07
26. PAQ – Femininity [b]	.16**	-.06	-.03	-.13*	.01	-.01	.09
27. PAQ – Masculinity [b]	-.03	-.03	-.02	-.05	.02	-.02	-.07
28. CVD social exposure [c]	.19***	.23***	.07	.08	.06	.48***	.05
29. BRCA social exposure [c]	—	.28***	.02	-.03	-.03	.06	.42***
30. LUCA social exposure [c]		—	.09	.10*	.00	.02	.05
31. CVD stigma			—	.45***	.42***	.02	.01
32. BRCA stigma				—	.35***	-.08	-.06
33. LUCA stigma					—	.07	.04
34. CVD family history [d]						—	.11*
35. BRCA family history [d]							—
36. LUCA family history [d]							
37. Age							
38. Body mass index [c]							
39. CVD health beliefs							
40. BRCA health beliefs							
41. LUCA health beliefs							
M	.72	.21	1.00	.73	1.48	.28	.19
SD	.97	.54	.77	.82	.86	.45	.39

Note. Table 1 continues on next page.

Table 1 (continued)

	36	37	38	39	40	41
1. CVD risk for self	.09	.16***	.13**	.07	.02	.00
2. CVD risk for avg. w.	.08	.01	.02	.17***	.07	.08
3. CVD prevalence	.03	.11*	-.03	.20***	.09*	.06
4. CVD mortality	.06	.14**	.01	.18***	.06	.03
5. BRCA risk for self	.11*	-.03	.09	.11*	.15***	-.01
6. BRCA risk for avg. w.	.00	-.04	-.04	.09	.11*	.02
7. BRCA prevalence	-.01	-.04	-.00	-.02	.05	-.05
8. BRCA mortality	.02	.01	.01	.01	.11*	-.09*
9. LUCA risk for self	.25***	.01	.03	.06	.10*	.01
10. LUCA risk for avg. w.	.16***	-.02	-.04	.15**	.17***	.18***
11. LUCA prevalence	.07	.03	.01	.08	.03	.13**
12. LUCA mortality [a]	.11*	.05	.02	.08	.05	.13**
13. Exercise	.14**	.00	.06	.15***	.12**	.15**
14. Cigarette use	.15***	.00	.02	.06	.08	.07
15. Alcohol use	.16***	.06	.04	.12*	-.01	.09
16. Vitamin use	.05	-.04	-.06	.09	.06	-.01
17. Monitor calories	.06	.13**	.16***	.10*	.12*	.12*
18. Balanced diet	.04	.08	-.02	.07	.15***	.13**
19. Breast self-exams	-.01	.05	.12*	.10*	-.06	.05
20. Mammograms	-.03	.04	-.06	-.09	.01	.01
21. Physicals from doctor	-.03	-.11*	.08	.04	.03	.05
22. Optimism	-.01	.05	-.01	.05	.08	.04
23. MHLC – Internal	.02	.07	.03	.13**	.20***	.18***
24. MHLC – Chance	.04	.08	-.04	-.13**	-.03	-.05
25. MHLC – Others	.02	.03	.05	.05	.00	.10*
26. PAQ – Femininity [b]	.03	.05	.09	.08	-.02	.15**
27. PAQ – Masculinity [b]	.04	.04	.03	.12*	.03	.04
28. CVD social exposure [c]	.08	.09	.01	.10*	.03	-.01
29. BRCA social exposure [c]	.12*	-.02	.06	.09*	.01	.04
30. LUCA social exposure [c]	.47***	-.02	-.03	.08	.09*	.01
31. CVD stigma	-.00	.10*	.03	.09	.12**	-.00
32. BRCA stigma	.01	.08	-.09*	-.06	.10*	-.03
33. LUCA stigma	-.03	.10*	-.02	.12*	.11*	.21***
34. CVD family history [d]	.07	.09	.11*	.07	-.00	.08
35. BRCA family history [d]	.13**	-.05	-.00	.05	.08	.04
36. LUCA family history [d]	—	.06	.00	.04	.08	.06
37. Age		—	.06	.05	.03	.07
38. Body mass index [c]			—	.10*	-.02	-.03
39. CVD health beliefs				—	.24***	.29***
40. BRCA health beliefs					—	.12**
41. LUCA health beliefs						—
M	.17	19.27	22.51	3.26	2.26	3.56
SD	.37	1.62	4.06	.71	.97	.73

Note. Table 1 continues on next page.

Table 1 (continued)

Note. CVD = cardiovascular disease; BRCA = breast cancer; LUCA = lung cancer; avg. w. = average woman; MHLC – Internal = Internal subscale of the Multidimensional Health Locus of Control Scale; MHLC – Chance = Chance subscale of the Multidimensional Health Locus of Control Scale; MHLC – Others = Powerful Others subscale of the Multidimensional Health Locus of Control Scale; PAQ – Femininity = Femininity subscale of the Personal Attributes Questionnaire; PAQ – Masculinity = Masculinity subscale of the Personal Attributes Questionnaire; health beliefs = beliefs in the role of health behaviors in disease etiology.

[a] = Variable was square root transformed to correct for nonnormality; correlations and descriptive statistics computed with the original variable.
[b] = For this variable, $n = 376$.
[c] = Variable was log transformed to correct for nonnormality; correlations and descriptive statistics computed with the original variable.
[d] = Variable coded as 0 = "no", 1 = "yes".

* = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$

Table 2

Correlations Among Major Study Variables in Older Women (n = 180)

	1	2	3	4	5	6	7
1. CVD risk for self	—	.17*	.17*	.17*	.17*	.10	-.04
2. CVD risk for avg. w.		—	.44***	.33***	.26***	.23**	.08
3. CVD prevalence			—	.73***	.09	.01	.42***
4. CVD mortality				—	.15	.09	.38***
5. BRCA risk for self					—	.51***	.26***
6. BRCA risk for avg. w.						—	.38***
7. BRCA prevalence							—
8. BRCA mortality							
9. LUCA risk for self							
10. LUCA risk for avg. w.							
11. LUCA prevalence							
12. LUCA mortality [a]							
13. Exercise							
14. Cigarette use							
15. Alcohol use							
16. Vitamin use							
17. Monitor calories							
18. Balanced diet							
19. Breast self-exams							
20. Mammograms							
21. Physicals from doctor							
22. Optimism							
23. MHLC – Internal							
24. MHLC – Chance							
25. MHLC – Others							
26. PAQ – Femininity [b]							
27. PAQ – Masculinity [b]							
28. CVD social exposure [c]							
29. BRCA social exposure							
30. LUCA social exposure [c]							
31. CVD stigma							
32. BRCA stigma							
33. LUCA stigma							
34. CVD family history [d]							
35. BRCA family history [d]							
36. LUCA family history [d]							
37. Age [a]							
38. Body mass index [c]							
39. CVD health beliefs							
40. BRCA health beliefs							
41. LUCA health beliefs							
M	2.11	2.51	47.00	32.61	1.94	2.28	38.18
SD	.86	.58	18.55	16.99	.80	.56	20.07

Note. Table 2 continues on next page.

Table 2 (continued)

	8	9	10	11	12	13	14
1. CVD risk for self	-.08	.33***	.07	-.01	-.00	-.20**	.14
2. CVD risk for avg. w.	.08	.11	.19*	.11	.08	-.13	-.11
3. CVD prevalence	.34***	.10	.09	.37***	.29***	.09	.00
4. CVD mortality	.44***	.11	.15*	.35***	.38***	.05	-.04
5. BRCA risk for self	.24***	.27***	.19**	.17*	.22**	.08	-.01
6. BRCA risk for avg. w.	.33***	.17*	.36***	.28***	.25***	-.03	-.09
7. BRCA prevalence	.82***	.08	.20**	.65***	.57***	-.06	.10
8. BRCA mortality	—	.05	.17*	.61***	.63***	-.02	.04
9. LUCA risk for self		—	.44***	.25***	.20**	-.11	.44***
10. LUCA risk for avg. w.			—	.42***	.34***	-.02	.23**
11. LUCA prevalence				—	.88***	-.08	.18*
12. LUCA mortality [a]					—	-.02	.06
13. Exercise						—	-.18*
14. Cigarette use							—
15. Alcohol use							
16. Vitamin use							
17. Monitor calories							
18. Balanced diet							
19. Breast self-exams							
20. Mammograms							
21. Physicals from doctor							
22. Optimism							
23. MHLC – Internal							
24. MHLC – Chance							
25. MHLC – Others							
26. PAQ – Femininity [b]							
27. PAQ – Masculinity [b]							
28. CVD social exposure [c]							
29. BRCA social exposure							
30. LUCA social exposure [c]							
31. CVD stigma							
32. BRCA stigma							
33. LUCA stigma							
34. CVD family history [d]							
35. BRCA family history [d]							
36. LUCA family history [d]							
37. Age [a]							
38. Body mass index [c]							
39. CVD health beliefs							
40. BRCA health beliefs							
41. LUCA health beliefs							
M	22.80	1.69	1.87	25.68	19.35	2.34	.54
SD	16.57	.92	.60	17.35	16.12	1.40	1.26

Note. Table 2 continues on next page.

Table 2 (continued)

	15	16	17	18	19	20	21	22
1. CVD risk for self	-.02	-.02	.02	-.07	-.14	-.01	.16*	-.17*
2. CVD risk for avg. w.	.17*	.20**	.11	.17*	.12	.04	.06	-.08
3. CVD prevalence	-.04	.10	.10	-.00	-.02	-.04	.05	-.23**
4. CVD mortality	-.03	.16*	.10	.04	-.02	.02	.01	-.29***
5. BRCA risk for self	.01	.19**	-.03	.12	.11	-.01	-.00	-.08
6. BRCA risk for avg. w.	-.09	.03	.09	-.01	-.02	.07	.05	-.14
7. BRCA prevalence	-.04	.02	.02	.02	.03	-.11	-.08	-.16*
8. BRCA mortality	-.04	-.02	.06	.01	.04	-.01	-.02	-.12
9. LUCA risk for self	-.07	.02	.00	.02	-.06	-.12	.04	-.26***
10. LUCA risk for avg. w.	-.12	.15	.05	.01	-.08	-.12	.06	-.01
11. LUCA prevalence	-.09	.05	.08	.03	.10	-.18*	-.03	-.10
12. LUCA mortality [a]	-.07	.06	.12	.10	.08	-.07	.02	-.10
13. Exercise	.23**	.11	.18*	.32***	.03	.06	.01	.09
14. Cigarette use	.01	.02	-.12	-.15*	-.02	-.14	.02	-.11
15. Alcohol use	—	-.01	-.19**	.13	-.02	.08	-.05	.03
16. Vitamin use		—	.09	.23**	-.02	.01	.08	.08
17. Monitor calories			—	.23**	.13	.07	.14	.08
18. Balanced diet				—	.18*	-.03	.06	.15*
19. Breast self-exams					—	.09	.02	.18*
20. Mammograms						—	.08	-.04
21. Physicals from doctor							—	.05
22. Optimism								—
23. MHLC – Internal								
24. MHLC – Chance								
25. MHLC – Others								
26. PAQ – Femininity [b]								
27. PAQ – Masculinity [b]								
28. CVD social exposure [c]								
29. BRCA social exposure								
30. LUCA social exposure [c]								
31. CVD stigma								
32. BRCA stigma								
33. LUCA stigma								
34. CVD family history [d]								
35. BRCA family history [d]								
36. LUCA family history [d]								
37. Age [a]								
38. Body mass index [c]								
39. CVD health beliefs								
40. BRCA health beliefs								
41. LUCA health beliefs								
M	1.41	2.39	1.84	2.92	4.27	.83	1.41	29.28
SD	1.14	1.49	1.24	.85	4.33	.53	1.09	5.11

Note. Table 2 continues on next page.

Table 2 (continued)

	23	24	25	26	27	28	29
1. CVD risk for self	-.07	-.01	-.04	.11	-.08	.29***	.15*
2. CVD risk for avg. w.	.14	-.15*	.06	.10	.08	.17*	.16*
3. CVD prevalence	.09	.05	-.11	-.03	.01	.23**	.06
4. CVD mortality	.01	-.03	-.02	.07	-.02	.08	-.00
5. BRCA risk for self	-.01	.04	-.07	.12	.04	-.05	.10
6. BRCA risk for avg. w.	-.10	-.01	.13	.05	-.01	-.08	-.04
7. BRCA prevalence	-.06	.09	.00	.09	-.01	-.06	-.12
8. BRCA mortality	-.10	.06	.01	.08	.05	-.10	-.16*
9. LUCA risk for self	.00	.08	-.03	-.05	-.09	.02	-.06
10. LUCA risk for avg. w.	.10	-.05	.12	.05	.01	-.02	-.13
11. LUCA prevalence	-.00	.06	.08	.00	.05	-.03	-.28***
12. LUCA mortality [a]	-.06	.11	.11	.04	.00	-.03	-.18*
13. Exercise	.01	-.07	.03	.05	.09	-.10	.15*
14. Cigarette use	.01	.09	-.01	-.11	.00	.01	-.14
15. Alcohol use	.02	-.08	-.06	.02	.00	.02	-.05
16. Vitamin use	.09	-.03	.00	-.01	.02	.11	.14
17. Monitor calories	.03	-.06	-.03	.18*	.01	.04	-.07
18. Balanced diet	.20**	-.16*	-.08	.12	.07	.03	.15
19. Breast self-exams	.23**	-.13	-.16*	.02	.25***	-.08	.12
20. Mammograms	-.10	.05	-.00	-.03	.00	-.03	.09
21. Physicals from doctor	.06	-.05	.06	.05	.08	.11	.11
22. Optimism	.18*	-.23**	-.02	.13	.40***	-.07	.06
23. MHLC – Internal	—	-.05	.03	.12	.26***	.04	.02
24. MHLC – Chance		—	.20**	-.07	-.13	.09	-.09
25. MHLC – Others			—	-.04	.04	.12	-.08
26. PAQ – Femininity [b]				—	.11	.03	.13
27. PAQ – Masculinity [b]					—	-.08	.04
28. CVD social exposure [c]						—	.34***
29. BRCA social exposure							—
30. LUCA social exposure [c]							
31. CVD stigma							
32. BRCA stigma							
33. LUCA stigma							
34. CVD family history [d]							
35. BRCA family history [d]							
36. LUCA family history [d]							
37. Age [a]							
38. Body mass index [c]							
39. CVD health beliefs							
40. BRCA health beliefs							
41. LUCA health beliefs							
M	25.40	16.69	19.05	25.30	20.15	1.19	2.29
SD	4.30	4.95	4.43	4.15	4.33	1.42	1.75

Note. Table 2 continues on next page.

Table 2 (continued)

	30	31	32	33	34	35	36
1. CVD risk for self	.18*	.06	-.01	.02	.38***	.13	.19**
2. CVD risk for avg. w.	.12	.08	-.02	-.06	.11	.11	.06
3. CVD prevalence	.16*	-.02	-.02	-.09	.11	.08	.07
4. CVD mortality	.13	.04	-.01	.00	.10	.10	.06
5. BRCA risk for self	.04	.11	-.00	.04	.04	.43***	.09
6. BRCA risk for avg. w.	-.06	.10	.09	.05	.10	.17*	-.11
7. BRCA prevalence	-.02	-.00	.00	-.08	.04	.10	-.03
8. BRCA mortality	-.03	.02	-.02	.00	.02	.11	-.04
9. LUCA risk for self	.11	.06	.01	-.00	.08	.18*	.15*
10. LUCA risk for avg. w.	-.01	.08	.16*	-.01	.09	.24***	-.03
11. LUCA prevalence	-.03	.05	.04	.00	.05	.02	-.00
12. LUCA mortality [a]	.02	.08	.07	.10	.05	.08	.03
13. Exercise	-.04	-.01	-.02	-.05	-.16*	.05	.02
14. Cigarette use	-.04	.03	.01	.08	-.05	.03	-.05
15. Alcohol use	-.06	-.03	.02	.11	-.05	.10	.03
16. Vitamin use	.07	.12	.10	-.03	-.02	.20**	-.02
17. Monitor calories	-.17*	.04	.01	-.05	.17*	-.06	-.02
18. Balanced diet	-.03	.02	-.03	-.06	-.03	.05	-.05
19. Breast self-exams	-.13	-.08	-.18*	-.01	-.00	.10	-.03
20. Mammograms	.00	-.05	.00	.09	-.01	.13	-.12
21. Physicals from doctor	-.06	.04	-.01	.11	.16*	.05	-.07
22. Optimism	-.00	-.14	-.10	-.03	-.18*	-.01	.03
23. MHLC – Internal	-.03	.10	.05	.13	-.01	.11	.11
24. MHLC – Chance	-.01	.04	-.02	-.03	.03	.08	.05
25. MHLC – Others	-.05	.07	.09	.01	.02	.02	-.13
26. PAQ – Femininity [b]	-.09	-.00	-.06	-.11	.25***	.16*	.05
27. PAQ – Masculinity [b]	-.08	.09	-.07	.11	-.04	.04	-.04
28. CVD social exposure [c]	.29***	.06	.10	.08	.36***	.05	.06
29. BRCA social exposure	.35***	-.05	-.06	.04	.11	.15*	.08
30. LUCA social exposure [c]	—	.03	.05	-.08	.09	.04	.20**
31. CVD stigma		—	.51***	.39***	-.00	.06	-.15*
32. BRCA stigma			—	.43***	.06	-.04	-.11
33. LUCA stigma				—	.04	-.04	-.12
34. CVD family history [d]					—	.14	.07
35. BRCA family history [d]						—	.02
36. LUCA family history [d]							—
37. Age [a]							
38. Body mass index [c]							
39. CVD health beliefs							
40. BRCA health beliefs							
41. LUCA health beliefs							
M	.54	1.14	.87	1.32	.51	.29	.16
SD	.79	.84	.82	.89	.50	.46	.37

Note. Table 2 continues on next page.

Table 2 (continued)

	37	38	39	40	41
1. CVD risk for self	-.00	.32***	.07	.02	.03
2. CVD risk for avg. w.	.02	-.04	.28***	.11	.08
3. CVD prevalence	-.09	-.01	.13	.13	.08
4. CVD mortality	-.05	-.03	.11	.15	.03
5. BRCA risk for self	.04	.03	.17*	.18*	.09
6. BRCA risk for avg. w.	.13	.12	.10	.07	.04
7. BRCA prevalence	.10	-.06	.07	.10	.01
8. BRCA mortality	.06	-.06	.07	.12	.03
9. LUCA risk for self	.01	.08	.03	.12	.13
10. LUCA risk for avg. w.	.16*	-.08	.08	.10	.07
11. LUCA prevalence	.11	-.08	.18*	.14	.07
12. LUCA mortality [a]	.11	-.09	.19*	.13	.12
13. Exercise	-.10	-.22**	.03	.08	.04
14. Cigarette use	-.07	.04	-.05	.04	-.12
15. Alcohol use	-.20**	-.24***	.09	.04	.04
16. Vitamin use	.21**	-.07	.10	.17*	-.03
17. Monitor calories	-.04	.08	.12	.02	.04
18. Balanced diet	.02	-.04	.09	.19*	.00
19. Breast self-exams	-.00	.08	.09	.04	.08
20. Mammograms	.09	-.15*	.03	.05	.07
21. Physicals from doctor	.15*	.35***	.01	.09	.05
22. Optimism	.00	.02	.08	.06	.08
23. MHLC – Internal	-.05	-.01	.07	.25***	-.01
24. MHLC – Chance	-.02	.01	-.20**	-.06	-.07
25. MHLC – Others	.16*	.01	.03	.14	.04
26. PAQ – Femininity [b]	-.23**	-.03	.20**	.21**	.12
27. PAQ – Masculinity [b]	-.01	.03	.05	.23**	-.02
28. CVD social exposure [c]	.08	.05	.01	.01	.13
29. BRCA social exposure	.04	.11	.11	.13	.16*
30. LUCA social exposure [c]	.10	-.09	.10	.04	.17*
31. CVD stigma	.14	.03	.01	-.06	-.19**
32. BRCA stigma	.17*	-.09	-.07	.13	-.12
33. LUCA stigma	.04	-.03	.09	.07	.08
34. CVD family history [d]	-.03	.09	.01	-.04	.04
35. BRCA family history [d]	.03	.06	.03	.16*	.12
36. LUCA family history [d]	-.03	-.03	.06	-.01	.08
37. Age [a]	—	.00	.03	.06	-.07
38. Body mass index [c]		—	-.15*	-.08	-.08
39. CVD health beliefs			—	.28***	.41***
40. BRCA health beliefs				—	.21**
41. LUCA health beliefs					—
M	53.00	26.96	3.29	2.42	3.28
SD	8.26	6.24	.63	.82	.76

Note. Table 2 continues on next page.

Table 2 (continued)

Note. CVD = cardiovascular disease; BRCA = breast cancer; LUCA = lung cancer; avg. w. = average woman; MHLC – Internal = Internal subscale of the Multidimensional Health Locus of Control Scale; MHLC – Chance = Chance subscale of the Multidimensional Health Locus of Control Scale; MHLC – Others = Powerful Others subscale of the Multidimensional Health Locus of Control Scale; PAQ – Femininity = Femininity subscale of the Personal Attributes Questionnaire; PAQ – Masculinity = Masculinity subscale of the Personal Attributes Questionnaire; health beliefs = beliefs in the role of health behaviors in disease etiology.

[a] = Variable was square root transformed to correct for nonnormality; correlations and descriptive statistics computed with the original variable.
[b] = For this variable, $n = 179$.
[c] = Variable was log transformed to correct for nonnormality; correlations and descriptive statistics computed with the original variable.
[d] = Variable coded as 0 = "no", 1 = "yes".

* = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$

Table 3

Participant Characteristics

Demographic variables	Younger women (n = 454)	Older women (n = 180)
Annual household income		
Less than $10,000	3.5%	1.1%
$10,000 to $30,000	15.2%	5.6%
$30,000 to $50,000	21.8%	14.4%
$50,000 to $70,000	22.9%	16.1%
$70,000 or more	36.6%	62.8%
Race or ethnicity		
African-American or Black	12.3%	2.2%
Asian or Pacific Islander	31.7%	3.9%
Latino or Hispanic	10.1%	6.1%
Multiethnic	4.6%	1.7%
Native American	0.2%	0%
White or European American	41.0%	86.1%
Highest level of education completed		
Some high school or less	0.4%	2.8%
Completed high school	24.2%	23.3%
Some college / Associate's degree / Trade school	70.9%	37.2%

Note. Table 3 continues on next page.

Table 3 (continued)

Demographic variables	Younger women (n = 454)	Older women (n = 180)
Completed college / Bachelor's degree	4.2%	14.4%
Some graduate school	0%	3.3%
Master's degree	0%	18.3%
Ph.D. or M.D.	0.2%	0.6%

Table 4

Differences in Risk Perceptions by Disease

Risk perceptions	Cardiovascular disease *M* ± *SD*	Breast cancer *M* ± *SD*	Lung cancer *M* ± *SD*	*F* value
Younger women (*n* = 454)				
Perceived risk for self	$1.83 \pm .87_a$	$1.97 \pm .85_b$	$1.57 \pm .96_c$	30.46
Perceived risk for average woman	$2.34 \pm .58_a$	$2.35 \pm .58_a$	$1.98 \pm .57_b$	69.66
Perceived prevalence	$37.33 \pm 18.68_a$	$41.55 \pm 18.83_b$	$32.48 \pm 18.92_c$	54.22
Perceived mortality	$22.72 \pm 14.91_a$	$23.58 \pm 15.65_a$	$21.11 \pm 16.51_b$	5.96
Older women (*n* = 180)				
Perceived risk for self	$2.11 \pm .86_a$	$1.94 \pm .80_b$	$1.69 \pm .92_c$	14.60
Perceived risk for average woman	$2.51 \pm .58_a$	$2.28 \pm .56_b$	$1.87 \pm .60_c$	73.79
Perceived prevalence	$47.00 \pm 18.55_a$	$38.18 \pm 20.07_b$	$25.68 \pm 17.35_c$	113.34
Perceived mortality	$32.61 \pm 16.99_a$	$22.80 \pm 16.57_b$	$19.35 \pm 16.12_c$	59.80

Note. Values within a row that share the same subscript do not differ significantly using Tukey's HSD test ($p > .05$).

Response scale for perceived risk for self and perceived risk for average woman is 0-4; response scale for perceived prevalence and perceived mortality is 0-100.

All *F* values significant at $p < .01$.

Table 5

Differences in Risk Perceptions by Subsample

Risk perceptions	Younger women (n = 454) $M \pm SD$	Older women (n = 180) $M \pm SD$	t-test
Cardiovascular disease			
Perceived risk for self	1.83 ± .87	2.11 ± .86	$t(632) = -3.66$***
Perceived risk for average woman	2.34 ± .58	2.51 ± .58	$t(632) = -3.20$***
Perceived prevalence	37.33 ± 18.68	47.00 ± 18.55	$t(632) = -5.89$***
Perceived mortality	22.72 ± 14.91	32.61 ± 16.99	$t(632) = -7.23$***
Breast cancer			
Perceived risk for self	1.97 ± .85	1.94 ± .80	$t(632) = .41$
Perceived risk for average woman	2.35 ± .58	2.28 ± .56	$t(632) = 1.37$
Perceived prevalence	41.55 ± 18.83	38.18 ± 20.07	$t(632) = 1.99$*
Perceived mortality	23.58 ± 15.65	22.80 ±16.57	$t(632) = .56$
Lung cancer			
Perceived risk for self	1.57 ± .96	1.69 ± .92	$t(632) = -1.34$
Perceived risk for average woman	1.98 ± .57	1.87 ± .60	$t(632) = 2.16$*
Perceived prevalence	32.48 ± 18.92	25.68 ± 17.35	$t(632) = 4.17$***

Note. Table 5 continues on next page.

Table 5 (continued)

Risk perceptions	Younger women (n = 454) $M \pm SD$	Older women (n = 180) $M \pm SD$	t-test
Perceived mortality	21.11 ± 16.51	19.35 ± 16.12	$t(632) = 1.21$

Note. Response scale for perceived risk for self and perceived risk for average woman is 0-4; response scale for perceived prevalence and perceived mortality is 0-100.

* = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$

Table 6

Differences in Risk Perceptions by Target

Risk perceptions	Self *M* ± *SD*	Average woman *M* ± *SD*	*F* value	Effect size (η^2)
Younger women (*n* = 454)				
Cardiovascular disease	1.83 ± .87	2.34 ± .58	160.04***	.26
Breast cancer	1.97 ± .85	2.35 ± .58	94.61***	.17
Lung cancer	1.57 ± .96	1.98 ± .57	84.61***	.16
			196.27*** [a]	.30 [a]
Older women (*n* = 180)				
Cardiovascular disease	2.11 ± .86	2.51 ± .58	31.59***	.15
Breast cancer	1.94 ± .80	2.28 ± .56	43.12***	.19
Lung cancer	1.69 ± .92	1.87 ± .60	8.58**	.05
			20.93*** [a]	.11 [a]

Note. Response scale for perceived risk for self and perceived risk for average woman is 0-4.

[a] = Results when the frequency of cigarette use was entered as a covariate in the analysis to control for the effect of this variable on risk perceptions for lung cancer.

** = $p \leq .01$; *** = $p \leq .001$

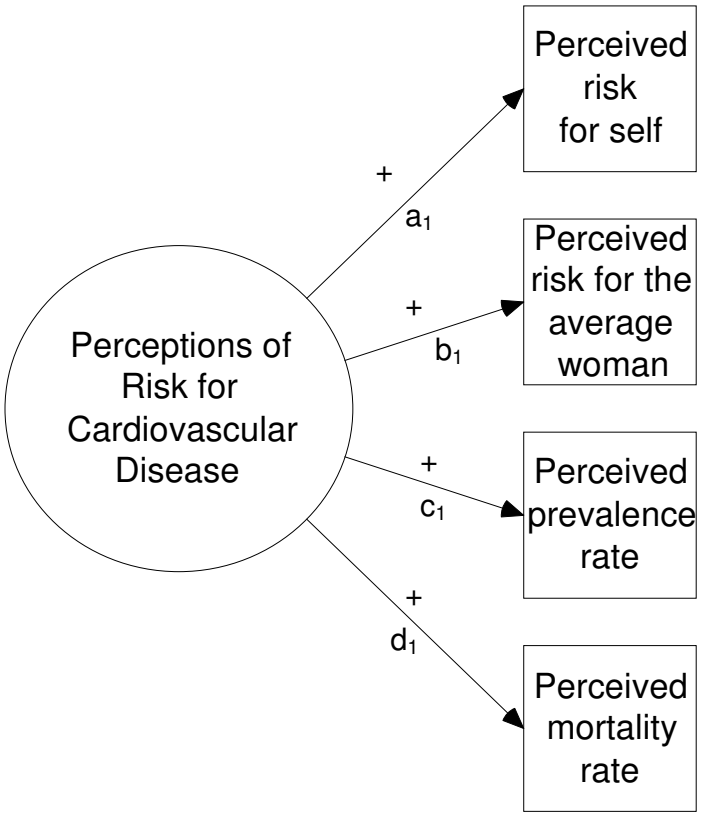


Figure 1.1. Hypothesized measurement model for the latent variable representing perceptions of risk for cardiovascular disease. Signs above the paths indicate the predicted direction of the association between study variables. Labels for each path are located below the path. For simplicity, error terms have been omitted.

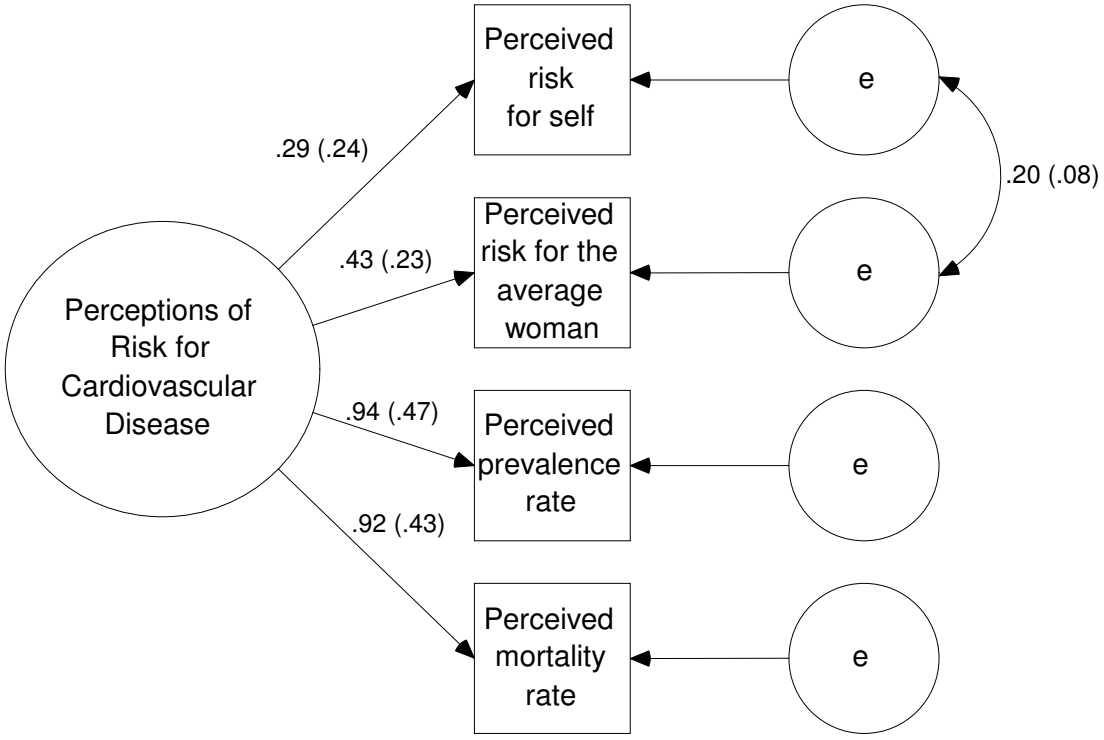


Figure 1.1a. Final measurement model for the latent variable representing perceptions of risk for cardiovascular disease in younger women. Model was a good fit to the data from 424 women ($\chi^2(1) = 2.13$, $p = .15$; CFI = 1.00; RMSEA = .05, 90% confidence limits for the RMSEA = .00 to .15). Values outside the parentheses are the standardized path coefficients; values inside the parentheses are the unstandardized path coefficients. All paths significant at $p < .001$.

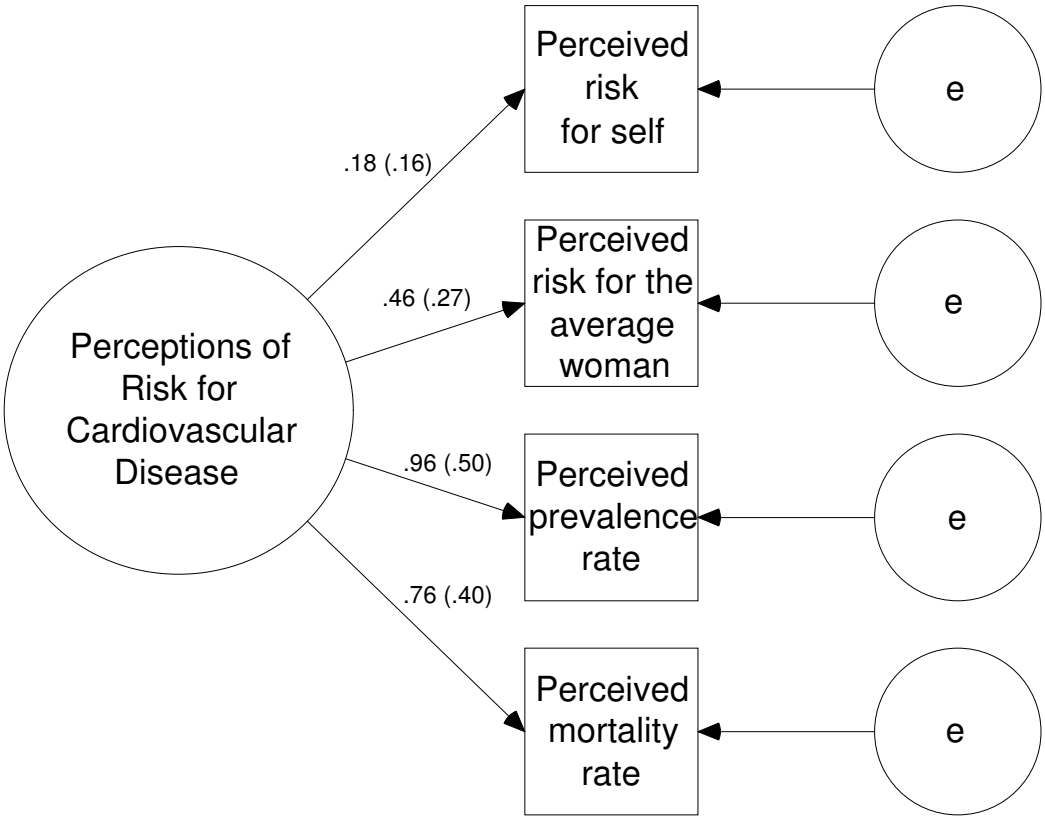


Figure 1.1b. Final measurement model for the latent variable representing perceptions of risk for cardiovascular disease in older women. Model was a good fit to the data from 180 women ($\chi^2(2) = 2.46$, $p = .29$; CFI = 1.00; RMSEA = .04, 90% confidence limits for the RMSEA = .00 to .16). Values outside the parentheses are the standardized path coefficients; values inside the parentheses are the unstandardized path coefficients. The path between the latent variable and perceptions of risk for self is significant at $p = .02$; all other paths significant at $p < .001$.

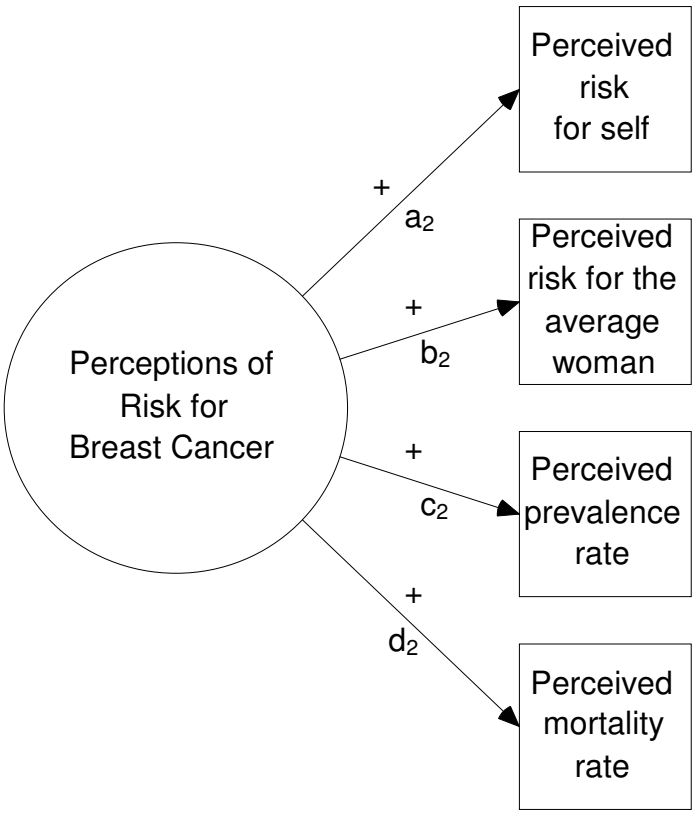


Figure 1.2. Hypothesized measurement model for the latent variable representing perceptions of risk for breast cancer. Signs above the paths indicate the predicted direction of the association between study variables. Labels for each path are located below the path. For simplicity, error terms have been omitted.

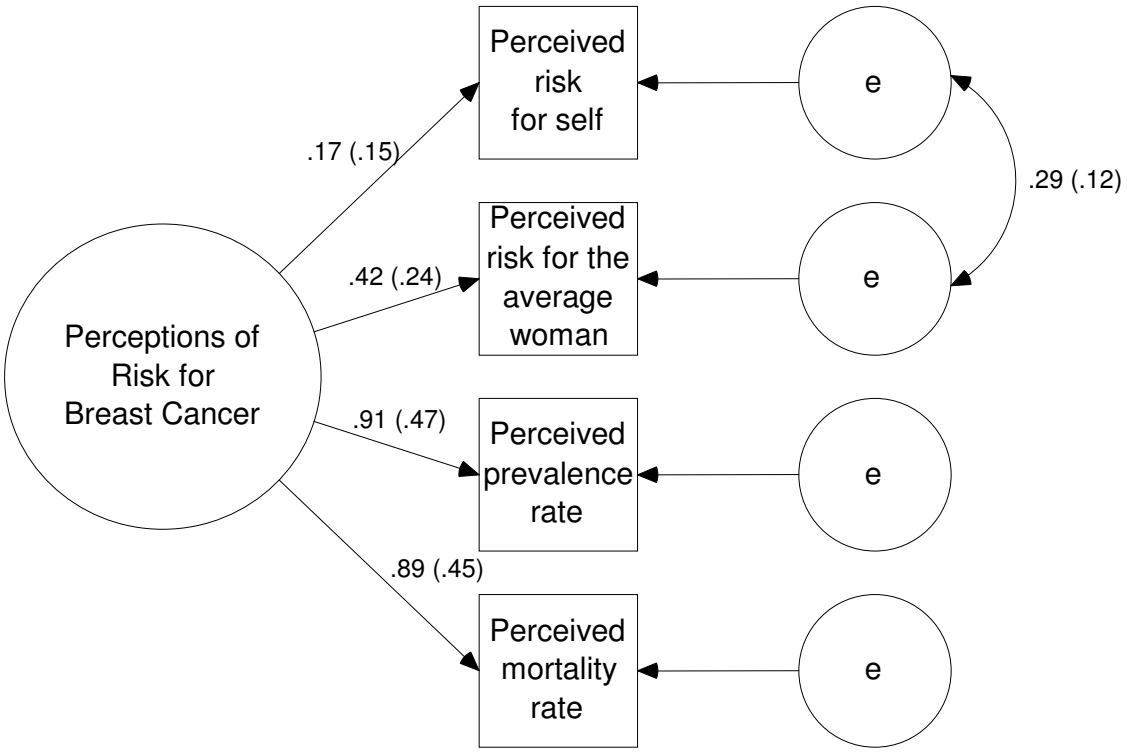


Figure 1.2a. Final measurement model for the latent variable representing perceptions of risk for breast cancer in younger women. Model was a good fit to the data from 450 women ($\chi^2(1) = 0.07$, $p = .79$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .08). Values outside the parentheses are the standardized path coefficients; values inside the parentheses are the unstandardized path coefficients. All paths significant at $p < .001$.

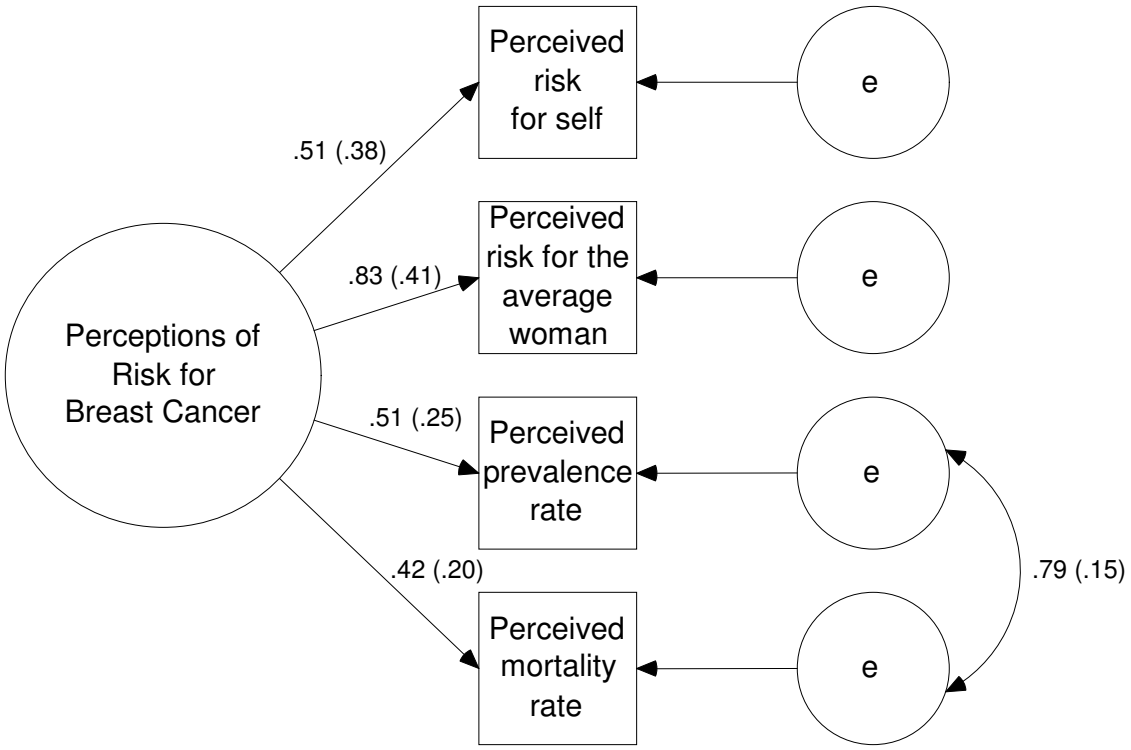


Figure 1.2b. Final measurement model for the latent variable representing perceptions of risk for breast cancer in older women. Model was a good fit to the data from 175 women ($\chi^2(1) = 0.17$, $p = .68$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .15). Values outside the parentheses are the standardized path coefficients; values inside the parentheses are the unstandardized path coefficients. All paths significant at $p < .001$.

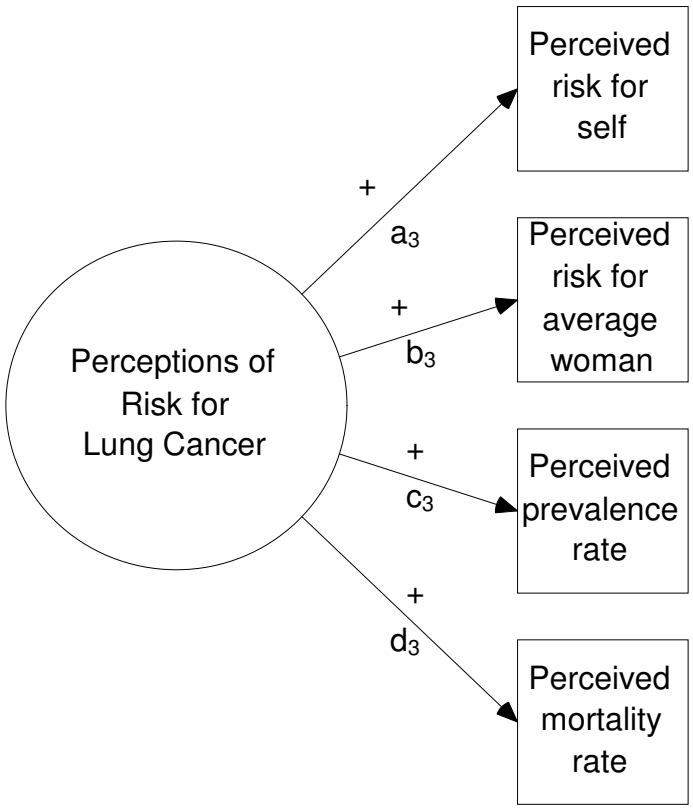


Figure 1.3. Hypothesized measurement model for the latent variable representing perceptions of risk for lung cancer. Signs above the paths indicate the predicted direction of the association between study variables. Labels for each path are located below the path. For simplicity, error terms have been omitted.

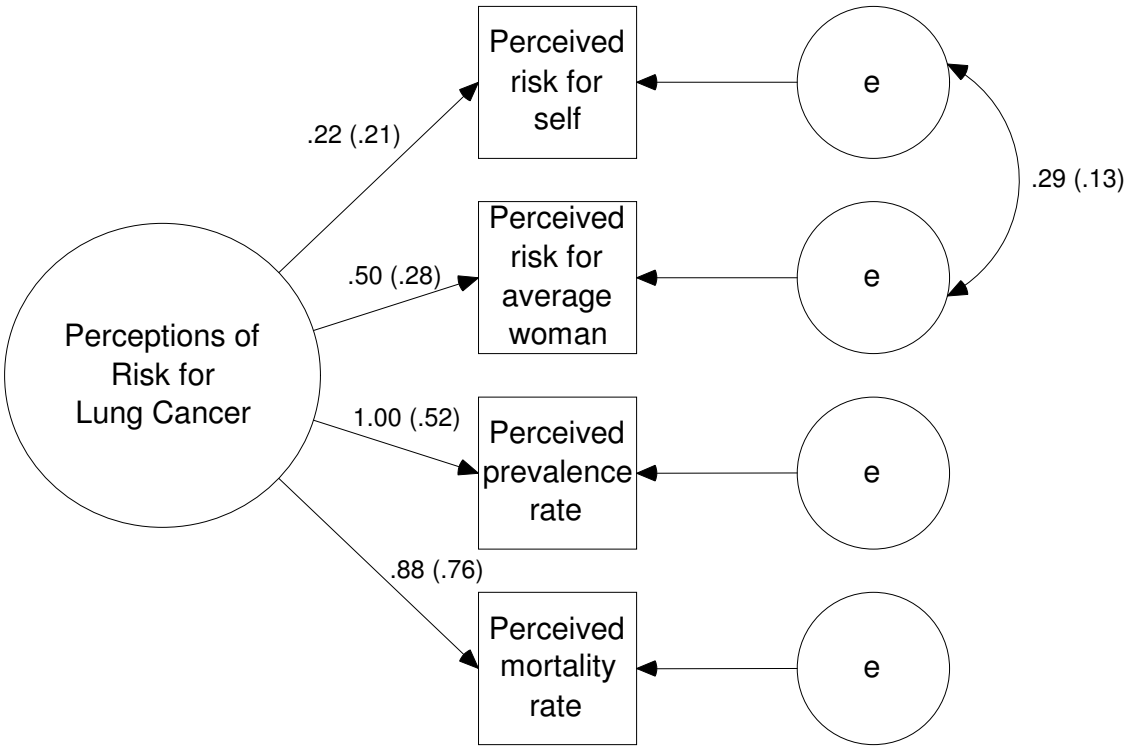


Figure 1.3a. Final measurement model for the latent variable representing perceptions of risk for lung cancer in younger women. Model was a good fit to the data from 449 women ($\chi^2(1) = 0.73$, $p = .39$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .12). Values outside the parentheses are the standardized path coefficients; values inside the parentheses are the unstandardized path coefficients. All paths significant at $p < .001$.

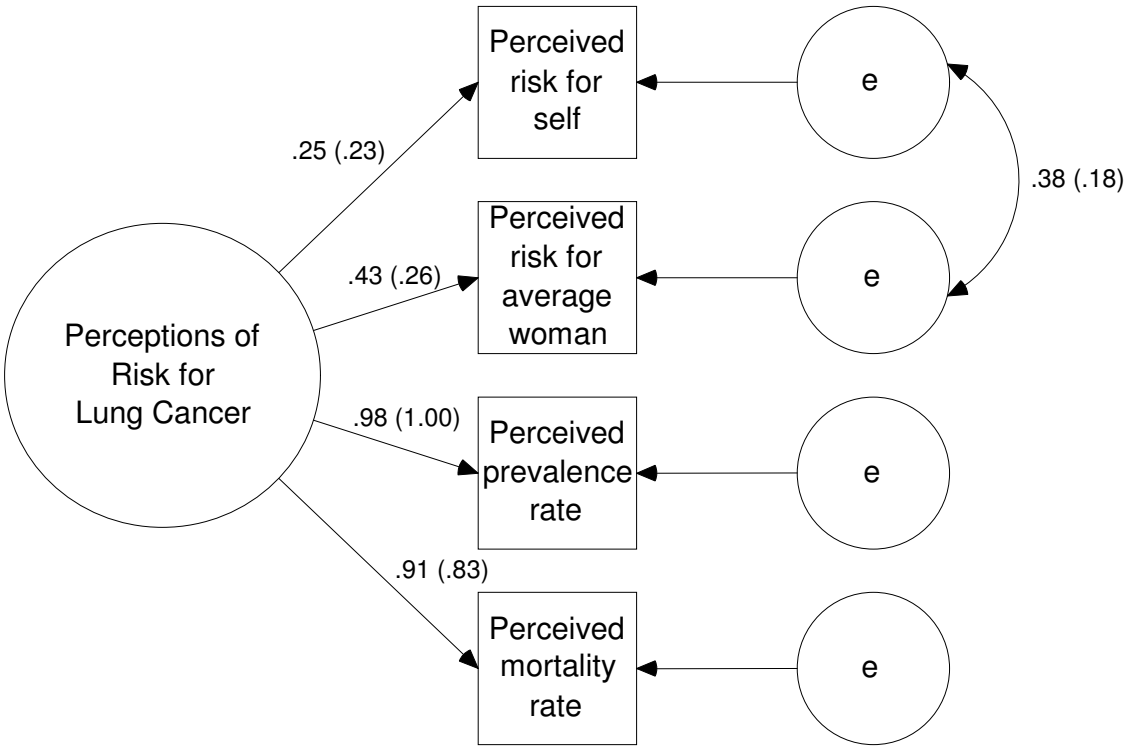


Figure 1.3b. Final measurement model for the latent variable representing perceptions of risk for lung cancer in older women. Model was a good fit to the data from 180 women ($\chi^2(1) = 1.08$, $p = .30$; CFI = 1.00; RMSEA = .02, 90% confidence limits for the RMSEA = .00 to .20). Values outside the parentheses are the standardized path coefficients; values inside the parentheses are the unstandardized path coefficients. All paths significant at $p < .001$.

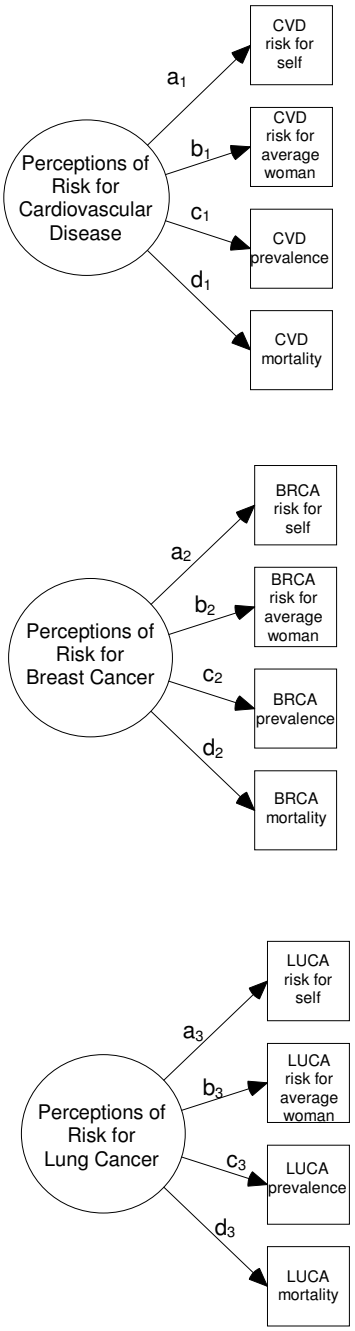


Figure 1.4. Hypothesized Disease-Specific Risk Model. Note that the latent variables representing perceptions of risk for each disease were expected to be independent of one another. For clarity, signs for the direction of the associations between variables have been omitted; they are not expected to differ from those depicted in Figures 1.1 through 1.3. Labels for each path are located above the path. For simplicity, error terms have been omitted.

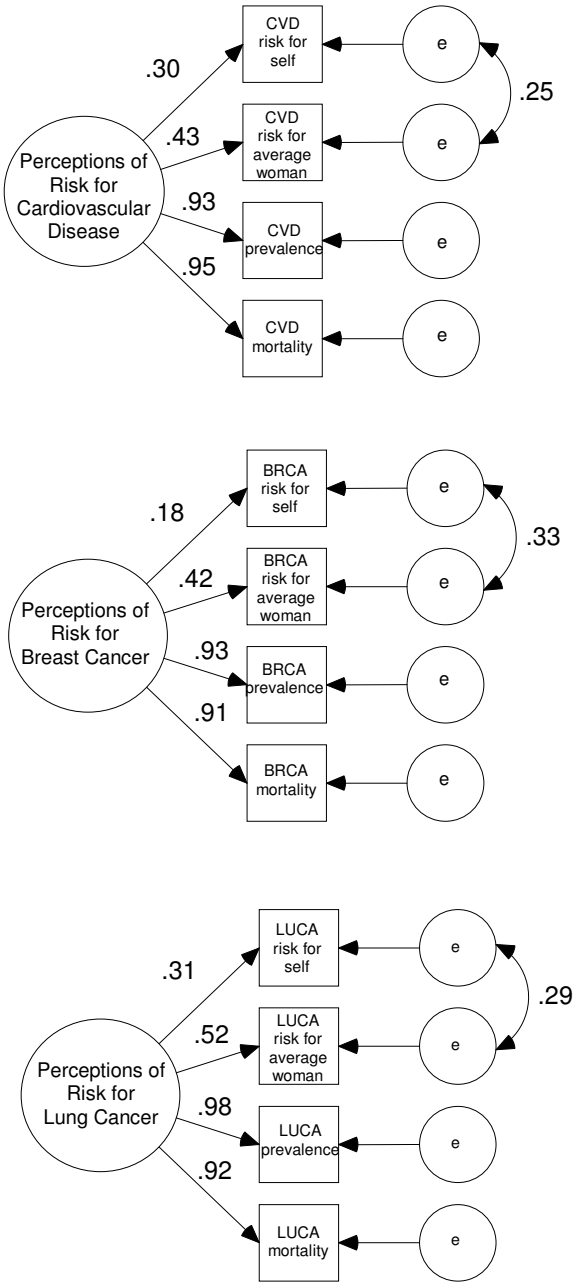


Figure 1.4a. Final Disease-Specific Risk Model in younger women. Results did not support this model, which was a poor fit to the data in a subsample of 374 women ($\chi^2(51) = 668.63$, $p < .001$; CFI = .77; RMSEA = .18, 90% confidence limits for the RMSEA = .17 to .19). All values are standardized path coefficients. All paths significant at $p < .001$.

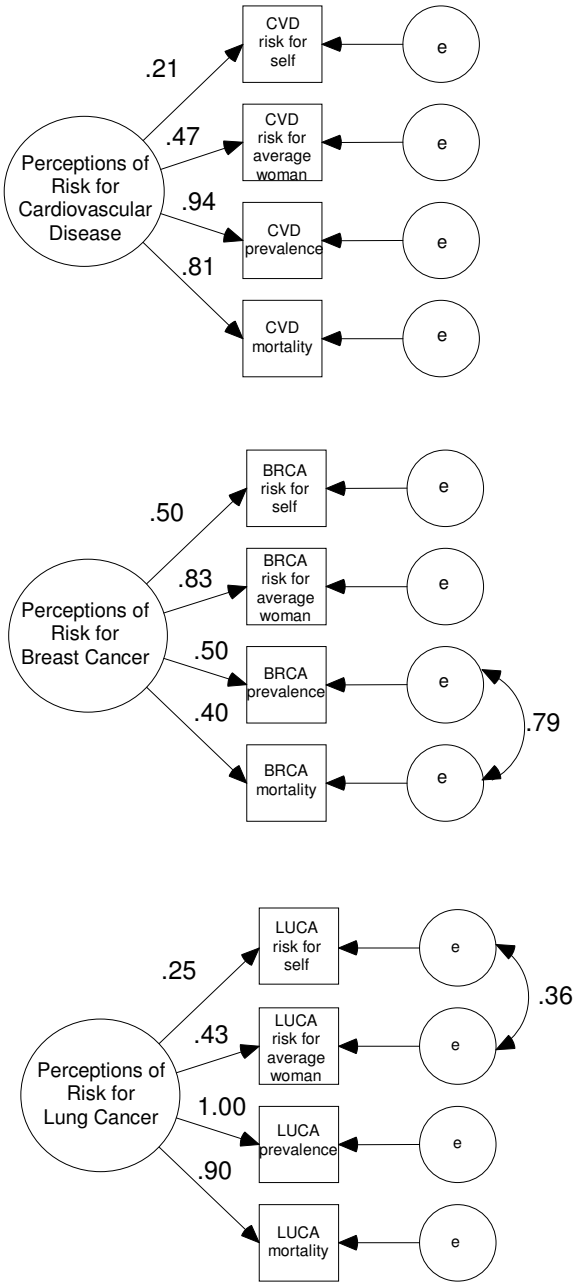


Figure 1.4b. Final Disease-Specific Risk Model in older women. Results did not support this model, which was a poor fit to the data in a subsample of 170 women ($\chi^2(52) = 294.99$, $p < .001$; CFI = .76; RMSEA = .17, 90% confidence limits for the RMSEA = .15 to .19). All values are standardized path coefficients. The path between the latent variable representing perceptions of risk for cardiovascular disease and perceptions of cardiovascular disease risk for self is significant at $p = .01$; all other paths significant at $p \leq .001$.

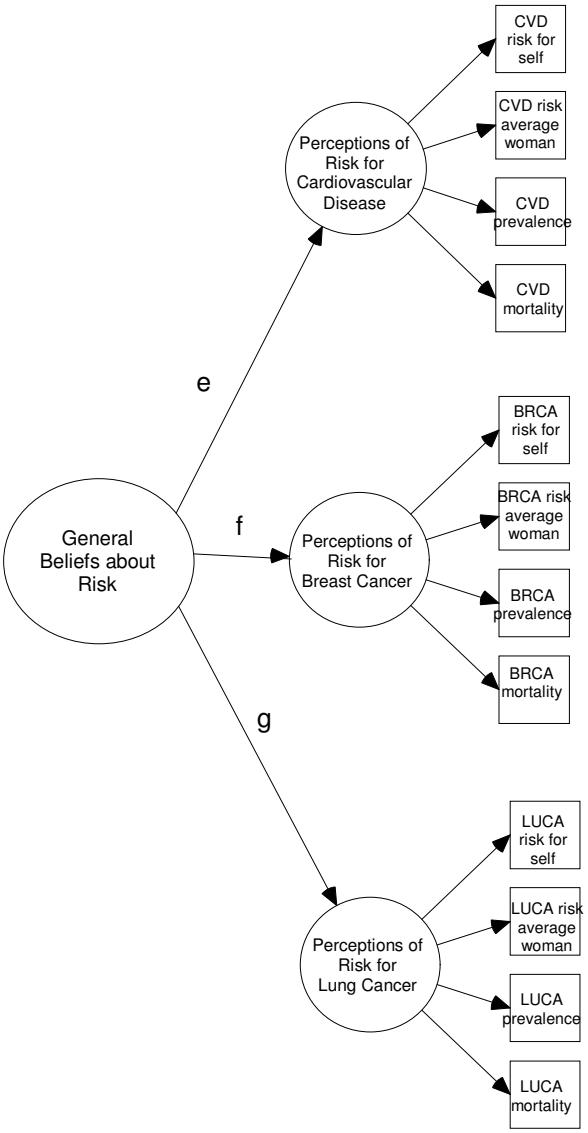


Figure 1.5. Hypothesized General Disease Risk Model. Note that the latent variables representing perceptions of risk for each disease are expected to load onto an underlying latent variable representing general beliefs about risk. For clarity, signs for the direction of the associations between variables have been omitted; they are not expected to differ from those depicted in Figures 1.1 through 1.3. Labels for each path are located above the path. For simplicity, error terms have been omitted.

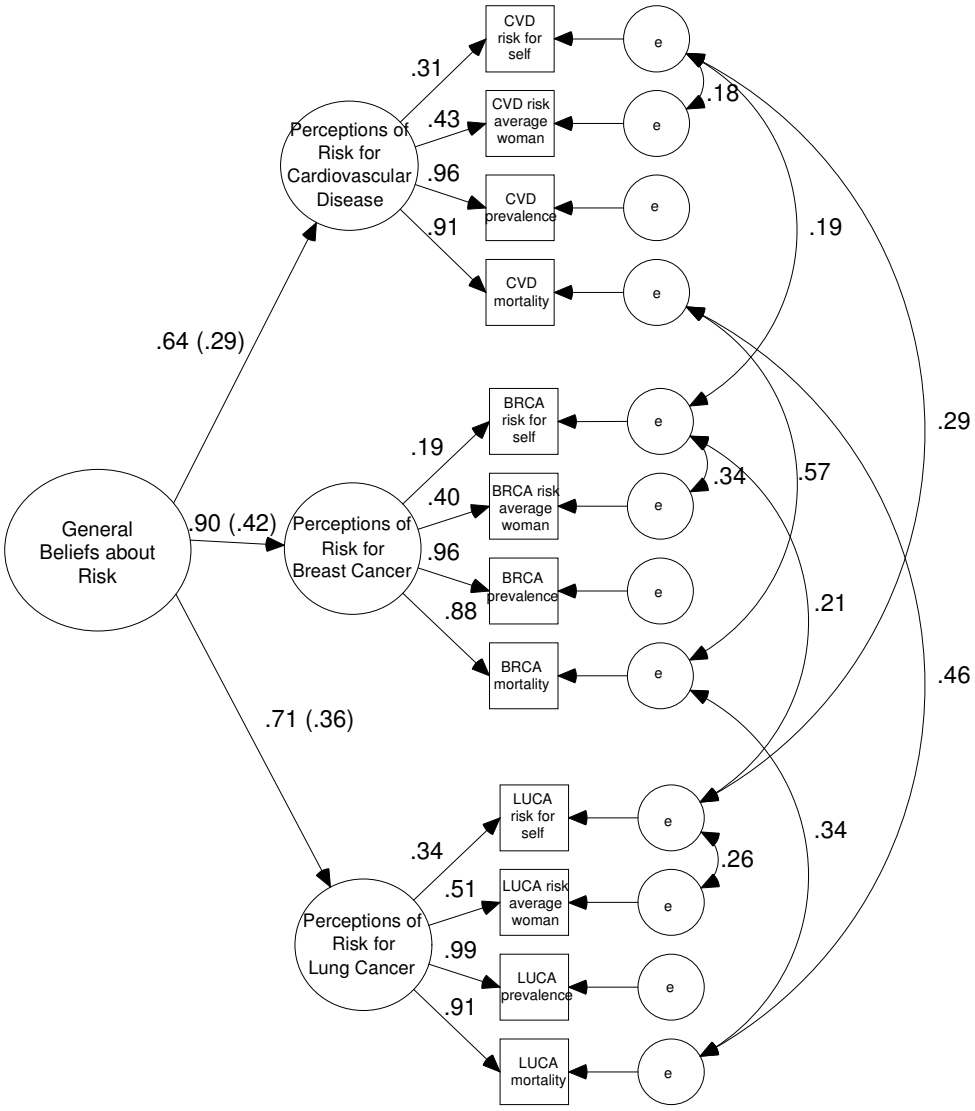


Figure 1.5a. Final General Disease Risk Model in younger women. Results supported this model, which was a good fit to the data in a subsample of 374 women ($\chi^2(42) = 141.88$, $p < .001$; CFI = .96; RMSEA = .08, 90% confidence limits for the RMSEA = .07 to .10). Values inside the parentheses are the unstandardized path coefficients; all other values are standardized path coefficients. All paths significant at $p \leq .001$.

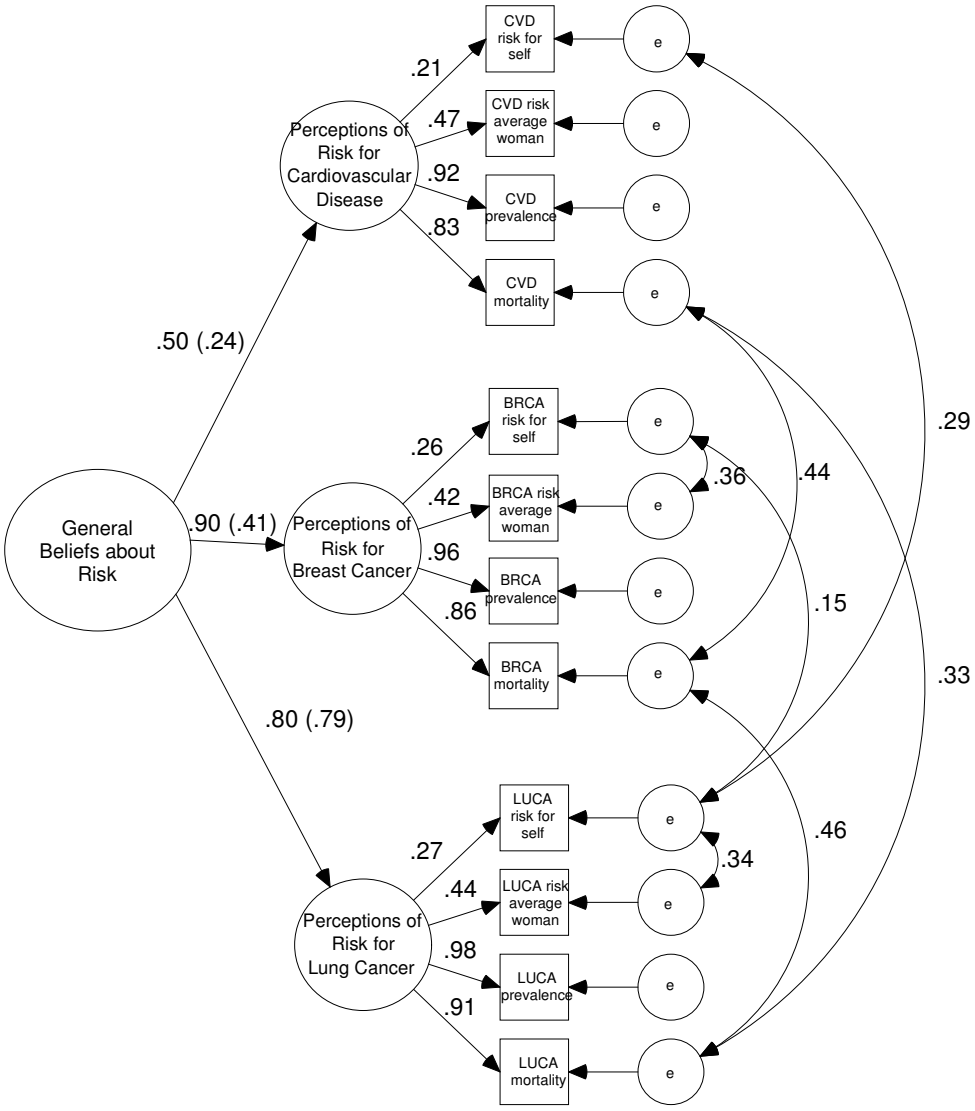


Figure 1.5b. Final General Disease Risk Model in older women. Results supported this model, which was a good fit to the data in a subsample of 170 women ($\chi^2(44) = 78.14$, $p = .001$; CFI = .97; RMSEA = .07, 90% confidence limits for the RMSEA = .04 to .09). Values inside the parentheses are the unstandardized path coefficients; all other values are standardized path coefficients. The path between the latent variable representing perceptions of risk for cardiovascular disease and perceptions of cardiovascular disease risk for the self is significant at $p = .006$; the correlation between the residuals for perceptions of breast cancer risk and lung cancer risk is significant at $p = .02$. All other paths significant at $p \leq .001$.

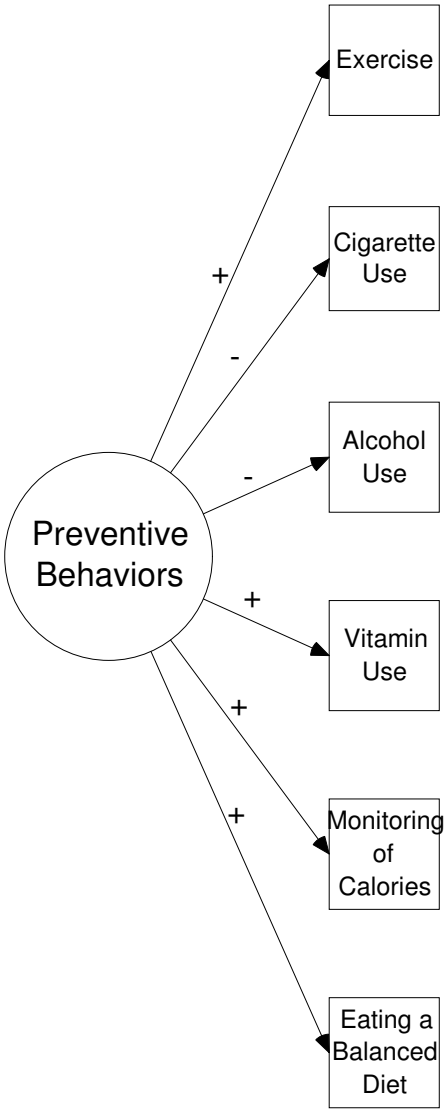


Figure 2.1. Hypothesized measurement model for the latent variable representing preventive behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

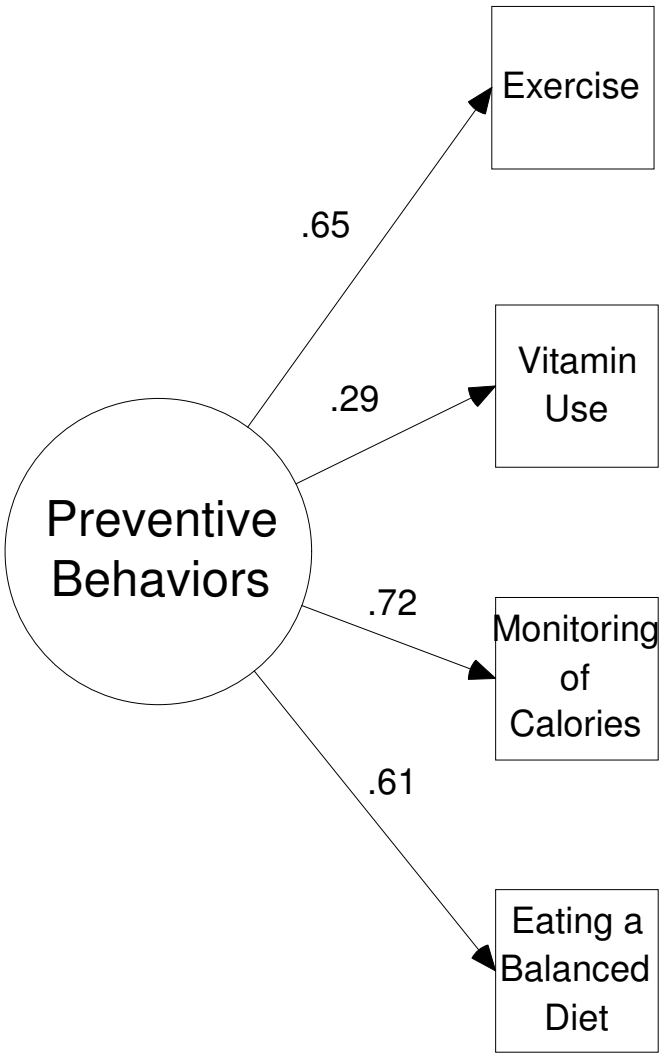


Figure 2.1a. Final measurement model (without cigarette use) for the latent variable representing preventive behaviors in younger women. The model was a good fit to the data in a subsample of 419 women ($\chi^2(2) = 4.18$, $p = .12$; CFI = .99; RMSEA = .05, 90% confidence limits for the RMSEA = .00 to .13). For simplicity, error terms have been omitted. All values are standardized path coefficients. All paths significant at $p < .001$.

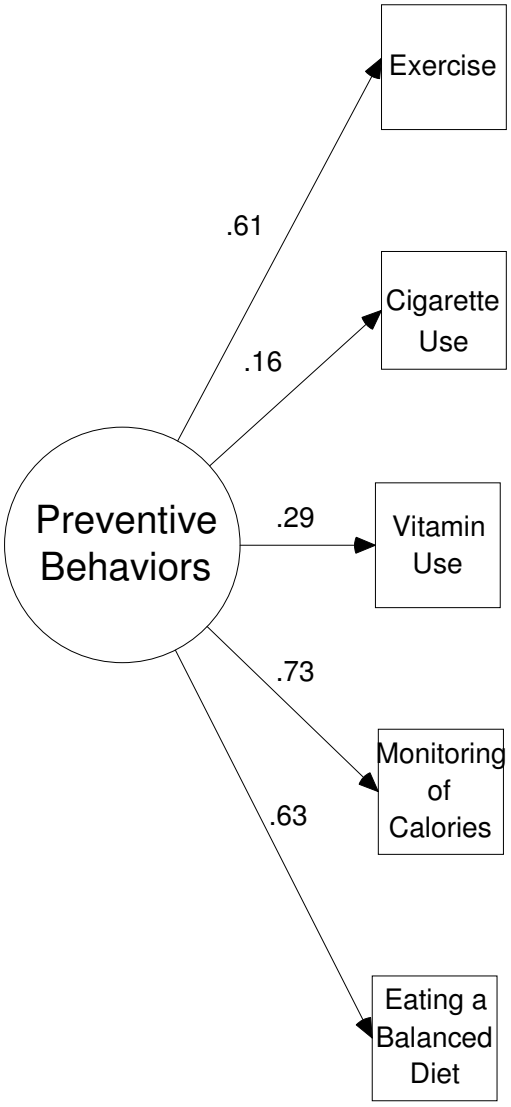


Figure 2.1b. Final measurement model (with cigarette use) for the latent variable representing preventive behaviors in younger women. The model was a good fit to the data in a subsample of 386 women ($\chi^2(5) = 6.41$, $p = .27$; CFI = .99; RMSEA = .03, 90% confidence limits for the RMSEA = .00 to .08). For simplicity, error terms have been omitted. All values are standardized path coefficients. The path between the latent variable and cigarette use is significant at $p = .007$; all other paths significant at $p < .001$.

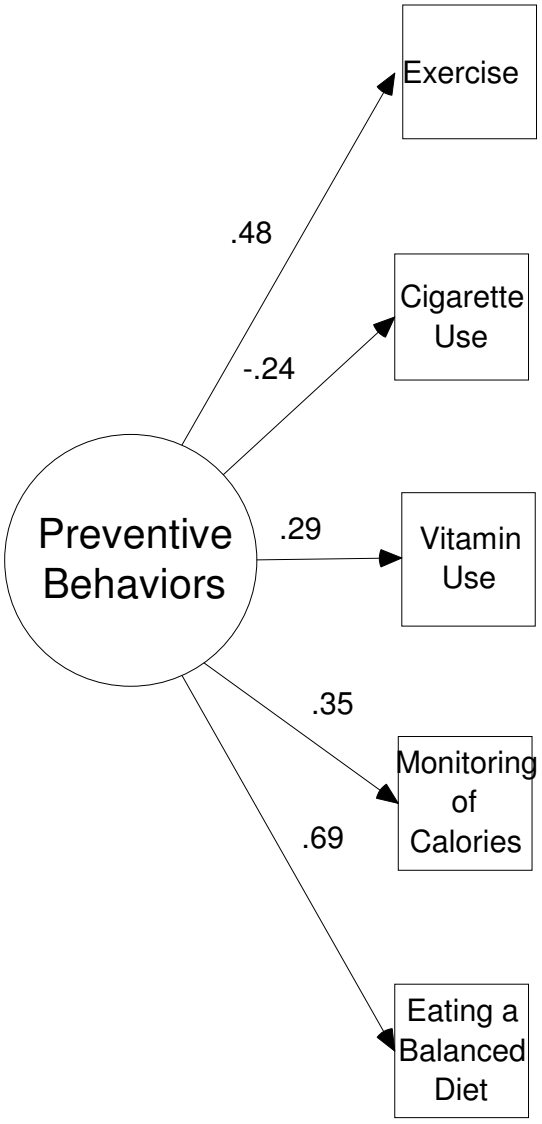


Figure 2.1c. Final measurement model for the latent variable representing preventive behaviors in older women. The model was a good fit to the data in a subsample of 175 women ($\chi^2(5) = 3.73$, $p = .59$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .09). For simplicity, error terms have been omitted. All values are standardized path coefficients. The path between the latent variable and vitamin use is significant at $p = .003$; the path between the latent variable and cigarette use is significant at $p = .02$. All other paths significant at $p < .001$.

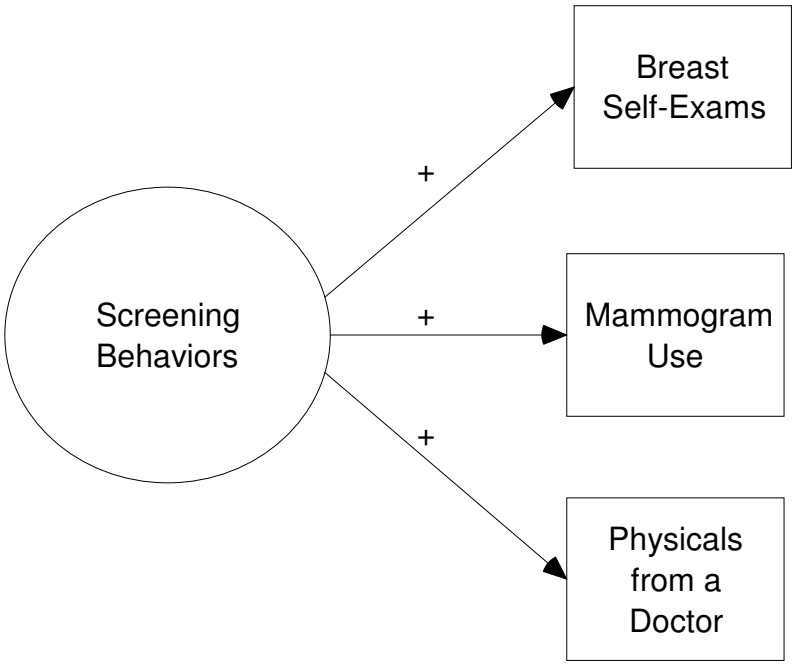


Figure 2.2. Hypothesized measurement model for the latent variable representing screening behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

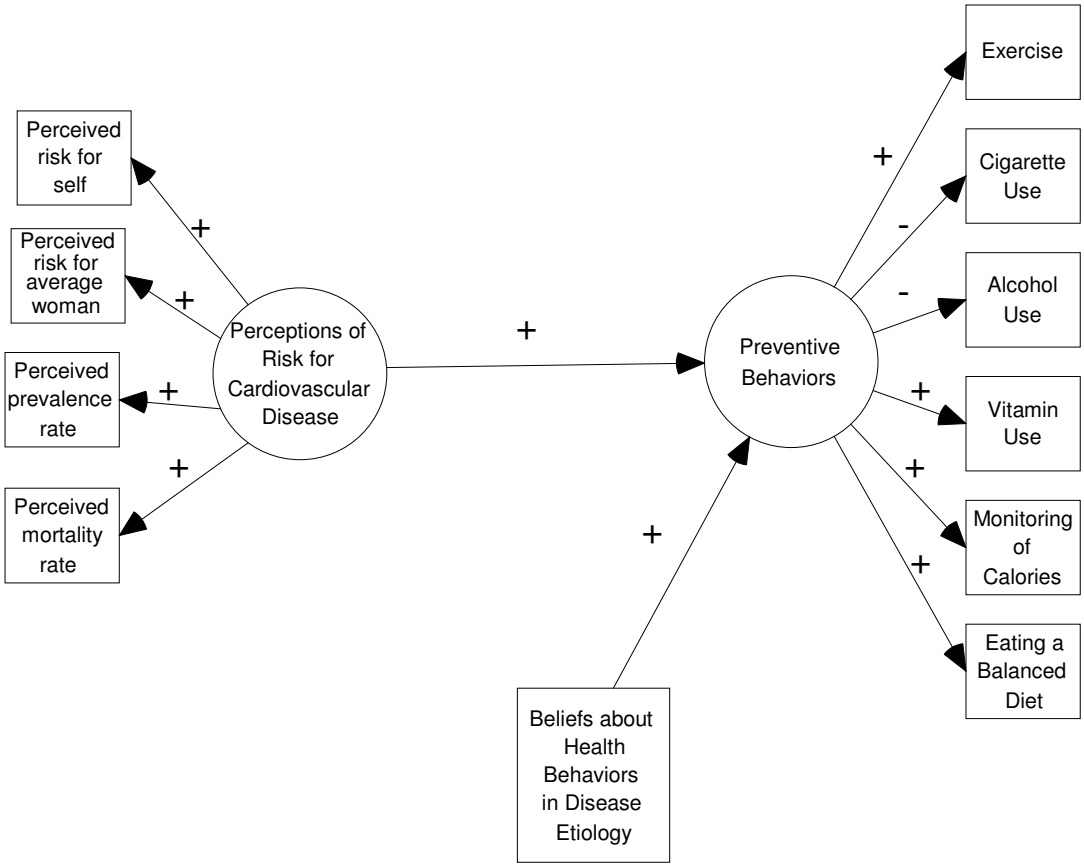


Figure 2.3.1. Hypothesized Main Effects Model for the relationship between perceptions of risk for cardiovascular disease, beliefs about the role of health behaviors in cardiovascular disease etiology, and preventive behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

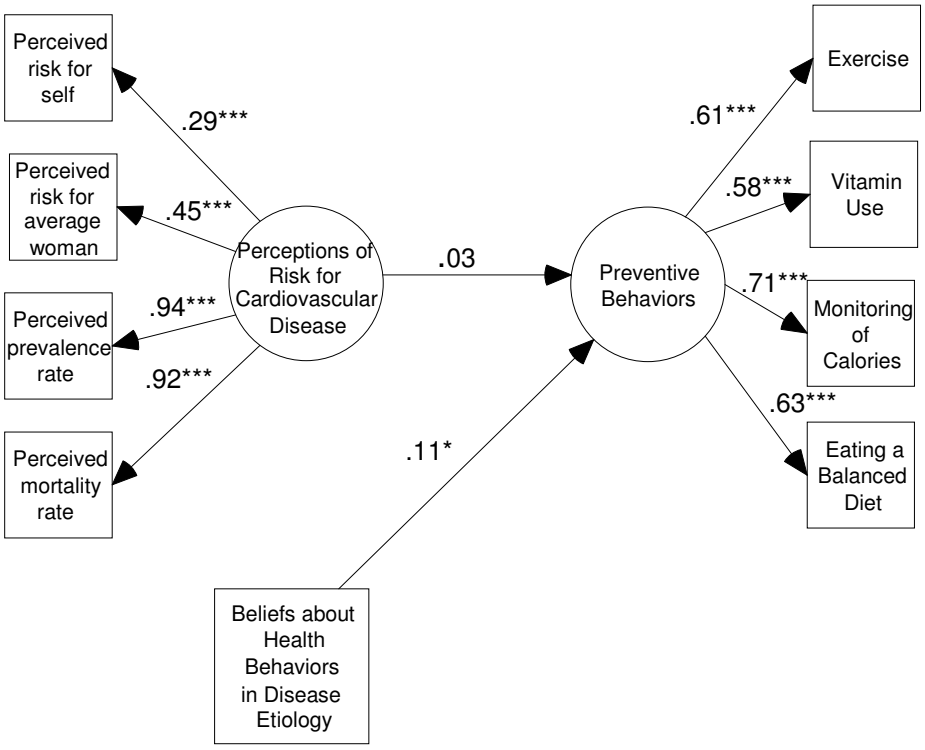


Figure 2.3.1a. Final Main Effects Model for the relationship between perceptions of risk for cardiovascular disease, beliefs about the role of health behaviors in cardiovascular disease etiology, and preventive behaviors in younger women. Results did not support this model, which was a poor fit to the data in a subsample of 418 women ($\chi^2(35) = 290.72$, $p < .001$; CFI = .87; RMSEA = .13, 90% confidence limits for the RMSEA = .12 to .15). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

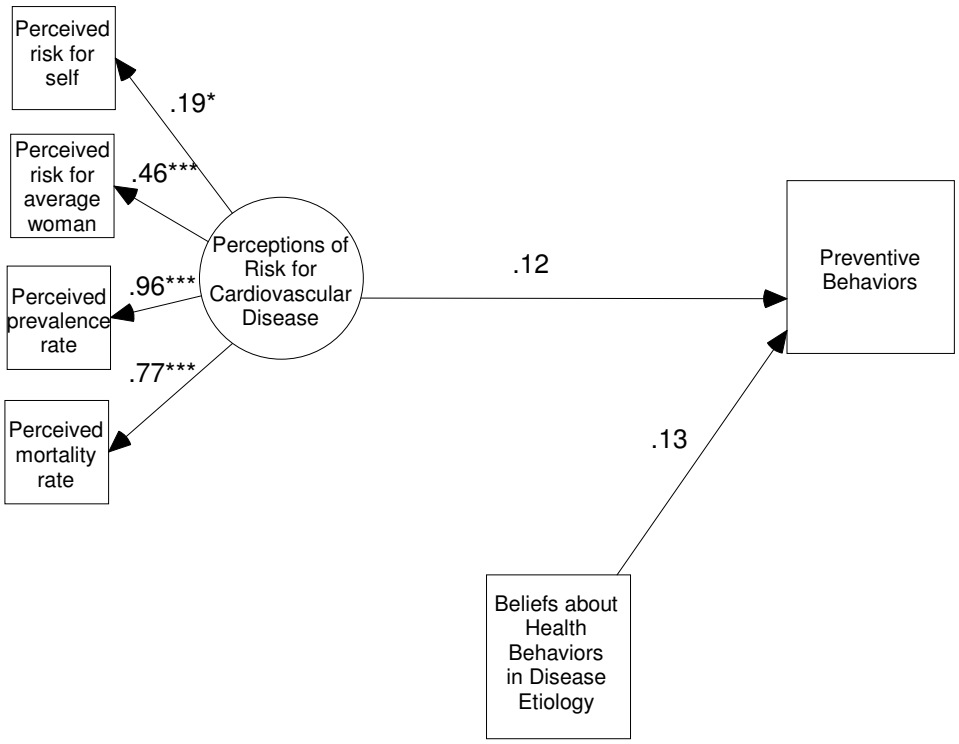


Figure 2.3.1b. Final Main Effects Model for the relationship between perceptions of risk for cardiovascular disease, beliefs about the role of health behaviors in cardiovascular disease etiology, and preventive behaviors in older women. Results did not support this model, which was a poor fit to the data in a subsample of 175 older women ($\chi^2(9) = 33.85$, $p < .001$; CFI = .88; RMSEA = .13, 90% confidence limits for the RMSEA = .08 to .17). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

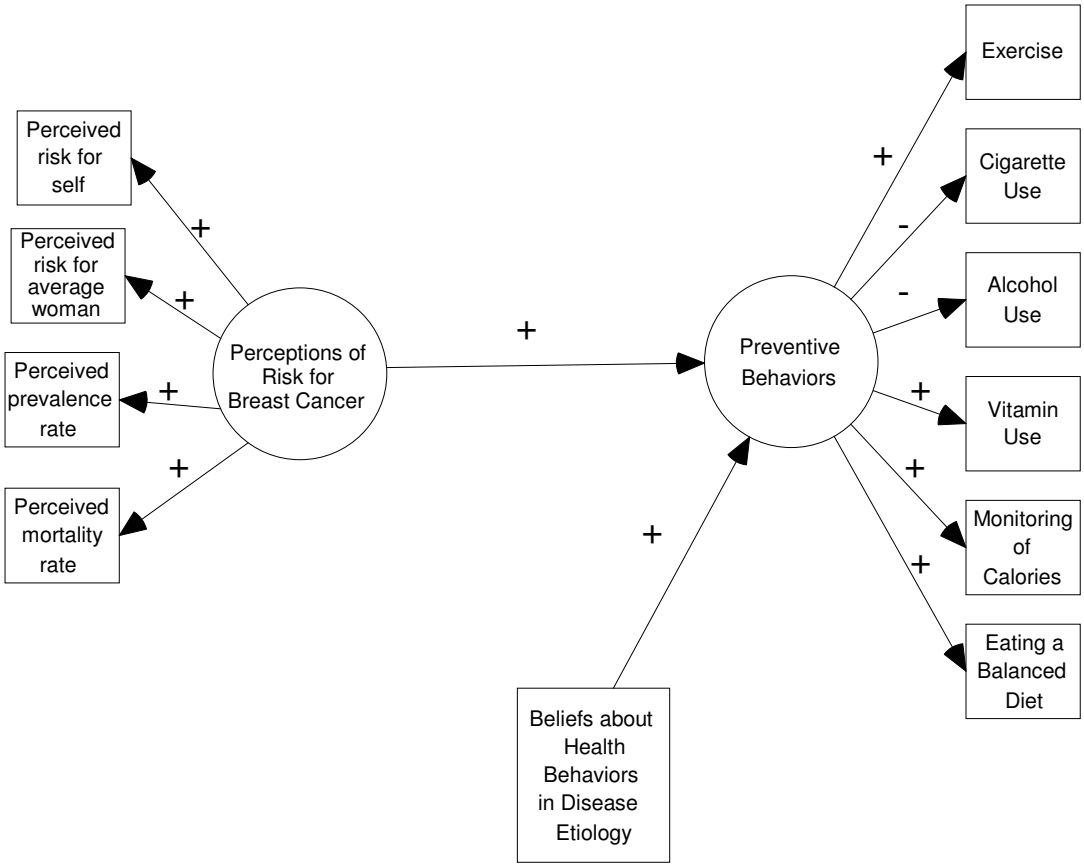


Figure 2.3.2. Hypothesized Main Effects Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and preventive behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

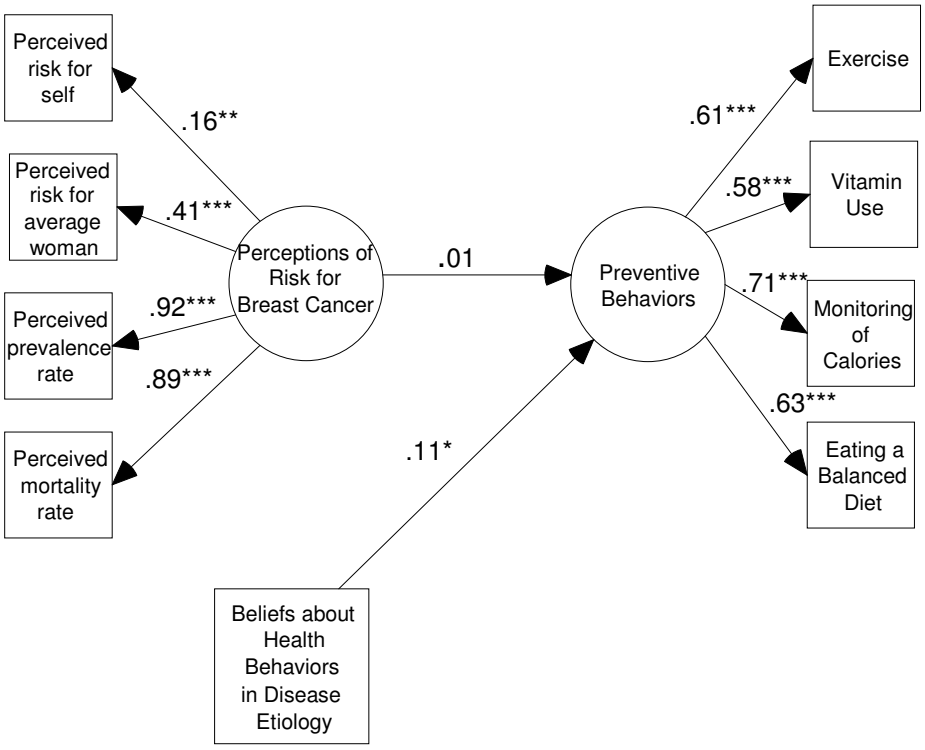


Figure 2.3.2a. Final Main Effects Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and preventive behaviors in younger women. Results did not support this model, which was a poor fit to the data in a subsample of 419 women ($\chi^2(35) = 258.84$, $p < .001$; CFI = .87; RMSEA = .12, 90% confidence limits for the RMSEA = .11 to .14). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

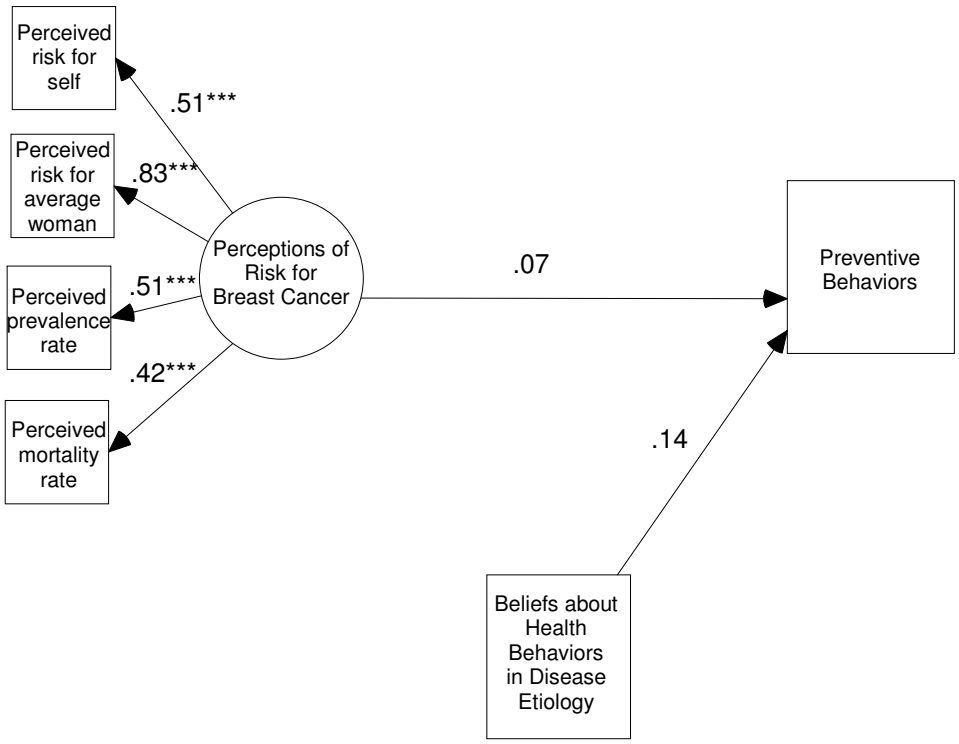


Figure 2.3.2b. Final Main Effects Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and preventive behaviors in older women. Results did not support this model, despite the fact that the model was a good fit to the data in a subsample of 175 women ($\chi^2(8) = 10.81$, $p = .21$; CFI = .99; RMSEA = .05, 90% confidence limits of the RMSEA = .00 to .11). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

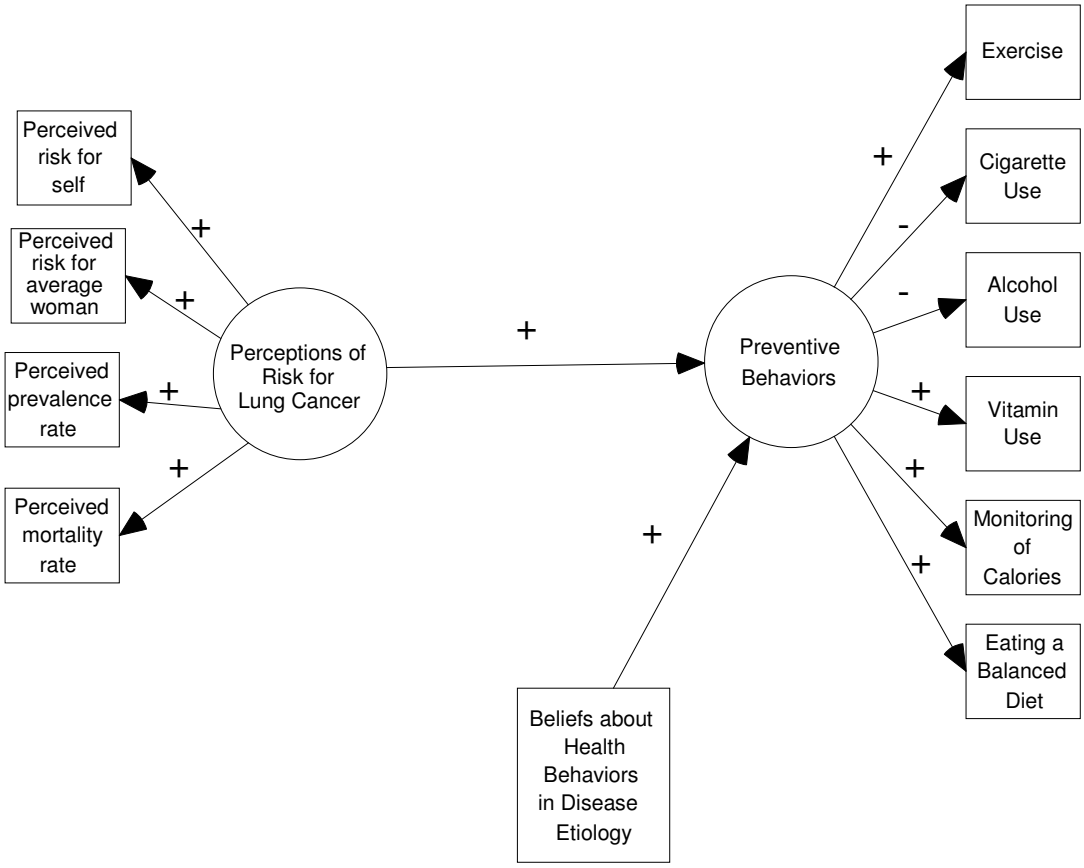


Figure 2.3.3. Hypothesized Main Effects Model for the relationship between perceptions of risk for lung cancer, beliefs about the role of health behaviors in lung cancer etiology, and preventive behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

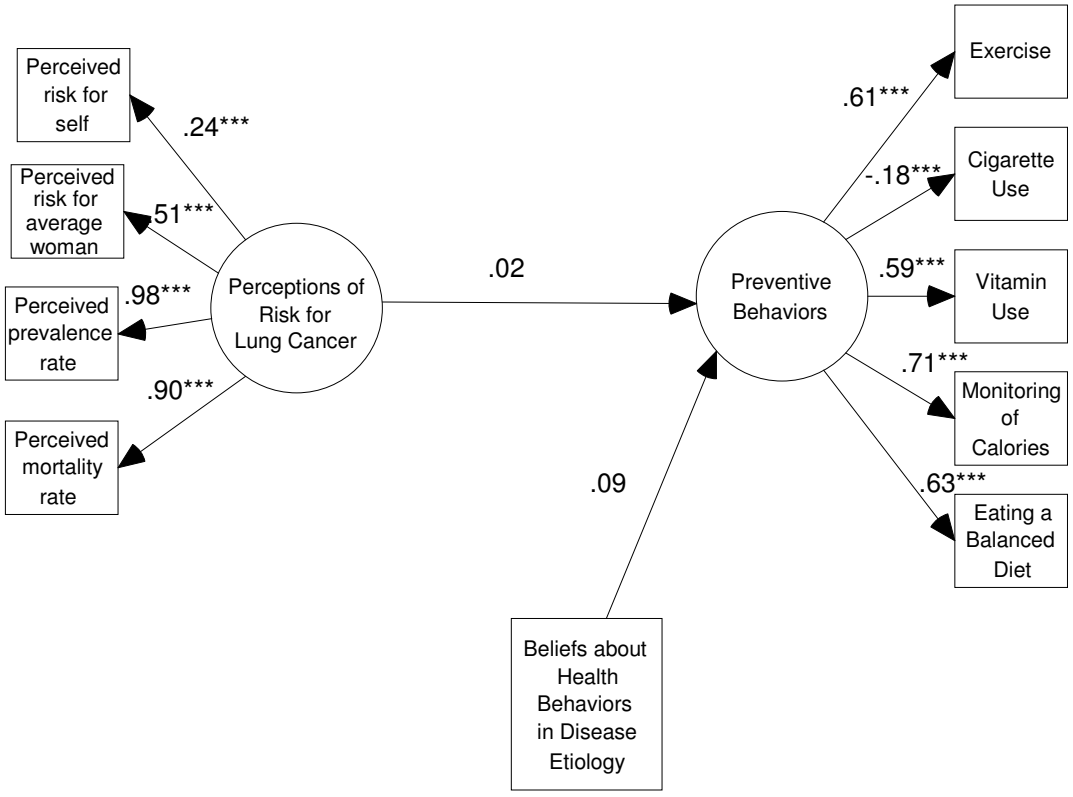


Figure 2.3.3a. Final Main Effects Model for the relationship between perceptions of risk for lung cancer, beliefs about the role of health behaviors in lung cancer etiology, and preventive behaviors in younger women. Results did not support this model, which was a poor fit to the data in a subsample of 414 women ($\chi^2(44) = 987.65$, $p < .001$; CFI = .65; RMSEA = .23, 90% confidence limits for the RMSEA = .22 to .24). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

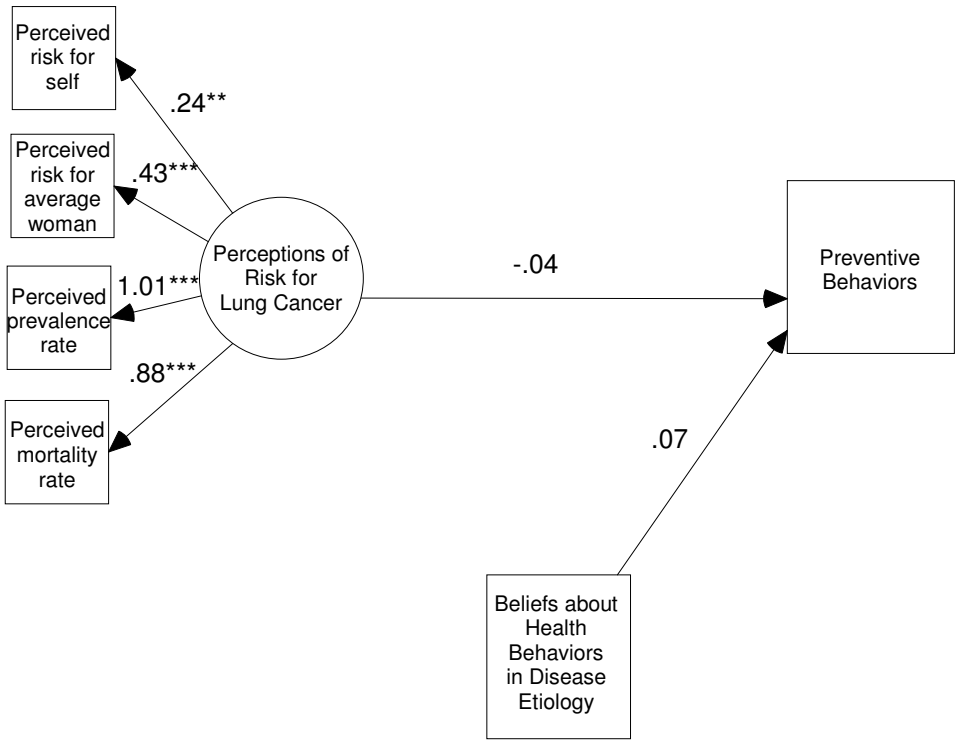


Figure 2.3.3b. Final Main Effects Model for the relationship between perceptions of risk for lung cancer, beliefs about the role of health behaviors in lung cancer etiology, and preventive behaviors in older women. Results did not support this model. The model was an adequate fit to the data in a subsample of 175 women ($\chi^2(8) = 22.68$, $p = .004$; CFI = .96; RMSEA = .10, 90% confidence limits for the RMSEA = .05 to .15); however, the model's solution was inadmissible because it resulted in a Heywood case. For simplicity, error terms have been omitted. All values are standardized path coefficients. ** = $p \leq .01$; *** = $p \leq .001$.

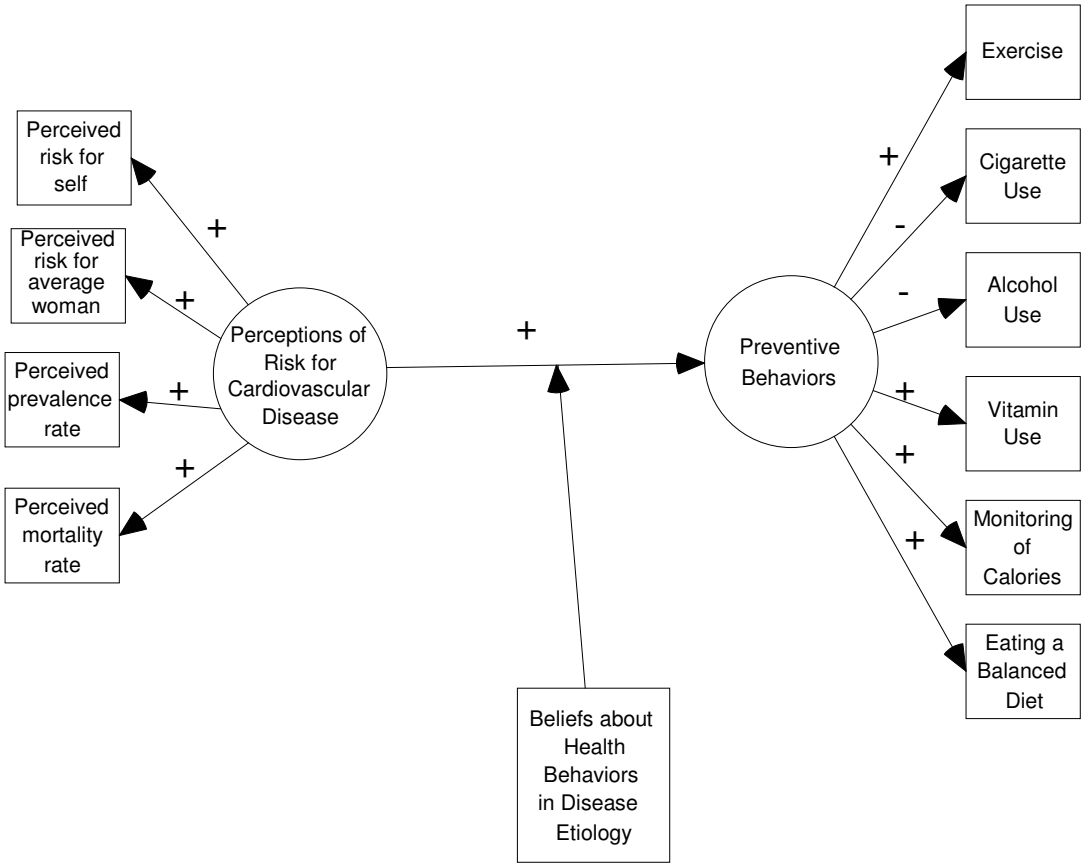
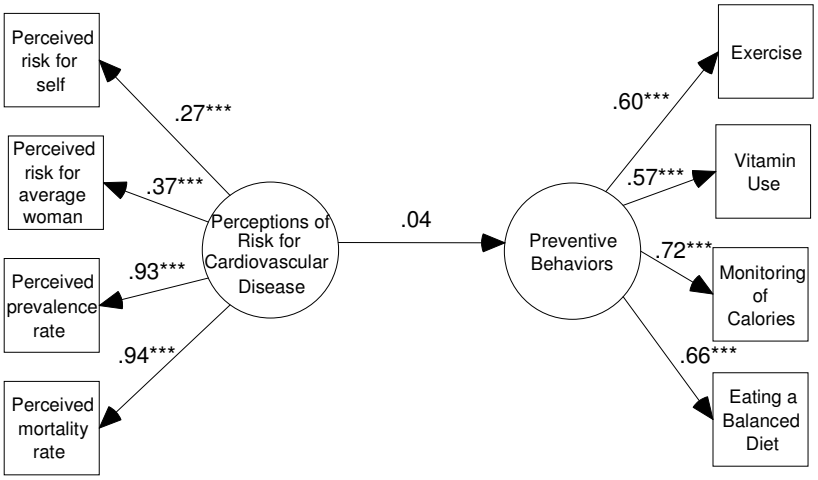


Figure 2.4.1. Hypothesized Interaction Model for the relationship between perceptions of risk for cardiovascular disease, beliefs about the role of health behaviors in cardiovascular disease etiology, and preventive behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

Weaker beliefs in the role of health behaviors in cardiovascular disease etiology:



Stronger beliefs in the role of health behaviors in cardiovascular disease etiology:

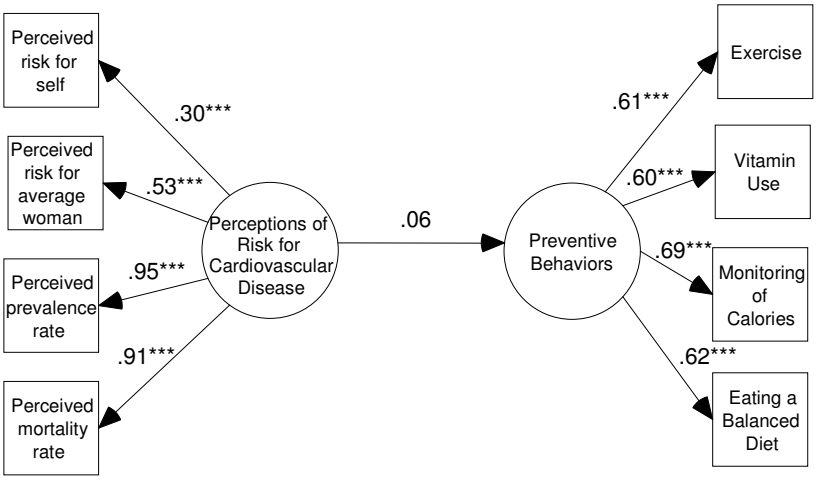
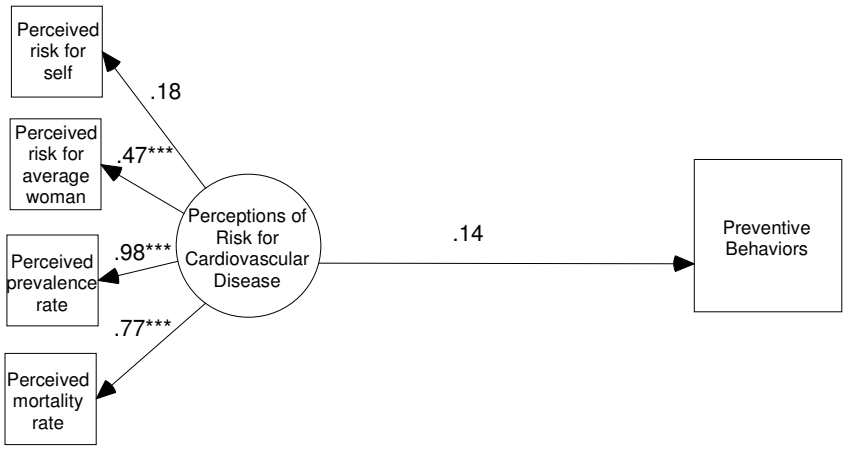


Figure 2.4.1a. Final Interaction Model for the relationship between perceptions of risk for cardiovascular disease, beliefs about the role of health behaviors in cardiovascular disease etiology, and preventive behaviors in younger women. Results did not support this model. The model was a poor fit to the data in a subsample of 251 women with weaker beliefs in the role of health behaviors in cardiovascular disease etiology ($\chi^2(27) = 162.28$, $p < .001$; CFI = .88; RMSEA = .14, 90% confidence limits for the RMSEA = .12 to .16), and in a subsample of 162 women with stronger beliefs in the role of health behaviors in cardiovascular disease etiology ($\chi^2(27) = 143.87$, $p < .001$; CFI = .85; RMSEA = .16, 90% confidence limits for the RMSEA = .14 to .19). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

Weaker beliefs in the role of health behaviors in cardiovascular disease etiology:



Stronger beliefs in the role of health behaviors in cardiovascular disease etiology:

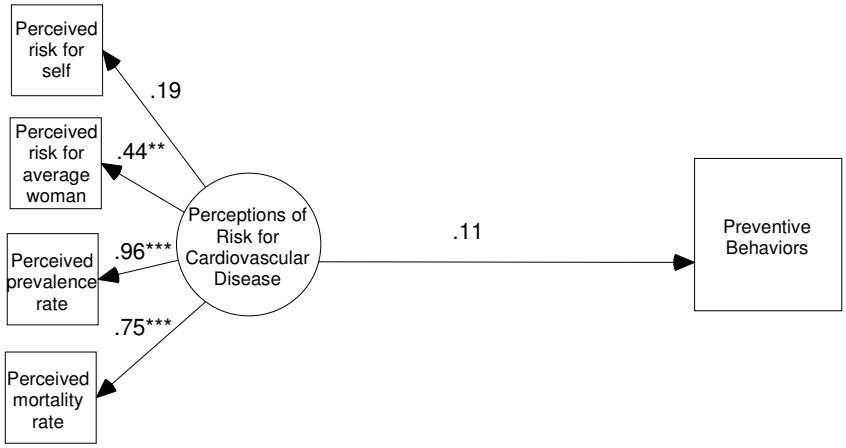


Figure 2.4.1b. Final Interaction Model for the relationship between perceptions of risk for cardiovascular disease, beliefs about the role of health behaviors in cardiovascular disease etiology, and preventive behaviors in older women. Results did not support this model. The model was an adequate fit to the data in a subsample of 110 women with weaker beliefs in the role of health behaviors in the etiology of cardiovascular disease ($\chi^2(5) = 8.23$, $p = .14$; CFI = .97; RMSEA = .08, 90% confidence limits for the RMSEA = .00 to .17), and a poor fit to the data in a subsample of 65 women with stronger beliefs in the role of health behaviors ($\chi^2(5) = 13.96$, $p = .02$; CFI = .86; RMSEA = .17, 90% confidence limits for the RMSEA = .07 to .27). For simplicity, error terms have been omitted. All values are standardized path coefficients. ** = $p \leq .01$; *** = $p \leq .001$.

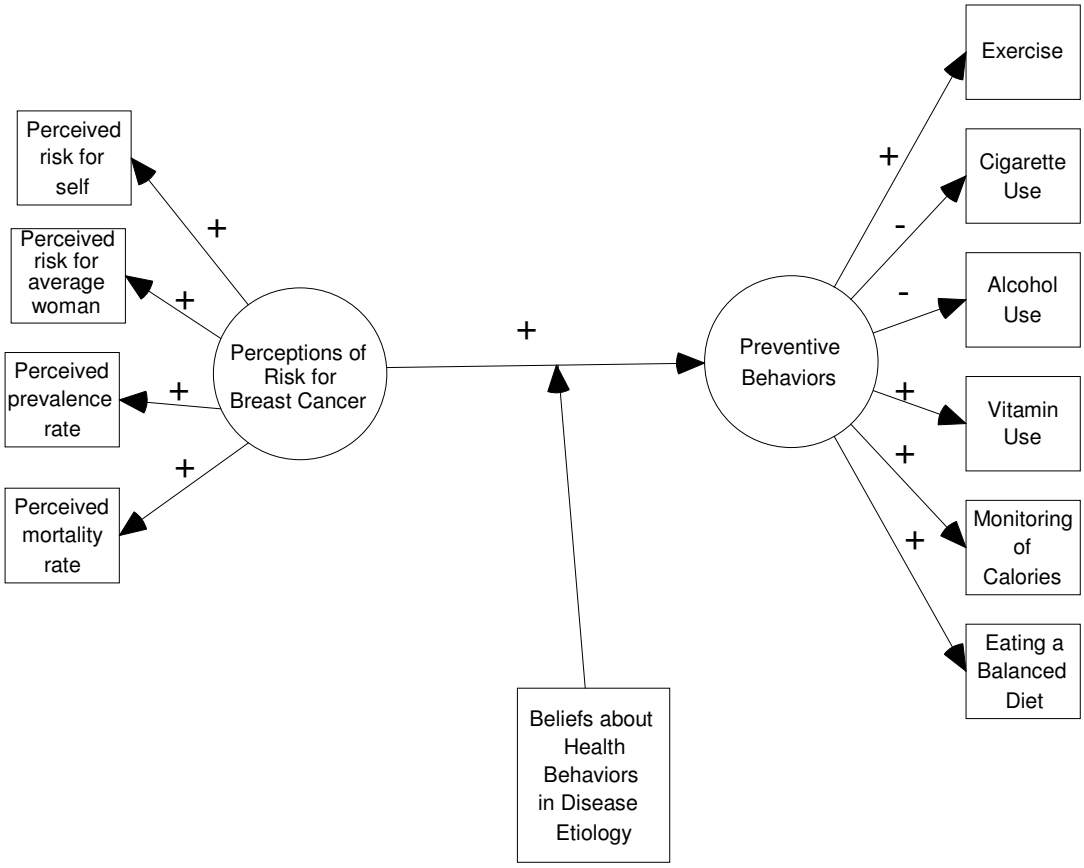
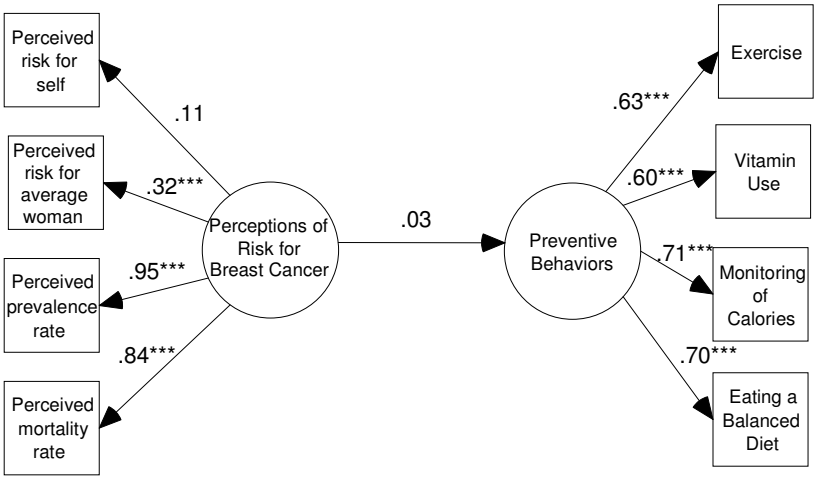


Figure 2.4.2. Hypothesized Interaction Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and preventive behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

Weaker beliefs in the role of health behaviors in breast cancer etiology:



Stronger beliefs in the role of health behaviors in breast cancer etiology:

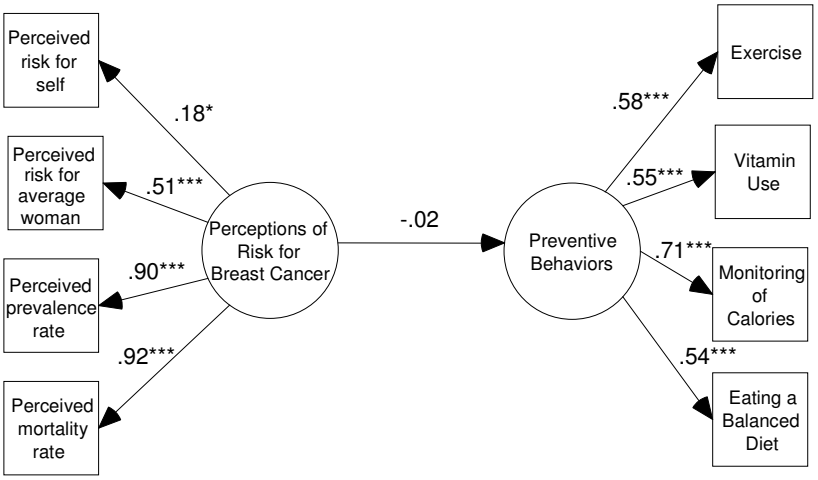
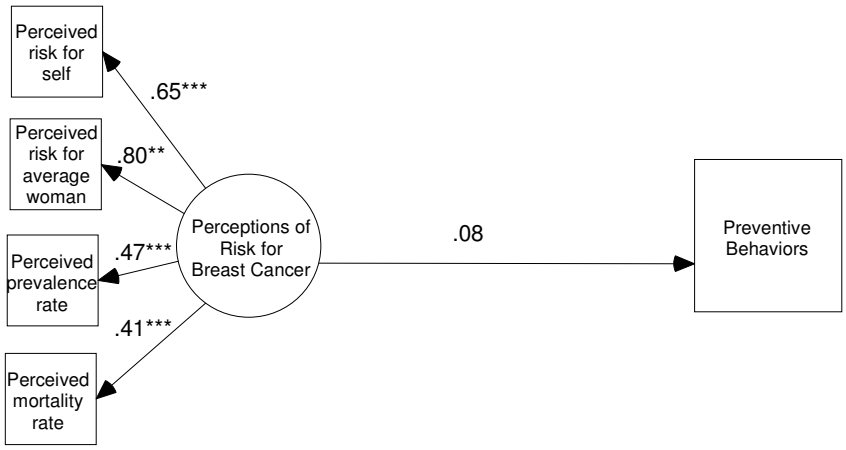


Figure 2.4.2a. Final Interaction Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and preventive behaviors in younger women. Results did not support the model. The model was a poor fit to the data in a subsample of 244 women with weaker beliefs in the role of health behaviors in breast cancer etiology ($\chi^2(27) = 176.35$, $p < .001$; CFI = .86; RMSEA = .15, 90% confidence limits for the RMSEA = .13 to .17), and in a subsample of 175 women with stronger beliefs in the role of health behaviors in breast cancer etiology ($\chi^2(27) = 114.59$, $p < .001$; CFI = .87; RMSEA = .14, 90% confidence limits for the RMSEA = .11 to .16). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

Weaker beliefs in the role of health behaviors in breast cancer etiology:



Stronger beliefs in the role of health behaviors in breast cancer etiology:

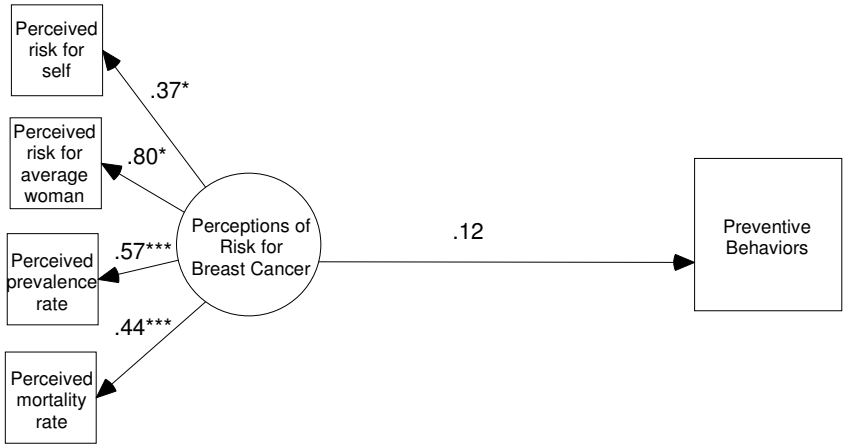


Figure 2.4.2b. Final Interaction Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and preventive behaviors in older women. Results did not support the model, despite the fact that the model was a good fit to the data in a subsample of 94 women with weaker beliefs in the role of health behaviors in breast cancer etiology ($\chi^2(4) = 10.32$, $p = .04$; CFI = .96; RMSEA = .13, 90% confidence limits for the RMSEA = .03 to .23), and in a subsample of 81 women with stronger beliefs in the role of health behaviors in breast cancer etiology ($\chi^2(4) = 0.82$, $p = .94$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .04). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

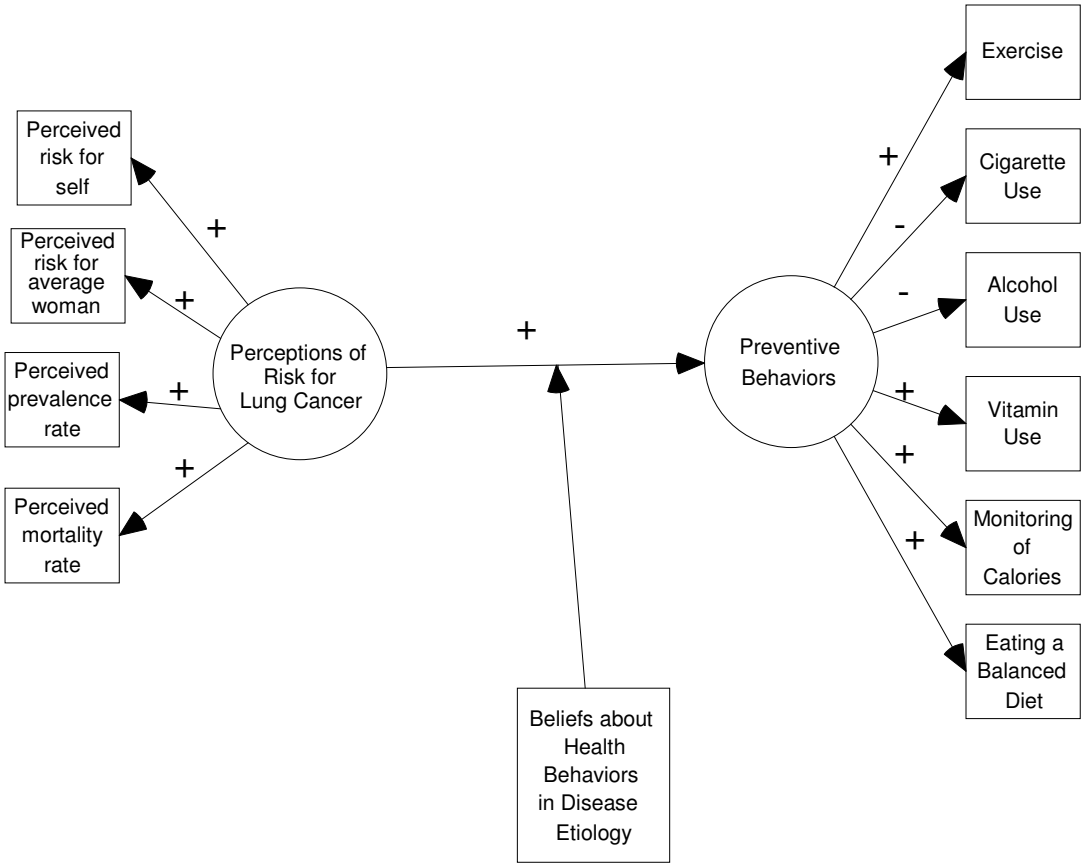
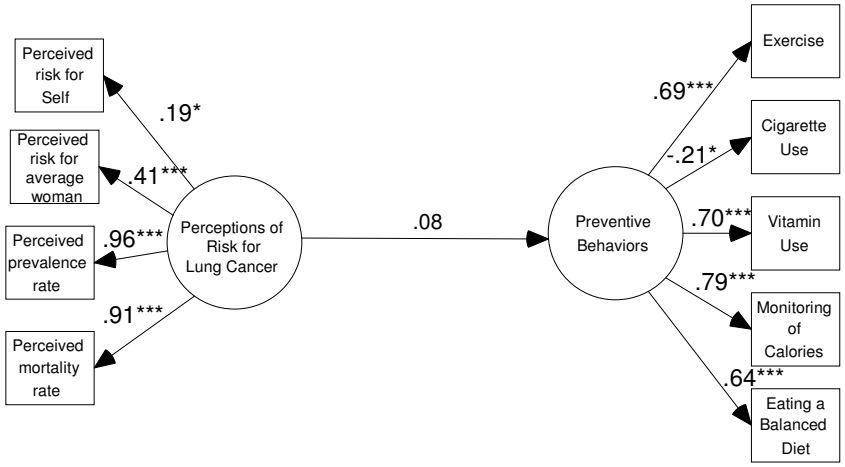


Figure 2.4.3. Hypothesized Interaction Model for the relationship between perceptions of risk for lung cancer, beliefs about the role of health behaviors in lung cancer etiology, and preventive behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

Weaker beliefs in the role of health behaviors in lung cancer etiology:



Stronger beliefs in the role of health behaviors in lung cancer etiology:

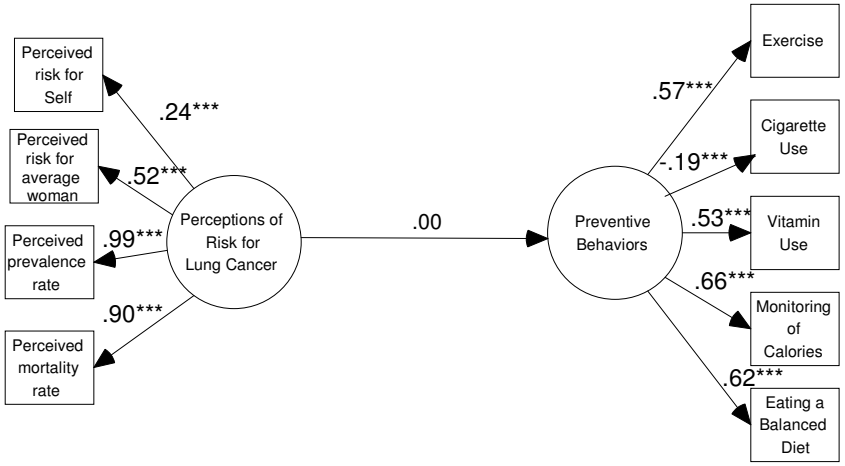
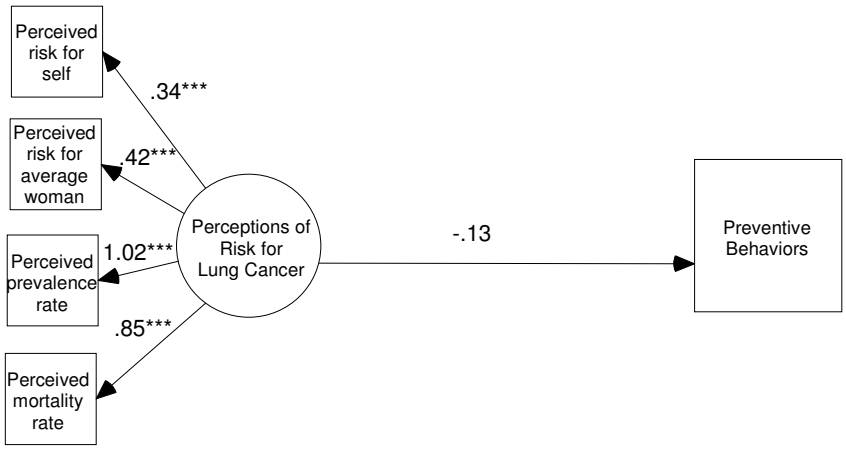


Figure 2.4.3a. Final Interaction Model for the relationship between perceptions of risk for lung cancer, beliefs about the role of health behaviors in lung cancer etiology, and preventive behaviors in younger women. Results did not support the model. The model was a poor fit to the data in a subsample of 135 younger women with weaker beliefs in the role of health behaviors ($\chi^2(35) = 348.77$, $p < .001$; CFI = .66; RMSEA = .26, 90% confidence limits for the RMSEA = .23 to .28), and in a subsample of 283 women with stronger beliefs in the role of health behaviors in the etiology of lung cancer ($\chi^2(35) = 634.51$, $p < .001$; CFI = .65; RMSEA = .25, 90% confidence limits for the RMSEA = .23 to .26). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

Weaker beliefs in the role of health behaviors in lung cancer etiology:



Stronger beliefs in the role of health behaviors in lung cancer etiology:

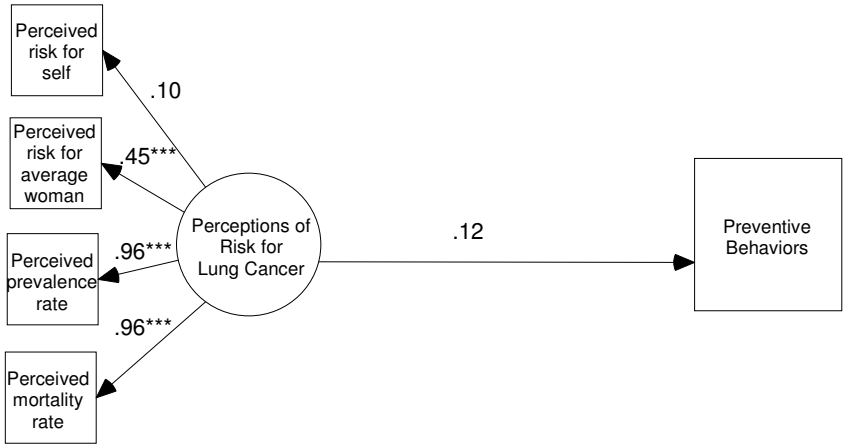


Figure 2.4.3b. Final Interaction Model for the relationship between perceptions of risk for lung cancer, beliefs about the role of health behaviors in lung cancer etiology, and preventive behaviors in older women. Results did not support the model. The model was an adequate fit to the data in a subsample of 98 women with weaker beliefs in the role of health behaviors in the etiology of lung cancer ($\chi^2(4) = 9.00$, $p = .06$; CFI = .97; RMSEA = .11, 90% confidence limits for the RMSEA = .00 to .21); however, the model's solution was inadmissible because it resulted in a Heywood case. The model was also an adequate fit in a subsample of 77 women with stronger beliefs in the role of health behaviors in the etiology of lung cancer ($\chi^2(4) = 7.36$, $p = .12$; CFI = .98; RMSEA = .11, 90% confidence limits for the RMSEA = .00 to .22). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

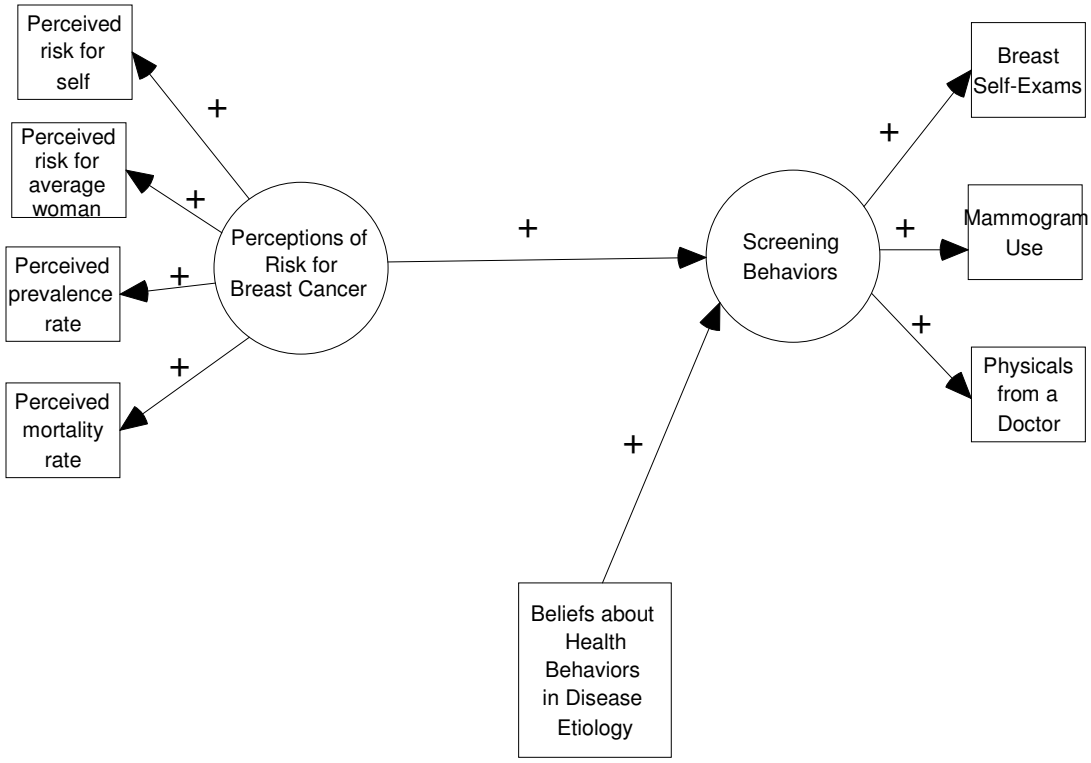


Figure 2.5. Hypothesized Main Effects Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and screening behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

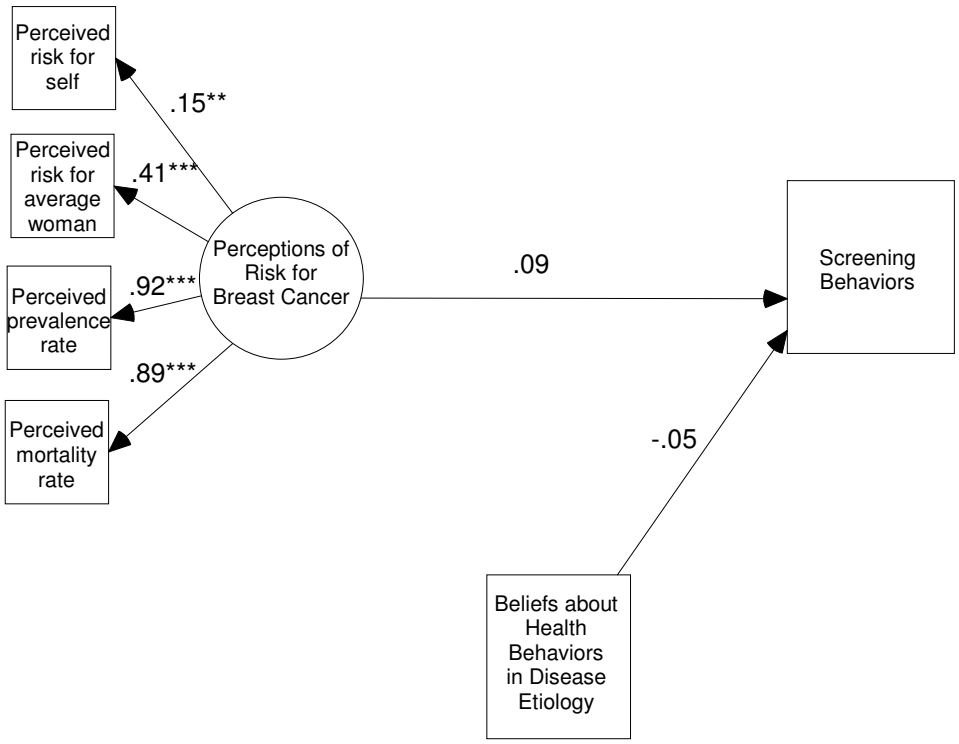


Figure 2.5a. Final Main Effects Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and screening behaviors in younger women. Results did not support the model, despite the fact that the model was a good fit to the data in a subsample of 418 women ($\chi^2(8) = 15.11$, $p = .06$; CFI = .99; RMSEA = .05, 90% confidence limits for the RMSEA = .00 to .08). For simplicity, error terms have been omitted. All values are standardized path coefficients. ** = $p \leq .01$; *** = $p \leq .001$.

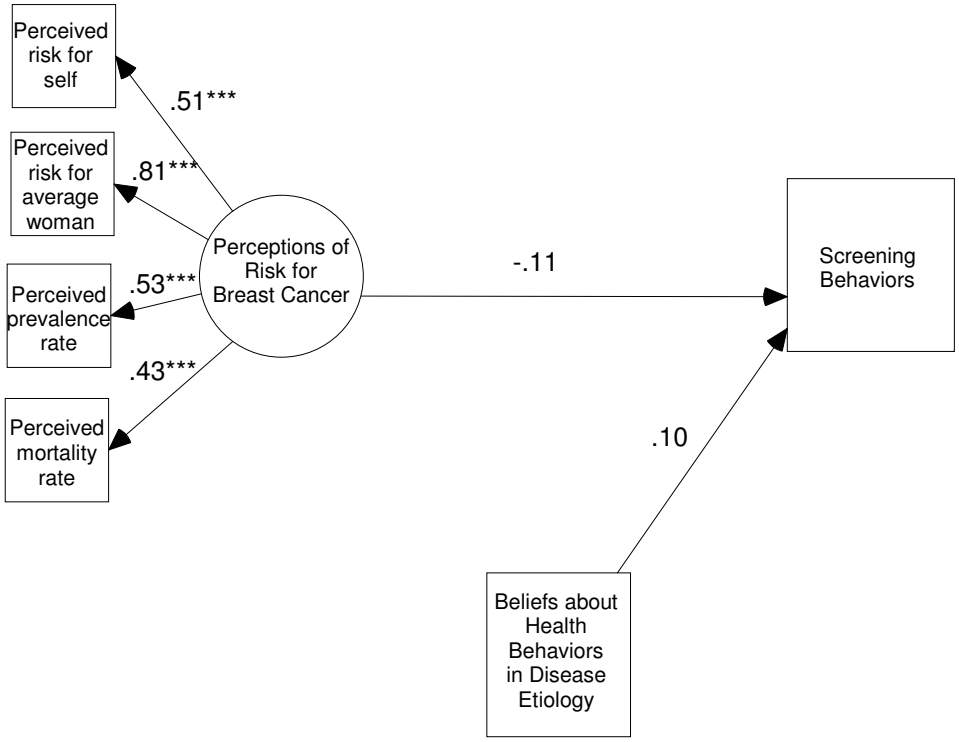


Figure 2.5b. Final Main Effects Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and screening behaviors in older women. Results did not support the model, despite the fact that the model was a good fit to the data in a subsample of 175 women ($\chi^2(8) = 9.66$, $p = .29$; CFI = .99; RMSEA = .04, 90% confidence limits for the RMSEA = .00 to .10). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

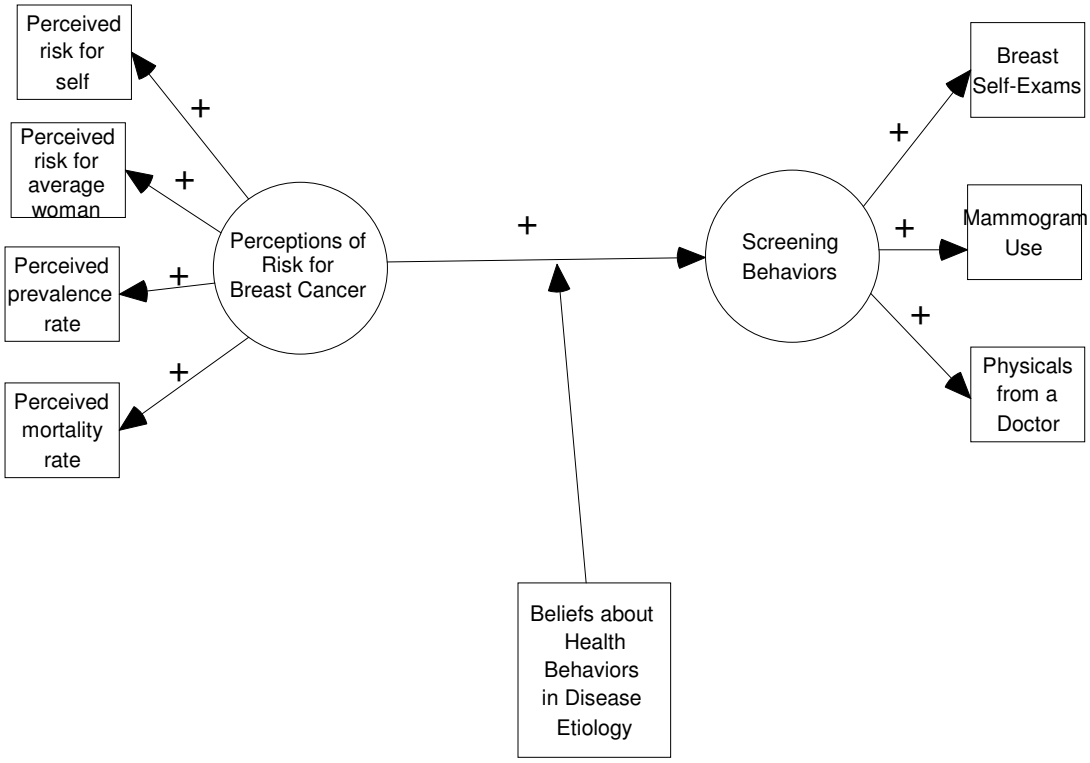
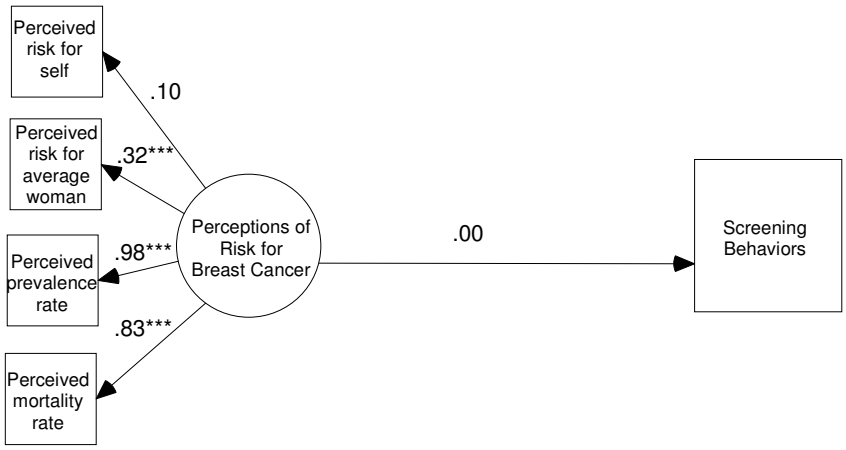


Figure 2.6. Hypothesized Interaction Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and screening behaviors. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

Weaker beliefs in the role of health behaviors in breast cancer etiology:



Stronger beliefs in the role of health behaviors in breast cancer etiology:

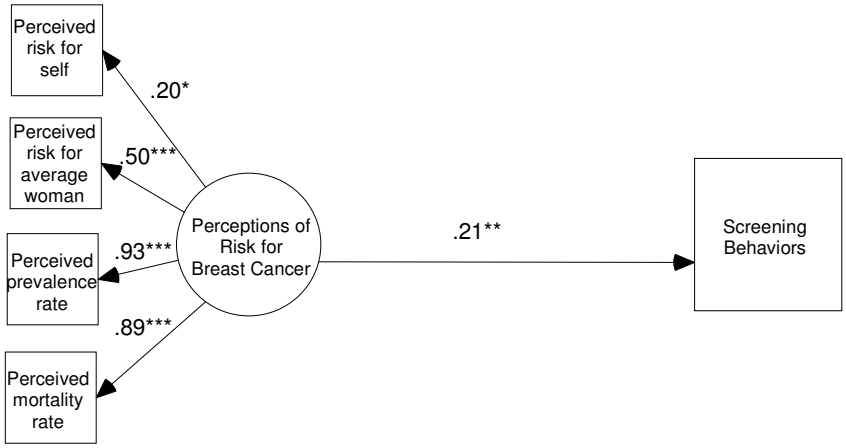
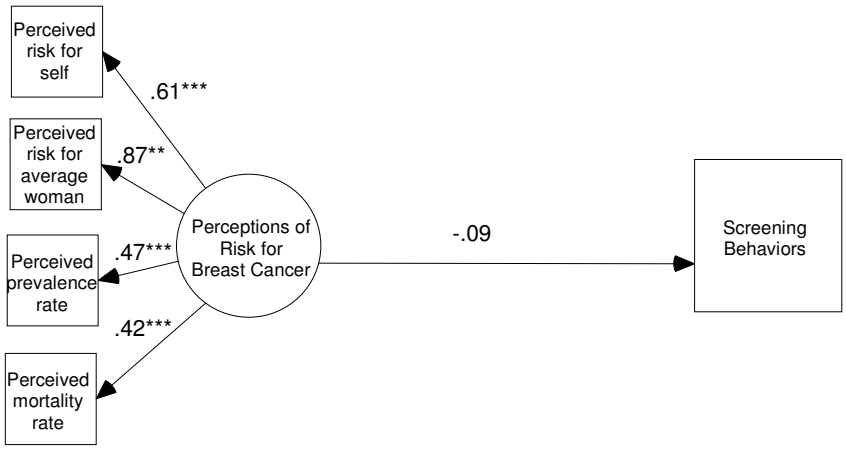


Figure 2.6a. Final Interaction Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and screening behaviors in younger women. Results supported the model. The model was a good fit to the data in a subsample of 241 women with weaker beliefs in the role of health behaviors in the etiology of breast cancer ($\chi^2(4) = 3.03$, $p = .55$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .09), and in a subsample of 175 younger women with stronger beliefs in the role of health behaviors in the etiology of breast cancer ($\chi^2(4) = 9.38$, $p = .05$; CFI = .98; RMSEA = .09, 90% confidence limits for the RMSEA = .00 to .16). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

Weaker beliefs in the role of health behaviors in breast cancer etiology:



Stronger beliefs in the role of health behaviors in breast cancer etiology:

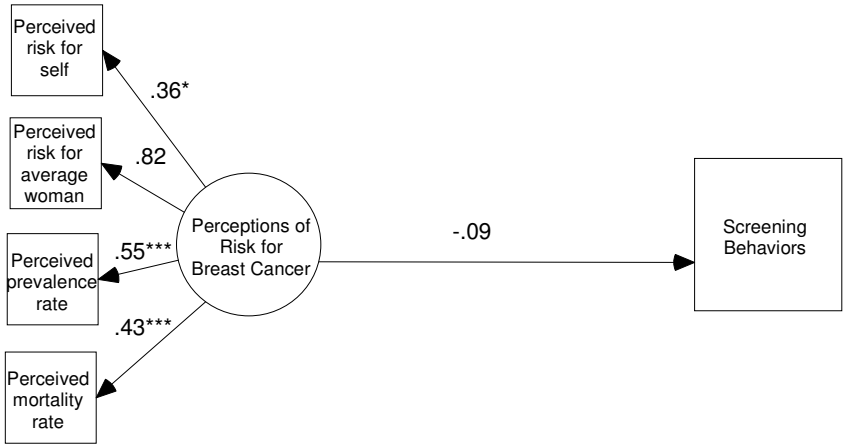


Figure 2.6b. Final Interaction Model for the relationship between perceptions of risk for breast cancer, beliefs about the role of health behaviors in breast cancer etiology, and screening behaviors in older women. Results did not support the model. The model was an adequate fit to the data in a subsample of 92 women with weaker beliefs in the role of health behaviors in the etiology of breast cancer ($\chi^2(4) = 5.66$, $p = .23$; CFI = .99; RMSEA = .07, 90% confidence limits for the RMSEA = .00 to .18), and was a good fit in a subsample of 81 women with stronger beliefs in the role of health behaviors in the etiology of breast cancer ($\chi^2(4) = 0.58$, $p = .97$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .00). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

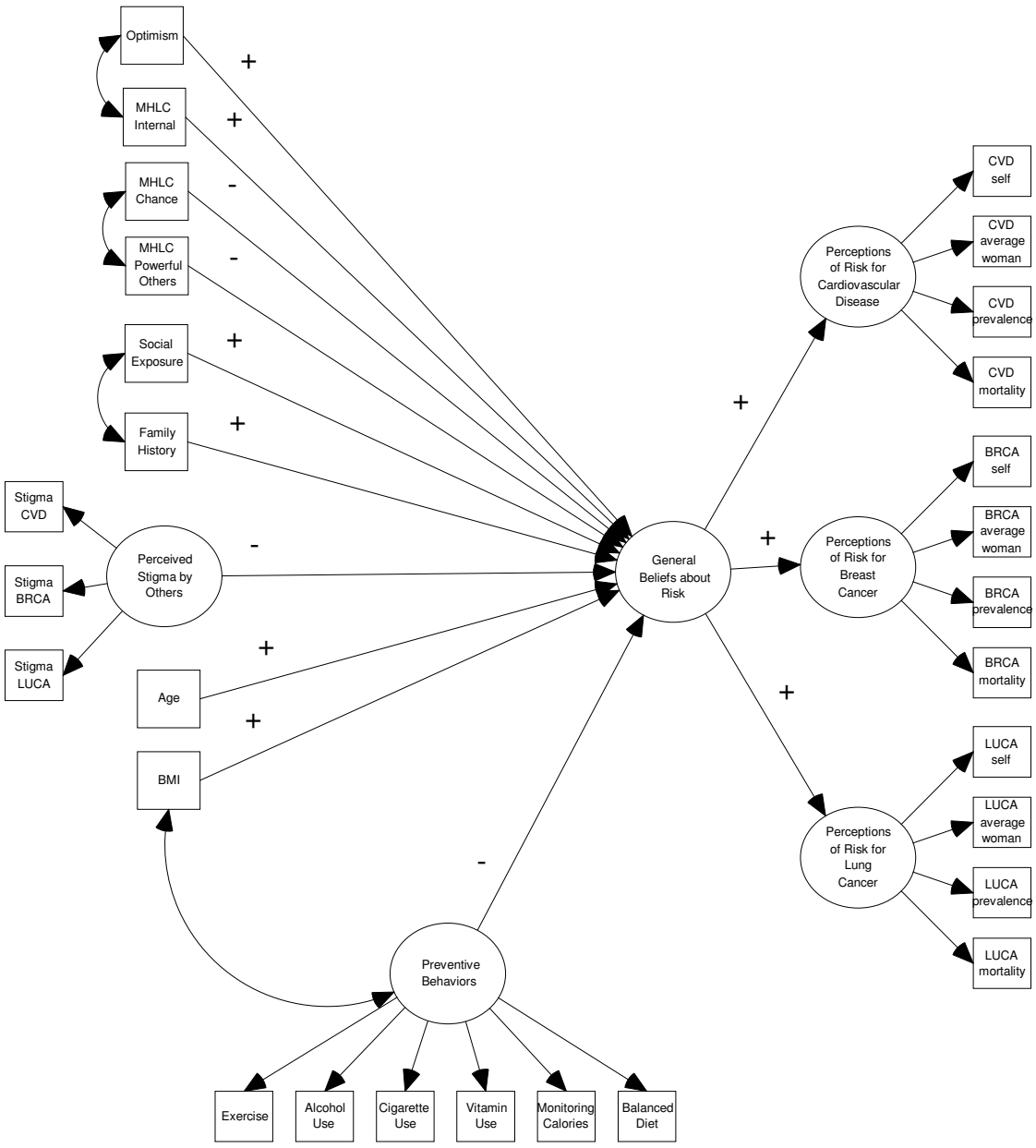


Figure 3.1. Hypothesized model of psychosocial correlates of perceptions of disease risk. Signs above the paths indicate the hypothesized direction of the associations between psychosocial variables and the criterion variable of general beliefs about risk. For simplicity, signs for the direction of the relationships between indicator variables and latent variables have been omitted, error terms have been omitted, and variable labels have been abbreviated.

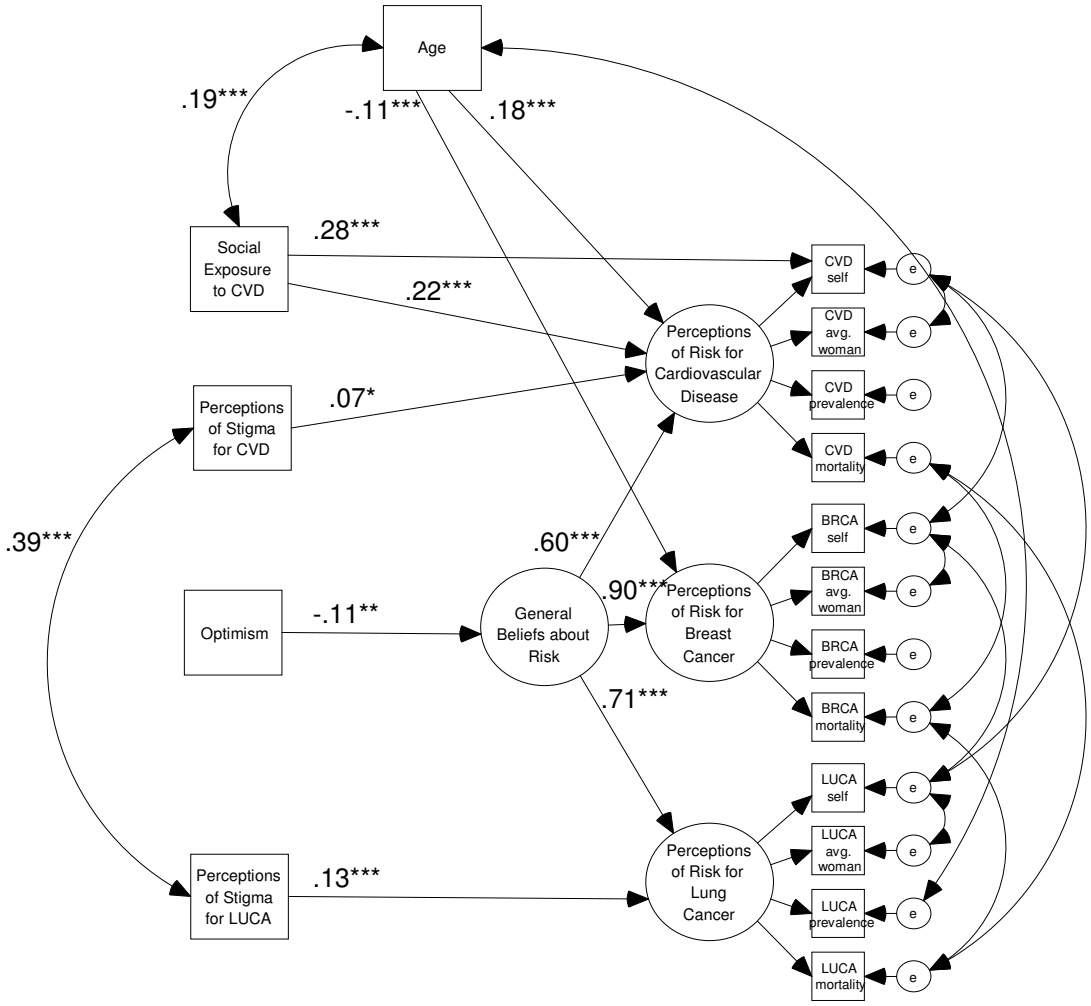


Figure 3.1a. Final model of psychosocial correlates of perceptions of disease risk in the full sample of younger and older women. The model was a good fit to the data in the sample of 634 women ($\chi^2(102) = 365.61$, $p < .001$; CFI = .94; RMSEA = .06, 90% confidence limits for the RMSEA = .06 to .07). The data were substantially nonnormal; however, bootstrapping with maximum likelihood estimation confirmed that parameter estimates were not biased. For simplicity, only standardized path coefficients for the relationships between psychosocial variables and perceptions of risk, between the latent variable representing general beliefs about risk and the latent variables representing perceptions about disease risk, and between the individual psychosocial variables are displayed. Variable labels have also been abbreviated. All unlabeled path coefficients are significant at $p \leq .001$. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

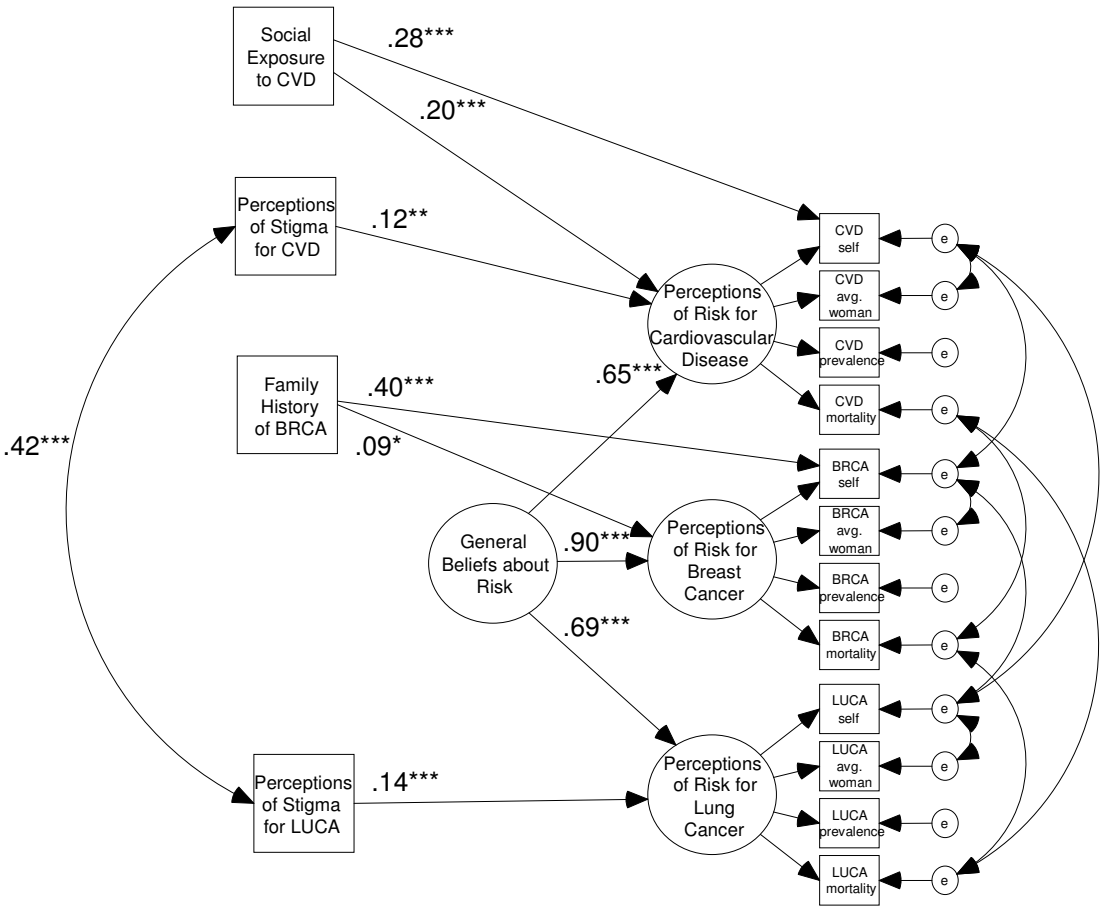


Figure 3.1b. Final model of psychosocial correlates of perceptions of disease risk in younger women. The model was a good fit to the data in the sample of 454 women ($\chi^2(89) = 229.14$, $p < .001$; CFI = .95; RMSEA = .06, 90% confidence limits for the RMSEA = .05 to .07). The data were substantially nonnormal; however, bootstrapping with maximum likelihood estimation confirmed that parameter estimates were not biased. For simplicity, only standardized path coefficients for the relationships between psychosocial variables and perceptions of risk, between the latent variable representing general beliefs about risk and the latent variables representing perceptions about disease risk, and between the individual psychosocial variables are displayed. Variable labels have also been abbreviated. All unlabeled path coefficients are significant at $p \leq .001$. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

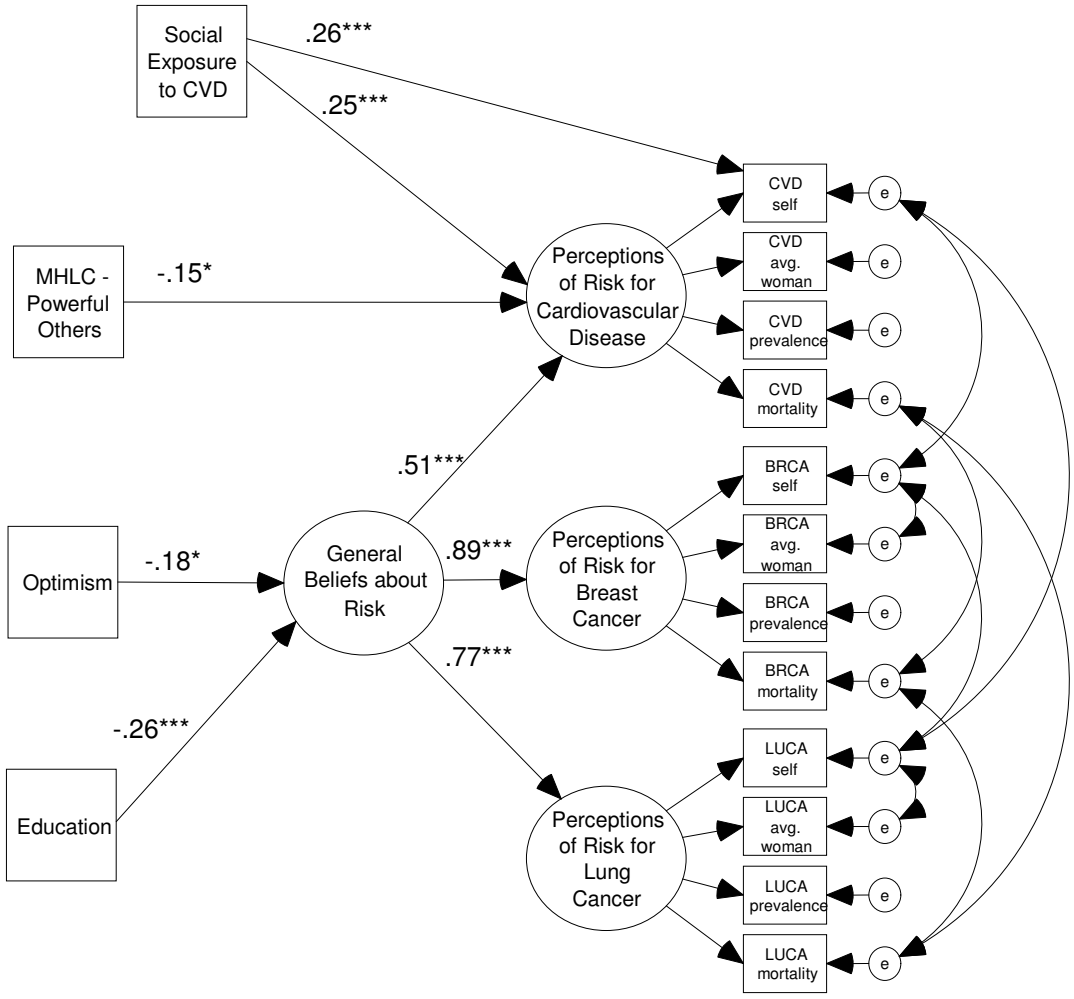


Figure 3.1c. Final model of psychosocial correlates of perceptions of disease risk in older women. The model was a good fit to the data in the sample of 180 women ($\chi^2(92) = 157.57$, $p < .001$; CFI = .94; RMSEA = .06, 90% confidence limits for the RMSEA = .05 to .08). Because the data were nonnormal and the sample size was small for a model of this complexity, bootstrapping with maximum likelihood estimation was used. Bootstrapping revealed that parameter estimates may be biased. For simplicity, only standardized path coefficients for the relationships between psychosocial variables and perceptions of risk, and between the latent variable representing general beliefs about risk and the latent variables representing perceptions about disease risk, are displayed. Variable labels have also been abbreviated. The path between the indicator variable of perceptions of cardiovascular disease risk for the self and the latent variable representing perceptions of risk for cardiovascular disease is not significant; all other unlabeled path coefficients are significant at $p < .04$. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

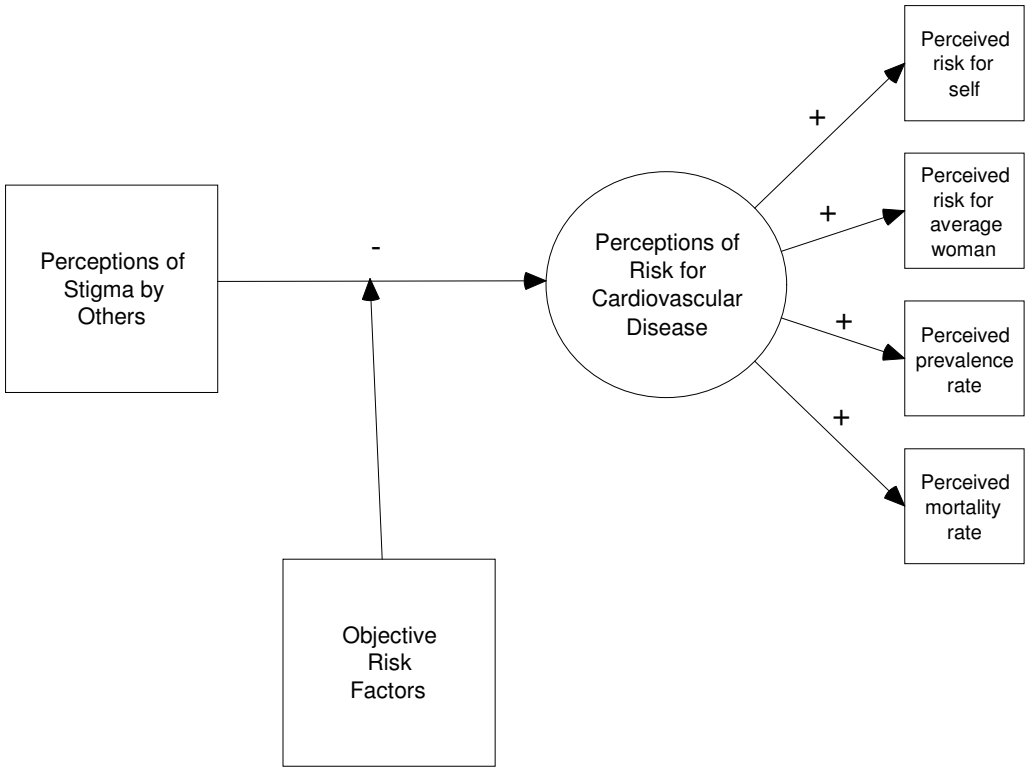
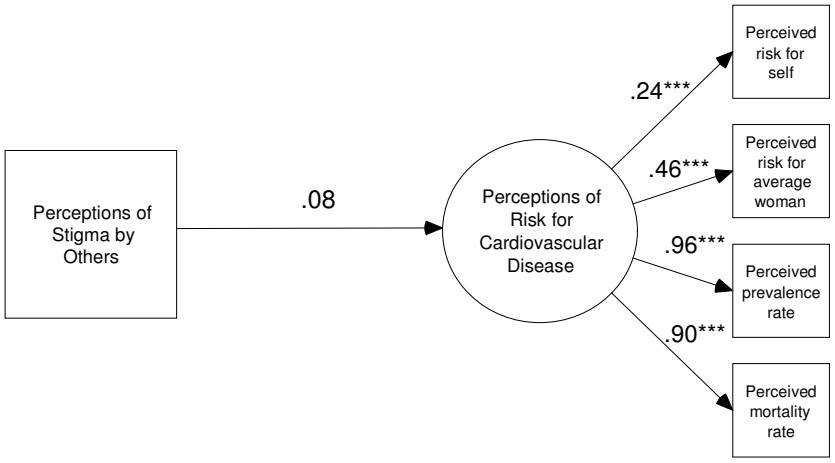


Figure 3.2.1. Hypothesized model of the moderating effect of objective risk factors on the association between perceptions of stigma by others and perceptions of risk for cardiovascular disease. Signs above the paths indicate the hypothesized direction of the associations between variables. For simplicity, error terms have been omitted.

Lower levels of objective risk:



Higher levels of objective risk:

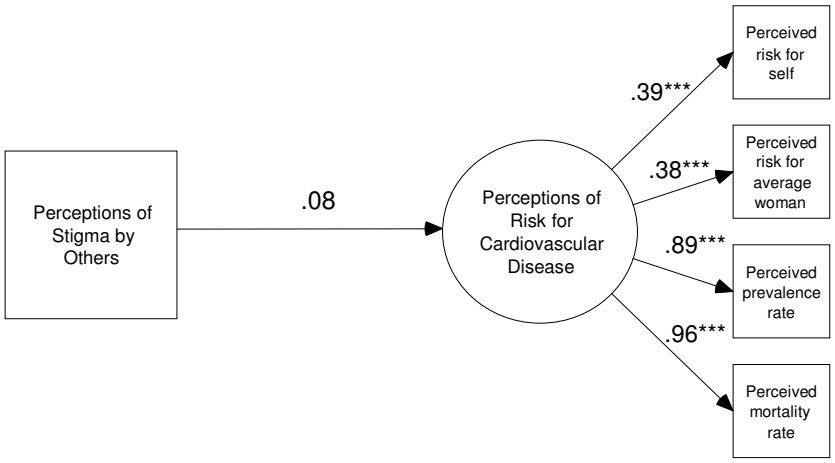
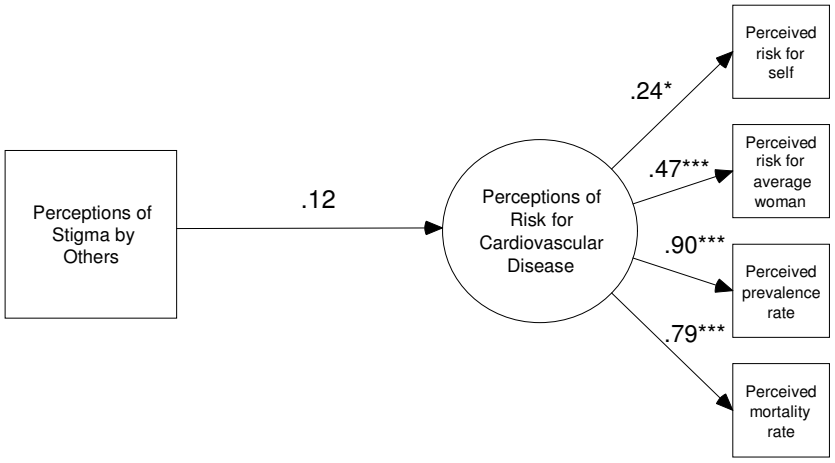


Figure 3.2.1a. Final model of the moderating effect of objective risk factors on the association between perceptions of stigma by others and perceptions of risk for cardiovascular disease in younger women. Results did not support this model, despite the fact that the model was a good fit in a subsample of 293 women with lower levels of objective risk ($\chi^2(4) = 4.69$, $p = .32$; CFI = 1.00; RMSEA = .02, 90% confidence limits for the RMSEA = .00 to .10), and was a good fit in a subsample of 146 women with higher levels of objective risk ($\chi^2(4) = 0.45$, $p = .98$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .00). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

Lower levels of objective risk:



Higher levels of objective risk:

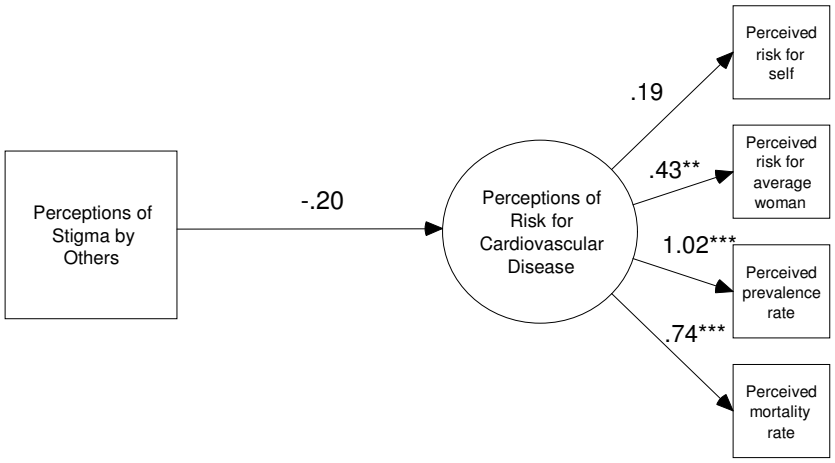


Figure 3.2.1b. Final model of the moderating effect of objective risk factors on the association between perceptions of stigma by others and perceptions of risk for cardiovascular disease in older women. Results did not support the model. The model was a good fit in a subsample of 113 women with lower levels of objective risk ($\chi^2(5) = 4.59$, $p = .47$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .13), and was a good fit in a subsample of 67 women with higher levels of objective risk ($\chi^2(5) = 5.91$, $p = .32$; CFI = .99; RMSEA = .05, 90% confidence limits for the RMSEA = .00 to .19). However, among women with higher levels of objective risk, the model's solution was inadmissible because it resulted in a Heywood case. For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

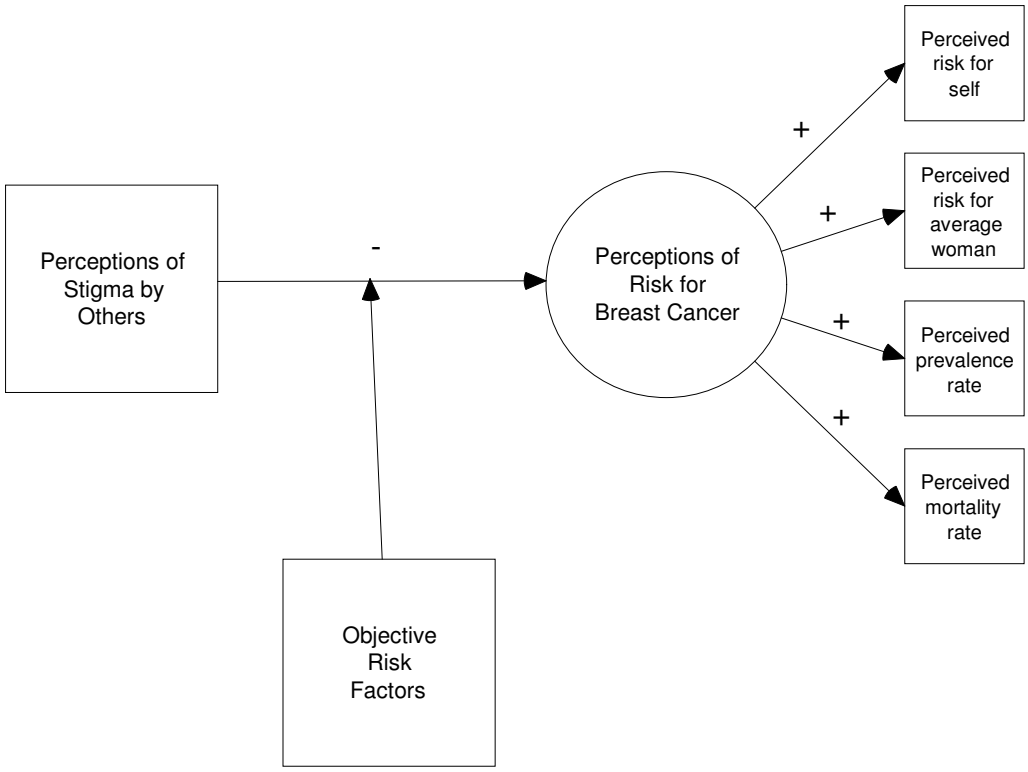
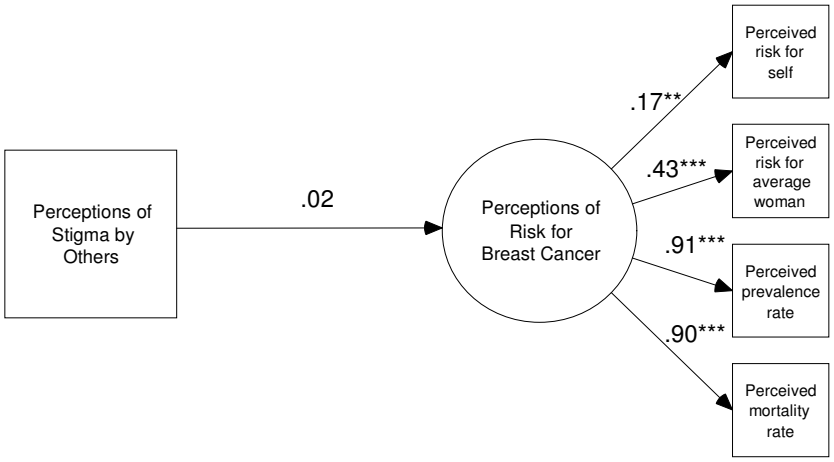


Figure 3.2.2. Hypothesized model of the moderating effect of objective risk factors on the association between perceptions of stigma by others and perceptions of risk for breast cancer. Signs above the paths indicate the hypothesized direction of the associations between variables. For simplicity, error terms have been omitted.

Lower levels of objective risk:



Higher levels of objective risk:

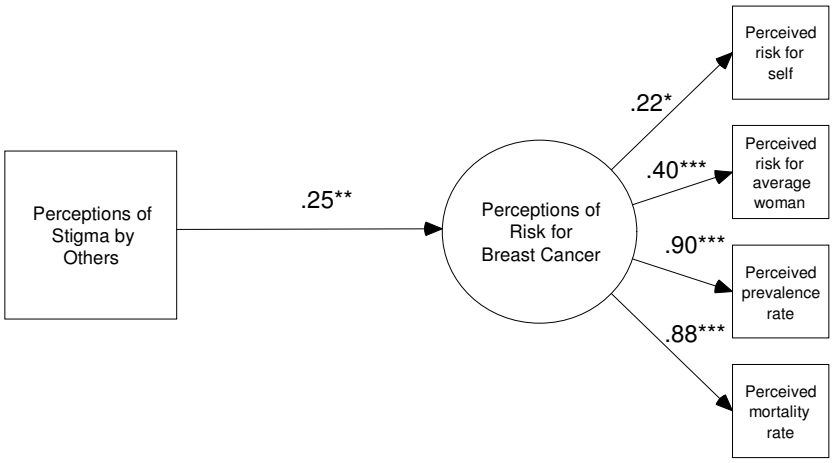
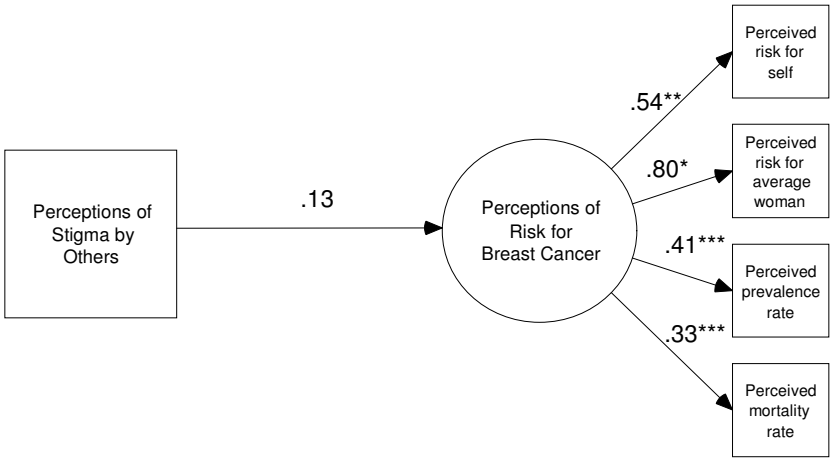


Figure 3.2.2a. Final model of the moderating effect of objective risk factors on the association between perceptions of stigma by others and perceptions of risk for breast cancer in younger women. Results supported the model. The model was a good fit in a subsample of 303 women with lower levels of objective risk ($\chi^2(4) = 3.96$, $p = .41$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .09), and was an adequate fit in a subsample of 149 women with higher levels of objective risk ($\chi^2(4) = 10.38$, $p = .03$; CFI = .97; RMSEA = .10, 90% confidence limits for the RMSEA = .03 to .18). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

Lower levels of objective risk:



Higher levels of objective risk:

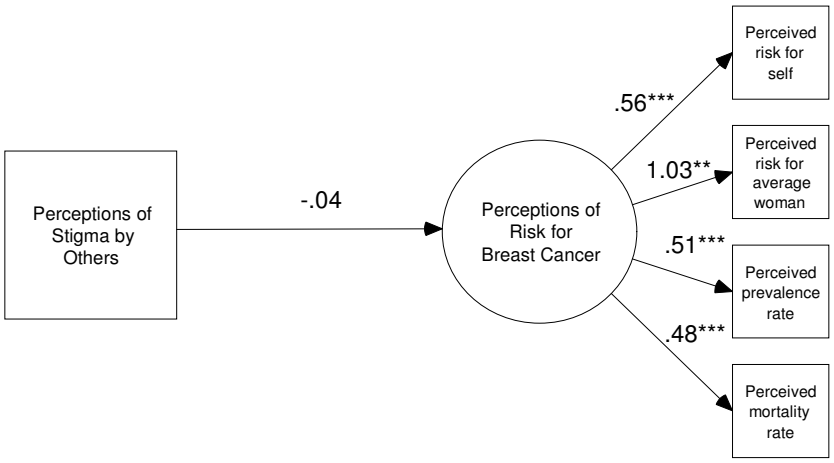


Figure 3.2.2b. Final model of the moderating effect of objective risk factors on the association between perceptions of stigma by others and perceptions of risk for breast cancer in older women. Results did not support the model. The model was a good fit in a subsample of 112 women with lower levels of objective risk ($\chi^2(4) = 4.01$, $p = .41$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .14), and was a good fit in a subsample of 67 women with higher levels of objective risk ($\chi^2(4) = 0.73$, $p = .95$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .02). However, among women with higher levels of objective risk, the model's solution was inadmissible because it resulted in a Heywood case. For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

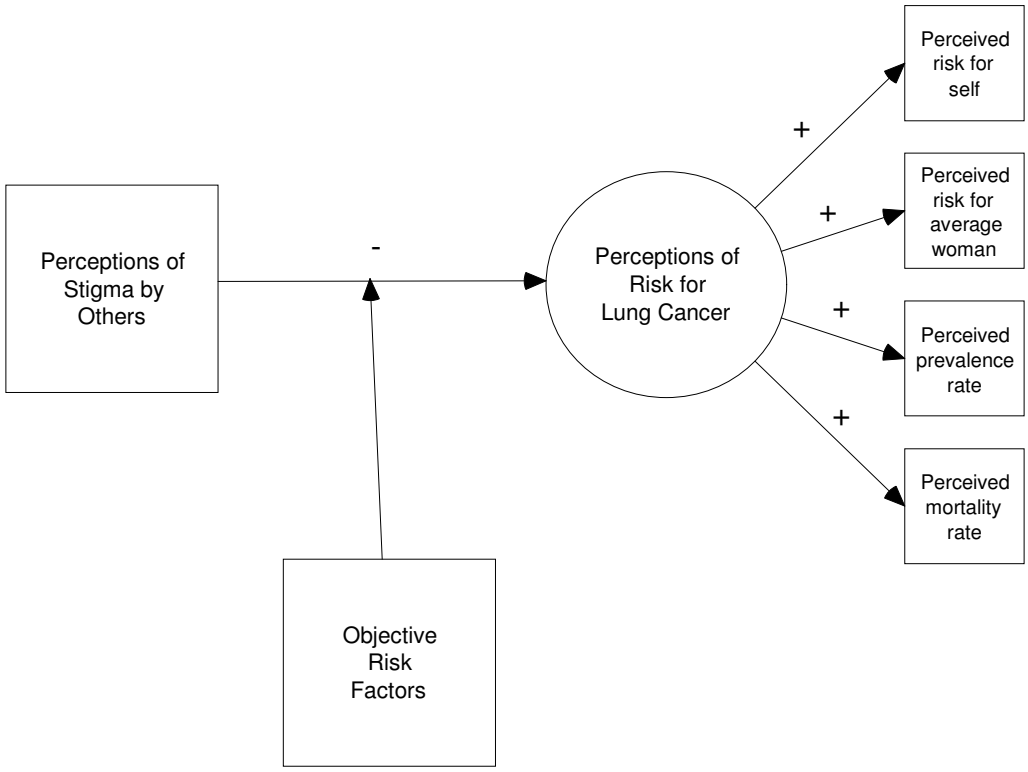
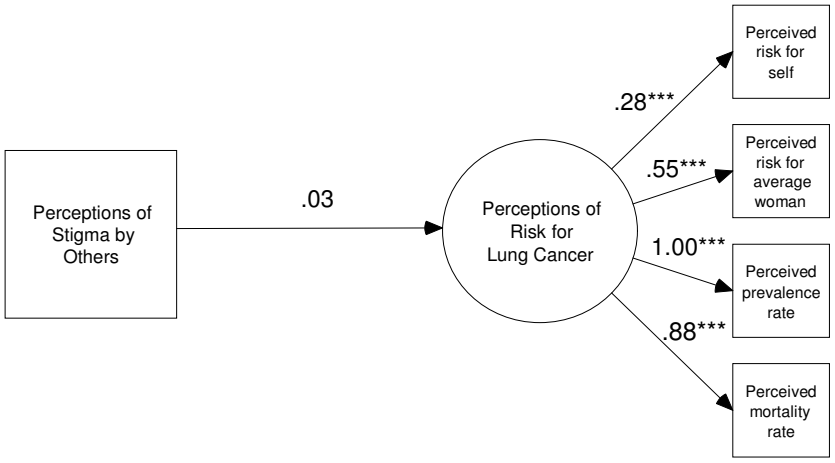


Figure 3.2.3. Hypothesized model of the moderating effect of objective risk factors on the association between perceptions of stigma by others and perceptions of risk for lung cancer. Signs above the paths indicate the hypothesized direction of the associations between variables. For simplicity, error terms have been omitted.

Lower levels of objective risk:



Higher levels of objective risk:

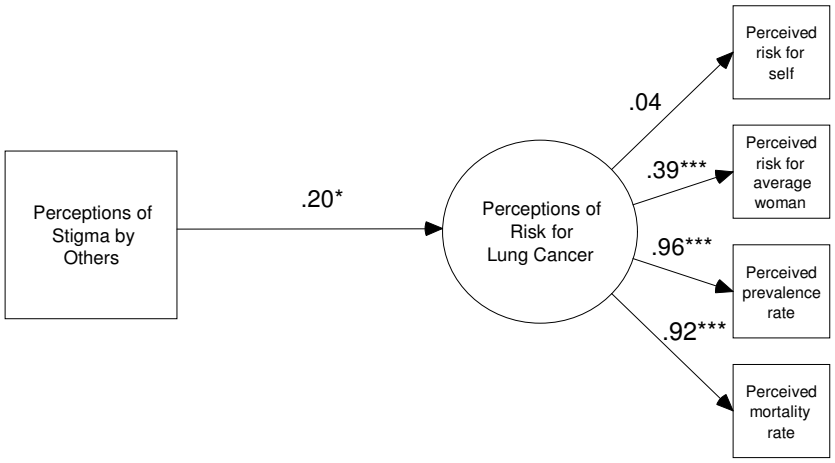
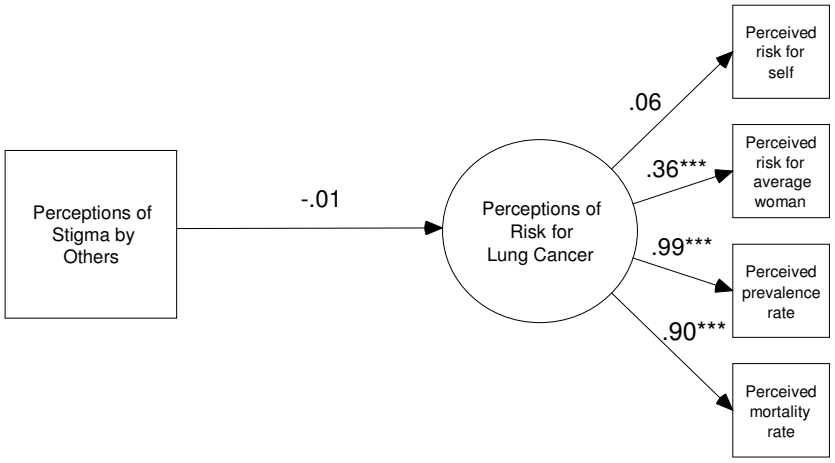


Figure 3.2.3a. Final model of the moderating effect of objective risk factors on the association between perceptions of stigma by others and perceptions of risk for lung cancer in younger women. Results supported the model. The model was an adequate fit in a subsample of 304 women with lower levels of objective risk ($\chi^2(4) = 16.63$, $p = .002$; CFI = .98; RMSEA = .10, 90% confidence limits for the RMSEA = .06 to .16), and was a good fit in a subsample of 149 women with higher levels of objective risk ($\chi^2(4) = 2.91$, $p = .57$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .11). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

Lower levels of objective risk:



Higher levels of objective risk:

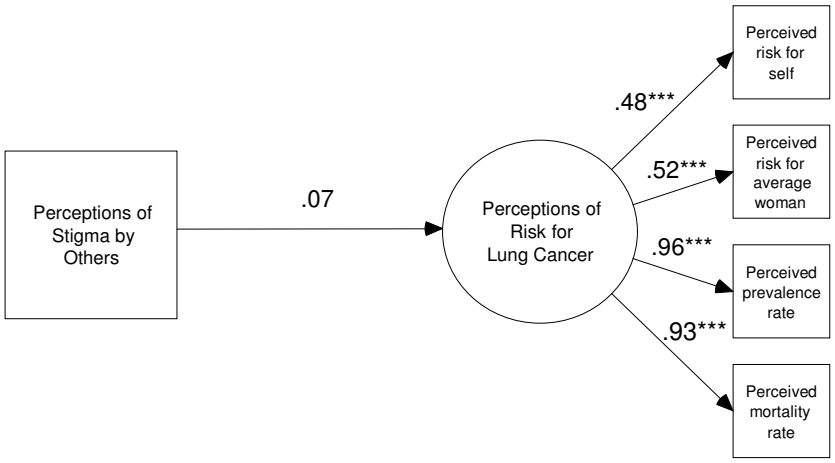


Figure 3.2.3b. Final model of the moderating effect of objective risk factors on the association between perceptions of stigma by others and perceptions of risk for lung cancer in older women. Results did not support the model, despite the fact that the model was a good fit in a subsample of 113 women with lower levels of objective risk ($\chi^2(4) = 3.71$, $p = .45$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .14), and was a good fit in a subsample of 67 women with higher levels of objective risk ($\chi^2(4) = 2.25$, $p = .69$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .14). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

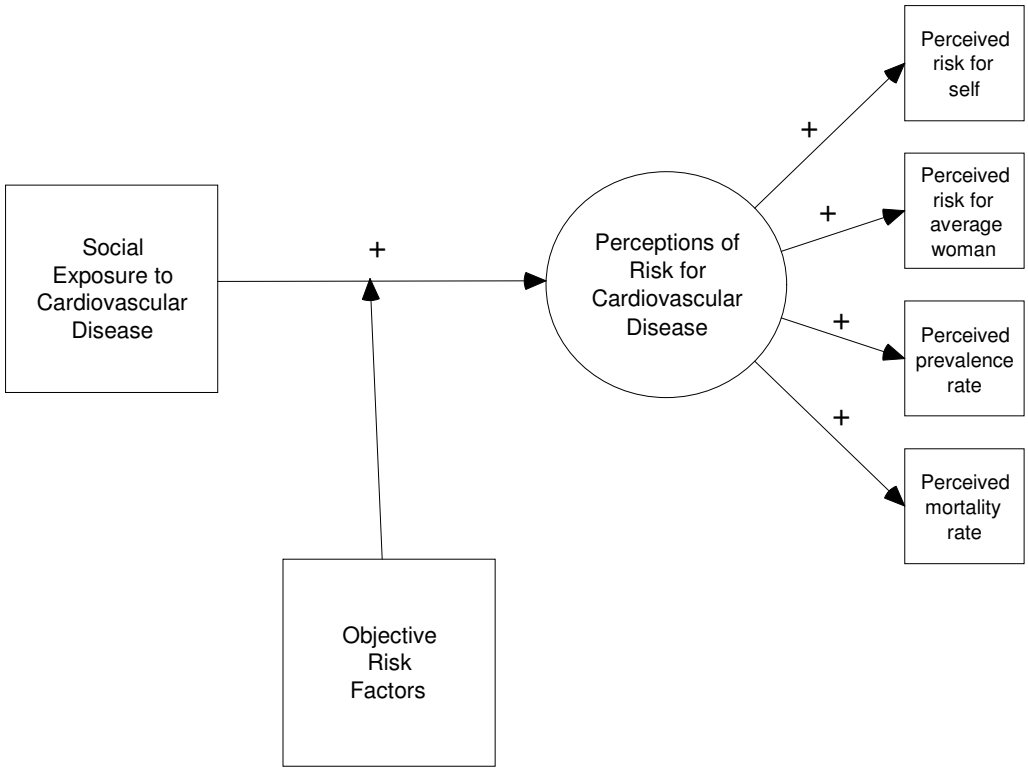
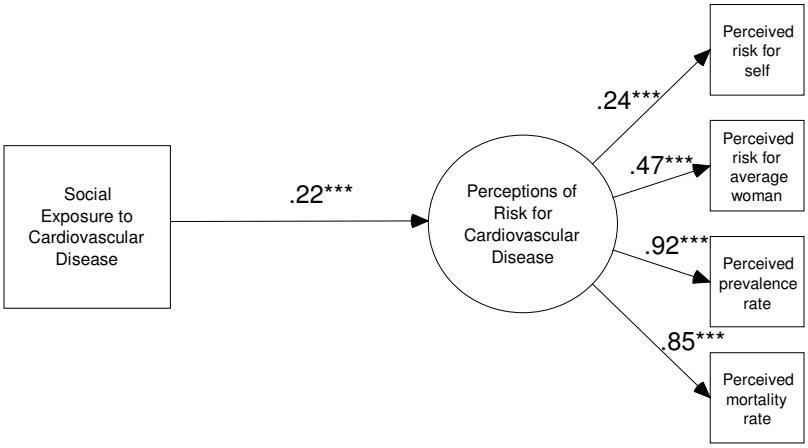


Figure 3.3.1. Hypothesized model of the moderating effect of objective risk factors on the association between social exposure to cardiovascular disease and perceptions of risk for cardiovascular disease. Signs above the paths indicate the hypothesized direction of the associations between variables. For simplicity, error terms have been omitted.

Lower levels of objective risk:



Higher levels of objective risk:

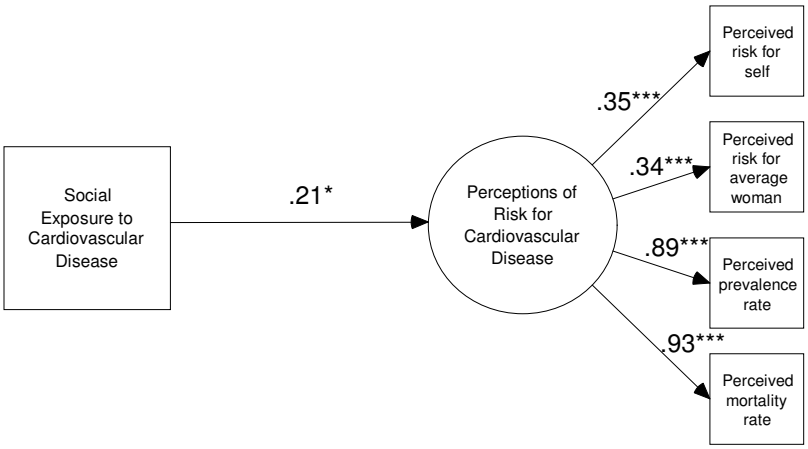
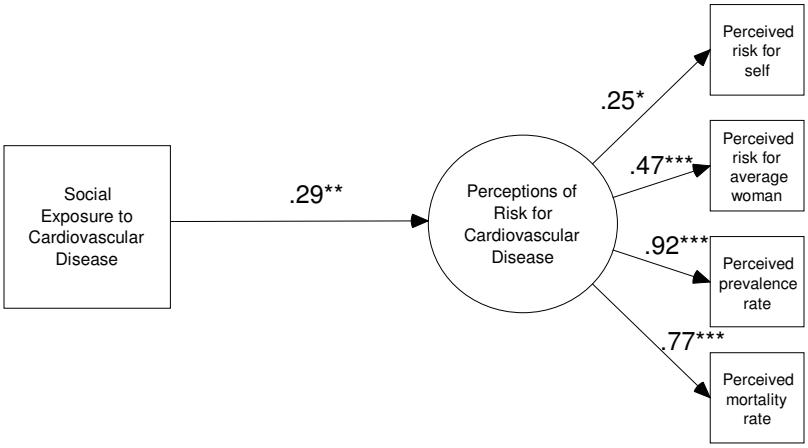


Figure 3.3.1a. Final model of the moderating effect of objective risk factors on the association between social exposure to cardiovascular disease and perceptions of risk for cardiovascular disease in younger women. Results did not support the model. The model was a poor fit in a subsample of 305 women with lower levels of objective risk ($\chi^2(4) = 29.46$, $p < .001$; CFI = .94; RMSEA = .15, 90% confidence limits for the RMSEA = .10 to .20), and was a poor fit in a subsample of 149 women with higher levels of objective risk ($\chi^2(4) = 20.32$, $p < .001$; CFI = .93; RMSEA = .17, 90% confidence limits for the RMSEA = .10 to .24). The data were substantially nonnormal; however, bootstrapping with maximum likelihood estimation confirmed that parameter estimates were not biased. For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

Lower levels of objective risk:



Higher levels of objective risk:

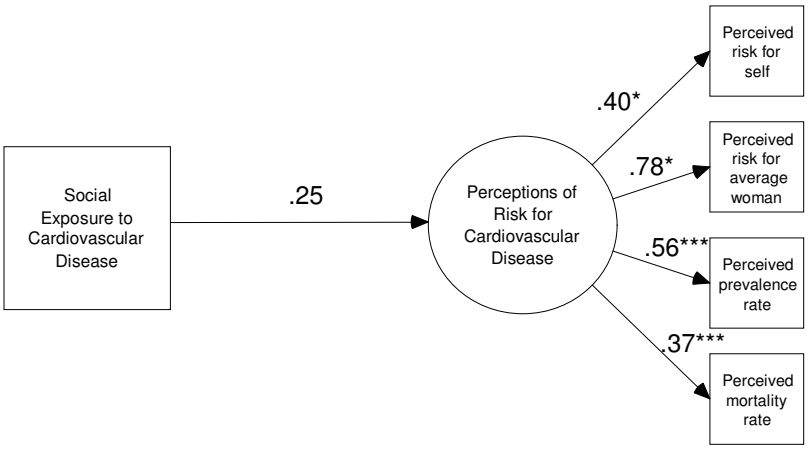


Figure 3.3.1b. Final model of the moderating effect of objective risk factors on the association between social exposure to cardiovascular disease and perceptions of risk for cardiovascular disease in older women. Results did not support the model. The model was a poor fit in a subsample of 113 women with lower levels of objective risk ($\chi^2(5) = 17.63$, $p = .003$; CFI = .90; RMSEA = .15, 90% confidence limits for the RMSEA = .08 to .23), and was a good fit in a subsample of 67 women with higher levels of objective risk ($\chi^2(4) = 2.15$, $p = .71$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .14). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; * = $p \leq .01$; *** = $p \leq .001$.

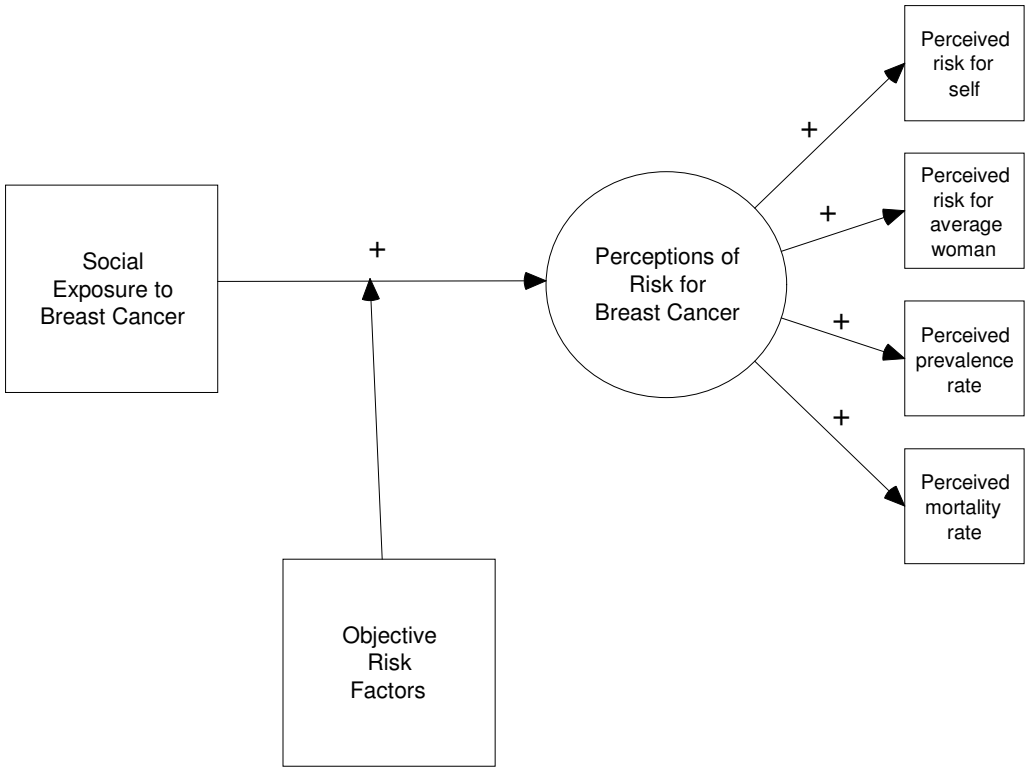
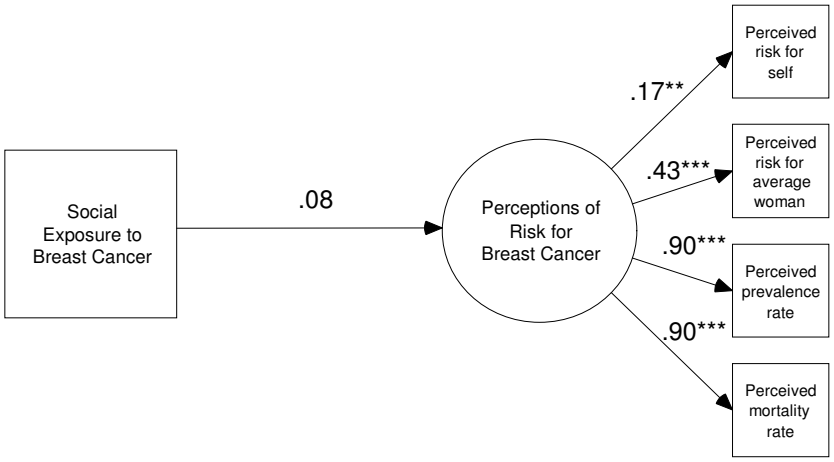


Figure 3.3.2. Hypothesized model of the moderating effect of objective risk factors on the association between social exposure to breast cancer and perceptions of risk for breast cancer. Signs above the paths indicate the hypothesized direction of the associations between variables. For simplicity, error terms have been omitted.

Lower levels of objective risk:



Higher levels of objective risk:

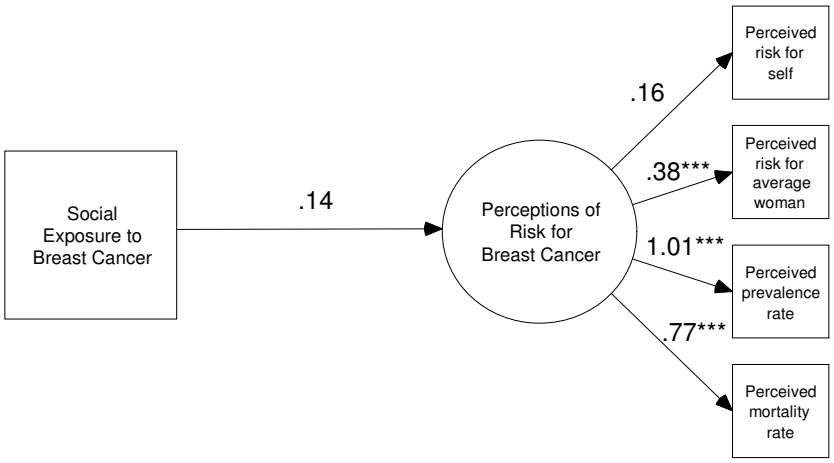
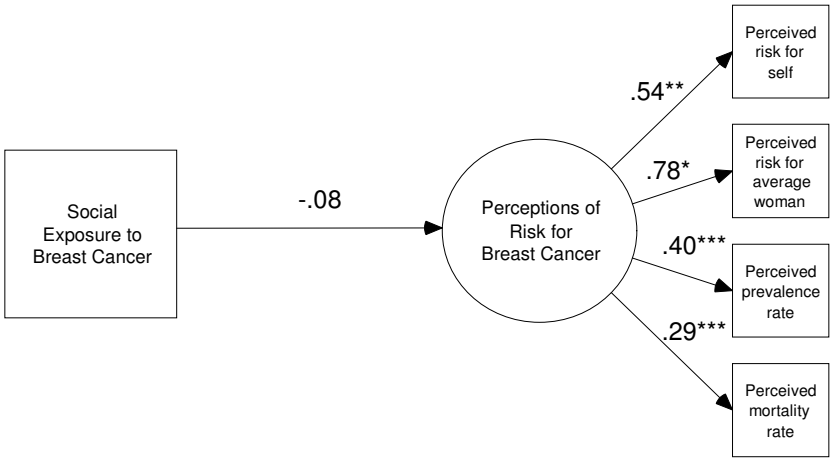


Figure 3.3.2a. Final model of the moderating effect of objective risk factors on the association between social exposure to breast cancer and perceptions of risk for breast cancer in younger women. Results did not support the model. The model was a poor fit in a subsample of 304 women with lower levels of objective risk ($\chi^2(4) = 33.58$, $p < .001$; CFI = .93; RMSEA = .16, 90% confidence limits for the RMSEA = .11 to .21), and was a poor fit in a subsample of 148 women with higher levels of objective risk ($\chi^2(4) = 10.63$, $p = .03$; CFI = .96; RMSEA = .11, 90% confidence limits for the RMSEA = .03 to .19). However, among women with higher levels of objective risk, the model's solution was inadmissible because it resulted in a Heywood case. For simplicity, error terms have been omitted. All values are standardized path coefficients. ** = $p \leq .01$; *** = $p \leq .001$.

Lower levels of objective risk:



Higher levels of objective risk:

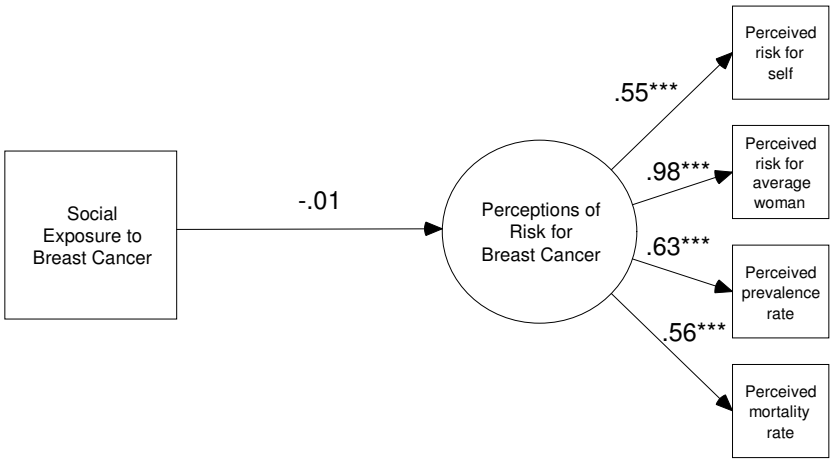


Figure 3.3.2b. Final model of the moderating effect of objective risk factors on the association between social exposure to breast cancer and perceptions of risk for breast cancer in older women. Results did not support the model. The model was a good fit in a subsample of 111 women with lower levels of objective risk ($\chi^2(4) = 6.46$, $p = .17$; CFI = .98; RMSEA = .08, 90% confidence limits for the RMSEA = .00 to .18), and was a poor fit in a subsample of 66 women with higher levels of objective risk ($\chi^2(4) = 12.55$, $p = .01$; CFI = .94; RMSEA = .18, 90% confidence limits for the RMSEA = .08 to .30). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; ** = $p \leq .01$; *** = $p \leq .001$.

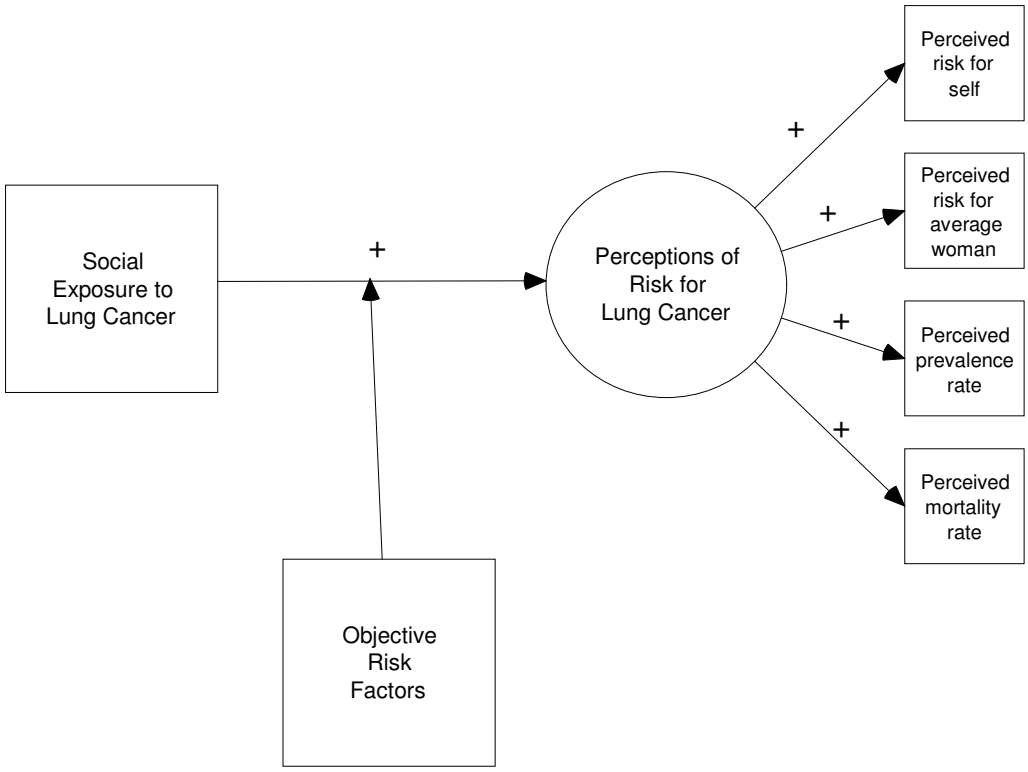
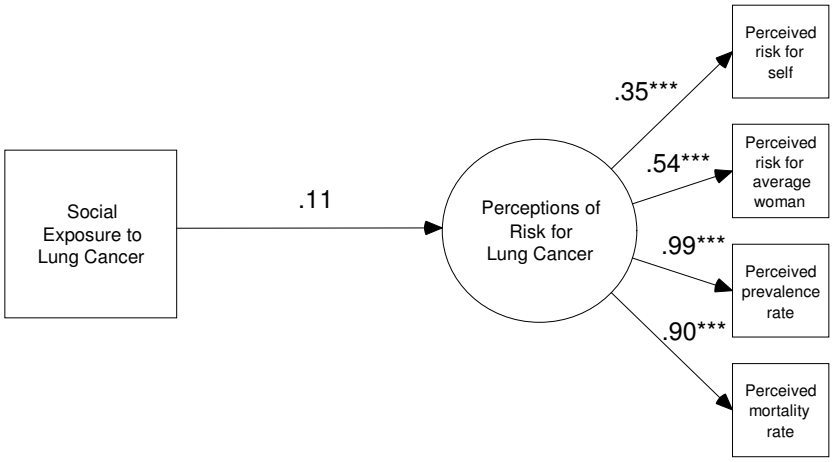


Figure 3.3.3. Hypothesized model of the moderating effect of objective risk factors on the association between social exposure to lung cancer and perceptions of risk for lung cancer. Signs above the paths indicate the hypothesized direction of the associations between variables. For simplicity, error terms have been omitted.

Lower levels of objective risk:



Higher levels of objective risk:

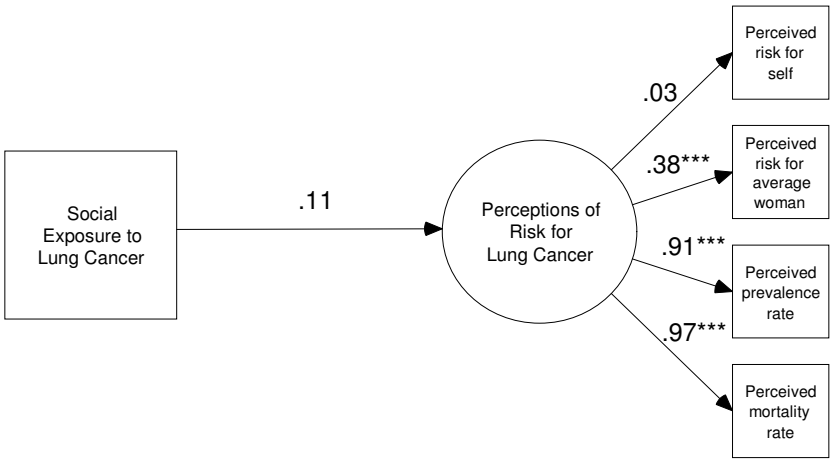
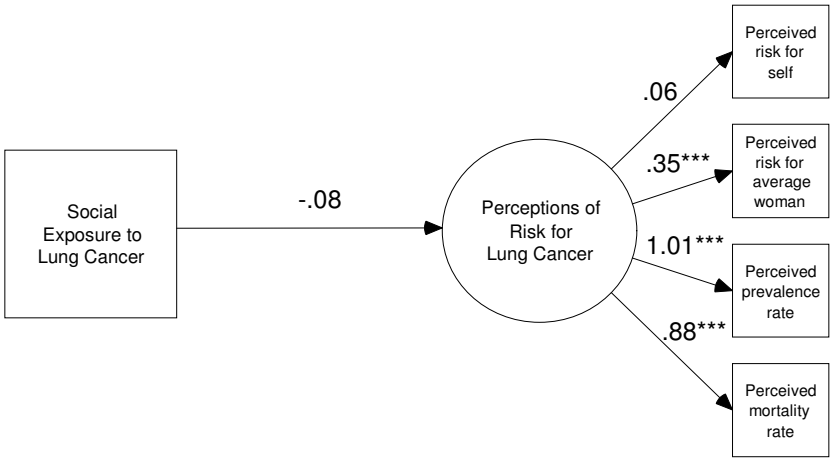


Figure 3.3.3a. Final model of the moderating effect of objective risk factors on the association between social exposure to lung cancer and perceptions of risk for lung cancer in younger women. Results did not support the model, despite the fact that the model was a good fit in a subsample of 285 women with lower levels of objective risk ($\chi^2(4) = 3.57$, $p = .47$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .09), and was an adequate fit in a subsample of 146 women with higher levels of objective risk ($\chi^2(4) = 10.15$, $p = .04$; CFI = .98; RMSEA = .10, 90% confidence limits for the RMSEA = .02 to .18). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

Lower levels of objective risk:



Higher levels of objective risk:

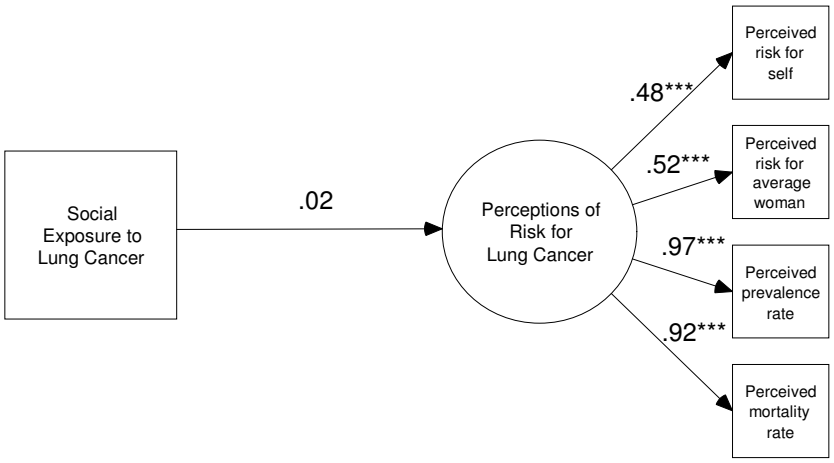


Figure 3.3.3b. Final model of the moderating effect of objective risk factors on the association between social exposure to lung cancer and perceptions of risk for lung cancer in older women. Results did not support the model. The model was a good fit in a subsample of 113 women with lower levels of objective risk ($\chi^2(4) = 6.40$, $p = .17$; CFI = .99; RMSEA = .07, 90% confidence limits for the RMSEA = .00 to .17), and was a good fit in a subsample of 67 women with higher levels of objective risk ($\chi^2(4) = 0.75$, $p = .95$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .03). However, among women with lower levels of objective risk, the model's solution was inadmissible because it resulted in a Heywood case. For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

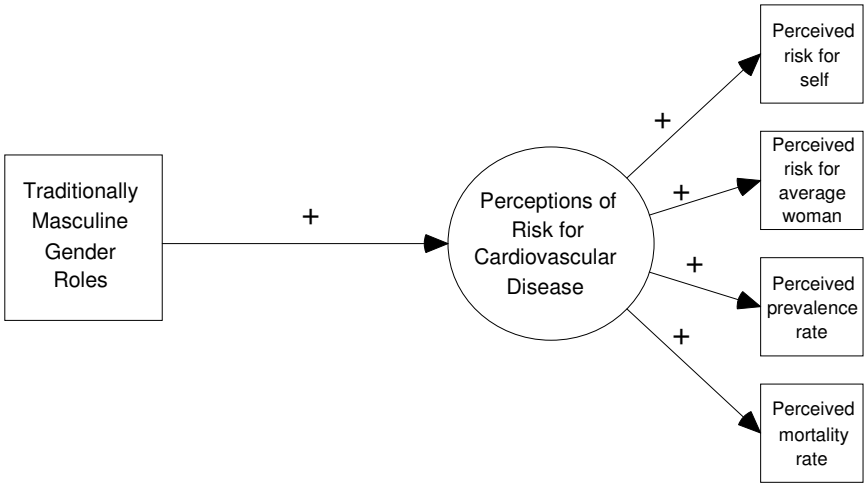


Figure 4.1. Hypothesized model for the relationship between identification with traditionally masculine gender roles and perceptions of risk for cardiovascular disease. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

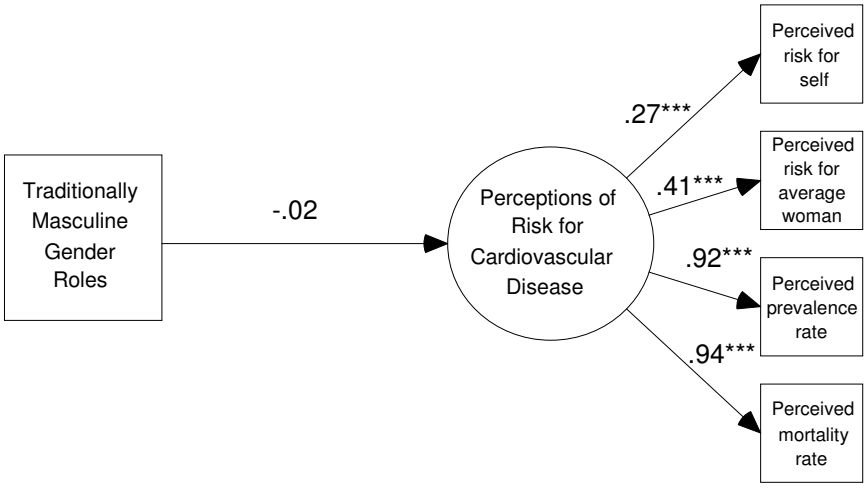


Figure 4.1a. Final model for the relationship between self-identification with traditionally masculine gender roles and perceptions of risk for cardiovascular disease in younger women. Results did not support the model, although the model was an adequate fit to the data in a subsample of 357 women ($\chi^2(4) = 13.90$, $p = .008$; CFI = .98; RMSEA = .08, 90% confidence limits for the RMSEA = .04 to .13). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

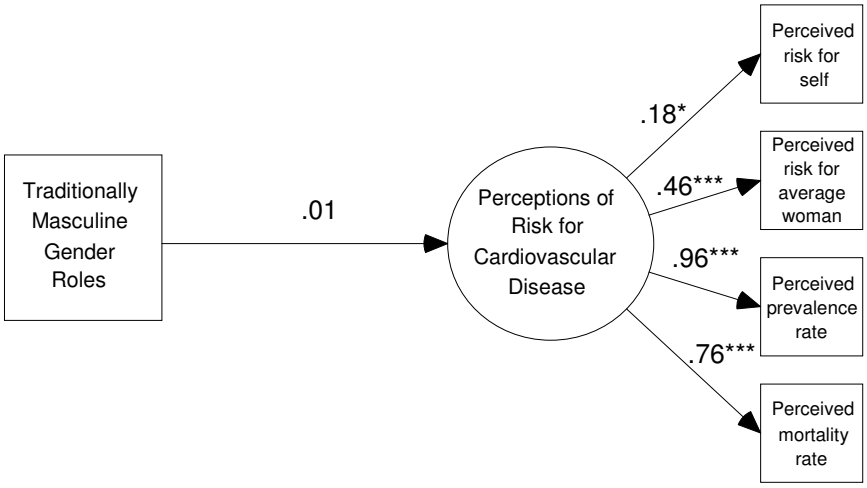


Figure 4.1b. Final model for the relationship between identification with traditionally masculine gender roles and perceptions of risk for cardiovascular disease in older women. Results did not support the model, although the model was a good fit to the data in a subsample of 179 women ($\chi^2(5) = 5.52$, $p = .36$; CFI = 1.00; RMSEA = .02, 90% confidence limits for the RMSEA = .00 to .11). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

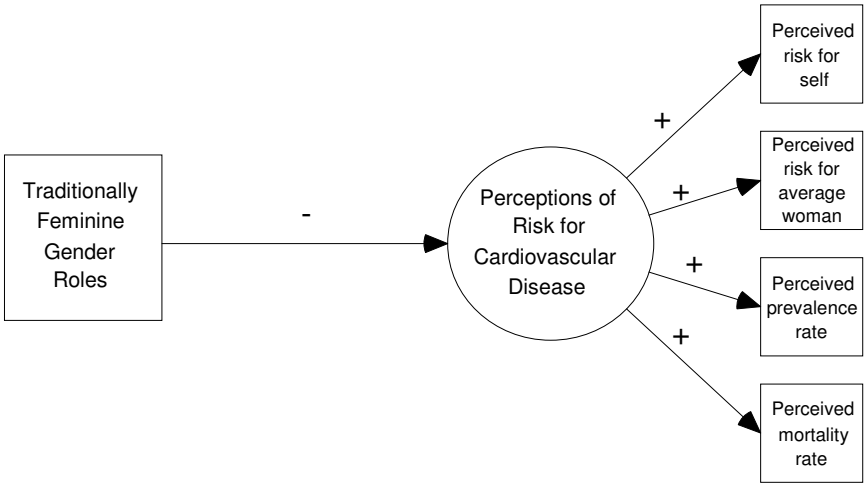


Figure 4.2. Hypothesized model for the relationship between identification with traditionally feminine gender roles and perceptions of risk for cardiovascular disease. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

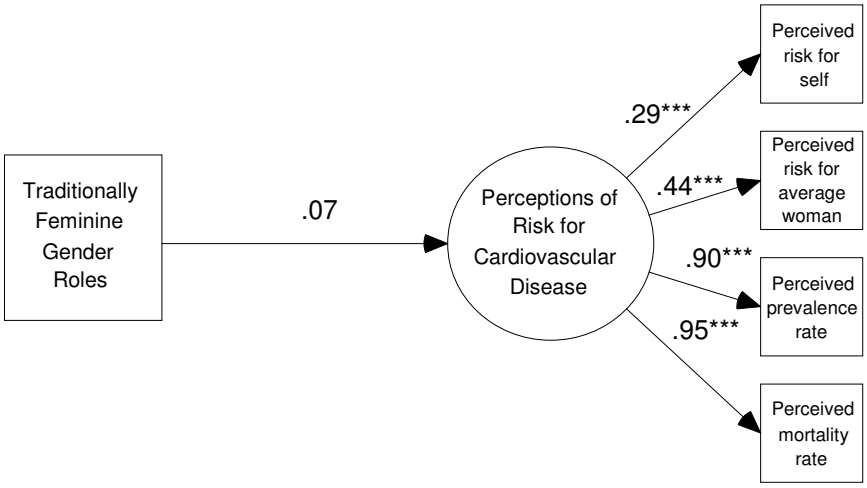


Figure 4.2a. Final model for the relationship between identification with traditionally feminine gender roles and perceptions of risk for cardiovascular disease in younger women. Results did not support the model, although the model was a good fit to the data in a subsample of 356 women ($\chi^2(4) = 2.40$, $p = .66$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .06). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

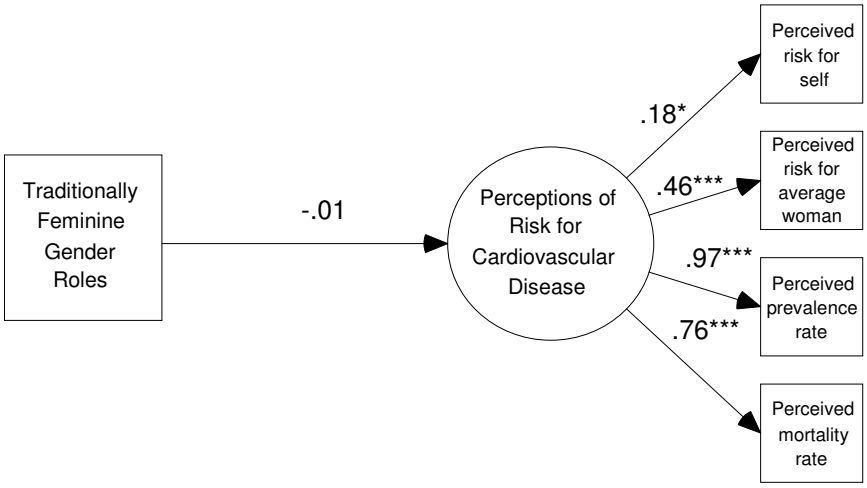


Figure 4.2b. Final model for the relationship between identification with traditionally feminine gender roles and perceptions of risk for cardiovascular disease in older women. Results did not support the model, although the model was a good fit to the data in a subsample of 179 women ($\chi^2(5) = 10.12$, $p = .07$; CFI = .97; RMSEA = .08, 90% confidence limits for the RMSEA = .00 to .14). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

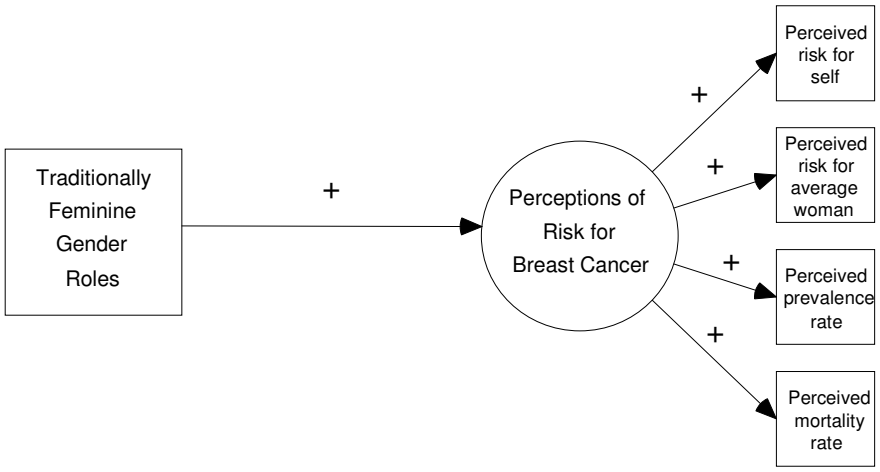


Figure 4.3. Hypothesized model for the relationship between identification with traditionally feminine gender roles and perceptions of risk for breast cancer. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

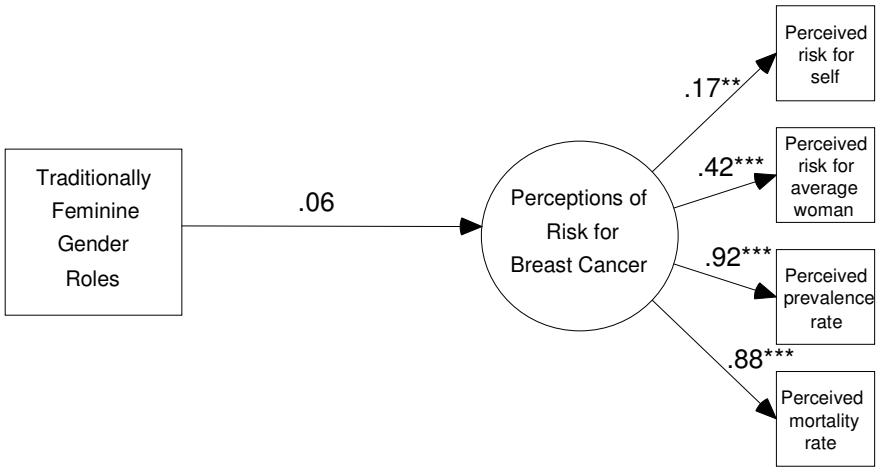


Figure 4.3a. Final model for the relationship between identification with traditionally feminine gender roles and perceptions of risk for breast cancer in younger women. Results did not support the model, although the model was a good fit to the data in a subsample of 374 women ($\chi^2(4) = 1.05$, $p = .90$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .03). For simplicity, error terms have been omitted. All values are standardized path coefficients. ** = $p \leq .01$; *** = $p \leq .001$.

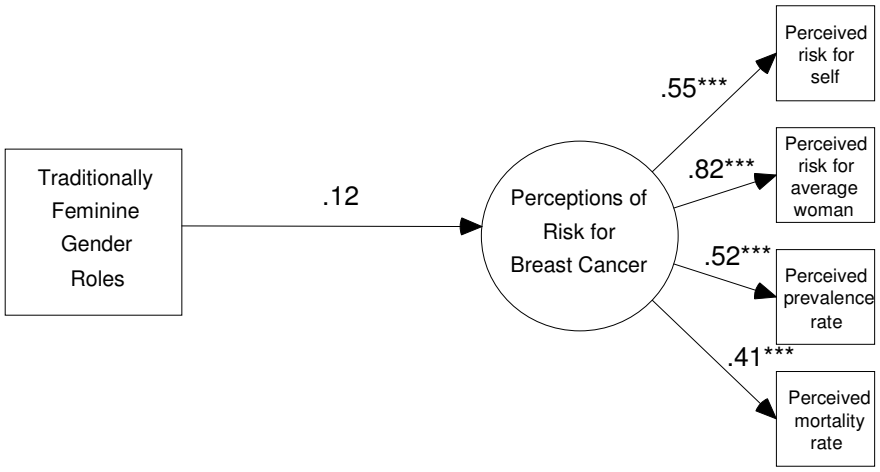


Figure 4.3b. Final model for the relationship between identification with traditionally feminine gender roles and perceptions of risk for breast cancer in older women. Results did not support the model, although the model was a good fit to the data in a subsample of 174 women ($\chi^2(4) = 2.28$, $p = .69$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .09). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

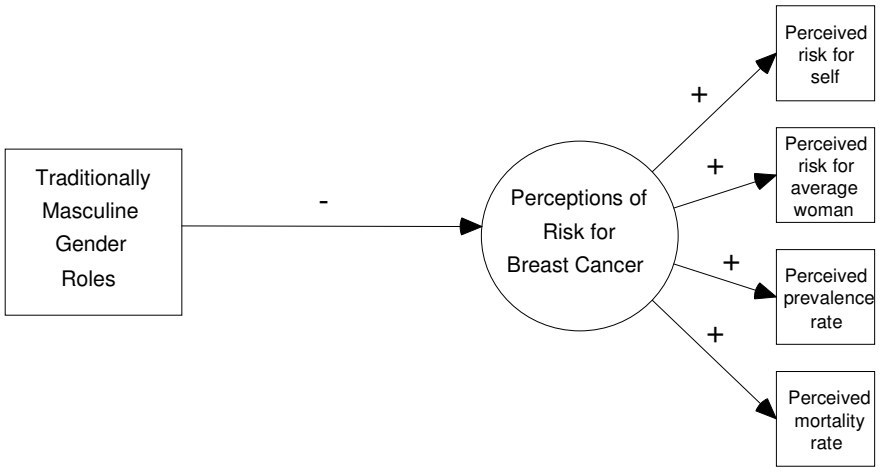


Figure 4.4. Hypothesized model for the relationship between identification with traditionally masculine gender roles and perceptions of risk for breast cancer. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

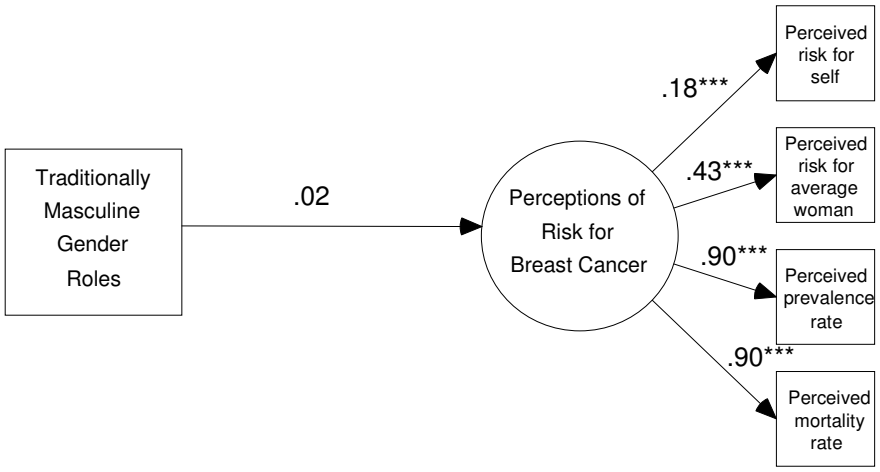


Figure 4.4a. Final model for the relationship between identification with traditionally masculine gender roles and perceptions of risk for breast cancer in younger women. Results did not support the model, although the model was an adequate fit to the data in a subsample of 375 women ($\chi^2(4) = 15.45$, $p = .004$; CFI = .98; RMSEA = .09, 90% confidence limits for the RMSEA = .04 to .14). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

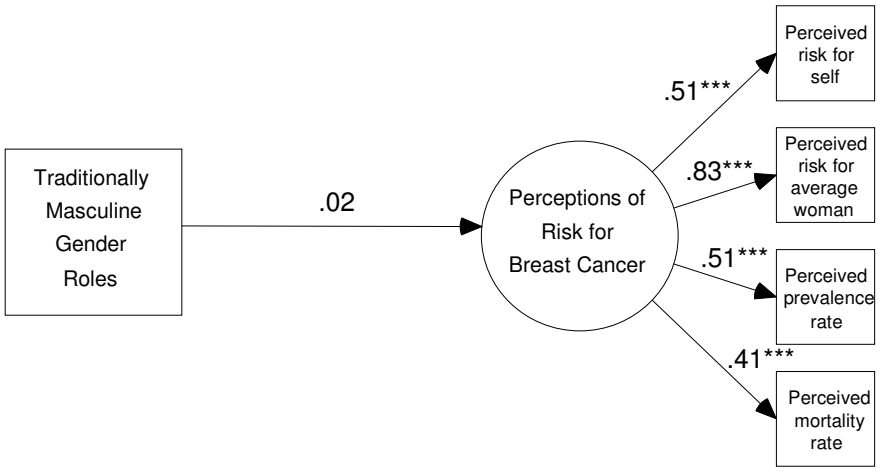


Figure 4.4b. Final model for the relationship between identification with traditionally masculine gender roles and perceptions of risk for breast cancer in older women. Results did not support the model, although the model was a good fit to the data in a subsample of 174 women ($\chi^2(4) = 1.43$, $p = .84$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .07), identification with traditionally masculine gender roles was not significantly related to perceptions of risk for breast cancer. For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

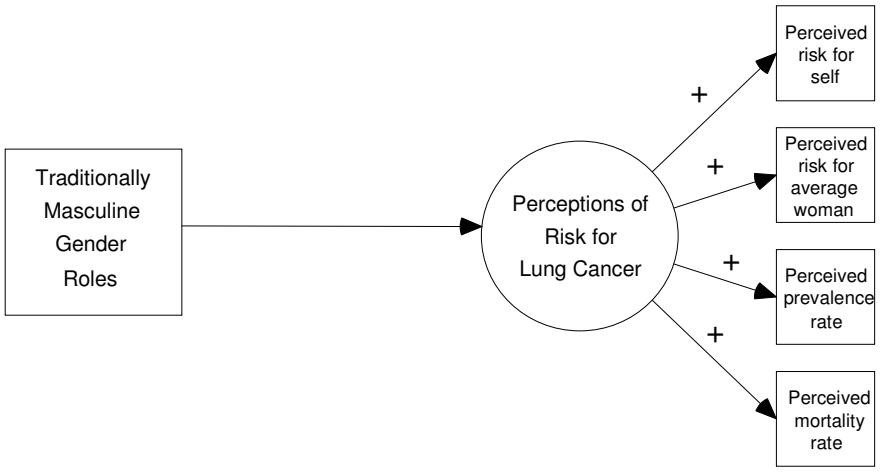


Figure 4.5. Exploratory model for the relationship between identification with traditionally masculine gender roles and perceptions of risk for lung cancer. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

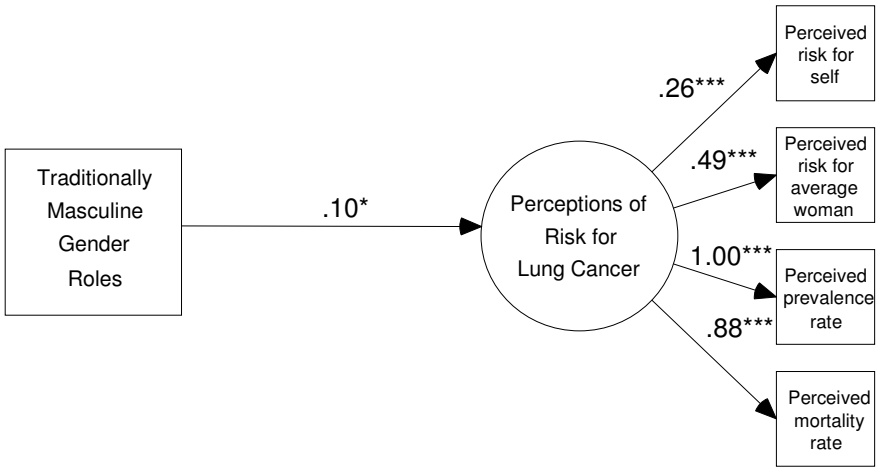


Figure 4.5a. Final model for the relationship between identification with traditionally masculine gender roles and perceptions of risk for lung cancer in younger women. Results supported the model, which was a good fit to the data in a subsample of 374 women ($\chi^2(4) = 9.30$, $p = .05$; CFI = .99; RMSEA = .06, 90% confidence limits for the RMSEA = .00 to .11). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

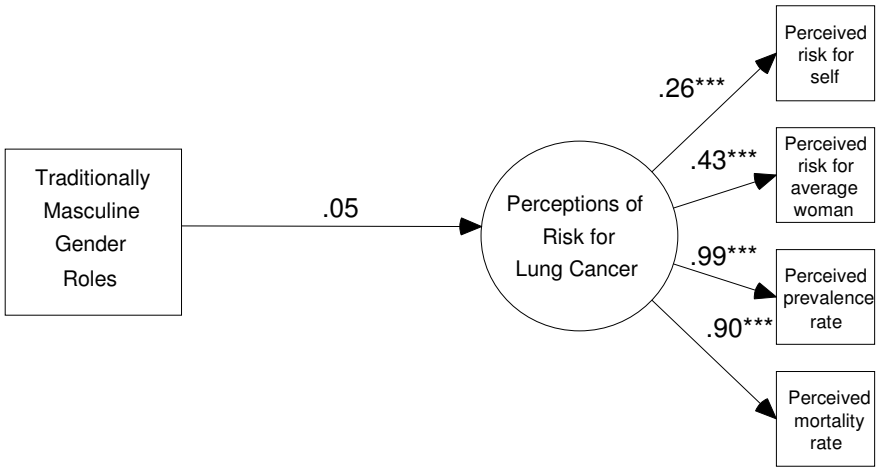


Figure 4.5b. Final model for the relationship between identification with traditionally masculine gender roles and perceptions of risk for lung cancer in older women. Results did not support the model, although the model was a good fit to the data in a subsample of 179 women ($\chi^2(4) = 6.74$, $p = .15$; CFI = .99; RMSEA = .06, 90% confidence limits for the RMSEA = .00 to .14). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

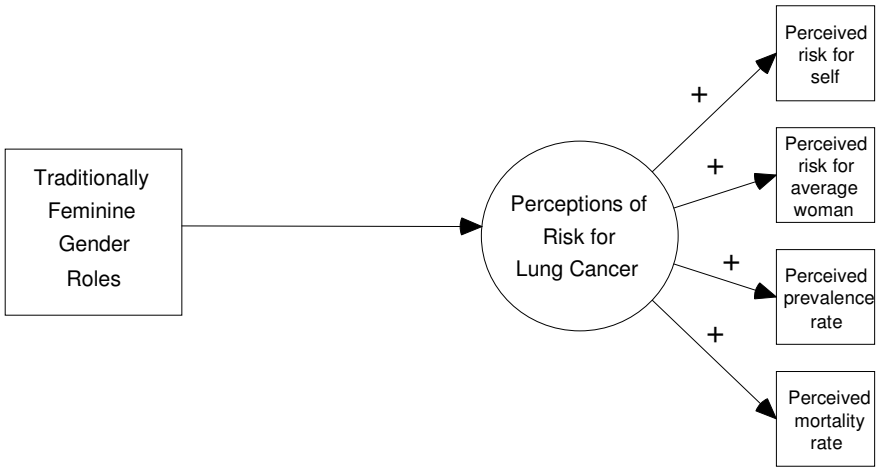


Figure 4.6. Exploratory model for the relationship between identification with traditionally feminine gender roles and perceptions of risk for lung cancer. Signs above the paths indicate the predicted direction of the association between study variables. For simplicity, error terms have been omitted.

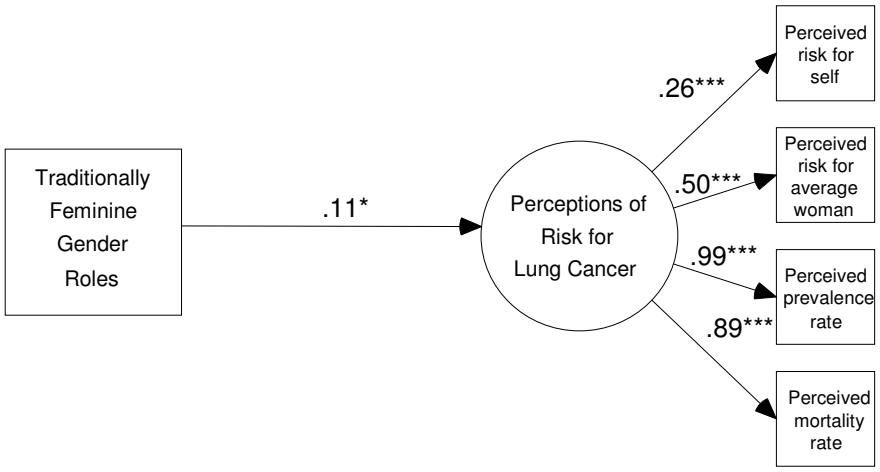


Figure 4.6a. Final model for the relationship between identification with traditionally feminine gender roles and perceptions of risk for lung cancer in younger women. Results supported the model, which was a good fit to the data in a subsample of 370 women ($\chi^2(4) = 3.07$, $p = .55$; CFI = 1.00; RMSEA = .00, 90% confidence limits for the RMSEA = .00 to .07). For simplicity, error terms have been omitted. All values are standardized path coefficients. * = $p \leq .05$; *** = $p \leq .001$.

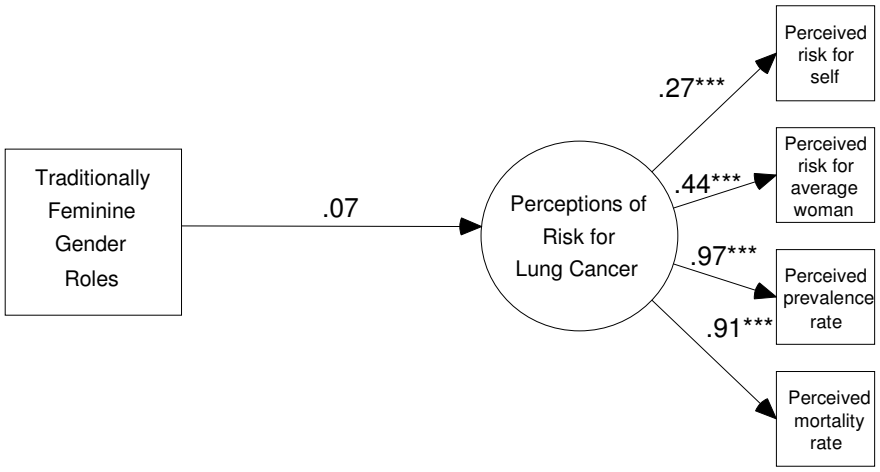


Figure 4.6b. Final model for the relationship between identification with traditionally feminine gender roles and perceptions of risk for lung cancer in older women. Results did not support the model, although the model was a good fit to the data in a subsample of 178 women ($\chi^2(4) = 7.57$, $p = .11$; CFI = .99; RMSEA = .07, 90% confidence limits for the RMSEA = .00 to .15). For simplicity, error terms have been omitted. All values are standardized path coefficients. *** = $p \leq .001$.

Appendix A: Consent Form for Younger Women (Ages 18 to 25)

Project Title: Women's Perceptions and Beliefs about Health
Principal Investigator: Dr. Marci Lobel
Co-Investigator: Jada Hamilton – Graduate Student

RESEARCH CONSENT FORM

Dear Participant,

You are being asked to be a volunteer in a research study.

PURPOSE

- The purpose of this study is to investigate women's thoughts about a variety of topics, including health issues and life events. You have been asked to participate in this study because we are interested in the thoughts of women between the ages of 18 and 25.

PROCEDURES

- If you decide to be in this study, your part will involve carefully and honestly answering the survey questions on the following pages.
- This survey should take you approximately 30 minutes to complete.

RISKS / DISCOMFORTS

- There are no foreseeable risks or discomforts associated with your participation in this study.

BENEFITS

- There may be no foreseeable benefit to you as a result of being in this study.

PAYMENT TO YOU

- You will receive one research credit for your participation, in accordance with the Department of Psychology Subject Pool guidelines.

CONFIDENTIALITY

- Your identity will be completely anonymous. We will not collect any information that would allow us to identify you. All of the collected information will be kept in a secure location.

COSTS TO YOU

- There are no foreseeable costs to you as a result of being in this study.

SUBJECT RIGHTS

- Your participation in this study is voluntary. You do not have to be in this study if you don't want to be.

- You have the right to change your mind and leave the study at any time without giving any reason, and without penalty.
- Any new information that may make you change your mind about being in this study will be given to you.
- You will get a copy of this letter to keep.

QUESTIONS ABOUT THE STUDY OR YOUR RIGHTS AS A RESEARCH SUBJECT

- If you have any questions about the study, you may contact Dr. Marci Lobel at telephone number (631) 632-9208.
- If you have any questions about your rights as a research subject, you may contact Ms. Judy Matuk, Committee on Research Involving Human Subjects, (631) 632-9036.

If you complete the attached survey, it means that you have read (or have had read to you) the information contained in this letter, and would like to be a volunteer in this research study.

Thank you,

_____

Dr. Marci Lobel, Principal Investigator

_____

Jada Hamilton, Co-Investigator

Appendix B: Cover Letter for Older Women (Ages 40 and Above)

Dear Study Participant,

We are contacting you about the "Women's Perceptions and Beliefs about Health" study because you provided your contact information to a Stony Brook University student indicating that you would like to participate. In this study, women over the age of 40 answer simple questions about health. This packet contains:

- A **consent letter** with details about your involvement in this study.
- The study **questionnaire**. Please read all directions and answer all questions as honestly and completely as possible.
- A **stamped and addressed envelope** for you to return your completed questionnaire to us. When you mail us the completed questionnaire, please be sure that you do not include your name or address on any of the materials. We want to keep your responses completely anonymous.

Please complete the questionnaire and return it to us as soon as possible. If you have any questions about the study, please feel free to contact Dr. Marci Lobel in the Department of Psychology at Stony Brook University at (631) 632-9208.

By participating in this study, you are contributing to important scientific research on women's health. We appreciate your contribution very much!

_____
Dr. Marci Lobel, Principal Investigator

_____
Jada Hamilton, Co-Investigator

Appendix C: Consent Letter for Older Women (Ages 40 and Above)

Project Title: Women's Perceptions and Beliefs about Health
Principal Investigator: Dr. Marci Lobel
Co-Investigator: Jada Hamilton – Graduate Student

RESEARCH CONSENT FORM

Dear Participant,

You are being asked to be a volunteer in a research study.

PURPOSE

- The purpose of this study is to investigate women's thoughts about a variety of topics, including health issues and life events. You have been asked to participate in this study because we are interested in the thoughts of women over the age of 40.

PROCEDURES

- If you decide to be in this study, your part will involve carefully and honestly answering the questions on the following pages.
- This questionnaire should take you approximately 30 minutes to complete.

RISKS / DISCOMFORTS

- There are no foreseeable risks or discomforts associated with your participation in this study.

BENEFITS

- There may be no foreseeable benefit to you as a result of being in this study.

PAYMENT TO YOU

- You will not be paid for your involvement in this study.

CONFIDENTIALITY

- Your identity will be completely anonymous. We will not collect any information that would allow us to identify you, such as your name or address. All of the collected information will be kept in a secure location.

COSTS TO YOU

- There are no foreseeable costs to you as a result of being in this study.

SUBJECT RIGHTS

- Your participation in this study is voluntary. You do not have to be in this study if you don't want to be.

- You have the right to change your mind and leave the study at any time without giving any reason, and without penalty.
- Any new information that may make you change your mind about being in this study will be given to you.
- You will get to keep your copy of this letter.

QUESTIONS ABOUT THE STUDY OR YOUR RIGHTS AS A RESEARCH SUBJECT

- If you have any questions about the study, you may contact Dr. Marci Lobel at telephone number (631) 632-9208.
- If you have any questions about your rights as a research subject, you may contact Ms. Judy Matuk, Committee on Research Involving Human Subjects, (631) 632-9036.

If you complete the attached survey, it means that you have read (or have had read to you) the information contained in this letter, and would like to be a volunteer in this research study.

Thank you,

_____

Dr. Marci Lobel, Principal Investigator

_____

Jada Hamilton, Co-Investigator