
[All ETDs from UAB](#)

[UAB Theses & Dissertations](#)

2012

Cardiovascular Disease Knowledge and Risk Perception among Women with Recent Preeclampsia: Interventional Education in Disease Management and Prevention

Patsy M. Spratling
University of Alabama at Birmingham

Follow this and additional works at: <https://digitalcommons.library.uab.edu/etd-collection>

Recommended Citation

Spratling, Patsy M., "Cardiovascular Disease Knowledge and Risk Perception among Women with Recent Preeclampsia: Interventional Education in Disease Management and Prevention" (2012). *All ETDs from UAB*. 3020.

<https://digitalcommons.library.uab.edu/etd-collection/3020>

This content has been accepted for inclusion by an authorized administrator of the UAB Digital Commons, and is provided as a free open access item. All inquiries regarding this item or the UAB Digital Commons should be directed to the [UAB Libraries Office of Scholarly Communication](#).

CARDIOVASCULAR DISEASE KNOWLEDGE AND RISK PERCEPTION AMONG
WOMEN WITH RECENT PREECLAMPSIA: INTERVENTIONAL EDUCATION IN
DISEASE MANAGEMENT AND PREVENTION

by

PATSY M. SPRATLING

ERICA R. PRYOR, COMMITTEE CHAIR
ASHLEY L. HODGES
JAMES N. MARTIN, Jr.
LINDA D. MONEYHAM
CONNIE WHITE-WILLIAMS

A DISSERTATION

Submitted to the graduate faculty of The University of Alabama at Birmingham,
in partial fulfillment of the requirements for the degree of
Doctor of Philosophy

BIRMINGHAM, ALABAMA

2012

Copyright by
Patsy M. Spratling
2012

CARDIOVASCULAR DISEASE KNOWLEDGE AND RISK PERCEPTION AMONG
WOMEN WITH RECENT PREECLAMPSIA: INTERVENTIONAL EDUCATION IN
DISEASE MANAGEMENT AND PREVENTION

PATSY M. SPRATLING

SCHOOL OF NURSING

ABSTRACT

There is a growing body of evidence linking preeclampsia to future development of cardiovascular disease (CVD). Although CVD is well-known as the leading cause of death in women, a lack of evidence exists demonstrating that women with preeclampsia are routinely informed of their risks for future CVD. The specific aims of this study were to: (1) examine the levels of CVD knowledge and perception of CVD risk pre-CVD education; (2) explore relationships among age, race, parity, marital status, previous preeclampsia, income, education, CVD knowledge, and perception of CVD risk; and (3) examine the effect of a postpartum CVD educational intervention on personal perception of CVD risk in women with new-onset preeclampsia.

Using telephone-based interviews, baseline levels of CVD knowledge and CVD risk perception were examined among 64 women with recent, new-onset preeclampsia. Relationships among CVD knowledge, CVD risk perception, and several covariates (age, race, parity, marital status, previous preeclampsia, income, and education) were also examined. The intervention of CVD education was provided after baseline data were collected; CVD risk perception was reexamined post-CVD education.

At baseline, CVD knowledge was found to be a significant predictor of CVD risk perception, accounting for 8.4% of explained variance ($p = 0.011$). Although none of the covariates significantly influenced CVD risk perception, the factors of age and income

significantly influenced CVD knowledge ($R^2 = 0.226$; $p = .001$). After CVD education, levels of CVD risk perception were significantly higher than at baseline (paired $t = - 2.3$; $p = 0.003$).

Accurate perceptions of CVD risk have been associated with demonstrable behaviors suggestive of risk reduction. As an intervention, CVD education, provided by telephone, served as a practical and effective approach to reaching women with recent, new-onset preeclampsia. Results of the CVD risk perception scale from baseline to post-CVD education demonstrated that CVD education significantly increased perceptions of personal CVD risk among women with recent, new-onset preeclampsia.

Keywords: cardiovascular disease in women; preeclampsia

DEDICATION

I dedicate this dissertation to my husband, Otis. Otis, you were a constant strength throughout. You unselfishly made sacrifices so that I could complete this journey. Thank you. I would also like to dedicate this dissertation to my mother, Mrs. Katie Miller and in memory of my father, Mr. Levell Miller. You have been a constant source of support throughout my entire life; I will always be indebted to you.

ACKNOWLEDGEMENTS

I would like to acknowledge my dissertation committee for all of your time, effort, and support throughout this entire process. Each time that I reached out to one of you for guidance, you were always there.

Dr. Erica Pryor, you have been with me through my entire program of study. You were my advisor, statistics professor, and dissertation committee chair; you encouraged my efforts throughout. Your kind words and reassurance gave me the boost that I needed to stay motivated.

Dr. Linda Moneyham, you have supported my efforts from day one in the doctoral program. I am truly grateful for all of the support and encouragement that you provided during the course of my studies. You stimulated my thoughts beyond what I ever imagined.

Dr. James N. Martin, Jr., I am truly thankful to you for the giving me the opportunity to collaborate with the University of Mississippi Medical Center for my dissertation study. With your help, I was able to accomplish an important goal: promoting cardiovascular disease knowledge and awareness in women. The nurses in your clinic, Belinda Ellis and Laura Bufkin, were awesome! I am grateful to both of them for assisting me in recruiting my study population.

Dr. Connie White-Williams and Dr. Ashley Hodges, you have been an inspiration. Your constructive feedback was invaluable. I thank both of you and recognize the time and effort that you devoted to my project.

I am grateful to UAB School of Nursing for being awarded the Graduate Assistance in Areas of National Need (GAANN) Fellowship. Without this financial support, completing this journey would not have been possible.

TABLE OF CONTENTS

	<i>Page</i>
ABSTRACT.....	iii
DEDICATION	v
ACKNOWLEDGEMENTS	vi
LIST OF TABLES	xi
LIST OF FIGURES	xii
LIST OF ABBREVIATIONS	xiii
CHAPTER	
1 INTRODUCTION	1
Definition of Terms.....	2
Background of the Problem	5
Statement of the Problem	7
Significance of the Problem	8
Study Purpose	12
Study Aims.....	13
Research Questions	14
Theoretical Framework	14
Personal Factors, Behavior, and Environmental Influences	15
Self- Regulation and Motivation	17
Knowledge and Risk Perception.....	19
Assumptions	22
2 LITERATURE REVIEW	23
Relationships between Preeclampsia and CVD	24
Preeclampsia and Future Risk of CVD.....	24
Risk Factors for Preeclampsia	31
Modifiable Risk Factors for both Preeclampsia and CVD	39

2	LITERATURE REVIEW (continued)	
	Pathophysiologic Effects of Preeclampsia.....	44
	Summary of Relationships between Preeclampsia and CVD.....	52
	Postpartum Care after Preeclampsia.....	54
	Summary of Postpartum Care after Preeclampsia.....	60
	Health Promotion, CVD Education, and Risk Perception in Women.....	61
	Adoption of Postpartum Lifestyle Changes.....	68
	Summary of Health Promotion, CVD Education, and Risk Perception in Women.....	71
3	METHODOLOGY	74
	Study Design	74
	Study Sample	75
	Sample Size and Power Analysis	75
	Study Procedures	77
	Ethical Considerations	77
	Informed Consent.....	77
	Recruitment.....	78
	Data Collection	80
	Study Measures	83
	Demographics.....	83
	CVD Knowledge.....	84
	CVD Risk Perception.....	85
	Reliability of Research Instruments.....	86
	Data Management	88
	Data Analysis	88
	Missing Data.....	91
4	STUDY RESULTS	92
	Data Screening.....	92
	Demographic Characteristics of Sample.....	93
	Description of Main Study Outcome Variables.....	95
	Specific Aims/Research Questions.....	96
	Aim 1	96
	Research Question 1	96
	Research Question 2	98
	Research Question 3	99
	Research Question 4	100
	Aim 2	100
	Research Question 5	100
	Aim 3	103
	Research Question 6	103

5 DISCUSSION AND CONCLUSIONS	105
Results.....	105
Demographic Characteristics of the Study Sample	105
CVD Knowledge and CVD Risk Perception.....	106
Effect of CVD Education Intervention	111
Discussion and Implications	111
Recommendations.....	115
Nursing Education	115
Clinical Practice.....	115
Research.....	116
Limitations	118
Summary.....	120
REFERENCES	121
APPENDICES	132
A IRB APPROVAL LETTER	132
B LETTER OF SUPPORT	134
C STUDY RECRUITMENT MATERIALS	137
D PERMISSION TO USE RESEARCH INSTRUMENTS	143
E TELEPHONE INTERVIEW SCRIPT	146
F STUDY QUESTIONNAIRES	149
G TELEPHONE EDUCATION INTERVENTION SCRIPT	153
H GUIDELINES FOR CVD PREVENTION IN WOMEN MAIL-OUT BROCHURE POST STUDY	155

LIST OF TABLES

<i>Table</i>	<i>Page</i>
1 Manifestations Shared between Preeclampsia and CVD.....	53
2 Implications and Recommendations for CVD Risk Reduction in Women with Previous Preeclampsia	65
3 Demographic Questionnaire	83
4 Reliability of Study Instruments	87
5 Demographic Characteristics of the Study Sample	94
6 Descriptive Statistics of Major Study Outcome Variables	96
7 Baseline Composite Scores of CVD Knowledge.....	97
8 CVD Knowledge Levels at Baseline	97
9 CVD Knowledge in Women with and Without Preeclampsia.....	98
10 Baseline Composite Scores of CVD Risk Perception.....	99
11 CVD Risk Perception in Women with and Without Previous Preeclampsia	100
12 Univariate Analysis of CVD Risk Perception Regressed on Study Covariates.....	102
13 Univariate Analysis of CVD Knowledge Regressed on Study Covariates.....	102
14 CVD Knowledge Regressed on Age and Education.....	103
15 Paired Differences of CVD Risk Perception at Baseline and Post-CVD Education.....	104
16 Item Responses for the CHDK Tool for Women.....	107

LIST OF FIGURES

<i>Figure</i>	<i>Page</i>
1 CVD Education Conceptual Model	22
2 Recruitment Flow Chart.....	80

LIST OF ABBREVIATIONS

ACOG	American College of Obstetricians and Gynecologists
BMI	Body Mass Index
CAD	Coronary Artery Disease
CDC	Centers for Disease Control and Prevention
CHD	Coronary Heart Disease
CHDK	Coronary Heart Disease Knowledge Tool for Women
CI	Confidence Interval
CVD	Cardiovascular Disease
FMD	Flow Mediated Dilation
HDL	High Density Lipoprotein
HELLP	Hemolysis, Elevated Liver Enzymes, and Low Platelet Counts
HOMA _{IR}	Homeostasis Model Assessment of Insulin Resistance
HR	Hazard Ratio
IOM	Institute of Medicine
IRB	Institutional Review Board
IRR	Incidence Rate Ratio
IUGR	Intrauterine Growth Restriction
LDL	Low Density Lipoprotein
OB/GYN	Obstetrician/Gynecologist

LIST OF ABBREVIATIONS (continued)

OR	Odds Ratio
PIGF	Placental Growth Factor
sFlt-1	Soluble fms-like tyrosine kinase
UAB	University of Alabama at Birmingham
UMMC	University of Mississippi Medical Center
USDA	United States Department of Agriculture
USDHHS	United States Department of Health and Human Services
VEGF	Vascular Endothelial Growth Factor
WHO	World Health Organization

CHAPTER 1

INTRODUCTION

There is a growing body of evidence that links preeclampsia to future development of cardiovascular disease (CVD). In comparison to women with uncomplicated pregnancies, women who have experienced preeclampsia during pregnancy are considered as having an increased risk for developing CVD later in life (Garovic & Hayman, 2007; McDonald, Malinowski, Zhou, Yusuf, & Devereaux, 2008). Although preeclampsia is believed to be an indicator for increasing risks of CVD later in life, recent studies have reported that follow-up care for these women is inadequate (Nijdam et al., 2009).

CVD is the leading cause of death, in both men and women, worldwide (Centers for Disease Control [CDC], 2010; Institute of Medicine [IOM], 2010). In 2006, of all American deaths related to CVD, 151,000 of these deaths were among people younger than 65 years of age (CDC, 2010). Consequently, heart disease and stroke, respectively, are the first and third leading cause of death and disability in the United States (US) and are responsible for nearly 3 million people being disabled (CDC, 2010). Although rates of death and disability due to CVD are staggering, these are not the only burdens that CVD imposes. An estimated \$503 billion were spent in 2010 in healthcare expenditures and lost productivity due to CVD-related disability and death (CDC, 2010).

In addition to the economic impact, CVD also carries a tremendous social burden. Although the social impact of CVD has not been quantified to the degree of the economic impact, there are many circumstances that must be considered relative to the social impact of CVD. Socially, the burden of CVD may include loss of employment for both the affected individual and caregiver, relocation due to loss of employment, the need to be closer to healthcare centers, or school-aged children dropping out of the education system to assume the role of caregiver (Gazlano, 2007).

Definition of Terms

The hypertensive disorders of pregnancy are inclusive of preeclampsia, eclampsia, chronic hypertension, pre-eclampsia superimposed on chronic hypertension, and gestational hypertension, (Garovic & Hayman, 2007). However in the present study, only women who experienced recent new-onset episodes of pure preeclampsia were examined. Conceptual and operational definitions of the major study variables follow.

Cardiovascular Disease (CVD)

CVD refers to diseases caused by disorders of the heart and blood vessels, and includes coronary heart disease, cerebrovascular disease, hypertension, peripheral artery disease, rheumatic heart disease, congenital heart disease and heart failure (WHO, 2009). The major causes of cardiovascular disease are tobacco use, physical inactivity, an unhealthy diet and harmful use of alcohol. Because coronary artery disease (CAD) and

coronary heart disease (CHD) are included as conditions of CVD, these terms, as used in the study, also refer to CVD.

CVD Risk Factors

CVD risk factors are classified as either modifiable or non-modifiable. Non-modifiable risk factors include: previous stroke or myocardial infarction, race, family history, presentation of symptoms, and increasing age (Gholizadeh et al., 2010; Hart, 2005). Tobacco use, hypertension, obesity, diabetes, physical inactivity, and dyslipidemia are each considered as modifiable CVD risk factors; each modifiable condition is considered as self-imposed (Gholizadeh et al., 2010; Hart, 2005; WHO, 2009). CVD risk was operationalized by informing study participants of modifiable CVD risk factors and CVD risk reduction approaches. In addition, a brochure, *Guidelines for Reducing Cardiovascular Disease in Women*, was mailed to each participant after completing the study (see Appendix H).

CVD Knowledge

CVD knowledge refers to the understanding that women have regarding accurate recognition of cardiovascular disease risk factors and healthy lifestyle practices (Thanavaro, Thanavaro, & Delicath, 2008). The *Coronary Heart Disease Knowledge* (CHDK) tool for women (Thanavaro, et al., 2008), was used, with permission, to measure

CVD knowledge in the proposed study. CVD knowledge was operationalized as the score on the CHDK scale (see Appendix F).

CVD Risk Perception

Risk perception, described by Healthy People 2020 as self-assessed health status, refers to the way in which an individual describes his or her health (United States Department of Health and Human Services [USDHHS], 2010). Because individual self-reports of health status allow for wide-ranging comparisons to be made among various populations and health conditions, self-assessed health status is recognized as a useful indicator of health (USDHHS, 2010). CVD risk perception was measured in the study using the *CVD Risk Perception* instrument (Schwarzer & Renner, 2000). CVD risk perception was operationalized as the score on the CVD Risk Perception instrument (see Appendix F).

Hypertensive Pregnancy Disorders

Women who recently experienced pure preeclampsia were the population examined in the present study. Women with known histories of hypertension and other hypertensive pregnancy-related disorders were not included. A description of preeclampsia and its severe forms of eclampsia, severe preeclampsia and HELLP syndrome follows.

Preeclampsia. Preeclampsia is described as a pregnancy-specific syndrome characterized by hypertension (blood pressure $\geq 140/90$ mm Hg) and proteinuria (protein ≥ 0.3 g present in urine) that occurs after 20 weeks gestation in women who were previously normotensive (American College of Obstetricians & Gynecologists [ACOG], 2002).

Severe Preeclampsia. Severe preeclampsia is considered an advanced stage of preeclampsia and is characterized by blood pressure $\geq 160/110$ mm Hg and proteinuria ≥ 5 g on two occasions at least six hours apart (ACOG, 2002).

Eclampsia. Eclampsia refers to new-onset grand mal seizures that occur in women with preeclampsia and who have no other known causes for seizures (ACOG, 2002).

HELLP Syndrome. HELLP syndrome refers to a severe form of preeclampsia characterized by hemolysis, elevated liver enzymes, and low platelet counts. Among the various forms of preeclampsia, HELLP syndrome is associated with some of the highest rates of significant maternal and perinatal morbidity and mortality (Martin, 2012).

Background of the Problem

Historically, cardiovascular epidemiologic research has focused on men. Early studies of patients with heart disease were performed almost exclusively with men.

However this pattern changed by the late 1980's when a number of studies on heart disease were conducted exclusively on female samples (Hart, 2005). In spite of the change in pattern for heart disease studies to include women, many studies that included both male and female patients still did not explicitly focus on gender differences (Emslie, 2005).

As early as the beginning of the 19th century, studies revealed data to support relationships between preeclampsia and the subsequent risk of developing hypertension; however those studies had substantial limitations, including: small sample sizes, follow-up durations that were inadequate, insufficient documentation, inconsistent methods of data collection, and changes in the definition of, and diagnostic criteria for, pregnancy-related hypertensive disorders over time (Garovic & Hayman, 2007). In addition, retrospective approaches, used in many studies, resulted in difficulty establishing a diagnosis. Consequently, the specific effects of various pregnancy-related hypertensive disorders were ignored. Studies, conducted between the 1950's and the early 1970's, further supported relationships between preeclampsia and higher blood pressure later in life and results of these studies indicated that children born to preeclamptic mothers were more likely to be born with low birth weight. Today, low birth weight is also recognized as being associated with increased risks for CVD later in life (Garovic & Hayman). Preeclampsia, low birth weight, and preterm delivery, especially in combination, are considered a first manifestation of atherosclerosis, which is considered a primary CVD risk (van Pampas, 2005).

Statement of the Problem

Research has demonstrated that women who have been affected by preeclampsia are at greater risk for developing CVD later in life than that of women who have not been affected by preeclampsia during pregnancy (Garovic & Hayman, 2007; Nijdam et al., 2009). Currently, CVD is the leading cause of death in women (Christian, Rosamond, White, & Mosca, 2007) regardless of the presence or absence of preeclampsia during pregnancy. Women diagnosed with coronary heart disease (CHD) have a greater morbidity and mortality rate than men; for example, 38% of women who suffer from a heart attack die within one year as compared to only 22% of men (Hart, 2005). In men, total deaths from CVD declined considerably during years 1979-2001, however the number of CVD-related deaths in women has remained constant (Christian et al., 2007).

For women who experience hypertensive disorders during pregnancy, a lack of evidence exists to support promotion of CVD risk-reduction specifically targeted to this group (Garovic & Hayman, 2007; Nijdam et al., 2009). Although hypertensive disorders of pregnancy, mainly preeclampsia, are believed to be indicators for increasing risks of CVD later in life, recent studies have reported that follow-up care for these women is inadequate (Nijdam et al.). In a recent study that compared cardiovascular follow-up between postpartum normotensive women and postpartum women who had preeclampsia in their most recent pregnancy, 42.9% of previously preeclamptic women did not have documented evidence of blood pressures being checked in the postpartum period (Nijdam

et al.). In addition, Nijdam et al. noted that few women were aware of the long-term risks of CVD subsequent to preeclampsia and other hypertensive disorders during pregnancy.

Significance of the Problem

CVD risk factors, such as: diabetes, smoking, obesity, family history, and increasing age, are also associated with preeclampsia (Silva et al., 2008) and although some CVD risk factors are not modifiable, others are. Because modifiable risk factors associated with CVD are inclusive of self-imposed conditions, CVD is believed to be mainly a preventable condition (CDC, 2010; IOM, 2010). The metabolic syndrome (a variety of disorders associated with type-2 diabetes, obesity, hypertension, dyslipidemia, and atherosclerosis) is said to be present in both preeclampsia and CVD (Harskamp & Zeeman, 2007); the metabolic syndrome is also inclusive of self-imposed conditions.

Some researchers (Alladin & Harrison, 2012; Magnussen et al. 2007; Romundstad, Magnussen, Smith, & Vatten 2010; Wen et al., 2012) suggest that preeclampsia development during pregnancy results from factors existing pre-pregnancy. Pre-pregnancy obesity (Romundstad et al., 2010); genetic thrombophilia (Irgens, Reisaeter, Irgens & Lie, 2001); unrecognized hypertension, and diabetes (Alladin & Harrison, 2012) are noted as preexisting maternal factors that possibly contribute to preeclampsia development. However, according to Ness and Hubel (2005), lifestyle interventions, aimed at improving diet and promoting physical activity, have demonstrated effectiveness in preventing the onset of type-2 diabetes mellitus and when

initiated early, can delay the onset of coronary artery disease (CAD). Thus, CAD prevention approaches, among women who experience preeclampsia, should be offered rigorously and early (Ness & Hubel, 2005).

Hypertensive disorders are said to affect 10% of all pregnancies worldwide, and preeclampsia is believed to be present in about one-half of pregnancies affected by hypertensive disorders (Craici, Wagner, & Garovic, 2008). According to Bellamy, Casas, Hingorani, and Williams (2008), globally, pregnancy-related hypertensive disorders account for approximately 12% of maternal mortality during pregnancy and in the puerperium. Accordingly, pregnancy-related hypertensive disorders are a leading cause of both maternal and fetal mortality (Garovic & Hayman, 2007). In women who are of reproductive age, hypertension is the most common diagnosis (Yoder, Thornburg, & Bisognano, 2009). The hypertensive disorders of pregnancy are inclusive of preeclampsia, eclampsia, chronic hypertension, pre-eclampsia superimposed on chronic hypertension, and gestational hypertension, (Garovic & Hayman). However, preeclampsia is considered to be the hypertensive disorder of pregnancy that is most problematic long-term because it contributes most to CVD later in life (Garovic & Hayman; Nijdam et al., 2009). In addition, a syndrome of hemolysis, elevated liver enzymes, and low platelet counts (HELLP syndrome) is associated with conditions of severe preeclampsia; this condition increases risks for both maternal and fetal outcomes (ACOG, 2002).

Some researchers have proposed that even during normal pregnancy, there is an associated accumulation of fatty deposits and plaque in the arteries (Mankuta et al., 2010; Nijdam et al., 2009). According to Mankuta et al., during the second trimester of pregnancy, total cholesterol, low density-lipoprotein (LDL) or so-called *bad cholesterol*, and triglycerides undergo a marked increase, but return to pre-pregnancy states during the second and third years postpartum. In contrast, high density-lipoprotein (HDL), or so-called *good cholesterol*, undergoes a steep elevation during the second trimester, but the level of HDL cholesterol significantly decreases within the third trimester. In addition, with each subsequent pregnancy, HDL cholesterol is lower than previous levels (Mankuta et al.). Thus, dyslipidemia is viewed as a significant factor linking parity to CVD prevalence (Catov et al., 2008). These data accentuate how pregnancy alone, even though uncomplicated, increases CVD risks for parous women later in life.

Preeclampsia and CVD share the risk factors of endothelial dysfunction, endothelial damage, hypertension, and diabetes (Bauer & Cleary, 2009). Physiologic changes associated with preeclampsia are inclusive of increased inflammatory markers, dyslipidemia, insulin resistance, endothelial dysfunction, and oxidative stress; each of these physiologic changes shares a relationship with CVD later in life (Bauer & Cleary; Garovic & Hayman, 2007). While most hypertensive disorders of pregnancy abate if pregnancy terminates, recent studies have demonstrated that even with normalization of blood pressure, women who have experienced hypertensive disorders during pregnancy continue to be affected by the adverse physiologic changes consequential to pregnancy.

The amount of time that it takes for preeclampsia to resolve is uncertain (Berks, Stegers, Molas, & Visser, 2009; Smith et al., 2009). Adverse physiologic changes resulting from pregnancy are believed to increase the overall risk of developing future CVD (Berks et al., 2009; Garovic & Hayman).

Inconsistent findings have been reported among researchers regarding the interrelatedness of pregnancy-related hypertensive disorders and CVD. Valdés et al. (2009) reported that CAD was not significantly associated with hypertensive pregnancies; however Smith et al. (2009) reported that risks for cardiovascular events increased by two to three fold for women who experience preeclampsia as compared to women who experience normotensive pregnancies. Accordingly, Catov et al. (2008) and Garovic and Hayman (2007) acknowledged the associations of hypertensive pregnancies and future CVD development. However, Diehl (2008) emphasized the systemic nature of preeclampsia and indicated that because of systemic involvement with hypertensive pregnancies, other conditions may be responsible for CVD development. According to van Pampus (2005), women with histories of preeclampsia should be considered as having increased risk for developing severe preeclampsia in subsequent pregnancies and at increased risk for chronic hypertension.

There are unmistakable gaps in the literature relative to the relationships of preeclampsia during pregnancy and future development of CVD. However, understanding relationships between preeclampsia and CVD are necessary so that women affected by preeclampsia and other pregnancy-related hypertensive disorders may be

identified earlier and receive appropriate modalities to prevent and reduce CVD development.

Accurate perceptions of CVD risk have been associated with demonstrable behaviors that are suggestive of risk reduction. Thus accurate perceptions of CVD risks are necessary for health behavior to be aligned with measures to treat and prevent CVD (Christian, Mochari, & Mosca, 2005). Behavior change has been described as a difficult and multifaceted processes nonetheless accurate perceptions of CVD risk are necessary for behavior change to occur (Gholizadeh, Davidson, Salamonson, & Worrall-Carter, 2010; USDHHS, 2010). According to Bandura (2004), in order for behavior change to take place, knowledge is essential; change is unlikely if a lack of knowledge exists regarding how lifestyle habits affects health.

When social and environmental factors are taken into consideration, seemingly simple and straightforward actions that are believed to prevent and treat CVD become very complicated (IOM, 2010). The interrelationships shared between health status, individual behavior, and social dynamics are increasingly recognized as factors that influence health and health behavior (USDHHS, 2010).

Study Purpose

The overarching purpose of this study was to promote CVD knowledge and awareness among women with recent new-onset pure preeclampsia, using CVD education as an intervention, so that this population of women may develop accurate

perceptions of their personal CVD risk. Because accurate perceptions of CVD risk have been associated with demonstrable behaviors that are suggestive of risk reduction, CVD education is necessary to foster CVD knowledge and promote awareness of CVD risk. Intervention research is useful for examining effectiveness of nursing interventions for achieving desired outcomes (Burns & Grove, 2009). In this study, CVD education was provided as an intervention. Knowledge of CVD risk, along with accurate perceptions of CVD risks, provides the necessary foundation for health behavior to be aligned with actions aimed at reducing and/or preventing CVD (Christian, et al., 2005).

Study Aims

The aims of the project were to study women with recent, new-onset, pure preeclampsia to: (1) examine levels of CVD knowledge and perceptions of CVD risk pre-CVD education; (2) explore relationships among age, race, parity, marital status, previous preeclampsia, income, education, CVD knowledge, and perception of CVD risk; and (3) examine the effect of a CVD educational intervention on levels of personal perception of CVD risk post-education. Women with known histories of hypertension or other pregnancy-related hypertensive disorders were not included in the present study.

Research Questions

Research questions guiding the study were:

1. Among women with recent, new-onset preeclampsia, what is the baseline level of CVD knowledge?
2. Among women with recent, new-onset preeclampsia, what is the awareness of CVD as being the leading cause of death and healthcare problems in women?
3. Among women with recent, new-onset preeclampsia, what is the baseline level of CVD risk perception?
4. Among women with recent, new-onset preeclampsia, what is the relationship, at baseline, between CVD knowledge and CVD risk perception?
5. Among women with recent, new-onset preeclampsia, what is the relationship, at baseline, among levels of CVD knowledge and perceptions of CVD risk and the variables of age, race, parity, marital status, previous preeclampsia, income, and education?
6. Among women with recent, new-onset preeclampsia, compared with baseline perceptions of CVD risk, is there a significant change in perception of CVD risk after receiving CVD education?

Theoretical Framework

In the present study, levels of CVD knowledge and risk perception were examined at baseline among women with recent new-onset pure preeclampsia. In

addition, CVD education, as intervention, served to determine the influence of education on perceptions of CVD risk. Inadequate understanding of how conditions such as hypertension, diabetes, obesity, and lifestyle habits such as diet, smoking and being physically inactive increase risks for CVD development, negatively influences perceptions of CVD risk (King & Arthur, 2003; Homko et al., 2008). Social cognitive theory explains approaches to developing interventions that may positively influence health outcomes through strategies that modify expectations and motivation. Several fundamental constructs of social cognitive theory served as the framework to guide this study.

The organizing principle of social cognitive theory is that of reciprocal determination (Redding, 2000) where human behavior is explained in a three-dimensional model of causal determinism (Bandura, 1989). Behavior, cognition and other personal factors, and environmental influences are labeled as determinants that act together, in a bi-directional manner, where each determinant impacts the other (Bandura). Although this triad of determinants work in concert, social cognitive theory emphasizes that their influences are neither of equal strength; nor do they all occur at the same time (Bandura).

Personal Factors, Behavior, Environmental Influences

According to social cognitive theory, relationships between personal factors and behavior are demonstrated by the way in which people behave (Bandura, 1989).

Behavior is said to be molded by people's expectations, beliefs, self-perceptions, goals, and intentions. Therefore, what people think, believe, and feel impacts how they act (Bandura). In the present study, women with recent preeclampsia, including its severe forms of eclampsia and HELLP syndrome, were examined; these health-related conditions represent personal factors in the present study. In addition, parity, the presence of preeclampsia in pregnancies other than the most recent pregnancy, and multiple births during pregnancy were also viewed as personal factors in the present study.

Relationships between personal factors and environmental influences are said to develop through human expectations, emotional shifts, and cognitive abilities (Bandura, 1989). As well, relationships between personal factors and environmental influences are modified by social influences. Social influences act to link information and stimulate emotional reactions through modeling, teaching, and social persuasion. In addition, physical characteristics, such as: age, physical size, race, and sex tend to induce reactions from their social environment. Thus, people activate different social reactions depending on their social roles and/or statuses. For example, those with a reputation of being belligerent will induce a different reaction from peers than those believed to be unassertive (Bandura). In the present study, relationships among age, income, marital status, and race were examined as personal and environmental factors.

The behavior/environment relationship is demonstrated by the manner in which behavior adjusts to environmental conditions and is, in turn, transformed by the actual circumstances that it creates. Thus, people are both products and producers of their

environment (Bandura, 1989). Through their actions, people both select and create their environment. For example aggressive people tend to create hostile environments; whereas those who behave in a more pleasant manner tend to create a pleasant environment (Bandura). Behavior was not overtly examined in the present study.

Self-Regulation and Motivation

According to social cognitive theory, dominant sociocultural influences, as well as unexpected life events, explain the directions that personal lives take. Consequently, having knowledge of factors (both planned and unexpected) that may change the direction of life paths, serves as a guide to support values. At the individual level, developing competencies, which foster self-confidence in attaining goals and self-regulatory capabilities for influencing one's own motivation and actions, are acquired through knowledge. In the present study, CVD education, as an intervention, equipped participants with CVD knowledge and support for personal goal-setting. In social cognitive theory, people are neither motivated by inner forces nor are they inevitably shaped and controlled by the environment. Rather, people contribute to their own motivation, behavior, and development within a system of mutually interacting influences (Bandura, 1989).

Although personal efficacy was not examined as a study variable in the present study, in social cognitive theory, personal efficacy is considered as the foundation of human motivation and action. The principle of personal efficacy holds that if people do

not believe that they are able to achieve favorable results through their actions, then there will be a lack of motivation to take action (Bandura, 2004); personal efficacy is therefore subjective in nature. Social cognitive theory embraces the belief that personal motivation is intensified when people receive support that facilitates the realization that habit or lifestyle changes are in their best interest and are inclusive of their personal goals. Such support, in the present study, was generated through CVD education. In addition, study participants received printed CVD guidelines to foster CVD knowledge and awareness. Personal goals, embedded in a value system, promote goal-setting for health habits (Bandura). However, massive conflicting influences may be present; thus long-range goals to control current behavior become challenging. For that reason short-term goals, that are realistic, facilitate individuals' abilities to be successful with behavior change by inducing support and regulating current actions (Bandura). In the present study, CVD knowledge was assessed at baseline and CVD education was provided as an intervention to promote CVD knowledge and to foster decision-making processes.

Perceived facilitators and obstacles are also influences of health habits; some constraints to performing healthful behavior are personal in nature. Personal barriers are said to be counterbalanced through personal self-efficacy. Bandura (2004) asserts that regulating health behavior is a joint venture and acknowledges that some of the obstacles to healthful living stem from health systems rather than from personal or situational factors. In addition to verbal CVD education, each study participant received printed CVD risk reduction guidelines to foster motivation and self-regulation. In the social

cognitive perspective, people actively contribute to their own motivation. In this light, standards which are somewhat easy are insufficient for arousing much interest or effort; whereas moderately difficult standards tend to produce high effort and create fulfillment through achievement of goals. However, when standards are set far beyond a person's reach, discouragement and/or demotivation occurs and a sense of inefficacy or feelings of inadequacy in one's ability to produce the desired goal develops (Bandura, 1989).

Knowledge and Risk Perception

CVD knowledge and CVD risk perception were the central variables of interest in the present study. CVD knowledge was examined as a baseline variable and CVD education served as an intervention in the present study. Knowledge of health risks and benefits of various health behaviors, an individual's perceived-self-efficacy in his/her ability to control over his/her own health routines, and outcome expectancies about the costs and benefits for various health behaviors are described by social cognitive theory as a collective set of contributing factors that must be considered with health promotion and disease prevention efforts (Bandura, 2004). According to social cognitive theory, knowledge is the prerequisite for change because change is implausible if a lack of knowledge exists regarding how lifestyle habits affects health. However, other factors, such as perceived-self-efficacy, are also necessary for most people to overcome barriers that prevent them from embracing, implementing, and sustaining new lifestyle habits (Bandura).

Because knowledge and thinking abilities provide the tools necessary for cognitive problem-solving, people first test possible solutions to problems in thought. Before acting, possible solutions are then discarded or retained based on consequences. However, faulty judgments may be made when reasoning is based on incomplete or erroneous information (Bandura, 1989). This process, in social cognitive theory, is referred to as outcome expectancies. In short, outcome expectancies are subjective and refer to what an individual expects personal actions to produce (Bandura, 2004). Outcome expectations may be: (a) physical outcomes, including both pleasant and unpleasant effects of behavior, such as related material losses and benefits; (b) social outcomes, such as the approval or disapproval that individuals receive through social relations; and (c) the personal reactions that are experienced relative to individual health behaviors or health statuses. Thus, people embrace personal standards that promote personal satisfaction and self-confidence, but relinquish those behaviors that do not (Bandura, 2004).

According to Bandura (1994), factual information, combined with an extension of practical risk reduction approaches, creates good results; people learn and develop effective ways of behaving under realistic situations. According to Schwarzer (2011), risk perception or perceiving a health threat, represents the most obvious motivation for overcoming a risk behavior. General perceptions of risk (e.g., high blood pressure may lead to heart disease) and personal perceptions of risk (e.g., I am at risk for heart disease because I have high blood pressure), tend to differ vastly. In this light, although

individuals may be well-informed of the general aspects that a particular health risk holds, they may not feel personally at risk. In the present study, risk perception was examined, both at baseline and after the CVD education intervention, by asking study participants to compare themselves to others of their same age. Schwarzer (2011) points out that when an individual compares him/herself to similar others, individual risk perception becomes biased; people typically view themselves as being less likely than others to experience health problems in the future. Thus, adopting health behaviors should not be viewed simply as a natural response to a health threat because risk information, by itself, will not change risky behavior; risk information alone does not provide meaningful information on how to manage behavior (Schwarzer, 2011). Initial risk perception, however is beneficial in that it helps people become motivated to change. Accordingly, Bandura (2004) points out that in order for health communication to assist people with adopting health-promoting behaviors, it must be structured in a way that it provides the self-management knowledge, skills, and confidence necessary to take control of health behaviors. In order to improve human health, identifying cognitive predictive factors, along with effective guides on how to manage them, must operate in concert (Bandura, 2005). A model depicting variables used in the study and their interconnectedness follows (see Figure 1).

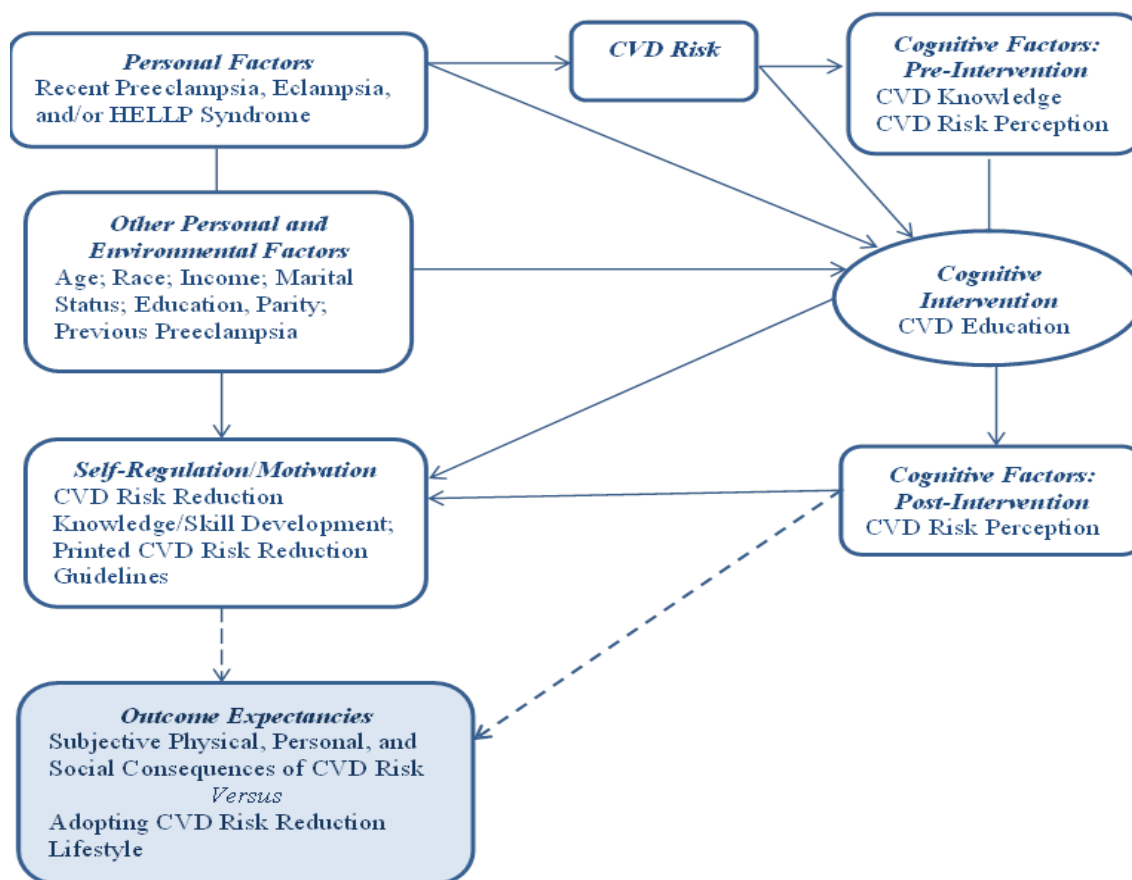


Figure 1. CVD Education Conceptual Model

Assumptions

Assumptions of the study were that:

1. The study variables of CVD knowledge and CVD risk perception, could be measured with available instruments; and
2. Women participating in this study responded accurately and truthfully to the instrument questions.

CHAPTER 2

LITERATURE REVIEW

Because CVD is increasingly recognized as an impending risk for women who experience preeclampsia during pregnancy, this study examined women with recent, new-onset, pure preeclampsia to explore perceptions of personal CVD risk, both pre and post the CVD educational intervention. Because levels of CVD knowledge have been reported as lacking among women in general (Mosca, Mochari-Greenberger, Dolor, Newby, & Robb, 2010), CVD knowledge was also examined. In addition, age, race, parity, history of previous preeclampsia, income, marital status, and education were compared with levels of CVD knowledge to determine if relationships existed. Subsequent to CVD education, CVD risk perception was reassessed to determine if CVD education impacted levels of CVD risk perception.

Although CVD is well documented as the leading cause of death in women, (Mosca et al., 2010), evidence supporting that women who have experienced preeclampsia have been informed of their risks for developing CVD is lacking. According to Mosca et al., CVD awareness is positively correlated with actions to reduce CVD risk. Consistently, Bandura (2004) emphasizes knowledge as the precondition necessary for positive lifestyle habits, aimed at reducing CVD risk, to occur. Likewise, accurate perceptions of CVD risk have been associated with demonstrable behaviors

suggestive of risk reduction (Christian et al., 2005). Therefore, knowledge and accurate perceptions of CVD risks are necessary in order for lifestyle habits to be aligned with measures aimed at reducing/preventing CVD risk.

The purpose of this literature review was to examine the current state of the science relative to associations of preeclampsia and subsequent CVD and to determine current educational needs of women who have experienced preeclampsia during pregnancy. This literature review is divided into three primary areas: (1) relationships between preeclampsia during pregnancy and subsequent CVD; (2) postpartum status and care after preeclampsia; and (3) health promotion, CVD education, and CVD risk perception in women.

Relationships between Preeclampsia and CVD

Preeclampsia and Future Risk of CVD

Several groups of researchers (Bellamy, Casas, Hingorani, & Williams 2007; also see Evans et al. 2011; McDonald, Malinowski, Zhou, Yusuf, & Devereaux, 2008; Ray, Vermeulen, Schull, & Redelmeier, 2005; Smith et al., 2009) have reported that women who experience preeclampsia during pregnancy are at increased risk for developing future CVD. Further, the evidence supports a direct relationship between the severity of the preeclampsia and subsequent risk of CVD. In addition, there is evidence that CVD in this population develops earlier as compared to those who have not experienced preeclampsia.

Bellamy et al. (2007) conducted a systematic review and meta-analysis to measure future risks for CVD, cancer, and all-cause mortality among women with histories of preeclampsia and eclampsia. Study results demonstrated that at a weighted mean follow-up time of 14.1 years, relative risk (RR) values for chronic hypertension in women with preeclampsia was greater than threefold than that of women who did not have preeclampsia [RR=3.70; 95% confidence interval (CI): 2.70 -5.05]. As a risk factor for ischemic heart disease later in life, preeclamptic women demonstrated more than twice the risk of ischemic heart disease as compared to women without preeclampsia (RR= 2.16; 95% CI: 1.86-2.52). Women diagnosed with preeclampsia before 37 weeks gestation were at higher risk for stroke (RR=5.08; 95% CI: 1.2.09-12.35) in comparison to women who did not have preeclampsia. Conversely, women diagnosed with preeclampsia after 37 weeks gestation did not have a demonstrated increase in risk (RR= 0.98; 95% CI: 0.50-1.92). The mean follow-up for stroke was 10.4 years. In relation to all-cause mortality, women with preeclampsia had an increased risk (RR=1.49; 95% CI: 1.05-2.14) at an average weighted follow-up of 14.5 years. The risk for death from any cause was even greater in women 37 weeks or less gestation with preeclampsia (RR=2.71; 95% CI: 1.99-3.68). Bellamy et al. determined that a history of previous preeclampsia increases the risk for future hypertension, ischemic heart disease, stroke, venous thromboembolism, and death from any cause. Although causal mechanisms that link preeclampsia and CVD development remain obscure, a history of preeclampsia should serve as a cue to evaluate women's risk for CVD (Bellamy et al.).

McDonald et al. (2008) conducted a systematic review and meta-analysis of the literature to determine if women, beyond six weeks postpartum and who experienced preeclampsia/eclampsia during pregnancy ($n = 118,990$), were at increased risk for developing cardiac disease, cerebrovascular disease, peripheral artery disease, or cardiovascular mortality when compared to women without histories of preeclampsia/eclampsia ($n = 2,259,576$). McDonald et al. determined that women who experience preeclampsia have almost double the risk of future cardiac disease as compared with women who experience uncomplicated pregnancies; even when controlling for confounding factors. In addition, a *dose response* relationship among women with severe preeclampsia was said to exist; women with severe preeclampsia were found to be at higher risk for CVD. Because 11 of 14 studies reported cardiac events in women less than 56 years of age, McDonald et al. established that women who experience preeclampsia are at risk for early-onset cardiac disease. Conditions of cerebrovascular and peripheral arterial diseases, as well as cardiovascular mortality, were determined to be associated with higher long-term risks in women who previously experienced preeclampsia and eclampsia (McDonald et al.).

Wikström, Haglund, Olovsson, and Lindeberg (2005) investigated whether the risk of developing future ischemic heart disease increases with severity and/or recurrence of hypertensive disease during pregnancy. In comparison to non-hypertensive women, adjusted incidence rate ratios (IRR) of hospitalizations due to dying from ischemic heart disease were much higher ($p < 0.02$) in women with severe preeclampsia (IRR=2.8; 95%

CI: 2.2-3.7) than in women with gestational hypertension (IRR=1.6; 95% CI: 1.3-2.0) and mild preeclampsia (IRR=1.9; 95% CI: 1.6-2.2) ($p < 0.36$). In comparison to women with two non-hypertensive pregnancies, significant differences were found when compared to women with hypertensive pregnancies. Adjusted IRRs for women with hypertensive pregnancies were: hypertensive first pregnancy (IRR=1.9; 95% CI: 1.5-2.4); hypertensive second pregnancy (IRR=2.4; 95% CI: 1.5-2.4); and both hypertensive pregnancies (IRR=2.8; 95% CI: 2.0-3.9). Wikström et al. (2005) determined that severe hypertensive disease during pregnancy (severe preeclampsia) has a stronger link with ischemic heart disease development than mild hypertensive diseases (gestational hypertension and mild preeclampsia). In addition, recurrent hypertensive disease during pregnancy demonstrated a stronger association with ischemic heart disease than pregnancies complicated by hypertensive disease only once. Because severe preeclampsia and episodes of recurrent hypertensive disorders during pregnancy were strongly linked to developing ischemic heart disease, Wikström et al. stated that only women with severe preeclampsia should be advised of this risk because providing this information to all women who experience hypertensive pregnancies could “create much unnecessary anxiety in a large number of women who in fact will never develop ischemic heart disease” (p. 1490).

In a Canadian population-based retrospective cohort study, maternal-placental syndromes (preeclampsia, and placental abruption/infarction) were studied to examine relationships between premature CVD, hypertensive disorders of pregnancy, and placental abruption/infarction in combination with poor fetal growth and intrauterine fetal

death (Ray et al., 2005). In this study, CVD was substantiated based on hospitalizations due to coronary, cerebrovascular and/or peripheral vascular disease. In addition, revascularization of either the carotid artery or lower extremity arteries were also used as defining criteria for CVD. To lessen the influence of pregnancy or delivery on immediate CVD risk, study outcome assessments began 90 days after hospital discharge for an obstetrical delivery. Hypertensive disorders of interest were preeclampsia ($n = 36,982$) and gestational hypertension ($n = 20,942$) and participants' ages ranged from 14 to 50 years. Time-to event analyses were used to develop hazard ratios of both composite and individual study outcomes among women with and without maternal placental syndromes. Ray et al. reported that risk for premature maternal cardiovascular disease after maternal placental syndrome in the index pregnancy was increased in comparison to women not affected by maternal placental syndrome. Adjusted hazard ratios (HR) with 95% confidence intervals for those affected were: cardiovascular disease (HR=2.0; 95% CI: 1.7 to 2.2); coronary artery disease (HR=2.0; 95% CI: 1.7 to 2.3); cerebrovascular disease (HR=1.9; 95% CI: 1.4 to 2.5); and peripheral artery disease (HR=3.0; 95% CI: 1.9 to 4.8). The preeclampsia/premature maternal CVD relationship demonstrated a HR=2.1; with 95% CI: 1.8 to 2.4 (Ray et al.). The researchers concluded that because the average age of onset for cardiovascular events was 38 years, women with maternal placental syndromes may benefit from early recognition of risk factors. In addition, Ray et al. stated that whether or not women who experience maternal placental syndromes will benefit from lifestyle modification is uncertain.

Similarly, Lykke, Langhoff-Ross, Triche, and Paidas (2010) reported severe preeclampsia as being a marker of early death from cardiovascular causes. Because pregnancy outcomes such as preeclampsia, preterm birth, small for gestational age offspring, placental abruption, and/or stillbirth frequently take place concurrently, Lykke et al. (2010) conducted a retrospective registry-based cohort study to determine rates of death from cardiovascular and non-cardiovascular causes. The HR for preeclampsia relative to death from all causes was not significant (HR=1.10; 95% CI: 0.97-1.24); but when combined with small for gestational age, the hazard ratio increased markedly (HR=2.02; 95% CI: 1.55-2.63). Specifically relative to death from cardiovascular causes, the HR for preeclampsia was even higher (HR=2.08; 95% CI: 1.63-2.64), and when preeclampsia and small for gestational age were combined, HRs for death from cardiovascular causes increased even further (HR=4.67; 95% CI: 2.89-7.55). Lykke et al. reported a mean age of 27 years at delivery and a mean age of 42 years at death.

According to Melchiorre, Sutherland, Liberati, and Thilaganathan (2011), preeclampsia is associated with persistent cardiovascular impairment postpartum. Because preeclampsia has been associated with significantly increased risk for developing heart failure, ischemic and hypertensive heart disease, and CVD-related mortality, Melchiorre et al. (2011) conducted a prospective longitudinal case-control study to examine the natural history and clinical significance of left ventricular abnormalities in women with previous preeclampsia. Acute preeclampsia, specifically, has been linked to asymptomatic left ventricular abnormal function and myocardial

injury. Data were collected over a three-year period of time: at baseline, at one-year, and at two-years after baseline. Study results demonstrated that at two-years postpartum, women who experienced preeclampsia had significantly higher incidence of asymptomatic left ventricular abnormalities and higher prevalence of hypertension as compared to controls. In women who experienced preterm or acute preeclampsia, these findings were even more pronounced (Melchiorre et al.). Consistent with these findings, Bauer and Cleary (2009) reported that women who experience early-onset, recurrent or severe preeclampsia appear to at higher risk for developing CVD; including the premenopausal period. Bauer and Cleary stated that “these women may have unrecognized chronic hypertension, an inherited thrombophilia, or other genetic or environmental factors predisposing them to hypertension during and after pregnancy” (p. 164).

Mongraw-Chaffin, Cirillo, and Cohn (2010) reported that traditional risk factors, such as obesity, hypertension, and diabetes have been indicated as underlying causes of both diseases. However, based on results of their prospective cohort study, the researchers determined that higher risks of CVD-related death for women with preeclampsia are independent of BMI, gestational hypertension, preexisting hypertension, and persisting diabetes mellitus. Mongraw-Chaffin et al. (2010) concluded that preeclampsia may be indicative of more than just a combination of these risk factors and added that even though causal factors, such as immune function, oxidative stress, and vascular influences may explain the association between preeclampsia and CVD none

have yet been solidly identified. However, the researchers stated that early CVD screening in preeclamptic women may be beneficial; even in the absence of other risk factors (Mongraw-Chaffin et al.).

Risk Factors for Preeclampsia

Valdés et al. (2009) conducted a retrospective case-control study to determine if women who have had hypertensive pregnancies developed cardiovascular lesions earlier or more extensively than women who had normotensive pregnancies. Results of coronary angiography were used to establish the presence or absence of cardiovascular lesions. Results of the study indicated that frequencies of smoking habits and occurrences of diabetes mellitus were not significantly different among hypertensive pregnancies and normotensive pregnancies; however hypertension, hyperlipidemia, and a family history of premature cardiovascular disease were more frequent in women with previous hypertensive pregnancies. Women with hypertensive pregnancies were also found to have higher BMIs; however there were no significant findings related to the number of stenotic lesions found among the hypertensive and normotensive pregnancy groups. Correlations between age and the number of diseased coronary vessels revealed the number of stenotic arteries increased with age for both groups (Valdés et al.). In contrast to other researchers, Valdés et al. reported that CAD was not significantly associated with hypertensive pregnancies. However, their results did show an association between

diabetes mellitus, a family history of premature CVD, and current smoking. These results are consistent with findings from other studies.

Other researchers have also reported family history as being associated with preeclampsia. According to Chesley (1980), familial history of preeclampsia/eclampsia and diabetes predispose to preeclampsia. Cincotta and Brennecke (1998) reported family history as being associated with a fourfold increased risk of severe preeclampsia. In a recent intergenerational study, Berends et al. (2008) determined that fasting glucose levels, waist circumferences, and levels of systolic blood pressures all demonstrated significantly higher results in mothers of preeclamptic women. Likewise, the metabolic syndrome was found to be more widespread both in women with a history of preeclampsia and their mothers ($p < 0.05$). Diastolic blood pressures were reported as being higher among women who developed preeclampsia later during pregnancy, yet significantly more women ($p = 0.04$) who developed early-onset preeclampsia were diagnosed with hypertension (Berends et al.).

According to Andersgaard et al. (2012), family history is a significant independent risk factor for CHD. Results from a population-based study indicated that family histories of stroke, hypertension, angina pectoris, and myocardial infarction were more frequently reported among women who experienced hypertensive disorders of pregnancy than those who did not. The researchers determined that the rates at which this information is reported supports that similarities exist in profiles of CHD and preeclampsia (Andersgaard et al., 2012). In addition, the researchers determined that

recurrence of hypertensive disorders increase substantially with subsequent pregnancies. In women who experience preeclampsia in the first pregnancy, rates of preeclampsia in the second pregnancy demonstrated a RR = 6.6 (95% CI: 5.5-7.9) and after non-proteinuric hypertension in the first pregnancy, RR = 1.2 (95% CI: 0.8-1.9) was demonstrated in second pregnancies (Andersgaard et al.).

Roes, Sieben, Raijmakers, Peters, and Steegers (2005) studied associations between family history of CVD and severe preeclampsia and/or HELLP syndrome and determined that history of familial CVD in women with histories of HELLP syndrome and/or preeclampsia was not significantly different from that of controls. However, when compared to controls, the odds more than doubled (OR= 2.6; 95% CI: 1.5-4.3) for women with severe preeclampsia and/or HELLP syndrome to have one or more first-degree relatives with hypertension and/or hypercholesterolemia before 60 years of age. The prevalence of hypertension and hypercholesterolemia among first-degree relatives, regardless of age, was also higher in women with HELLP syndrome and/or severe preeclampsia (OR=2.0; 95% CI: 1.2-3.4) than in controls (Roes, et al., 2005).

Irgens, Reisaeter, Irgens and Lie (2001) determined that long-term risk for death, due to cardiovascular causes, is likely due to a maternal genetic predisposition among women who experience preeclampsia. The researchers examined 626,272 recorded births between 1967 and 1992; births represented the mother's first delivery. In this Norwegian-based cohort study, groups were stratified based on whether or whether not the mother had preeclampsia during pregnancy. Because severe preeclampsia was believed to be

more prevalent in preterm pregnancies, groups were further stratified based on whether the birth was term or preterm (Irgens et al., 2001). Of the 626,272 first-births which occurred in women between 1967 and 1992, there were 4350 deaths among those women. Women who had *term* deliveries and preeclampsia accounted for 6.6 deaths per 1000; however women who had *preterm* deliveries and preeclampsia accounted for 15.5 deaths per 1000. In comparison, there were 6.7 deaths per 1000 and 10.9 deaths per 1000 in who did not experience preeclampsia for term and preterm deliveries respectively (Irgens et al., 2001). Regarding cardiovascular mortality, women who had preterm deliveries, but did not have preeclampsia, experienced higher mortality rates than did women who had term births and experienced preeclampsia (HR = 2.95; 95% CI: 2.12-4.11 versus HR = 1.65; 95% CI 1.01-2.70 respectively). In addition, there was a marked increase in cardiovascular-related deaths among women who had preeclampsia and preterm births (HR = 8.12; 95% CI: 4.31-15.33) as compared to women who term deliveries without preeclampsia. Women who had term deliveries and preeclampsia also demonstrated an increase in death from cardiovascular causes (HR = 1.62; 95% CI: 1.01-2.70) (Irgens et al.). The researchers concluded that increased cardiovascular mortality in women who experience preeclampsia may be related to genetic thrombophilia and in circumstances of preterm deliveries, cardiovascular mortality may be related to other preexisting factors, such as smoking.

Kaaja and Greer (2010) describe pregnancy as a thrombophilic state and stated that thrombophilia is attributable to the risk of venous thrombosis and other pregnancy

complications. In addition, complications experienced during pregnancy are commonly the first signs of hereditary thrombophilia (Kaaja & Greer, 2010). Because smoking is believed to mask preeclampsia symptoms, Irgens et al. determined that instances of preeclampsia may be underreported and that this further substantiates the contribution of genetic thrombophilia to cardiovascular mortality (Irgens et al.). According to Kaaja and Greer, awareness of thrombophilia, identified during pregnancy, may lead to better preventive measures, both in at-risk situations, such as surgery, and in subsequent pregnancies.

Magnussen, Vatten, Smith, and Romundstad (2009) also indicated that women with histories of hypertensive pregnancies, especially women with recurrent hypertensive episodes during pregnancy, are at risk for early CVD development and should be candidates for interventions aimed at preventing premature CVD. Magnussen et al. (2009) studied associations between hypertensive pregnancies and modifiable risk factors for CVD and metabolic disease using blood pressures, lipid profiles, and BMIs as standardized measures. Preeclampsia was significantly associated with diabetes development (OR=3.8; 95% CI: 2.1-6.6). In addition, in situations where hypertensive episodes were recurrent or occurred later in pregnancy, women were deemed as having more than 10 times greater odds of using anti-hypertensive medications (OR=11.6; 95% CI: 7.1-26.6). Because adjusted BMIs fully attenuated the association with serum lipids for gestational hypertension and partly attenuated associations with preeclampsia,

Magnussen et al. determined that the unfavorable lipid profiles were likely mediated by higher BMIs.

Based on the hypothesis that premature CVD and preeclampsia share common underlying causes and that preeclampsia may be the first indication of a woman's risk for future CVD, Smith et al. (2009) began a longitudinal, matched, prospective cohort study. The study evaluated physical and biochemical cardiovascular risk markers with the belief that studying women prospectively, beginning at one-year postpartum, would facilitate the ability to determine the timeframe in which cardiovascular risk factors progress and cardiovascular disease develops. This 2009 report represents the first analysis of the prospective study and consists of 70 women who developed preeclampsia during pregnancy and 70 women who were normotensive during pregnancy. Study results demonstrated no differences among groups relative to age, BMI, and family history of cardiovascular risks and/or CVD. However, significant differences existed among women in the preeclampsia group ($p = 0.05$) relative to self-reported family histories of pregnancies complicated by hypertension. In addition, women in the previous preeclampsia group demonstrated significantly higher levels ($p = 0.01$) of blood pressure, total cholesterol, LDL cholesterol, and microalbumin/creatinine levels; insulin levels and HOMA indexes were also significantly higher ($p = 0.05$) among women in the previous preeclampsia group as compared to women in the normotensive group (Smith et al.). The researchers determined that at one-year postpartum, risks for cardiovascular events increased by two-to-three fold for women with previous preeclampsia as compared to

women who had normotensive pregnancies (Smith et al.). The researchers acknowledged that although renal pathological lesions of preeclampsia begin to resolve within three months after delivery for most women with preeclampsia and that proteinuria and hypertension usually resolve over a period of months in this population, the amount of time that it takes for cardiovascular risks and/or disease to develop is unknown; however in many women, the initial presentation of hypertensive disorders is during pregnancy and hypertension may persist into the postpartum period (Smith et al.).

Evans et al. (2011) hypothesized that differences existed among women with histories of preeclampsia and women who experienced uncomplicated first pregnancies; especially with regard to cardiovascular and biochemical markers. They compared 50 women with previously uncomplicated pregnancies and 18 women with previous preeclampsia at approximately 16 months postpartum. Study results demonstrated higher total vascular resistance levels ($p = 0.027$); higher diastolic blood pressure levels ($p = 0.037$); and higher mean arterial blood pressure levels ($p = 0.038$) in women who previously experienced preeclampsia versus women who experienced uncomplicated pregnancies (Evans et al.). The researchers concluded that at approximately 16 months postpartum, functional differences exist among women who have experienced preeclampsia as compared to women who experienced uncomplicated pregnancies and that the differences observed among the two groups are consistent with increased CVD risk for the women with previous preeclampsia. In addition, Evans et al. commented that although some alterations were noted to remain in the postpartum state in previous

preeclamptic women, whether or not these women are at a higher risk for CVD later in life than women without previous preeclampsia is unknown.

Brown et al. (2009) examined women 15-44 years of age to examine relationships between preeclampsia and the likelihood of ischemic stroke greater than 42 days after pregnancy. Cases ($n = 40$) were identified as women hospitalized with first cerebral infarcts and controls ($n = 43$) were identified as women free of stroke histories. Study results demonstrated that women with histories of preeclampsia were more likely to: be obese ($p \leq 0.01$); have histories of hypertension ($p \leq 0.01$); have diabetes ($p \leq 0.01$); have elevated cholesterol levels ($p \leq 0.01$); and current smokers ($p < 0.001$) than women in the control group; each of these conditions is associated with stroke. However, associations between preeclampsia and ischemic stroke were not statistically significant (OR=1.35; 95% CI: 0.15-11.99). Because of attenuation of the OR after adding non-pregnancy related hypertension, Brown et al. suggested that the association between histories of preeclampsia and ischemic stroke may be mediated by hypertension to some extent. Recommendations for close risk factor monitoring and blood pressure control beyond the postpartum period were proposed (Brown et al.).

Although Haukkamaa et al. (2004) pointed out distinct differences in preeclampsia and gestational hypertension, Robbins, Dietz, Bombard, and Valderrama (2011) labeled gestational hypertension as a *neglected* CVD risk indicator. Consistently, Valdiviezo, Garovic, and Ouyang (2012) indicate that gestational hypertension *or* preeclampsia is a risk factor for CVD later in life. In addition, Bhattacharya et al. (2010)

posits that women who experience both preeclampsia and gestational hypertension are at higher risk for CVD as well as CVD-related mortality.

Modifiable Risk Factors for both Preeclampsia and CVD

According to Wen et al. (2012), because the clinical syndrome of preeclampsia seemingly disappears after placental delivery, the pathogenesis of preeclampsia has traditionally focused on a placental pathology. However, the current belief is that foundations for both preeclampsia and gestational diabetes mellitus are established pre-pregnancy, but remain undetected in the absence of pregnancy. It is believed that pregnancy activates these conditions and allows the pathologies to become clinically evident (Wen et al.). Magnussen et al. (2007) and Romundstad, Magnussen, Smith, and Vatten (2010) reported cardiovascular risk factors as being consistently present pre-pregnancy. Magnussen et al. implied that unfavorable cardiovascular and metabolic profiles are likely the main causes of preeclampsia; unfavorable cardiovascular and metabolic profiles predispose to both preeclampsia and subsequent CVD. According to Harskamp and Zeeman (2007), obesity underlies the metabolic syndrome and because obesity has become epidemic worldwide, an increase in the incidence of preeclampsia is expected.

According to Roberts and Hubel (2010), because preeclampsia is defined arbitrarily and because few studies have been conducted which provide details of women's pre-pregnancy characteristics, misdiagnosis is common. It is possible that

hypertension may pre-date pregnancies complicated by preeclampsia, but because blood pressure decreases in early in the second trimester of pregnancy and because women may not begin prenatal care until after 20 weeks gestation (ACOG, 2001), existing hypertension may be masked. Factors believed to contribute to preeclampsia are also believed to contribute to cardiovascular disease. Therefore, preeclampsia is believed to be a *forerunner of*, but *not the cause of* vascular disease (Roberts & Hubel, 2010).

Although preeclampsia and gestational diabetes mellitus are distinct diseases and each has its own characteristic pathophysiology, the two diseases are said to share similarities (Wen, et al., 2012). For example, both conditions develop during pregnancy as clinical syndromes, seemingly disappear after delivery, and share similar risk factors, such as obesity, elevated blood pressure, dyslipidemia, insulin resistance, and hyperglycemia. Likewise, having either preeclampsia or gestational diabetes mellitus predisposes to CVD (Wen et al.). According to Wen and colleagues, preeclampsia is likely a two-stage disorder. In stage-one, there is decreased placental perfusion and this is likely to occur during the late portion of the first trimester or the early portion of the second trimester. In stage-two, the maternal syndrome of preeclampsia, due to systemic endothelial dysfunction, occurs; typically early in the third trimester (Wen et al.).

Newstead, von Dadelszen, and Magee (2007), describe pregnancy, even an uncomplicated pregnancy, as a metabolic and vascular *stress test* for women. For those who *fail the stress test*, (for example, women with preeclampsia), risks for cardiovascular complications increase (Newstead et al., 2007). Globally, CVD is said to be the cause of

death for > 50% of women; the majority of CVD deaths are said to be attributable to CHD (23%) and stroke (18 %) (Newstead et al.).

Risk markers for both preeclampsia and CVD share several similarities. However, non-modifiable risk markers, such as age, family history, and other ethnically-related conditions, are said to be of least concern, whereas modifiable or treatable risk factors, which include hypertension, diabetes mellitus, renal disease, obesity, dyslipidemia, metabolic syndrome, microalbuminuria, thrombophilia, and elevated antibodies to *Chlamydia pneumoniae* and cytomegalovirus, represent areas of greatest concern (Newstead et al., 2007). Thus, the notion of preeclampsia as being viewed as a *failed stress test* and viewing pregnancy, even uncomplicated pregnancy, as a form of the metabolic syndrome facilitates understanding of the relationship between CVD and preeclampsia (Newstead et al.).

The presence of dyslipidemia during pregnancy was examined in a study by Mankuta et al. (2010). Over a three-year period of time, lipid profiles were measured in 1752 women aged 20-45 to examine the effects of plasma lipids in up to three pregnancies. Study results indicated that that during the second trimester of pregnancy, total cholesterol, low density-lipoprotein (LDL) or so-called *bad cholesterol*, and triglycerides undergo a marked increase, but return to pre-pregnancy states during the second and third years postpartum (Mankuta et al.). In contrast, high density-lipoprotein (HDL) or so-called *good cholesterol* undergoes a steep elevation during the second trimester, but the level of HDL cholesterol significantly decreases within the third

trimester. In addition, with each subsequent pregnancy, HDL cholesterol was determined to be lower than previous levels (Mankuta et al.). These data accentuate how pregnancy alone, even though uncomplicated, may increase CVD risks for parous women later in life.

Catov et al. (2008) studied 540 women to determine if parity was associated with a higher prevalence of CVD among older women and how CVD risk factors were related to parity, pregnancy complications, and maternal CVD risk. Study results demonstrated that parous women without pregnancy complications were twice as likely to have CVD as compared with nulliparous women. In addition, women with at least one complicated pregnancy experienced rates of CVD almost three times higher than did nulliparous women (Catov et al.). Because of the high percentage of parous women, with both complicated and uncomplicated pregnancies, who were on statin therapy during this study, Catov et al. determined that dyslipidemia was a significant factor linking parity to cardiovascular disease prevalence.

Hauth et al. (2011) analyzed a subgroup of nulliparous women at low-risk for CVD with the aim of reducing pregnancy-associated hypertension. The researchers specifically examined the role of insulin resistance. Insulin resistance was measured from fasting maternal plasma glucose and insulin concentration levels were obtained between weeks 22 and 36 gestation. Study results demonstrated that mid-trimester maternal insulin resistance was significantly higher in women with higher BMIs in Hispanic and African American women; mid-trimester insulin resistance is associated with

preeclampsia (Hauth et al., 2011). Rodie, Freeman, Sattar, and Greer (2004) explain that even in normal pregnancies, a state of insulin resistance exists. However, increased insulin resistance reaches a maximum level in the third trimester and improves after delivery (Rodie et al., 2004). Conditions associated with insulin resistance, such as polycystic ovarian syndrome, increased weight gain, and obesity, are also associated with preeclampsia. Abdominal obesity, in particular, is associated with insulin resistance and elevated triglycerides leading to dyslipidemia; insulin resistance and dyslipidemia are also associated with CVD (Rodie et al.).

According to Wolf et al. (2004), both altered angiogenesis and insulin resistance are linked to preeclampsia and CVD. As a result, women who experience preeclampsia have increased risks for developing CVD. Wolf et al. hypothesized that factors associated with altered angiogenesis and insulin resistance were evident in postpartum preeclamptic women; despite the fact that they may be asymptomatic. Vascular endothelial growth factor (VEGF) is recognized as promoting angiogenesis; it also plays a role in decreasing vascular tone and blood pressure (Maynard et al., 2002). However, when VEGF's circulating inhibitor, soluble fms-like tyrosine kinase (sFlt-1), binds VEGF and placental growth factor (PlGF), the placenta's ability to thrive becomes limited. Thus, altered angiogenesis occurs and results in hypertension and proteinuria (Alladin & Harrison, 2012).

Wolf et al. (2004) measured fasting insulin levels, glucose, VEGF, and sFlt-1 in 29 normotensive women with histories of preeclampsia and 32 women with previous

normotensive pregnancies; homeostasis model assessment of insulin resistance (HOMA_{IR}) scores were also calculated. Study results demonstrated that sFlt-1 ($p < 0.01$) and HOMA_{IR} ($p = 0.04$) were both significantly higher in women with previous preeclampsia versus women with previous normotensive pregnancies. In addition, fasting insulin levels bordered statistical significance ($p = 0.05$); women who experienced previous preeclampsia had higher fasting insulin levels. The authors reported greater odds of prior preeclampsia for women with the greatest levels of both sFlt-1 and HOMA_{IR} and that at one-year postpartum, these factors may contribute to their risks of future CVD (Wolf et al.). Although an increase in sFlt-1 along with a decrease in circulating VEGF and PlGF has been reported weeks before preeclampsia develops (Alladin & Harrison, 2012), Wolf et al. reported a tendency toward increased free VEGF levels in the previous preeclampsia group. Because increased sFlt-1 was found to be independent of BMI, Wolf et al. determined that observable characteristics of women with prior preeclampsia apparently “include not only the obesity-insulin resistance syndrome, but also alterations in angiogenesis factors” (p. 6243).

Pathophysiologic Effects of Preeclampsia

Because differences may exist between women who experience early-onset versus late-onset preeclampsia, Yinon et al. (2010) compared women who developed preeclampsia at less than 34 weeks gestation ($n = 15$); at greater than 34 weeks gestation ($n = 9$); and a control group ($n = 16$). In addition, women with previous intrauterine

growth restriction (IUGR) were also analyzed ($n = 9$). Obesity, diabetes, progestational renal disease, past hypertension, multiple gestations in the index pregnancy, and past or present history of smoking (as well as living with a smoker) were exclusion criteria for all participants. Mean arterial pressures in the greater than 34 week gestation preeclamptic group were significantly higher ($p = 0.04$). None of the biochemical parameters (including cholesterol levels, HOMA index scores, glucose levels, insulin levels, microalbuminuria ratios) demonstrated statistical significance. Endothelial dysfunction was defined as flow mediated dilation (FMD) $< 4.5\%$, and although all groups demonstrated some degree of endothelial dysfunction, women in the early-onset preeclamptic group (93%) and IUGR group (89%) demonstrated greater prevalence ($p < 0.0001$) of endothelial dysfunction than women in the late preeclampsia group (22%) and control group (12.5%). In addition, radial arterial stiffness was found to be significantly increased, but only among the early-onset preeclampsia group. Based on study results, Yinon et al. determined that women who experience early-onset preeclampsia and normotensive IUGR pregnancies are regarded as having endothelial dysfunction, but that women with late-onset preeclampsia do not have evidence of vascular dysfunction.

According to Chambers et al. (2001), relationships between preeclampsia and endothelial dysfunction are independent of risk factors that are usually associated with vascular disease. In a case-control study, Chambers et al. examined whether or not endothelial function was impaired in non-pregnant women who previously experienced preeclampsia and whether endothelial dysfunction was mediated by oxidative stress. One

hundred thirteen women with previous preeclampsia ($n = 35$ with recurrent episodes of preeclampsia and $n = 78$ with a single episode of preeclampsia) along with 48 women (control group) with previously uncomplicated pregnancies were examined; all women were at least three months postpartum. Clinical and biochemical analyses demonstrated that on average, women with recurrent and single episodes of preeclampsia had current hypertension ($p = 0.001$); whereas none of the controls demonstrated hypertension. Notably, a higher incidence of hypertension was demonstrated in women with recurrent preeclampsia. In addition, a higher incidence of family history of hypertension ($p = 0.004$); higher BMIs ($p = 0.01$); higher waist-hip girth ratio ($p = 0.004$); and higher total cholesterol to HDL cholesterol ratios ($p = 0.02$) were reported for women with previous preeclampsia as compared to controls (Chambers et al.). Specific to vascular endothelial function, brachial artery diameter was lower ($p < 0.001$) in women with previous preeclampsia as compared to controls; women with recurrent preeclampsia had significantly lower brachial artery diameters ($p = 0.02$) as compared to those with a single episode. Flow-mediated endothelium-dependent dilation was also lower among women with previous preeclampsia. With this these finding, Chambers et al. recognized that at a median three-year interval, women with previous preeclampsia have impaired vascular endothelial function (Chambers et al.).

Impaired endothelial function has also been noted in women with severe preeclampsia episodes such as HELLP syndrome. According to Martin, Rose, and Briery (2006) HELLP syndrome likely stems from abnormal placental development, function,

and ischemia-generating oxidative stress. This combination of factors likely activates the release of factor(s) which injure the endothelium and result in loss of normal pregnancy vasculature relaxation (Martin et al., 2006). Habli et al. (2009) reported that women with histories of HELLP syndrome are at increased risk for preeclampsia and HELLP syndrome in future pregnancies as well as chronic hypertension and depression.

To determine if endothelial dysfunction was present 6-12 months after delivery, Agatisa et al. (2004) compared 50 women: 16 with histories of preeclampsia; 14 with histories of normal pregnancies; and 20 controls who had never been pregnant. Study results demonstrated that although baseline heart rates, blood pressures, and forearm blood flows were comparable among all groups, heart rate and forearm blood flow values were highest in the preeclamptic group. However, mean arterial pressure differed significantly ($p = 0.007$); the preeclamptic group had higher values. Stress-induced forearm blood flow demonstrated similar results. According to Agatisa et al., at approximately 10 months postpartum, preeclamptic women demonstrated evidence of endothelial dysfunction in forearm vasculature. In addition 31% of preeclamptic women were obese and had higher BMIs; these factors were believed to play a role in the onset of preeclampsia. Agatisa et al. suggested that because endothelial dysfunction may be an antecedent to clinically evident CVD, preeclamptic women may benefit from intensified observation and early preventive interventions such as weight loss, blood pressure control, and lipid and glycemic control.

According to Bilhartz, Bilhartz, Bilhartz, and Bilhartz, (2011), endothelial dysfunction plays a critical role in both preeclampsia and CVD. Metabolic and vascular abnormalities are present with both conditions; these markers, in either condition, closely resemble that of the other condition. Inflammatory changes which occur in normal pregnancy are said to be inflated with hypertensive pregnancies and may be intensified by maternal factors such as obesity, lipid abnormalities, and insulin resistance; these factors predispose to endothelial dysfunction, preeclampsia, and potentially CVD later in life (Bilhartz et al., 2011).

Haukkamaa et al. (2004) reported similar findings relative to endothelial dysfunction. To determine the contribution of hypertensive pregnancies (especially preeclampsia) to CAD, Haukkamaa et al. (2004) conducted a study among women less than 66 years of age using documented angiography as a measure of comparison. There were 141 women in the CAD group and all had $\geq 50\%$ stenosis in 1 to 3 coronary arteries. There were two control groups: (1) an outpatient group ($n = 99$); and (2) a hospital group ($n = 112$). Study results demonstrated that women with documented CAD had more child deliveries and were younger during their first pregnancy in comparison to women in both control groups ($p = < 0.001$). Risk factors for CAD, such as smoking, body mass index, and hypercholesterolemia were also significantly higher ($p = < 0.001$) among women with documented CAD as compared to controls in both groups. Systemic hypertension and type-2 diabetes mellitus also were significantly higher ($p = < 0.001$) in women with documented CAD as compared with the outpatient control group only; data

for the hospital control group were not available. Results relative to hormone replacement therapy usage demonstrated that women in the hospital control group had significantly higher usage ($p = 0.003$) than women in the CAD group or outpatient group. Rates of preeclampsia, during both first pregnancy and *any* pregnancy, were significantly higher ($p = < 0.001$) among women in the CAD group versus women in either of the control groups. Rates of pregnancy-induced hypertension during first pregnancy did not demonstrate significant results, however when this variable was measured for *any* pregnancy, women in the CAD group demonstrated significantly higher ($p = 0.005$) rates of pregnancy-induced hypertension as compared to women in both control groups (Haukkamaa et al.).

According to Haukkamaa et al. (2004), although endothelial dysfunction is a key pathophysiologic finding in preeclampsia, its presence remains after delivery. Thus, preeclampsia, during *any* pregnancy, is an independent risk factor for subsequent CAD. Furthermore, although many view preeclampsia and hypertensive pregnancies as a continuous sequence of the same disease, endothelial dysfunction is present in preeclampsia, but is not evident in gestational hypertension. Consequently, coronary endothelial dysfunction was named as an independent predictor of acute cardiovascular events; regardless of the presence or absence of clinically evident CAD. Therefore, women with preeclampsia should be advised to avoid obesity, stop smoking, and prevent or manage hypertension and hyperlipidemia (Haukkamaa et al.).

Mangos, Spann, Pirabhahar, and Brown (2012) wanted to determine if women who had experienced preeclampsia or gestational hypertension during pregnancy had a larger group of cardiovascular risk abnormalities as compared to women who had a normal pregnancy. They evaluated 101 women who were 2 to 12 years postpartum. There were three groups in the study: (1) women with prior gestational hypertension ($n = 27$); (2) women with prior preeclampsia ($n = 39$); and (3) women who experienced normal pregnancies ($n = 35$). Study results demonstrated significantly higher ($p < 0.01$) ambulatory blood pressure measurements among women with previous preeclampsia and gestational hypertension as compared to women who experienced normal deliveries. HOMA scores also demonstrated significantly higher results ($p = 0.01$) in the gestational hypertension group and preeclampsia group ($p = 0.05$) as compared with women who experienced normal pregnancies. In addition, women with previous preeclampsia and gestational hypertension demonstrated significantly higher ($p = 0.01$) insulin levels as compared to women in the normal pregnancy group (Mangos et al.). According to Mangos et al., endothelial dysfunction, sympathetic overactivity, and renal dysfunction have been reported in postpartum preeclamptic women; however the researchers did not find evidence of either of these conditions in their sample. However, Mangos et al. indicated that results of this study should portray a strong clinical message to consider all women who experience either preeclampsia or gestational hypertension as having increased cardiovascular risks and that they should therefore be counseled to adopt healthy lifestyles, including medical follow-up.

To evaluate the contribution of pre-pregnancy factors to CVD among women who experienced both preeclampsia and gestational hypertension, Romundstad, Magnussen, Smith, and Vatten (2010) analyzed longitudinal data, from two consecutive population-based studies. Women with histories of preeclampsia ($n = 168$), gestational hypertension ($n = 93$), and normotensive pregnancies ($n = 2964$) were compared; a total of 3225 women were included in the study. Romundstad et al. hypothesized that if relationships between hypertension during pregnancy with ensuing CVD risk factors were substantially attenuated after adjusting for pre-pregnancy risk factors; the CVD/hypertensive pregnancy would primarily be the result of a risk profile that was present prior to pregnancy. However, if attenuation was not present or was weakly present, this may suggest that pregnancy-induced hypertension may well increase the risk of later CVD. Study results demonstrated that after adjusting for pre-pregnancy BMI, the variation in BMI after pregnancy was 67%. In addition, age-adjusted systolic blood pressures were higher among women with histories of preeclampsia and gestational hypertension; however differences were attenuated by almost 50% after adjusting for pre-pregnancy blood pressures. Also, women with hypertensive pregnancies were found to have more unfavorable serum lipid levels and after adjusting for pre-pregnancy BMI, variations in serum triglycerides was almost fully attenuated (Romundstad et al.). Differences in types of instruments used to measure blood pressure at time-point one and time-point two were acknowledged as possibly contributing to these findings. In conclusion, Romundstad et al. determined that the contribution of cardiovascular risks is smaller than that of factors

existing pre-pregnancy. Thus, the presence of cardiovascular risk factors may be present before a hypertensive pregnancy occurs; pre-pregnancy BMIs and blood pressure levels may be important determinants of future cardiovascular risks. However, Romundstad et al. acknowledged that study results could not dismiss that a relationship exists between later cardiovascular disease development and preeclampsia and gestational hypertension; irrespective of pre-pregnancy factors.

Summary of Relationships between Preeclampsia and CVD

Exact causal pathways linking CVD and preeclampsia remain obscure. However, researchers have proposed many conditions shared by both diseases (see Table 1).

Although viable explanations, based on research, have been offered to explain associations between preeclampsia and CVD, gaps and inconsistencies in the literature are evident. For example, Valdés et al. (2009) reported that CAD was not significantly associated with hypertensive pregnancies; however Smith et al. (2009) reported that preeclampsia increases risk for cardiovascular events by two to three fold.

Overwhelmingly, endothelial dysfunction was named as existing in both CVD and preeclampsia. According to Haukkamaa et al. (2004), although endothelial dysfunction is a key pathophysiologic finding in preeclampsia, its presence remains after delivery. In addition, Haukkamaa et al. labeled coronary endothelial dysfunction as an independent predictor of acute cardiovascular events; regardless of the presence or absence of clinically evident CAD. However, Mangos et al. (2012) reported that there is

no evidence that endothelial dysfunction exists in women with previous preeclampsia.

Thus, inconsistencies remain relative to the exact linkage of preeclampsia and CVD.

Table 1

Manifestations Shared between Preeclampsia and CVD

Health Condition	Researchers
Dyslipidemia (Including Atherosclerosis)	Bilhartz et al., 2011; Brown et al., 2009; Catov et al., 2008; Mankuta et al., 2010; Roes, et al., 2005; Rodie et al., 2011; Romundstad et al., 2010; Smith et al., 2009; Valdés et al., 2009; Wen et al., 2012
Endothelial Dysfunction	Agatasa et al., 2004; Bilhartz et al., 2011; Chambers et al., 2011; Haukkamaa et al., 2004; Martin et al., 2006; Wen et al., 2012; Yinon et al., 2010
Family History	Andersgaard et al., 2012; Berends et al., 2008; Chesley 1980; Cincotta & Brennecke, 1998; Roes, et al., 2005; Valdés et al., 2009
Hypertension	Agatasa et al., 2004 ; Bauer & Cleary, 2009; Bilhartz et al., 2011; Brown et al., 2009; Evans et al., 2011; Melchiorre et al., 2011; Roberts & Hubel, 2010; Romundstad et al., 2010; Smith et al., 2009; Wen et al., 2012
Metabolic Disorders (Including: Insulin Resistance; Hyperinsulinemia; Diabetes; and the Metabolic Syndrome)	Bilhartz et al., 2011; Brown et al., 2009; Hauth et al., 2011; Mangos et al., 2012; Smith et al., 2009; Valdés et al., 2009; Wen et al., 2012; Wolf et al., 2004
Obesity	Agatasa et al., 2004; Bilhartz et al., 2011; Berends et al., 2008; Haukkamaa et al., 2004; Hauth et al., 2011; Romundstad et al., 2010; Valdés et al., 2009; Wen et al., 2012

Some researchers (Irgens et al., 2001; Magnussen et al. 2007; Newstead et al., 2007; Roberts & Hubel, 2010; Romundstad et al., 2010; Wen et al., 2012) hypothesize that maternal factors, existing prior to pregnancy, herald the onslaught of preeclampsia. In this light, pregnancy activates the maternal syndrome of preeclampsia. In fact, Newstead et al. (2007) refers to the maternal syndrome of preeclampsia as a *failed stress test*. Hypertension and diabetes are noted as preexisting maternal factors contributing to preeclampsia development (Alladin & Harrison, 2012). Evans et al. (2011), McDonald et al. (2008), Ray et al. (2005), and Smith et al. (2009) reported that women who experience preeclampsia during pregnancy are not only at risk for developing future CVD, but that CVD in this population develops earlier as compared to those who have not experienced preeclampsia. In light of these findings, Wikström et al. (2005) stated that only women with severe preeclampsia should be advised of this risk; however, most researchers support a broader approach to patient advisement.

Postpartum Care after Preeclampsia

The postpartum period has been identified as an opportune time for educating women who have experienced preeclampsia regarding risks for future CVD development and benefits adopting a heart-healthy lifestyle (Firoz & Melnik, 2011). However, as Smith et al. (2009) indicated, complications associated with preeclampsia do not always abate with child delivery. This underscores the necessity of postpartum education and follow-up within this population of women so that the identification of lingering CVD-

related complications may be identified and so that health promotion efforts, through education, may be initiated. To determine factors associated with postpartum medical visit compliance in women who experience complications, Bryant, Hass, McElrath, and McCormick (2006) hypothesized that individual social factors, as well as enabling and inhibiting factors in the healthcare environment, would be significant indicators in predicting use of postpartum medical services. Of 1637 women included in the study, 37% had heart disease and hypertension (Bryant et al.). Study results demonstrated that the presence of maternal chronic health conditions was a significant predictor of compliance with postpartum visits. However, enabling factors such as telephone reminders, contributed significantly to compliance with postpartum visits (Bryant et al.).

Edlow, Srinivas, and Elovitz (2009) examined women with histories of preeclampsia ($n = 79$) and controls ($n = 140$), using telephone interviews, to determine the prevalence of maternal hypertension and other cardiovascular risk factors and if the severity of preeclampsia modified relationships between preeclampsia and the short-term prevalence of hypertension 6-13 months after pregnancy. Of the 79 women who experienced preeclampsia, 21.5% experienced preterm preeclampsia (less than or equal to 34 weeks gestation). Study results demonstrated that at 6-13 months after delivery, the presence of hypertension or use of anti-hypertensive medications was more prevalent among women who experienced preeclampsia ($p < 0.0001$) than among controls. In addition, the presence of hypertension and use of medications other than anti-hypertensives was also more prevalent among women with preeclampsia ($p < 0.0001$).

than in controls. Furthermore, histories of preeclampsia in more than one pregnancy was significantly higher among women with preeclampsia ($p = 0.002$) than in controls. Of the cardiovascular risk factors examined (hypertension, diabetes, and dyslipidemia), only hypertension was significantly higher in the preeclamptic group (adjusted OR = 10.5; 95% CI: 5.2-37.4; $p = < 0.0001$). Examination of the same cardiovascular risk factors among women who experienced preterm preeclampsia revealed an 18-fold higher incidence of hypertension when compared with controls and after controlling for confounders (adjusted OR = 18.31; 95% CI: 5.1-65.5; $p = < 0.001$) (Edlow et al.). The researchers determined that the large relative risk of hypertension in women with histories of preeclampsia versus that of controls suggests a true association. Because the existence of cardiovascular risk factors in preeclamptic women was present as early as six months postpartum, the post-delivery period was identified as an opportune time for identifying at-risk women and engaging them in receiving routine health care, including lifestyle modification and therapeutic interventions, which may significantly influence long-term cardiovascular morbidity and mortality (Edlow et al.).

Samwiil, Mercer, Jarrett, and O'Malley (2004) recruited a cohort of women previously affected by preeclampsia to determine if preeclampsia was resolved. A total of 260 women were asked to recall: (1) the location of their routine six-week postnatal examination; (2) whether or not blood pressures were measured; and (3) whether or not urine was tested (Samwiil et al., 2004). Study results demonstrated that: (a) 257 of the 260 women (99%) reported attending postnatal check-ups; (b) 16 of the 257 women (6%)

reported not having blood pressures measured; and (c) 174 of the 257 women (68%) reported not having urine tested. Further examination revealed that 28 of the 257 women (11%) presented signs of unresolved preeclampsia (Samwiil et al.). The authors established that although routine follow-up examinations six weeks after childbirth are designed to assess physical and psychological wellbeing, a considerable percentage of women are screened for unresolved preeclampsia during routine postnatal visits; especially urinalysis screenings for proteinuria (Samwiil et al.).

Berks, Stegers, Molas, and Visser (2009) studied 116 women to determine the amount of time that is required for hypertension and proteinuria to resolve postpartum. At two-years postpartum, study results showed that 18 % had persistent hypertension; the incidence for proteinuria was 14% at three months postpartum. The researchers determined that these findings were likely due to lack of endothelial recovery. Levels of endothelial cell injury during preeclampsia were reflected by highest blood pressure readings and levels of proteinuria and were directly correlated with the time to postpartum recovery (Berks et al.). In addition, the researchers stated that this association in itself implies that preeclampsia has an effect on future cardiovascular risk and that delaying treatment may also increase remote cardiovascular risk (Berks et al.).

To determine how cardiovascular risk factors are managed by general practitioners after delivery, Nijdam et al. (2009) examined women who experienced preeclampsia during pregnancy. Selection criteria were based on actual diagnoses of preeclampsia, HELLP syndrome, or eclampsia. In addition, women suspected of

preeclampsia, due to the presence of substantial proteinuria in combination with hypertension, were also selected. There were 35 women in the preeclampsia group and 150 women in the control group. Mean follow-up time was 2.9 years for the preeclampsia group and 2.5 years for the control group. Study results indicated that blood pressure measurements were more often recorded in pregnancies complicated by preeclampsia than in uncomplicated pregnancies (57.1% vs. 12% respectively, $p < 0.001$). However, recorded blood pressure measurements continued after three months in only 8 of 30 women without signs of chronic hypertension, although six of these women still had one or more hypertensive blood pressure measurements after this period of time (Nijdam et al.). Of the 18 women coded as having previous preeclampsia, only 61.1% had blood pressure measurements recorded after delivery and of these, 54.5% had blood pressures measured within three months of delivery. In women coded incorrectly, 52.9% had recorded blood pressure readings after delivery; 44.4% within three months of delivery. In the preeclampsia group, the reasons given for blood pressure measurement were history of preeclampsia (70%) and non-resolved hypertension (30%). In the control group, several reasons were recorded for blood pressure measurements, including routine controls after birth, hypertension, complaints of chest pains, and screening for family history of vascular disease. Based on documented information, nine women in the preeclampsia group had one or more cardiovascular risk factors present before pregnancy. However, after delivery, only two women (22%) were evaluated for these preexisting risks. Fifteen of the 35 women (42.9%) in the preeclampsia group did not

have recorded blood pressure readings; glucose and cholesterol assessments were said to be rare and similar in both the preeclampsia and control groups. The researchers implied that a weakness in knowledge base and communication exists among maternity care providers and general practitioners, and that identification and follow-up of women experiencing preeclampsia is not taking place (Nijdam et al.).

Similar conclusions were made by Young, Hacker, and Rana (2012). Unlike the study conducted by Nijdam et al. (2009), this study was based in an academic medical center in the northern part of the United States. Young et al. (2012) hypothesized that providers caring for women with histories of preeclampsia were unaware of their risks for future CVD and that few health care providers provide risk-reduction counseling to these women despite their high-risk for CVD development. Because most female patients present to internal medicine care providers and obstetrician/gynecologists (OB/GYN) for annual well-woman examinations, both types of physicians were included in the study. The study's aim was to assess physician's knowledge of preeclampsia's association to future CVD and to determine if differences existed in levels of knowledge based on the provider's specialty area of practice (Young et al.). Using a web-based survey, 118 residents and internists and 53 residents and attending OB/GYNs were examined. Study results demonstrated that 56% of internists and 23% of OB/GYNs were unaware of increased risks for ischemic heart disease in women with previous preeclampsia ($p = 0.0001$). Relative to shorter life expectancies in women with previous preeclampsia, both internists (79%) and OB/GYNs (77%) were either unsure or incorrect regarding the

association. In addition, 48% of internists and 38% OB/GYNs were unaware of the association of previous preeclampsia with stroke. However, internists (77%) were more likely than OB/GYNs (62%) to be aware of the American Heart Association's guidelines for primary prevention of CVD and stroke ($p = 0.04$). Relative to knowledge of relationships between preeclampsia and future hypertension, most providers were aware of the association with only 6% of OB/GYNs and 17% of internists being unaware or answering incorrectly. Ninety-five percent of internists and 70% of OB/GYNs reported providing routine counseling for CVD risk reduction (Young et al.). The researchers concluded that knowledge relative to the association between preeclampsia and CVD was deficient, resulting in limited application of this risk to clinical care (Young et al.).

Summary of Postpartum Care after Preeclampsia

Berks et al. (2009), Nijdam et al. (2009), Samwiil et al. (2004), and Smith et al. (2009) identified that symptoms of preeclampsia may not resolve during the normal six-week postpartum period. Yet, follow-up during postpartum is not adequate; lack of knowledge relative to relationships between preeclampsia and CVD is evident among health care providers (Nijdam et al, 2009; Young et al., 2012). Thus, women who experience preeclampsia during pregnancy are likely unaware of their increased risk for future CVD.

Health Promotion, CVD Education, and Risk Perception in Women

Although the American Heart Association has issued *Guidelines on the Prevention of Cardiovascular Disease in Women* for quite some time, year 2011 was the first year in which pregnancy complications, namely preeclampsia, were listed as risk factors for CVD (Rich-Edwards, 2012). Several researchers (see Table 2) who studied relationships between preeclampsia and CVD, as well as postpartum statuses of preeclamptic women, have recommended lifestyle counseling and early intervention to promote CVD knowledge and lessen CVD risk in this population. However, research studies demonstrating that such actions are taking place were not found. This research represents one of the first efforts aimed at informing women with previous preeclampsia of their increased risk for CVD development. Along with educating previously preeclamptic women of their increased CVD risk, quantifying levels of personal risk perception serves as an important first-step to addressing health and education needs in this population. In accordance with social cognitive theory, providing factual information, combined with an extension of practical risk reduction approaches, is necessary; people learn and develop effective ways of behaving under realistic situations (Bandura, 1994).

Recent research has demonstrated that CVD educational interventions have positive influences (Nawathe, Glied, Weintraub, & Mosca, 2010). According to Strecher, De Vellis, Becker, and Rosenstock (1986), verbal persuasion, by way of education, is an effective approach for promoting efficacy expectations in individuals. In addition,

Strecher et al. (1986) describes education as a resourceful means to encourage people to persevere in their efforts to change behavior. Roberts and Catov (2012) point out that there is little information available that guides health care providers on next-steps relative to women who experience preeclampsia. However, based on what is known, lifestyle modification to reduce coextensive risk factors including: smoking, obesity, and sedentary lifestyles should be emphasized.

Because an instrument that measures CHD knowledge, exclusively designed for women, was not available, Thanavaro, Thanavaro, and Delicath (2008) developed the *Coronary Heart Disease Knowledge (CHDK)* tool for women. To evaluate the tool's reliability and validity, a pilot study was conducted with 39 women who were admitted to a chest pain center; none of the women in the pilot test had prior diagnoses of CHD. The CHDK tool's reliability and validity were further evaluated in another study where study participants were divided into three groups. Group one consisted of 49 women who were recruited from a primary care office. Women in this group were without prior CHD diagnoses; the CHDK tool was completed during their office visit. Group two consisted of 23 cardiovascular nurses. Group three consisted of the treatment group; 22 women who received a CVD education intervention. Although group two consisted of cardiovascular nurses, study results showed that the mean CHD knowledge score (range: 0 – 25) was higher for participants who received the CVD educational intervention than scores for cardiovascular nurses (22.4 ± 1.2 versus 21.8 ± 2.2 , respectively). In addition to validating the new CHDK tool for women, the researchers concluded that an organized

educational strategy could notably expand women's knowledge of CHD and health promoting behaviors (Thanavaro et al., 2008).

Homko et al., (2008) conducted a study to examine knowledge of CVD risk factors and risk perception among a group of patients at high risk for CVD. The study was conducted at two medical centers in a northern state in an area considered as medically underserved. There were 465 participants in the study and each participant was reported to have a greater than 10% CVD risk level. CVD risk perception was measured using a three-question risk perception scale that compares personal perception of risk to that of others of the same age. Study results demonstrated that those living in the inner city had lower perceived CVD risk than their actual CVD risk calculated by the Framingham 10-year risk prediction scale. Inner city participants were also significantly less knowledgeable of CVD than those who lived in rural areas. Factors that predicted knowledge and perception of CVD risk factors included: the presence of diabetes, white race, female gender, and advanced age (Homko et al., 2008). With regard to general CVD knowledge, higher income levels were determined to be the sole predictor (Homko et al.). According to Schwarzer (2011), when an individual compares him/herself to similar others, individual risk perception becomes biased; people typically view themselves as being less likely than others to experience health problems in the future.

To examine perceived CVD risk versus calculated CVD risk and to evaluate the effectiveness of educational interventions to improve knowledge Christian, Mochari, and Mosca (2005) conducted a study of 125 women. Perceived risk was conceptualized as the

woman's belief regarding personalized CVD risk and measured by asking the question: "In the next ten years, what do you think your chances of having a heart attack or dying of heart disease compared to a woman of similar age as you" (Christian et al., 2005, p. 1595). Actual CVD risk was estimated using the Framingham global 10-year risk estimate. CVD 10-year risk perception was measured at baseline ($n = 125$, immediately after educational intervention, and at a one-month follow-up ($n = 111$) using the same question (Christian et al.).

Results of the Framingham global risk estimate at baseline demonstrated that 59% of study participants had less than 10% 10-year risk; 10% of study participants had between 10% and 20% 10-year risk; and 21% of study participants had a greater than 20% 10-year risk of CVD (Christian et al., 2005). Interestingly, only 52% of those calculated to be at low risk accurately perceived their risk as being low at baseline (Christian et al.). Measurement of perceived risk immediately after educational intervention demonstrated that 67% of those calculated as being at low risk accurately perceived their risk for CVD as low; this was a significant increase ($p = .02$) compared with baseline results (Christian et al.). At one-month follow-up, however there was a decline in CVD risk perception for those calculated as low risk; only 52% accurately perceived their CV risk as low (Christian et al.). Among participants calculated as moderate to high risk ($\geq 10\%$), accurate risk perceptions gradually declined from baseline (71%), to post educational intervention (68%), to one-month follow-up (64%) Results of multiple regression analyses that examined family history, age, previous CVD risk

factors, and CVD knowledge demonstrated that only age (≥ 65 years) was as a significant predictor of CVD perception (Christian et al.).

Table 2

Implications and Recommendations for CVD Risk Reduction in Women with Previous Preeclampsia

Author(s)	Implications/Findings	Recommendations
Agatisa et al. (2004)	Women with histories of preeclampsia display impaired endothelial dysfunction at one-year postpartum; this may be a precursor to clinical disease.	Early preventative interventions, e.g. weight loss, blood control, lipid and glycemic control are warranted.
Andersgaard et al. (2012)	Previous preeclampsia increases risk for preeclampsia in subsequent pregnancies. These women have twice the risk of future hypertension and CAD. Family history of CVD was more common in preeclamptic women.	Women with Preeclampsia and non-proteinuria hypertension should be informed of risks for recurrence in future pregnancies and advised to control blood pressure after pregnancy, and receive lifestyle counseling.
Anderson (2007)	The challenges of pregnancy represent an opportunity to screen for CVD and establish prevention strategies prior to overt disease.	Nurses have a unique opportunity to educate women about cardiovascular risk and establish a plan for optimal cardiovascular health.
Bellamy et al. (2007)	Women who experience preeclampsia have a two-to-fourfold increased risk for CVD.	Preventive therapies targeting established risk factors for CVD should be initiated at a younger age than usual among this population of women.
Bilhartz et al. (2011)	Women with advanced maternal age, obesity, nulliparity, and multiple gestations are more likely to develop preeclampsia.	Collaborative efforts are needed to increase women's awareness and effectiveness of screenings.

(Continued)

Author(s)	Implications/Findings	Recommendations
Brown et al., (2006)	Women with preeclampsia may be at increased risk for ischemic stroke.	Targeting this population for early risk-factor monitoring, beyond the postpartum period, may be justified.
Edlow et al. (2009)	Women with preeclampsia are at increased risk for long-term cardiovascular morbidity and mortality.	Preeclampsia provides an opportunity to identify women at risk for cardiovascular morbidity years before disease onset; this allows a window of prevention and intervention.
Kaaja and Greer (2005)	Exaggerated physiologic responses, reflective of metabolic syndrome, are seen in preeclampsia and gestational diabetes and can signal future CVD and metabolic disease.	Primary prevention after pregnancy, including weight reduction (where appropriate), dietary advice and increased exercise.
Harskamp et al. (2007)	Women with histories of preeclampsia are at increased risk for CVD later in life. The worldwide epidemic of obesity is expected to increase and obesity underlies the existence of metabolic syndrome.	Lifestyle modification, in addition to diabetes screening, is worth considering for these women.
Lykke et al. (2009)	Hypertensive pregnancy disorders are strongly associated with subsequent cardiovascular morbidity.	Early identification of at-risk women allows for prompt interventions, including modification of traditional cardiovascular risk factors.
Magnussen et al. (2009)	Years after pregnancy, women with hypertensive pregnancy disorders, especially when recurrent, are likely to be at substantially higher risk for CVD due to increased body mass, blood pressure, and cholesterol levels.	Identifying and targeting high-risk women for early prevention is crucial.

(Continued)

Author(s)	Implications/Findings	Recommendations
Melchiorre et al. (2011)	Cardiovascular implications do not end with delivery of the infant and placenta. Preeclampsia, especially early-onset, predisposes to left ventricular dysfunction/preeclampsia within 1 to 2 years after delivery.	Preeclampsia represents an opportunity to identify women at high-risk before other risk factors or symptoms become clinically evident.
Mongraw-Chaffin et al. (2010)	Women with previous preeclampsia are at increased risk for cardiac death later in life independent of other risk factors.	Women with histories of preeclampsia should be targeted for early and intensive screening and intervention.
Ness and Hubel (2005)	Prior preeclampsia identifies reproductive-aged women at elevated risk for CAD.	Women with prior preeclampsia should be offered CAD prevention intensively and early.
Nijdam et al. (2009)	Women with preeclampsia are at increased risk for CVD.	Follow-up for women with preeclampsia is insufficient and undeveloped.
Roberts and Catov (2012)	There is little evidence-based information to guide recommendations for follow-up among preeclamptic women.	Based on what is known, lifestyle modification to reduce coextensive risk factors including: smoking, obesity, and sedentary lifestyles should be emphasized.
Robbins et al. (2011)	Gestational hypertension is a neglected CVD risk marker.	Multi-leveled CVD prevention approaches are indicated.
Smith et al. (2009)	Preeclampsia identifies one of the earliest clinical markers to identify women's elevated risk for CVD.	Women with preeclampsia should have screenings for CVD risk factors at one-year postpartum. Early identification and management of CVD risk factors has the potential to prevent long-term morbidity and mortality in these women.

(Continued)

Author(s)	Implications/Findings	Recommendations
Valdés et al. (2009)	The relationship between hypertensive pregnancies and CVD has been confirmed and highlights the need to detect both chemical and biochemical markers of CVD prior to gestation.	Early management of CVD risk is an effective approach to preventing or delaying CVD.
Van Pampas (2008)	Women with histories of preeclampsia, especially early-onset, should be informed of their increased risk for CVD.	These women should be advised on being examined for established risks for CVD and advised on lifestyle changes.
Young et al. (2012)	Knowledge of the association between future CVD and preeclampsia is deficient among health care providers.	Guidelines to assist providers in identifying and counseling women with histories of preeclampsia may help to reduce CVD mortality.

Adoption of Postpartum Lifestyle Changes

Hoedjes et al. (2012) conducted six focus group interviews centered on motivators and barriers to adopting health postpartum lifestyles. In addition, attitudes, subjective norms, perceived behavioral control, and intention, using the theory of planned behavior as a framework, were addressed. Four interviews were conducted with women who experienced either mild or severe preeclampsia or intrauterine growth restriction; one was with women with previous severe preeclampsia; and one was with women with previous gestational diabetes. Hoedjes et al. reported that despite positive attitudes, motivations and intentions to comply, women were unsuccessful in achieving healthy lifestyles due to low perceived behavioral control. Hoedjes et al. suggested that removing or lowering

personal barriers, by offering postpartum guidance, may promote health postpartum lifestyles.

Robbins et al. (2011) used data from the 2008 National Health Interview Survey (NHIS) to examine: (1) compliance with hypertension and cholesterol screening recommendations; and (2) knowledge of heart attack symptoms and CVD risk factors in women with histories of gestational hypertension; data were compared with women who did not have such histories. Notably, year 2008 was the first year that NHIS questioned female respondents regarding histories of pregnancy-related hypertension. Robbins et al. stratified women into three groups for comparison: (1) no hypertension ($n = 7915$); (2) gestational hypertension ($n = 301$); and (3) ever hypertension ($n = 3754$). Study results demonstrated that the levels of CVD knowledge and rates of recommended screenings were not statistically significant between groups. However, in an unadjusted analyses, Robbins et al. reported that women with both gestational and *ever* hypertension had greater prevalence of physical inactivity and obesity in comparison to the *no* hypertension group. Although this study portrayed a nationally representative sample of women, those with gestational hypertensive were no more likely than others to receive recommended blood pressure and cholesterol screenings or to have correct knowledge of heart attack symptoms; even though they had increased risk for developing CVD. Robbins et al. recommended that multi-leveled approaches, aimed at CVD prevention, are indicated. At the individual level, providing knowledge, focused on eliminating modifiable risks, for

example, smoking, obesity, physical inactivity, and excessive alcohol intake were offered as viable solutions to reducing overall CVD risk in women (Robbins et al.).

Kvehaugen, Andersen, and Staff (2010) investigated whether or whether not maternal or offspring differences existed in diet and physical activity 5-8 years after pregnancies complicated by preeclampsia or diabetes mellitus as compared to uncomplicated pregnancies. In the preeclampsia group, women performed high-intensity physical activity significantly less ($p = 0.045$) than women in the control group. Women in both the preeclampsia and diabetes groups were found to have higher prevalence of central obesity as compared to controls. Kvehaugen et al (2010) suggested that lifestyle interventions are needed for women whose pregnancies were complicated by diabetes or preeclampsia. Benefits of increasing physical activity were emphasized. Although weight loss may or may not be achieved with increased physical activity, improving insulin-sensitivity and HDL cholesterol levels are known benefits of adequate physical activity; improved insulin-sensitivity and HDL cholesterol lessens CVD risk (Robbins et al., 2011).

Because exercise has been associated with reduced rates of preeclampsia and other pregnancy-specific condition, Lewis, Martinson, Sherwood, and Avery (2011) evaluated the effectiveness of a telephone-based intervention for pregnant and postpartum women. Social cognitive theory and the transtheoretical model served as the conceptual frameworks for the study. Because of time constraints, child-care conflicts, and transportation limitations, known to exist in this population, a telephone-based

intervention was selected to facilitate study participation. Women less than 16 weeks gestation and women less than six months postpartum were recruited; all women were 23-40 years of age. Telephone-based counseling sessions were used to motivate pregnant ($n = 19$) and postpartum ($n = 18$) women to engage in increased amounts and durations of exercise. Sessions took place weekly for the first month and then biweekly in months two and three with each session lasting 10-15 minutes. In addition to telephone counseling, printed material relative to adopting and maintaining exercise, social support, self-rewards, etc. were also provided. At the end of three months, levels of physical activity significantly increased for both pregnant women ($p = 0.01$) and postpartum women ($p = 0.05$) when compared to baseline scores.

Summary of Health Promotion, CVD Education, and Risk Perception in Women

Dyslipidemia, endothelial dysfunction, family history, hypertension, the metabolic syndrome (including insulin resistance and overt diabetes), maternal predisposition, and obesity have all been noted as conditions occurring in both preeclampsia and CVD. Coincidentally, most of these conditions are also considered as modifiable. Although women with previous preeclampsia are identified as being at risk for CVD, approaches to increasing awareness and knowledge in this population are lacking.

Hoedjes et al. (2012) identified barriers to attaining and maintaining health lifestyles in the postpartum period. Accordingly, Bryant et al. (2006) identified barriers

encountered during the postpartum period that hinder healthful lifestyles.

Interrelationships between health status, individual behavior, and social dynamics are increasingly recognized as factors that influence health and health behavior (USDHHS, 2010).

Thanavaro et al. (2008) demonstrated the usefulness of a newly developed instrument for evaluating CVD knowledge in women. In addition, providing a well thought-out CVD education program for women was found to be an effective approach to increase levels of CVD knowledge in women. Similarly, Christian et al. (2005), reported CVD education, as an intervention, as being useful for increasing CVD knowledge and promoting accurate perceptions of CVD risk. Accurate perceptions of CVD risk have been associated with demonstrable behaviors suggestive of risk reduction. Thus accurate perceptions of CVD risks are necessary for health behavior to be aligned with measures to treat and prevent CVD. Further, Lewis et al. (2011) demonstrated that a telephone-based delivery format for CVD risk reduction counseling was effective.

In spite of these findings, research demonstrating that women experiencing preeclampsia are being informed of their risks for future CVD was not found. This study represents one of the first of such efforts. The postpartum period has been identified as an opportune time for educating women who have experienced preeclampsia regarding risks for future CVD development and benefits adopting a heart-healthy lifestyle (Firoz & Melnik, 2011). According to Dolmans, De Grave, Wolfhagen, and van der Vleuten (2005), learning takes place, more readily, in contextual situations. Likewise, Bandura

(2004) states that knowledge is the prerequisite for change because change is implausible if a lack of knowledge exists regarding how lifestyle habits affects health.

CHAPTER 3

METHODOLOGY

Knowledge of CVD risk, along with accurate perceptions of CVD risks, provides the necessary foundation for health behavior to be aligned with actions aimed at reducing and/or preventing CVD (Christian, et al. (2005). Behavior change has been described as a difficult and multifaceted processes; however accurate perceptions of CVD risk are necessary for behavior change to occur (Gholizadeh et al., 2010; USDHHS, 2010). According to Bandura (2004), in order for behavior change to take place, knowledge is essential; change is unlikely if a lack of knowledge exists regarding how lifestyle habits affects health. The overarching purpose of this study was to promote CVD knowledge and awareness among women with recent preeclampsia, through an educational intervention and to determine the effect of this education on accurate perceptions of personal CVD risk

Study Design

An exploratory cross-sectional repeated measures (pretest/posttest) design was used to address the study aims. The primary outcome variable in the study was CVD risk perception, which was measured both before and after the CVD educational intervention. In addition, CVD knowledge was measured prior to CVD education. Covariates in the

study included: age, race, parity, income, marital status, education, and history of previous preeclampsia. CVD education was provided as an intervention after measuring all study variables; only perception of CVD risk was reevaluated after CVD education.

Study Sample

Women who experienced recent, new-onset, pure preeclampsia during their most recent pregnancy were the study's target population. An accessible population, from a high-risk obstetrical practice, was used. Inclusion criteria for study participants were that participants: (1) were ≥ 19 years of age; (2) were pregnant within the last 12 months; (3) had a diagnosis of preeclampsia, including eclampsia and/or HELLP syndrome during the most recent pregnancy; (4) were able to read and speak English; (5) did not experience fetal demise during the most recent pregnancy; and (6) were willing to participate in the study. Exclusion criteria included women who: (1) were unable to read and speak English; (2) were < 19 years of age; (3) did not have a diagnosis of pure preeclampsia, eclampsia, and/or HELLP syndrome during their most recent pregnancy; (4) had existing diagnoses of hypertension; (5) were mentally and/or cognitively impaired; and (6) experienced fetal demise with the most recent pregnancy.

Sample Size and Power Analysis

According to Polit and Beck (2008), results from relevant earlier studies may be used directly as the estimated effect size (ES). In the present study, the CHD knowledge

tool (Thanavaro et al., 2010) was used to examine levels of CVD knowledge among women with previous preeclampsia. Thanavaro, et al. (2006) used a previous version of the instrument to examine heart disease knowledge in women without prior CHD with a value of $R^2 = 0.35$ for CHD knowledge. Homko et al. (2008) used the 7-point Likert scale CVD risk perception scale (Schwarzer & Renner, 2000) which was used in the present study to examine factors contributing to higher risk perceptions; CVD knowledge was a part of the model. The R^2 value obtained was 0.269 (Homko et al., 2008). Thus, both large and medium ESs were calculated for the present study with power = .80, seven independent variables (covariates), and $\alpha = .05$ to determine the necessary sample size.

Polit and Beck (2008) suggests the following approach to determine sample size: $N = (L / \lambda) + k + 1$, where N = estimated number of subjects needed, L = tabled value for the desired α and power, k = number of independent variables, and λ = estimated effect size. This estimate uses R^2 values where small = $R^2 .02$, medium = $R^2 .13$, and large = $R^2 .30$. The estimated ES, λ , is calculated by $R^2 / (1 - R^2)$. Using this formula, for a large ES, $N = (14.35/.429) + 7 + 1 = 41.44$. Therefore, for a large ES, the required sample size was 42. For a medium effect size, $N = (14.35 /.149) + 7 + 1 = 104.30$, thus the required sample size was 105.

According to Cohen (1992), in multiple regression/correlation analysis, ES indexes are: .02 = small, .15 = medium and .35 = large. Sample size values are calculated using the F^2 statistic and are listed for various combinations of values for power, α , and ES as tabled values. Using Cohen's (1992) tabled values with power = .80, seven

independent variables, $\alpha = .05$, and a large ES, the required sample size is 48. The corresponding sample size for a medium effect size was 102.

Study Procedures

Ethical Considerations

The study was approved by expedited review from the University of Alabama at Birmingham's (UAB) institutional review board (IRB). The study also was sanctioned by University of Mississippi Medical Center's (UMMC) IRB (see Appendices A and B).

Informed Consent

Informed consent was obtained so that participants' rights to self-determination were protected and so that participants could choose whether or not to participate in the study (Burns & Grove, 2009; Polit & Beck, 2008). During the informed consent process, the following elements were explained verbally: (a) the purpose of the study, (b) number of times data would be collected (c) the amount of time expected to complete questionnaires, (d) foreseeable risks, (e) expected benefits, (f) how anonymity and confidentiality would be protected, (d) who to contact with questions or concerns, (g) the voluntary nature of study participation, and (h) the right to refuse study participation. The study did not involve procedures for which consent would normally be required outside the context of research and only minimal risks were incurred. Therefore, a request for waiver of documentation of informed consent was submitted and approved by UAB IRB.

Recruitment

A total of 75 women were referred to the principle investigator (PI) as potential study participants. Of these 75 potential participants, the PI was able to contact 69 (92%) by telephone and 64 of 75 (85.33%) were consented to participate in the study. One potential participant was contacted, but excluded due to not being 19 or more years of age and another potential participant was excluded due to still being pregnant (see Figure 2).

Women meeting the study inclusion criteria were informed of the study and asked to participate by members of the UMMC maternal-fetal medicine research team. Through personal conversation with Dr. James N. Martin, Jr., Director of the maternal-fetal medicine department, UMMC maternal-fetal medicine department serves as a tertiary referral center to those living in the catchment service area and beyond. Thus, women diagnosed with preeclampsia frequently present for medical attention related to symptoms of these conditions and delivery of the fetus ensues shortly thereafter. Follow-up visits to UMMC in the postpartum period are inconsistent, as many women will resume health care services in their immediate geographic area. Therefore, the period of time when eligible participants are hospitalized presented the most opportune time for seeking study participation.

In addition, flyers and/or posters that announced the study were strategically placed within the clinic (see Appendix C). Announcements related to the upcoming study contained information related to: (1) the purpose of the study; (2) inclusion criteria for participants; and (3) the timeline in which the study would be conducted. In this way, all

women who visited the clinic during pregnancy were aware of the study. Because most eligible participants received medical care during pregnancy from the high-risk obstetrical clinic, placing flyers in strategic locations in the clinic likely fostered awareness of the study. Women eligible for study participation, but who did not routinely receive obstetrical care at this clinic, were informed of the study by the UMMC maternal-fetal medical research team during their hospitalization. An information sheet that contained details of the study was given to potential study participants in conjunction with their discharge information. Contact information for eligible participants was obtained by the researcher from the UMMC maternal fetal medicine research nurse. Information obtained included only the name and phone number of the eligible participant. The researcher used a dedicated cell phone to make telephone contact with potential participants. Up to three contact attempts were made. Eligibility for study participation was verified with each potential participant prior to obtaining informed consent. A pictorial depiction of recruitment follows (see Figure 2).

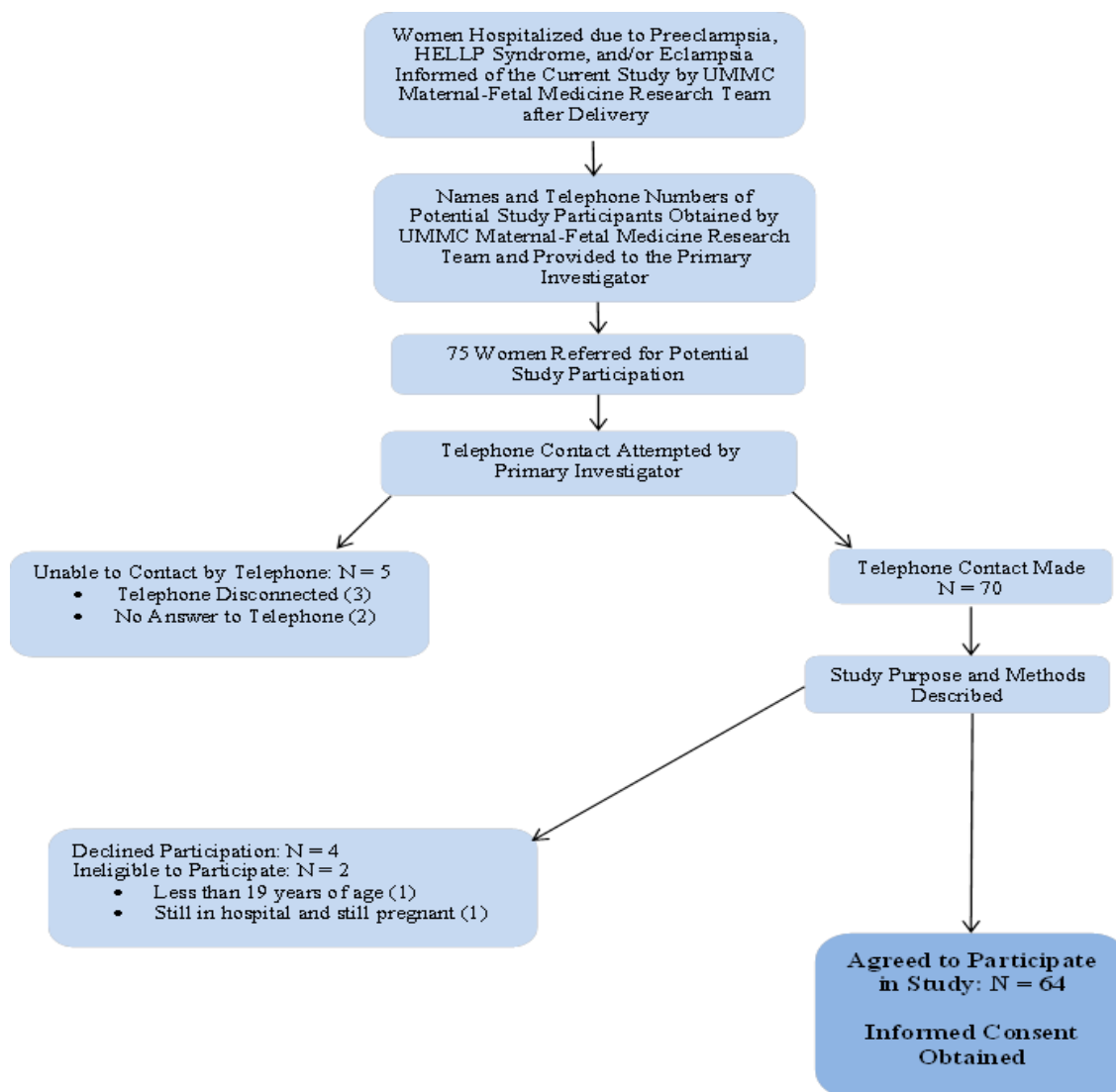


Figure 2. Recruitment Flow Chart

Data Collection

Data were collected using telephone interviews using a prepared script (see appendix E). Under normal circumstances, IRBs require that study participants are given 24 hours after being contacted by telephone and during this 24 hour period, potential

participants are able to decide whether or not they want to participate in the study. During the follow-up telephone call, verbal consent would be obtained and data may be collected. However, because members of the proposed study population are likely to be lost to follow-up, the traditional 24- hour requirement was waived. Thus, informed consent and data collection occurred during the initial telephone contact. Completing the three study questionnaires took approximately 30-45 for each participant.

After verbal consent was obtained, demographic information (age, race, parity, histories of previous preeclampsia, multiple birth, income, education, and marital status), CVD knowledge, and CVD risk perception were solicited from each participant (see Appendix F). Because participants in the proposed study experienced preeclampsia, eclampsia, and/or HELLP syndrome during their most recent pregnancies and because these conditions have been associated with CVD development, CVD education was then provided as an intervention using a prepared script (see Appendix G).

The purpose of providing education intervention was to promote CVD knowledge and awareness among this population. According to Dolmans et al. (2005), learning takes place more readily in contextual situations. Because study participants recently experienced conditions strongly associated with CVD, their recent circumstantial experiences provided an opportunity to introduce CVD knowledge and awareness at a time that may likely transform lifestyle behaviors. According to Bandura (2004), knowledge is the prerequisite for change because change is implausible if a lack of knowledge exists regarding how lifestyle habits affects health. Interrelationships between

health status, individual behavior, and social dynamics are increasingly recognized as factors that influence health and health behavior (USDHHS, 2010). Accurate perceptions of CVD risk have been associated with demonstrable behaviors suggestive of risk reduction. Thus accurate perceptions of CVD risks are necessary for health behavior to be aligned with measures to treat and prevent CVD (Christian et al., 2005).

According to Lloyd-Jones et al., (2010), dietary habits have the strongest evidence base of factors contributing most to cardiovascular events, diabetes, and/or obesity. Therefore, the CVD education was centered on: healthful meal planning, physical activity, medication compliance (if indicated), and the importance of regular blood pressure and cholesterol screenings. In addition, symptoms of heart attack were provided. The *Dietary Guidelines for Americans* (USDA & HHS, 2010) offer a framework for adopting and maintaining healthful meal planning. CVD education, relative to healthful meal planning, was based on the *Dietary Guidelines for Americans*. In addition, the CHDK tool for women (Thanavaro et al., 2008) was modified and used as a part of the education intervention. Elements from *Effectiveness-Based Guidelines for the Prevention of Cardiovascular Disease in Women* (Mosca et al., 2011) were also used to structure the CVD education intervention (see appendix G). After CVD education, CVD risk perception was reexamined to determine if the education intervention affected CVD risk perception. To reinforce CVD education, a brochure was mailed to each study participant after the interview was completed (see Appendix H). A \$5.00 gift card was included in the mailing as well.

Study Measures

Three questionnaires were used in the study; descriptions of each follow.

Demographics

A demographic questionnaire was developed by the researcher and was based on interests of the present study as well as information previously reported in the literature (see Appendix F and Table 3).

Table 3

Demographic Questionnaire

Question	Response Choices
1. Age	19-24; 25-29; 30-34; 35-39; or ≥ 40
2. Race	White/Caucasian; Black/African American; or Hispanic
3. Parity	1; 2; 3; 4; 5; or > 5
4. Multiple Births	No or Yes
5. Previous Preeclampsia	No; Yes; or N/A- First pregnancy
6. Level of Education	Less than High School; High School Graduate/GED; Some College; or College Graduate
7. Annual Household Income	\leq \$10,000; \$10,001-\$20,000; \$20,001-\$30,000; \$30,001-\$40,000; $>$ \$40,001; or Declined to Answer
8. Marital Status	Single/Never Married; Married; Divorced; Living with Significant Other; or Widowed

For regression analyses, several categorical demographic variables were recoded. Education was recoded as 1 = high school graduate or less and 2 = all education beyond high school. Marital status was recoded as 1 = single, never married and 2 = all others (married, separated, widowed, divorced, or living with significant other). Race was recoded where 1 = white and 2 = non-white.

CVD Knowledge

The Coronary Heart Disease Knowledge (CHDK) tool for women was used to assess knowledge of CVD (see Appendix D). The conceptual basis for this instrument is Pender's health promotion model, constructs from expectancy-value theory, and social cognitive theory (Thanavaro et al., 2008). The CHDK tool for women measures CHD knowledge and awareness in women and provides a measure to quantify the perceptions that women have related to coronary heart disease risks. The CHDK tool for women contains 25 multiple choice questions. Summated scores range from zero to 25; higher scores indicate better CHD knowledge (Thanavaro et al).

Cronbach's alpha coefficient of the CHDK tool was reported as .74 and .79, with a 1-to-2 week test-retest reported as .70. Thanavaro et al. (2008) reported inter-item correlations as ranging from -.20 to .57 and determined that because none of the inter-item correlations were .70, redundancies were not a problem.

In the present study, the CHDK tool was modified to facilitate its use and understanding during telephone interviews. The items on the instrument were asked as declarative statements and participants were asked to respond as true or false. The correct response for each item was true. Scores were summated based on the number of true responses from each participant. Thus, the number of correct responses was based on the number of statements that the participant responded to as *true*. As with the original version of the CHDK tool (Thanavaro et al., 2008), summated scores on the modified version of the instrument ranged from zero to 25; higher scores indicated better CHD knowledge (see Appendix F).

CVD Risk Perception

CVD risk perception was measured using a three-item CVD Risk Perception Scale developed by Schwarzer and Renner (2000), which in its original form, was measured on a -3 to +3 Likert scale (see Appendix D). In the present study, participants were asked to respond to each item on a scale of 1-7 (see Appendix F). Modifying the numbered responses to all positive integers was done to allay confusion and facilitate attaining accurate responses from participants. For analyses, value labels for the CVD Risk Perception Scale were recoded to their original values of -3 (much below average) to +3 (much above average), with zero indicating average risk as compared to others of the same age and gender (Schwarzer & Renner, 2000).

Scores from the CVD Risk Perception Scale were summated to estimate participants' perceptions of CVD risk; higher scores indicated higher perceived CVD risk. In order to compare CVD risk perception in the present study with results from previous studies, summated scores were divided by the number of items to obtain an average score. According to Schwarzer and Renner (2000), reliability estimates for the three-item risk perception scale were calculated using Cronbach's alpha internal consistency coefficients with a reported value of 0.78.

Reliability of Research Instruments

Reliability and homogeneity of internal consistency were measured for instruments used in the study using Cronbach's alpha coefficients (see table 4). Complete data were available at baseline; however one response was missing for the post-intervention assessment of risk perception. Reliability coefficients were compared from the present study to reliability coefficients obtained in previous studies that used the same research instruments.

Thanavaro, Thanavaro and Delicath (2010) used the CHD Knowledge Tool for Women to examine 39 women without previous histories of heart disease and reported Cronbach's alpha coefficients of 0.79 at baseline and 0.70 at 2-3 week retest. In the present study, the CHDK instrument was changed from a multiple-choice instrument measured on a 4-point Likert scale to a dichotomized true/false scale. The Cronbach's alpha for the CHD Knowledge Tool for Women was low for the present study ($\alpha = .56$);

however, as noted above, the scoring of the instrument was changed from its original format.

Homko et al., (2008) used the CVD Risk Perception Scale to evaluate medically underserved patients at high-risk for CVD; there were 465 participants with 205 being women. Homko et al. reported a Cronbach's alpha coefficient of 0.78. The CVD Risk Perception Scale was changed in the present study to reflect all positive integers.

Although values of 1 to 7 were used in the current study to facilitate data collection via telephone interviews, data were recoded to values from the original version of the scale (i.e., -3 to +3) for statistical analyses. The pre-intervention reliability was lower than the post-intervention reliability; the latter result is similar to the Cronbach's alpha reported by Homko et al. for their sample (see Table 4).

Table 4

Reliability of Study Instruments

Instrument	Sample Size	Cronbach's Alpha Coefficient
CHD Knowledge Tool for Women	N = 64	.56
CVD Risk Perception Scale	N = 64 (Baseline) N = 63 (Post-CVD Education)	.69 (Baseline) .76 (Post-CVD Education)

Data Management

Prior to collecting data, all instruments were numerically coded. Each participant was assigned a single number that was used for each instrument. The primary investigator had access to participants' contact information; however when participants' contact information was not in use by the primary investigator, information was stored and maintained, in a locked cabinet, by the research nurse in the maternal-fetal medicine department at the UMMC. Collected data were stored in a locked cabinet by the primary investigator. Participants' addresses were obtained so that printed CVD education information and gift cards could be mailed; however addresses were recorded only on the envelope used to mail the CVD education brochure and gift card and were not otherwise maintained.

Data Analysis

Data were analyzed using PASW® Statistics Grad Pack 18 (formerly SPSS). Descriptive statistics (means, frequencies, percentages, and standard deviations) were used, as appropriate, to describe the study sample. Other statistical analyses were performed to test the following study questions:

1. Among women with recent, new-onset preeclampsia, what is the baseline level of CVD knowledge? Summated scores were used to obtain a mean score of true responses. Thanavaro, Thanavaro and Delicath (2010) administered the CHDK Tool for Women with a mean summated score of 17.6 (70.5% total correct answers) and

considered the score as being low. In the present study, frequencies and percentages of correct responses on the modified CHD Knowledge Tool were examined to determine CVD knowledge in the study population at various levels, including: 48% (≥ 12 correct responses); 56% (≥ 14 correct responses); 64% (≥ 16 correct responses); 72% (≥ 18 correct responses); and 80% (≥ 20 correct responses).

Because women with histories of previous preeclampsia are considered as at-risk for recurring preeclampsia (Andersgaard et al., 2012; Barton & Sibai, 2008), independent samples t-tests were used to compare differences in levels of CVD knowledge among participants with and without histories of previous preeclampsia. Composite scores from the modified CHD Knowledge Test for Women were used to make the comparison.

2. Among women with recent, new-onset preeclampsia, what is the awareness of CVD as being the leading cause of death and healthcare problems in women? Item 25 of the CHDK Test for Women asks this question. Therefore, the dichotomized responses were evaluated to determine the percentage of women who were aware of CVD as being the leading cause of death and healthcare problems in women. In addition, frequencies of responses (true or false) were examined.
3. Among women with recent, new-onset preeclampsia, what is the baseline level of CVD risk perception? Summated scores from the CVD Risk Perception Scale were averaged to attain an average CVD risk perception score. A one-sample t-test was used to examine CVD risk perception and to determine if differences existed in mean

- scores for women with recent preeclampsia and other women examined using the same instrument as reported in previous studies. In addition, independent samples t-tests were conducted to evaluate differences in CVD risk perception among women with and without histories of previous preeclampsia.
4. Among women with recent, new-onset preeclampsia what is the relationship, at baseline, between CVD knowledge and CVD risk perception? A simple linear regression analysis was performed to examine CVD knowledge's influence on CVD risk perception.
 5. Among women with recent, new-onset preeclampsia what is the relationship, at baseline, among levels of CVD knowledge and perceptions of CVD risk and the variables of age, race, parity, marital status, previous preeclampsia, income, and education? Separate univariate regression analyses were performed to examine relationships between CVD knowledge and CVD risk and the study covariates: age, race, parity, marital status, previous preeclampsia, income, and education. A final model was constructed using covariates that had significant relationships with the dependent variable; multiple regression analyses were conducted to examine model fit.
 6. Among women with recent, new-onset preeclampsia, compared with baseline perceptions of CVD risk, is there a significant change in perception of CVD risk after receiving CVD education? A paired (dependent) t-test was used to compare mean scores of CVD risk perception before and after CVD education.

Missing Data

According to Tabachnick and Fidell (2007), the pattern of missing data is of more concern than the amount of missing data. Therefore, missing values scattered randomly throughout the data matrix posing a less serious problem. To decrease the potential of missing data, each questionnaire was checked for completeness. Although missing values did not pose a major problem in the present study, there was one participant who did not answer the CVD risk perception questions post-CVD education. Telephone connectivity was lost and the researcher was unable to reach the participant again to complete the questionnaire. Collecting data using telephone interviews possibly prevented excessive missing values; the primary investigator ensured that all questions were answered, to the extent possible, during data collection.

CHAPTER 4

STUDY RESULTS

In the present study, constructs from social cognitive theory served as a framework for exploring CVD knowledge and CVD risk perception among women with recent, new-onset, pure preeclampsia. In addition, the effects of CVD education, as an intervention, were examined. This chapter will describe results of the statistical analyses carried out to answer the study research questions. Descriptions of data cleaning/screening approaches, characteristics of the study participants, and results of analyses for each research question follow.

Data Screening/Cleaning

Prior to conducting statistical analyses, all data were inspected for accuracy by the principle investigator. Visual inspection of histograms, box plots, and Q-Q plots were used to assess normality of data distributions for the outcome variables. No apparent violations of the assumption were noted. In addition, tests of normality were conducted using the Shapiro-Wilk statistic. The results for the modified CHD Knowledge Tool for Women scale scores ($p = 0.099$), and the CVD Risk Perception Scale scores at baseline ($p = 0.086$) and post-intervention ($p = 0.053$) indicated that the assumption of normality for

these variables was tenable. Collinearity diagnostics were evaluated for the regression models; multicollinearity was not an issue in the present study.

Demographic Characteristics of Sample

There were a total of 64 participants in the study. Participants' age groups ranged from 19-24 years of age to greater than 40 years of age, with the majority of participants (45.3%) being in the 19-24 years of age group (see Table 5). Eighty-four percent of study participants were African American. Approximately one-third of participants were high school graduates; however, 13 participants (20%) were college graduates. Incomes of less than \$10,000 annually were reported by 55% of participants, and 61% of study participants were single and never married.

This study represented the first pregnancy for 41% of the sample; however 11% of participants reported their most recent pregnancy as being the fifth or more pregnancy. Although only seven participants (11%) reported histories of multiple births for the current or previous pregnancy, 13 of 38 multiparas (34%) reported histories of preeclampsia in pregnancies other than the most recent pregnancy (see Table 5).

Table 5

Demographic Characteristics of the Study Sample (N = 64)

Characteristic	Number	Percent
Age		
19-24	29	45.3
25-29	20	31.3
30-34	9	14.1
35-39	3	4.7
≥ 40	3	4.7
Race		
White/Caucasian	7	10.9
Black/African American	54	84.4
Hispanic	3	4.7
Parity		
1	26	40.6
2	15	23.4
3	9	14.1
4	7	10.9
5	5	7.8
> 5	2	3.1
Multiple Births		
No	57	89.1
Yes	7	10.9
Previous Preeclampsia		
No	25	39.1
Yes	13	20.3
N/A : First Pregnancy	26	40.6
Level of Education		
Less than High School	15	23.4
High School Graduate/GED	21	32.8
Some College	15	23.4
College Graduate	13	20.3

(Continued)

Characteristic	Number	Percent
Annual Household Income		
≤ \$10,000	35	54.7
\$10,001-\$20,000	14	21.9
\$20,001-\$30,000	5	7.8
\$30,001-\$40,000	5	7.8
> \$40,001	4	6.3
Declined to Answer	1	1.6
Marital Status		
Single/Never Married	39	60.9
Married	10	15.6
Divorced	4	6.3
Living with Significant Other	9	14.1
Widowed	2	3.1

Description of Main Study Outcome Variables

CVD knowledge and CVD risk perception were the primary variables of interest in the present study. Both the CHD Knowledge Tool for Women and the CVD Risk Perception Scale were modified for the present study to facilitate their use in telephone interviews. Table 6 provides descriptive statistics for the major study variables: CVD knowledge and CVD risk perception.

Table 6

Descriptive Statistics of Major Study Outcome Variables

Instrument	N	Mean	SD	Possible Range	Sample Range	Missing Values
CHD Knowledge Tool for Women	64	18.48	2.851	0 - 25	12 - 25	N/A
CVD Risk Perception (Pre-Education)	64	-.047	4.138	-9.00 – 9.00	-9.00 – 9.00	N/A
CVD Risk Perception (Post-Education)	63	2.37	4.437	-9.00 – 9.00	-9.00 – 9.00	1

Specific Research Aims and Research Questions

Aim 1

To examine baseline levels Of CVD knowledge and perceptions of CVD risk pre-CVD education.

Research Question One

Among women with recent, new-onset preeclampsia, what is the baseline level of CVD knowledge? Summated scores were used to obtain a mean score of true responses. The mean number of correct responses in the current study was 18.48 (73.92% correct responses), with a range of 12-25 (see Table 7). Thanavaro et al. (2010) administered the CHD Knowledge tool for women with a mean summated score of 17.6 (70.5% total correct answers) and considered the score as being low. In the present study, results from a one-sample t-test demonstrated that levels of CVD knowledge were significantly higher

($p = 0.018$) than mean total scores of CVD knowledge as reported by Thanavaro et al. In the present study, women responded correctly to 73.92% of items on the modified CHD Knowledge tool for women. Various levels of correct responses are reported in Table 8.

Table 7

Baseline Composite Scores of CVD Knowledge

N	Mean	Median	Mode	SD	Possible Range	Study Range
64	18.48	18.00	17	2.85	0-25	12-25

Table 8

CVD Knowledge Levels at Baseline

Number of Correct Responses	Percent of Items Answered Correctly (25 total items)	Response Rates for Correct Answers
≥ 20	80%	36 % ($n = 23$)
≥ 18	72%	56% ($n = 36$)
≥ 16	64%	86% ($n = 55$)
≥ 14	56%	97% ($n = 62$)
≥ 12	48%	100% ($n = 64$)

Because women with histories of previous preeclampsia are considered as at-risk for recurring preeclampsia (Barton & Sibai, 2008), an independent samples t-test was used to compare total (composite) CVD knowledge scores for women with and without histories of previous preeclampsia. Women with histories of previous preeclampsia

demonstrated no difference in CVD knowledge than women without histories of previous preeclampsia ($p=.81$) (see Table 9).

Table 9

CVD Knowledge in Women with and Without Previous Preeclampsia

Previous Preeclampsia	Mean	<i>t</i>	<i>p</i>-value
No ($n = 25$)	18.60	0.242	0.810
Yes ($n= 13$)	18.38		

Research Question Two

Among women with recent, new-onset preeclampsia, what is the awareness of CVD as being the leading cause of death and healthcare problems in women? Item 25 of the CHD Knowledge Tool for Women examined women's awareness of CVD as being the leading cause of death and healthcare problems among women. In the present study, 73% ($n = 47$) of women were aware of CVD as being the leading cause of death and healthcare problems among women; however 27% ($n = 17$) of women in the present study were not aware of CVD as being the leading cause of death and healthcare problems among women.

Research Question Three

Among women with recent preeclampsia, what is the baseline level of CVD risk perception? According to Schwarzer (2011), risk perception, or perceiving a health threat, represents the most obvious motivation for overcoming a risk behavior. The mean risk perception score for the current study was -0.5, on a scale of -3 to +3, with 0 representing average risk. In a previous study, Homko et al., (2008) reported a mean score of 0.35 for CVD risk perception using the same scale as used in the present study. The mean summated score of CVD risk perception was significantly lower ($p < 0.001$) than that reported by Homko et al. CVD risk perception was also evaluated among women with and without histories of previous CVD preeclampsia. Just as with CVD knowledge, there was no difference in levels of risk perception among women with and without histories of previous preeclampsia (See Tables 10 and 11).

Table 10

Baseline Composite Scores of CVD Risk Perception

N	Mean	Median	Mode	SD	Possible Range	Study Range
64	-0.05	0.00	-6.00	4.14	-9.00 - +9.00	-9.00 - +9.00

Table 11

CVD Risk Perception in Women with and Without Previous Preeclampsia

Previous Preeclampsia	Mean	<i>t</i>	<i>p</i>-value
No (<i>n</i> = 25)	-0.04	-0.189	0.851
Yes (<i>n</i> = 13)	0.23		

Research Question Four

Among women with recent, new-onset preeclampsia what is the relationship, at baseline, between CVD knowledge and CVD risk perception? In a regression analysis, CVD risk perception was regressed on CVD knowledge. CVD knowledge was found to be a significant ($p = 0.011$) predictor of CVD risk perception, accounting for 8.4% of variance ($R^2 = 0.084$).

Aim 2

To explore relationships between age, race, parity, marital status, previous preeclampsia, income, education, CVD knowledge, and perception of CVD risk.

Research Question Five

Among women with recent, new-onset preeclampsia what is the relationship, at baseline, among levels of CVD knowledge and perceptions of CVD risk and the variables: age, race, parity, marital status, previous preeclampsia, income, and education?

Separate univariate regression analyses were performed to determine the contribution of each study covariate for predicting levels of CVD risk perception and predicting CVD knowledge (see Tables 12 and 13). Covariates sharing a significant relationship with CVD knowledge or CVD risk perception (i.e., $p < .15$ in univariate analyses) were analyzed, as a set, in multiple regression analyses to examine the model fit.

Univariate regression analyses with CVD risk perception regressed on the study covariates demonstrated that none of the study covariates significantly influenced baseline levels of CVD risk perception. Univariate regression analyses of CVD knowledge regressed on the study covariates demonstrated significant relationships between CVD knowledge and age ($p = 0.010$); income ($p = 0.048$); and education ($p < 0.001$). As a set, age, education, and income significantly ($p = 0.001$) influenced CVD knowledge ($R^2 = 0.23$), although income became non-significant in the model with all three covariates. The final model, with age and education ($R^2 = .226$), is shown in Table 14.

Table 12

Univariate Analysis of CVD Risk Perception Regressed on Study Covariates (n=64)

	B	SE	β	p	F	R²	Adjusted R²
Age	.32	.475	.86	0.499	.464	.007	-.009
Recoded Race	-2.14	1.65	-.153	0.199	1.682	.026	.011
Parity	.059	.361	.021	0.870	.027	.000	-.016
Previous Preeclampsia	.25	1.061	.029	0.818	.053	.001	-.015
Recoded Education	1.04	1.04	0.13	0.324	.986	.016	.000
Annual Income	-0.80	0.43	-.02	0.853	.035	.001	-.016
Recoded Marital Status	1.32	1.06	.16	0.214	1.574	.025	.009

Table 13

Univariate Analysis of CVD Knowledge Regressed on Study Covariates (n = 64)

	B	SE	β	p	F	R²	Adjusted R²
Age*	.83	.31	.32	0.010	7.085	.103	.088
Recoded Race	-1.06	1.14	-.117	0.357	.860	.014	-.002
Parity	.13	.25	.07	0.603	.273	.004	-.012
Recoded Marital Status	1.04	.72	.18	0.155	2.074	.032	.017
Previous Preeclampsia**	-.22	.90	-.04	0.888	.020	.000	-.016
Income*	0.58	0.29	0.25	0.048	4.075	.063	.047
Recoded Education*	2.57	.65	.45	0.000	15.771	.203	.190

* $p < .05$ ** $n = 38$

Table 14

CVD Knowledge Regressed on Age and Education

	B	SE	β	<i>p</i>	<i>F</i>	R2	Adjusted R²
CVD Knowledge (constant)	13.982	1.037		0.000	7.04	.263	.226
Age in Years	.58	.31	0.222	0.067			
Education	2.388	.715	.415	0.001			

Aim 3

To provide a CVD education intervention and examine its effect on levels of personal perception of CVD risk post education.

Research Question Six

Among women with recent, new-onset preeclampsia, compared with baseline perceptions of CVD risk, is there a significant change in perception of CVD risk after receiving CVD education? A paired samples t-test was used to assess differences in CVD risk perception at baseline and post-CVD education. CVD risk perception was significantly different ($p = 0.003$) post-CVD education than at baseline demonstrating the effectiveness of CVD education, as an intervention for influencing CVD risk perception (see Table 15).

Table 15

Paired Differences of CVD Risk Perception at Baseline and Post-CVD Education

Paired Differences	Mean	N	SD	SE	95% CI Level of Difference		t	p- Value
					<i>Lower</i>	<i>Upper</i>		
	-2.3	63	4.42	0.72	-3.74	-0.84	-3.195	0.003

CHAPTER 5

DISCUSSION AND CONCLUSIONS

The overarching purpose of this study was to promote CVD knowledge and awareness among women with recent preeclampsia, through an education intervention, and to determine the effect of this education on accurate perceptions of personal CVD risk. This chapter will describe study results, study limitations, and implications for future research.

Results

Demographic Characteristics of the Study Sample

A total of 64 women with recent preeclampsia participated in the study. The sample was predominantly African-American (84%). Over three-fourths of the sample had at least a high school education and just over 20% were college graduates. Although approximately 44% of the sample had at least some college education, the annual incomes reported by participants placed a majority of these women below the poverty level. An annual household income of less than \$10,000 was reported by 55% of study participants. Only 14% of participants reported annual household incomes of \$30,000 or greater. Sixty-one percent of participants described themselves as single, never married.

In the present study, participants' age groups ranged from 19-24 years of age to 40 or greater years of age. As expected, the 19-24 year old age group represented the largest group among study participants (45.3%) and women 40 years of age or older accounted for approximately 5% of study participants. Participants were asked about the number of children they had given birth to, including the study pregnancy, and for many (41%), the most recent pregnancy was their first pregnancy. However, 11% of study participants ($n=7$) had given birth to five or more children, and 11% of study participants ($n=7$) had histories of multiple gestation pregnancies. According to Bilhartz et al. (2011), advanced maternal age, nulliparity, and multiple gestations each increase women's risks for developing preeclampsia. A history of previous preeclampsia also has been noted as a risk for recurrent preeclampsia (Andersgaard et al., 2012; Barton & Saibai, 2008). In the present study, 20% ($n = 13$) of study participants reported preeclampsia in previous pregnancies.

CVD Knowledge and CVD Risk Perception

At baseline, study participants demonstrated an overall low level of CVD knowledge; however, levels of CVD knowledge were significantly higher ($p = 0.018$) among this study population than levels of CVD knowledge reported in other populations of women where CVD knowledge was measured using the original instrument (Thanavaro et al., 2010). Several items on the modified CHD Knowledge Tool for Women were related to modifiable CVD risk factors. Participants demonstrated high

levels of knowledge relative to CVD modifiable risks such as: obesity, physical activity, smoking, and high-fat and cholesterol in the diet. However, participants demonstrated lower levels of understanding relative to how factors such as menopause and hormone therapy, alcohol consumption, and heredity relate to CVD (see Table 16). Questions relative to menopause and hormone therapy were the most problematic. Women in the present study demonstrated a lack of knowledge relative to how menopause and female hormones relate to CVD. A possible explanation for this is that this was a predominantly pre-menopausal sample. This finding underscores the need for increasing education and awareness among women relative to how female hormones and menopause affect CVD development (see Table 16).

Table 16

Item Responses from the CHDK Tool for Women

Item Number	Statement	True	False
1.	Heart disease, related to heart artery blockages, develops slowly over many years and can easily go undetected.	92%	8%
2.	Obesity may cause heart disease.	94%	6%
3.	Symptoms of heart pain or heart attack may include: chest pain; chest tightness; and unusual fatigue.	98%	2 %
4.	Women are more likely to get heart disease after menopause than before menopause.	27%	73 %
5.	African American women are more likely than White women to die from a heart attack or stroke.	67%	33%
6.	High cholesterol levels may cause heart artery blockages.	97%	3%

Item Number	Statement	True	False
7.	Symptoms of heart pain or heart attack may include: neck, shoulder, arm, or back pain, and dizziness.	83%	17%
8.	Once women are diagnosed or identified as having heart disease, they are more likely than men to become seriously ill or die.	41%	59%
9.	Stress may cause heart disease.	73%	27%
10.	There is no evidence that hormone therapy or hormone replacement prevents heart disease.	41%	59%
11.	High blood pressure may cause heart disease.	91%	9%
12.	Some forms of heart disease may result in stroke.	95%	5%
13.	A high fat diet may cause clogged heart arteries.	83%	17%
14.	Moderate alcohol use (1-2 drinks per day) may prevent heart disease.	34%	66%
15.	Smoking may cause heart artery blockages.	97%	3%
16.	Low levels of some female hormones may increase heart artery blockages in women.	59%	41%
17.	African American women are more likely to have heart disease than White women.	64%	36%
18.	Reducing dietary cholesterol may prevent clogged heart arteries.	89%	11%
19.	Routine exercise may prevent heart disease.	81%	19%
20.	Symptoms of heart pain or heart attack may include: shortness of breath; sweating; and nausea.	88%	12%
21.	Reducing dietary red meat may prevent heart artery blockages.	80%	20%

Item Number	Statement	True	False
22.	A family history of heart disease, from clogged heart arteries, may increase your risk of getting heart disease.	81%	19%
23.	Diabetes may increase the chance of having a heart attack.	72%	28%
24.	Heredity is a risk factor of heart disease, related to clogged heart arteries, that cannot be changed.	52%	48%
25.	Heart disease and stroke are the leading cause of health care problems and death in women.	73%	27%

Although 73% of participants in the present study were aware of heart disease and stroke as being the leading cause of death and healthcare problems among women, knowledge in this area still needs substantial improvement. According to Bandura (2004), knowledge is a requirement for health behavior change. One element influencing such behavior change is perception of risk.

Accurate perceptions of CVD risk have been associated with demonstrable behaviors suggestive of risk reduction. Thus accurate perceptions of CVD risks are necessary for health behavior to be aligned with measures to treat and prevent CVD (Christian et al., 2005). In the present study, CVD risk perception was regressed on CVD knowledge; CVD knowledge was a significant predictor ($p = 0.011$; $R^2 = 0.084$) of CVD risk perception. In this study, the sample as a whole perceived their risk of CVD as only average at baseline. Comparatively few women perceived their risk as being above that of

other women, in spite of the occurrence of preeclampsia in their most recent pregnancy. Although women with histories of preeclampsia are at-risk for recurrent preeclampsia and although preeclampsia has been identified as a risk factor for CVD (Andersgaard et al., 2012; Bellamy et al., 2007; Edlow et al., 2009; Kaaja & Greer, 2005; Harskamp et al., 2007; Lykke et al., 2009; Magnussen et al., 2009; Mongraw-Chaffin et al., 2010; Ness & Hubel, 2005; Smith et al., 2009; Van Pampas, 2008), in the present study, CVD knowledge ($p = 0.810$) and perceptions of CVD risk ($p = 0.851$) were not significantly different among women with and without histories of previous preeclampsia. This finding suggests that women with histories of previous preeclampsia may not be receiving appropriate counseling from their health care providers regarding their future CVD risk.

When CVD risk perception was regressed on CVD knowledge; CVD knowledge was a significant predictor ($p = 0.011$; $R^2 = 0.084$) of CVD risk perception. However, a substantial amount of the variability in risk perception remained unexplained by CVD knowledge. In separate univariate regression analyses, CVD knowledge and CVD risk perception were regressed on: age, race, education, income, marital status, histories of previous preeclampsia, and parity; none of the study covariates had a significant influence on CVD risk perception. However, age ($p = 0.010$), education ($p = 0.000$), and income ($p = 0.048$) were significant univariate predictors of CVD knowledge.

As a set, age and education were significant ($p = 0.001$; $R^2 = 0.226$) predictors of CVD knowledge, explaining almost 23% of the variability in that outcome. Participants who were older may have had more opportunities for education beyond high school and

higher levels of education may be positively associated with a higher general knowledge levels about CVD.

Effect of CVD Education Intervention

In the present study, CVD education, as an intervention, was provided to each study participant after baseline CVD knowledge and baseline CVD risk perception data were collected. CVD risk perception was measured again after the CVD education intervention to determine the influence of CVD education on CVD risk perception. Results of a paired-samples t-test demonstrated a significant difference ($p = 0.003$) in CVD risk perception post-CVD education when compared to CVD risk perception at baseline. Thus, CVD education, as an intervention, was effective in increasing levels of CVD risk perception among women with recent preeclampsia. According to Schwarzer (2011), risk perception or perceiving a health threat, represents the most obvious motivation for overcoming a risk behavior.

Discussion and Implications

Because preeclampsia is increasingly named as being a risk for future CVD in women, the present study examined women with recent preeclampsia, including its severe forms of severe preeclampsia and HELLP syndrome, to determine levels of CVD knowledge and perceptions of CVD risk among this population. According to researchers (Melchiorre et al., 2011), preeclampsia is associated with persistent cardiovascular

impairment. Based on results from a systematic review and meta-analysis, McDonald et al. (2008) established that women who experience preeclampsia have almost double the risk of future cardiac disease when compared to women who experience uncomplicated pregnancies. In addition, McDonald et al. determined that women who experience preeclampsia are at risk for early-onset cardiac disease.

Women who experience preeclampsia are found to be at higher risk for CVD development than women who have not experienced preeclampsia. Smith et al. (2009) determined that at one-year postpartum, when compared to women who have not experienced preeclampsia, women who experience preeclampsia have a two-to-three fold increased risk for cardiovascular disease events. In spite of these reports, in the present study, when comparing women with and without histories of previous preeclampsia, a difference in levels of both CVD knowledge and CVD risk perception was not found. This revelation demonstrates the need for CVD education among women who experience preeclampsia. Although many researchers (see Table 2) have recommended CVD education for women who experience preeclampsia, evidence that women with preeclampsia are being informed of their risks for future CVD is lacking. The present research represents one of the first efforts aimed at increasing CVD awareness among women who have experienced preeclampsia during pregnancy.

Social cognitive theory emphasizes knowledge as being integral to efforts aimed at health promotion and lifestyle behavior change. According to Bandura (2004), behavior change is implausible if lack of understanding exists for how lifestyle behaviors

affect health. In the present study, CVD knowledge shared a significant relationship with CVD risk perception. Although older age group, at least some college education, and higher income significantly predicted higher levels of CVD knowledge, none of the study covariates shared significant relationships with CVD risk perception. Nonetheless, providing women who experience preeclampsia with factual knowledge relative to their increased risk for CVD and approaches to reducing CVD risk would likely foster understanding and lead to more accurate personal perceptions of CVD risk.

The present study was aimed at providing a CVD education intervention as an effort promote CVD knowledge and awareness among women with recent preeclampsia. According to Dolmans et al. (2005), learning takes place, more readily, in contextual situations. Because participants in the present study recently experienced conditions strongly associated with CVD, their recent circumstantial experiences provided an opportunity to introduce CVD knowledge and promote awareness at a time that may likely alter lifestyle behaviors. Accurate perceptions of CVD risk are necessary so that lifestyle behaviors may be aligned with behaviors suggestive of CVD risk reduction (Christian et al., 2005).

The CVD education intervention in the present study centered on: healthful meal planning, physical activity, medication compliance (if indicated), and the importance of regular blood pressure and cholesterol screenings. In addition, symptoms of heart attack and factual information relative to heart disease in women were provided. According to Lloyd-Jones et al., (2010), dietary habits have the strongest evidence base of factors

contributing most to cardiovascular events, diabetes, and/or obesity. After CVD education, CVD risk perception was reexamined to determine if the CVD education intervention affected baseline levels of CVD risk perception. Bandura (2004) posits that in order for health communication to assist people with adopting health-promoting behaviors, it must be structured in a way that it provides the self-management knowledge, skills, and confidence necessary to take control of health behaviors. Results from the present study demonstrated that as an intervention, CVD education significantly influenced CVD risk perception from baseline to post-CVD education. Perception of CVD risk increased for this sample overall, with a higher proportion of women perceiving themselves at increased risk of CVD.

Schwarzer (2011) points out that initial risk perception is beneficial in that it helps people become motivated to change. Although CVD knowledge was not reassessed after the CVD education intervention, CVD education, as an intervention, was substantiated as an effective strategy for informing women with recent preeclampsia of their potential for future CVD development; practical approaches to ameliorating personal CVD risk profiles were provided as part of the CVD education intervention. According to Bandura (1994), factual information, combined with an extension of practical risk reduction approaches, creates good results; people learn and develop effective ways of behaving under realistic situations. To reinforce CVD education and to motivate participants to adopt lifestyle behavior changes consistent with CVD risk reduction, a brochure, which detailed CVD risk reduction guidelines for women, was mailed to each study participant.

Recommendations

Nursing Education

In order for women who experience preeclampsia to become aware of their risks for future CVD development and to become proactive in their efforts to reduce their personal risks for CVD, they must receive factual information which fosters understanding of the relationships between having preeclampsia during pregnancy and increased risk for future CVD development. Elements such as hypertension, obesity, lack of physical activity, smoking, high fat/cholesterol in the diet are essential components of CVD education. Because nurses are in close contact with women who experience preeclampsia, both during pregnancy and in the postpartum period, nurses are able to effectively communicate health promoting strategies to this population of women and provide patient education relative to fundamental elements associated with CVD risk reduction. The present study demonstrated that improvement in CVD knowledge is needed among this population of women.

Clinical Practice

Recent studies (Nijdam et al., 2009; Young et al., 2009) have determined follow-up for women with preeclampsia insufficient. In addition, knowledge of the association between future CVD and preeclampsia is said to be deficient among healthcare providers (Nijdam et al.; Young et al.). This report implies the need to healthcare providers to seek

further direction and guidance relative to the CVD/preeclampsia relationship so that they are able to appropriately inform women with preeclampsia of their risks for future CVD.

Dr. James N. Martin, Medical Director of the Maternal-Fetal Medicine Division of Obstetrics and Gynecology at University of Mississippi Medical Center described the study population as being a challenging population to reach in the postpartum period; many do not return for follow-up appointments due to factors such as lack of transportation and limited resources (personal communication, 2011). Bryant, et al. (2006) indicated that social factors, as well as inhibiting and enabling factors in the health care system were significant indicators of postpartum follow-up visits. However, according to Bryant et al., and Lewis et al., (2011), telephone-based interventions are effective approaches to reaching postpartum women. In the present study the telephone education intervention demonstrated usefulness as an approach for reaching women with recent preeclampsia and providing education about CVD risks and methods to reduce such risk. Use of telephone follow-up may be one strategy that could improve postpartum care for this high risk population. Special emphasis may be indicated for women who have experienced their first episode of preeclampsia to ensure appropriate medical follow up during any subsequent pregnancies.

Research

In spite of overwhelming evidence suggesting that women who experience preeclampsia are at increased risk for future CVD and although many researchers have

recommended informing women who experience preeclampsia of their increased CVD risk, evidence is lacking that women who experience preeclampsia are being informed of their increased risk for CVD. This research project represents one of the first such efforts aimed at informing women who have experienced preeclampsia of their increased risk for future CVD. In the present study, participants' intentions for adopting healthful lifestyle behaviors which promote CVD reduction were not examined, nor were specific questions about health behavior assessed. Further research studies aimed at examining motivation and intentions to adopt healthy lifestyle behaviors are needed. In addition, further studies are needed to examine whether improvements in CVD knowledge and risk perception promote subsequent CVD risk reduction behaviors.

Bandura (2004) points out that personal efficacy heavily influences behavior; people embrace efforts that they are able to accomplish, but relinquish those which are challenging. Self-efficacy was not examined in the present study. Therefore, future research which both informs women who experience preeclampsia of their risk and provides a structure which facilitates the ability to engage in risk-reduction behavior is needed.

Further studies are also needed to develop instruments to measure CVD knowledge in women. The inter-item correlations for the modified CHD Knowledge Tool for Women were generally quite low (less than .20 in many instances). Patterns of low inter-item correlations may occur when instrument items pertain to different fields of knowledge. For example, while the CHD Knowledge Tool for Women assesses

knowledge of known modifiable CVD risks (smoking, obesity, physical activity, diet, etc.), it also assesses other realms of CVD risks such as hormone therapy and menopause. Measuring CVD knowledge with an instrument that focuses on well-publicized risk factors such as hypertension, smoking, obesity, physical activity, etc. may produce an instrument containing fewer, but more homogenous items; thus, Cronbach's alpha would likely have been higher than seen in the current study ($\alpha = .56$).

Limitations

Although CVD education, as an intervention, proved useful for informing women with recent preeclampsia of their potential risk for future CVD, this study has several limitations, which are discussed below.

1. As noted above, the reliability of the study instrument used to assess CVD knowledge was low (Cronbach's alpha = .56).
2. The sample size in the present study was relatively small ($n = 64$), and the study was powered for a large effect size. Having a larger sample size may have provided more information relative to the study covariates contribution to CVD risk perception.
3. The cross-sectional design provided useful information as to participants' baseline CVD knowledge and CVD risk perception. While there was an increase in risk perception immediately following the educational intervention, it is not known whether this change in perception will be sustained over time. Studies with longer

- periods of follow-up are needed to evaluate changes in CVD knowledge and CVD risk perception over time.
4. Although the education intervention script was written at a comparatively low level of literacy (Flesh-Kincaid Grade Level = 7.5), many study participants voiced lack of understanding relative to items contained in the CHD Knowledge Tool for women. Therefore, in these instances, the education intervention script was altered so that simpler terms could be used and so that study participants could better understand what was being asked. Questions related to menopause and hormone therapy were the most problematic.
 5. Social cognitive theory served as a framework to guide the present study; however not all elements contained in the theory were tested. Because personal-efficacy (Bandura, 1989; Bandura, 2004) is considered as a major determinant of health behavior change, measuring self/personal-efficacy would have likely provided more valuable information relative to participants' beliefs in their abilities to carry out health-promoting lifestyle health behavior changes.
 6. Data collection and the CVD education intervention took place by telephone. According to Polit & Beck (2008), when the interviewer is unknown, participants may be uncooperative in answering questions; especially questions which are personal in nature. Thus, prior contact with participants or face-to-face communication during data collection and the education intervention would likely foster participants' levels of comfort in responding to questionnaire items. In

addition, face-to-face interviews would likely encourage participants to express misunderstandings during the CVD education intervention thereby providing an opportunity for the investigator to augment participants' understanding of CVD risk and risk reduction approaches; this is necessary for increasing CVD knowledge/awareness and developing accurate CVD risk perceptions.

Summary

The present study represented one of the first efforts aimed at informing women with recent preeclampsia of their risk for future CVD. As an intervention, CVD education was provided, by telephone, and served as a practical and effective approach to reaching women with recent preeclampsia. Results of the CVD risk perception scale from baseline to post-CVD education demonstrated CVD education, as an intervention, significantly increased perceptions of personal CVD risk among women with recent preeclampsia. Further studies are needed to determine whether educational interventions can lead to sustained increases in CVD knowledge and accurate perceptions of CVD risk.

References

- Agatisa, P. K., Ness, R. B., Roberts, J. M., Costantino, J. P., Kuller, L. H., & McLaughlin, M. K. (2004). Impairment of endothelial function in women with a history of preeclampsia: An indicator of cardiovascular risk. *American Journal of Physiology-Heart and Circulatory Physiology*, 286, H1389-H1393. doi: 10.1152/ajpheart.00298.2003
- Alladin, A. A., & Harrison, M. (2012). Preeclampsia: Systemic endothelial damage leading to increased activation of the blood coagulation cascade. *Journal of Biotech Research*, 4, 26-43. Retrieved from <http://www.btsjournals.com/Pages/JBR2012v4.aspx>
- American College of Obstetricians & Gynecologists [ACOG] (2001). ACOG practice bulletin no. 29: Chronic hypertension in pregnancy. *Obstetrics & Gynecology*, 98(1), 177-185. Retrieved from <http://mail.ny.acog.org/website/SMIPodcast/ChronicHypertension.pdf>
- American College of Obstetricians & Gynecologists [ACOG] (2002). ACOG practice bulletin no. 33: Diagnosis and management of preeclampsia and eclampsia. *Obstetrics & Gynecology*, 98(1), 159-167. Retrieved from <http://mail.ny.acog.org/website/SMIPodcast/DiagnosisMgt.pdf>
- Anderson, C. M., (2007). Preeclampsia: Exposing future cardiovascular risk in mothers and their children. *Journal of Obstetric, Gynecologic, & Neonatal Nursing*, 36, 3-8. doi: 10.1111/j.1552.6909.2006.00115x
- Andersgaard, A. B., Acharya, G., Mathiesen, E. B., Johnsen, S. H., Straume, B., & Øian, P. (2012). Recurrence and long-term maternal health risks of hypertensive disorders of pregnancy: A population-based study. *American Journal of Obstetrics & Gynecology*, 206, 143e1-8. doi: 10.1061/j.ajog.2011.09.032
- Bandura, A. (1989). Human agency in social cognitive theory. *American Psychologist*, 44(9), 1174-1185. doi: 10.1.1.152.23[2].pdf
- Bandura, A. (1994). Social cognitive theory and exercise of control over HIV infection. In R. J. DiClemente & J. L. Peterson (Eds.), *Preventing AIDS: Theories and methods of behavioral interventions* (pp. 25-99). New York: Plenum

- Bandura, A. (2004). Health promotion by social cognitive means. *Health Education and Behavior*, 31, 143-164. doi: 10.1144/1090198104263660
- Bandura, A. (2005). The primacy of self-regulation in health promotion. *Applied Psychology* 54(2), 245-254. doi: 10.1111/j.1464-0597.2005.00208.x.
- Barton, J. R., & Sibai, B. M. (2008). Prediction and prevention of recurrent preeclampsia. *Obstetrics & Gynecology*, 112(2), 359-372. doi: 10.1097/AOG.0B013E318101d56
- Bauer, S. T., & Cleary, K. L. (2009). Cardiopulmonary complications of pre-eclampsia. *Seminars in Perinatology*, 33, 158-165. doi: 10.1053/j.semperi.2009.02.08
- Bellamy, L., Casas, J. P., Hingorani, A. D., & Williams, D. J. (2007). Pre-eclampsia and risk of cardiovascular disease and cancer in later life: Systematic review and meta-analysis. *British Medical Journal*, 355(7627). doi: 10.1136/bmj.39335.385301BE
- Berends, A. L., de Groot, C. J. M., Sijbrands, E. J., Sie, M. P. S., Benneheij, S. H., Pal, P., Heydanus, R.,... Steegers, E. A. P. (2008). Shared constitutional risks for maternal vascular-related pregnancy complications and future cardiovascular disease. *Hypertension*, 51, 1034-1041. doi: 10.1161/HYPERTENSIONAHA.107.101873
- Berks, D., Steegers, E. A.P., Molas, M., & Visser, W. (2009). Resolution of hypertension and proteinuria after preeclampsia. *Obstetrics & Gynecology*, 114(6), 1307-1314. doi: 10.1097/AOG.0b013e3181c14e3e
- Bhattacharya, S., Prescott, G. J., Iversen, L., Campbell, D. M., Smith, W. C. S., & Hannaford, P. C. (2011). Hypertensive disorders of pregnancy and future health and mortality: A record linkage. *Pregnancy Hypertension: An International Journal of Women's Cardiovascular Health*, 2, 1-7. doi: 10.1016/j.preghy.2011.08.116
- Bilhartz, T. D., Bilhartz, P. A., Bilhartz, T. N., & Bilhartz, R. D. (2011). Making use of a natural stress test: Pregnancy and cardiovascular risk *Journal of Women's Health*, 20(5), 695-701. doi: 10.1089/jwh.2010.2291
- Brown, D. W., Ducker, N., Jamieson, D. J., Cole, J. W., Wozniak, M. A., Stern, B. J.,... & Kittner, S. J. (2006). Preeclampsia and risk of ischemic stroke among young women: Results from the stroke prevention in young women study. *Stroke*, 37, 1055-1059. doi: 10.1161/01.STR.0000206284.96739.ee

- Bryant, A. S., Haas, J. S., McElrath, T. F., & McCormick, M. C. (2006). Predictors of compliance with the postpartum visit among women living in healthy start project areas. *Maternal & Child Health Journal, 10*, 511-516. doi: 10.1007/s10995-006-0128-5
- Burns, N. & Grove, S. K. (2009). *The practice of nursing research: Appraisal, synthesis, and generation of evidence*. St. Louis: Saunders
- Craici, I., Wagner, S., & Garovic, V.D. (2008). Review: Preeclampsia and future risk of cardiovascular disease: Formal risk factor or failed stress test. *Therapeutic Advances in Cardiovascular Disease, 2*(4), 249-259. doi: 10.1177/1753944708094227
- Catov, J. M., Newman, A. B., Sutton-Tyrrell, K., Harris, T. B., Tylavsky, F., Visser, M., ...Ness, R. B. (2008). Parity and CVD risk among older women: How do pregnancy complications mediate the association? *Annals of Epidemiology, 18*, 873-879. doi:10.1016/j.annepidem.2008.09.009
- Centers for Disease Control and Prevention (2010). *Heart disease factsheet*. Retrieved from http://www.cdc.gov/dhdsdp/data_statistics/fact_sheets/fs_heart_disease.htm
- Chambers, J. C., Fusi, L., Malik, I. S., Haskard, D. O., De Swiet, M., & Kooner, J. S. (2001). Association of maternal endothelial dysfunction with preeclampsia. *The Journal of the American Medical Association, 285*(12), 1607-1612. doi: 10.1001/jama.285.12.1607
- Chesley, L. C., (1980). Hypertension in pregnancy: Definitions, familial factor, and remote prognosis. *Kidney International, 18*, 234-240. Retrieved from <http://www.nature.com/ki/journal/v18/n2/pdf/ki1980124a.pdf>
- Christian, A. H., Mochari, H. Y., & Mosca, L. (2005). Coronary heart disease in ethnically diverse women: Risk perception and communication. *Mayo Clinical Proceedings, 80*(12), 1593-1599. doi: 10.4065/80.12.1593
- 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*(1), 68-81. doi: 10.1089/jwh.2006.M072
- Cincotta, R. B., & Brennecke, S. P. (1998). Family history of pre-eclampsia as a predictor for pre-eclampsia in primigravidas. *International Journal of Gynecology and Obstetrics, 60*(1), 23-27. doi: 10.1016/S0020-7292(97)00241-5

- Cohen, J. (1992). A power primer. *Psychological Bulletin*, 112(1), 155-159. doi: 10.1037/0033-2909.1121.155
- Diehl, C. L., Brost, B. C., Hogan, M. C., Elesber, A. A., Offord, K. P., Turner, S. T., Garovic, V. D. (2008). Preeclampsia as a risk factor for cardiovascular disease later in life: Validation of a preeclampsia questionnaire. *American Journal of Obstetrics & Gynecology*, 108(5), e11-e13. doi: 10.1013/j.ajog.2007.09.038
- Dolmans, D. H. J. M., De Grave, W., Wolfhagen, I. H. A. P., & Van der Vleuten, C. P. M. (2005). Problem-based learning: future challenges for educational practice and research. *Medical Education*, 39(7), 732-741. doi: 10.1111/j.1365-2929.2005.02205.x
- Edlow, A. G., Srinivas, S. K., & Elovitz, M. A. (2009). Investigating the risk of hypertension shortly after pregnancies complicated by preeclampsia. *American Journal of Obstetrics & Gynecology*, 200(5), e60-62. doi: 10.1016/j.ajog.2008.10.012
- Emslie, C. (2005). Women, men and coronary disease: A review of the quantitative literature. *Journal of Advanced Nursing*, 51, 382-395. doi: 10.1111/j.1365-2648.2005.03509.x
- Evans, C. S., Gooch, L., Flotta, D., Powers, R. W., Landsittel, D., Roberts, J. M., & Shroff, S. G. (2011). Cardiovascular system during the postpartum state in women with a history of preeclampsia. *Hypertension*, 58, 57-62. doi: 10.1161/HYPERTENSIONAHA.111.193278
- Firoz, T., & Melnik, T. (2011). Postpartum evaluation and long term implications. *Best Practice & Research Clinical Obstetrics and Gynecology*, 25, 549-561. doi: 10.1016/j.bpogyn.2011.03.003
- Garovic, V. D., & Hayman, S. R. (2007). Hypertension in pregnancy: An emerging risk factor for CVD. *Nature Clinical Practice Nephrology*, 3, 316-622. doi: 10.1038/ncpneph0623
- Gazlano T. A. (2007). Reducing the growing burden of CVD in the growing world. *Health Affairs*, 26(1), 13-24. doi: 10.3777/hlthaff.26.1.13
- Gholizadeh, L., Davidson, P., Salamonson, Y., & Worrall-Carter, L. (2010). Theoretical considerations in reducing risk for cardiovascular disease: Implications for nursing practice. *Journal of Clinical Nursing*, 19, 2137-2145. doi: 10.1111/j.1365.2702.2009.03189.x

- Habli, M., Eftekhari, N., Wiebrach, E., Bombrys, A., Khabbaz, M., How, H., & Sibai, B. (2009). Long-term maternal and subsequent pregnancy outcome 5 years after hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome. *American Journal of Obstetrics & Gynecology*, *201*, 385e1-5. doi: 10.1016/j.ajog.2009.06.033
- Harskamp, R. E., & Zeeman, G. G. (2007). Preeclampsia: At risk for remote cardiovascular disease. *The American Journal of Medical Sciences*, *334*(4), 291-295. doi: 10.1097/MAJ.0b013e3180a6f094
- Hart, P. L. (2005). Women's perceptions of coronary heart disease. An integrative review. *Journal of Cardiovascular Nursing*, *20*(3), 170-176.
- Haukkamaa, L., Salminen, M., Laivuori, H., Leinonen, H., Hiilesmaa, V., & Kaaja, R. (2004). Risk for subsequent coronary artery disease after preeclampsia. *The American Journal of Cardiology*, *93*(6), 805-808. doi: 10.1016/j.amjcard.2003.11.065
- Hauth, J. C., Clifton, R. G., Roberts, J. M., Myatt, L., Spong, C. Y., Leveno, K. J.,...Anderson, G. D. (2011). Maternal insulin resistance and preeclampsia. *American Journal of Obstetrics & Gynecology*, *204*, 327e1-6. doi: 10.1016/j.ajog.2011.02.024
- Hoedjes, M., Berks, D., Vogel, I., Franx, A., Duvekot, J. J., Oenema, A.,...Raat, H. (2012). Motivators and barriers to a healthy postpartum lifestyle in women at increased cardiovascular and metabolic risk: A focus-group study. *Hypertension in Pregnancy*, *31*, 147-155. doi: 10.3109/10641955.2010.544803
- Homko, C. J., Santamore, W. P., Zamora, L., Shirk, G., Gaughan, J., Cross, R., Kashem, A.,...Bove, A. A. (2008). Cardiovascular disease knowledge and risk perception among underserved individuals at increased risk of cardiovascular disease. *Journal of Cardiovascular Nursing*, *23* (4), 332-337. doi: 10.1097/01.JCN.0000317432.44586.aa
- Irgens, H. U., Reisaeter, L., Irgens, L. M., & Lie, R. T. (2001). Long-term mortality of mothers and fathers after pre-eclampsia: Population based cohort study. *British Medical Journal*, *323*, 1213-1217. doi: 10.1136/bmj.323.7323.1213
- Institute of Medicine. (2010). *Promoting cardiovascular health in the developing world: A critical challenge to achieve global health*. Washington, D. C.: The National Academic Press

- Jahromi, B. N., & Husseini, Z. (2008). Pregnancy outcome at maternal age 40 and older. *Taiwanese Journal of Obstetrics & Gynecology*, 47(3), 318-321. doi: 10.1016/S1028-4559(08)60131-X
- Kaaja, R. J., Greer, I. A., (2005). Manifestations of chronic disease during pregnancy. *Journal of the American Medical Association*, 294(21), 2751-2757. doi: 10.1001/jama.294.21.2571
- Kvehaugen, A. S., Andersen, L. F., & Staff, C. A. (2010). Dietary intake and physical activity in women and offspring after pregnancies complicated by preeclampsia or diabetes mellitus. *Obstetricia et Gynecologica Scandinavica*, 89(11), 1486-1490. doi: 10.3019/00016349.2010.519378
- Lewis, B. A., Martinson, B. C., Sherwood, N. E., & Avery, M. D. (2011). A pilot study evaluating a telephone-based exercise intervention for pregnant and postpartum women. *Journal of Midwifery & Women's Health*, 56(2), 127-131. doi: 10.1111/j.1542-2011.2010.00016.x
- Lloyd-Jones, D., Hong, Y., Labarthe, D., Mozaffarian, D., Appel, L. J., Van Horn, L., Greenlund, K., ...Roasmond, W. D. (2010). Defining and setting national goals for cardiovascular health promotion and disease reduction: The American Heart Association's strategic impact goal through 2020 and beyond. *Circulation*, 121, 586-613. doi: 10.1161/CIRCULATIONAHA.109.192703
- Lykke, J. A., Langhoff-Ross, J., Lockwood, C. J., Triche, E. W., & Paidas, M. J. (2010). Mortality of mothers from cardiovascular and non-cardiovascular causes following pregnancy complications in first delivery. *Paediatric and Perinatal Epidemiology*, 24, 323-330. doi: 10.1111/j.1365-3016.2010.001120x
- Magnussen, E. B., Vatten, L. J., Lund-Nilsen, T. I., Salvesen, K. A., Smith, G. D., & Romundstad, P. R. (2007). Prepregnancy cardiovascular risk factors as predictors of pre-eclampsia: Population based cohort study. *British Medical Journal*, 355(7627). doi: 10.1136/bmj.39366.416817.BE
- Magnussen, E. B., Vatten, L. J., Smith, G. D., & Romundstad, P. R. (2009). Hypertensive disorders in pregnancy and subsequently measured cardiovascular risk factors. *Obstetrics & Gynecology*, 114(5), 961-970. doi: 10.1097/AOG.0b013e3181bb0dfc
- Mangos, G. J., Spann, J. J., Pirabhahar, S., & Brown, M. A. (2012). Markers of cardiovascular disease risk after hypertension in pregnancy. *Journal of Hypertension*, 30, 351-358. doi: 10.1097/HJH.0b013e32834e5ac7

- Mankuta, D., Elami-Suzin, M., Elhayani, A., & Vinker, S. (2010). Lipid profile in consecutive pregnancies. *Lipids in Health and Disease*, 9(58), 1-4. doi: 10.1186/1476-511X-9-58
- Martin, J. N. (2012). *HELLP Syndrome* (ID 1000). Retrieved from [https://online.epocrates.com/search/search.jsp?lang=en&query=HELLP Syndrome-Martin](https://online.epocrates.com/search/search.jsp?lang=en&query=HELLP+Syndrome-Martin)
- Martin, J. N., Rose, C. H., & Briery, C. M. (2006). Understanding and managing HELLP syndrome: The integral role of aggressive glucocorticoids for the mother and child. *American Journal of Obstetrics & Gynecology*, 195, 914-934. doi: 10.1016/j.ajog.2005.08.044
- Maynard, S. E., Min, J. Y., Merchan, J., Lim, K. H., Li, J., Mondal, S.,... Karumanchi, V. P. (2003). Excess placental soluble fms-like tyrosine kinase 1 (sFlt1) may contribute to endothelial dysfunction, hypertension, and proteinuria in preeclampsia. *The Journal of Clinical Investigation*, 111(5), 649-658. doi: 10.1172/JC1200317189.
- McDonald, S. D., Malinowski, A., Zhou, Q., Yusuf, S., Devereaux, P. J., (2008). Cardiovascular sequelae of preeclampsia/eclampsia: A systematic review and meta-analyses. *American Heart Journal*, 156(5), 918-930. doi:10.1016/j.ahj.2008.06.042
- Melchiorre, K., Sutherland, G. R., Liberati, M. & Thilaganathan, B. (2011). Preeclampsia is associated with persistent postpartum cardiovascular impairment. *Hypertension*, 58(4), 709-715. doi: 0.1161/HYPERTENSIONAHA.111.176537
- Mongraw-Chaffin, M. L., Cirillo, P. M., Cohn, B. A. (2010). Preeclampsia and cardiovascular disease death: Prospective evidence from the child health and development studies cohort. *Hypertension*, 56, 166-171. doi: 10.1161/HYPERTENSIONAHA.110.150078
- Mosca, L., Mochari-Greenberger, H., Dolor, R. J., Newby, L. K., & Robb, K. J. (2010). Twelve-year follow-up of American women's awareness of cardiovascular disease risk and barriers to heart health. *Circulation Cardiovascular Quality and Outcomes*, 3, 120-127. doi: 10.1161/CIRCOUTCOMES.109.915538

- Mosca, L., Benjamin, E. J., Berra, K., Bezanson, J. L., Dolor, R. J., Lloyd-Jones, D. M., ... & Wenger, N. K. (2011). Effectiveness-based guidelines for the prevention of cardiovascular disease in women-2011 update: A guideline from the American Heart Association. *Circulation, 123*, 1243-1262. doi: 10.1161/CIR.0b013e31820faaf8
- Nawathe, A. C., Glied, S. A., Weintraub, W. S., & Mosca, L. J. (2010). The effects of cardiovascular educational intervention on healthcare utilization and costs. *The American Journal of Managed Care, 16*(5), 339-346.
- Ness, R. B., & Hubel, C. A. (2005). Risk for coronary artery disease and morbid preeclampsia: A commentary. *Annals of Epidemiology, 15*, 726-733. doi: 10.1016/j.annepidem.2005.02.007
- Newstead, J., von Dadelszen, P., & Magee, L. (2007). Preeclampsia and future cardiovascular risk. *Expert Review of Cardiovascular Therapy, 5*(2), 283-294. doi: 10.1586/14779072.5.2.283
- Nijdam, M. E., Timmerman, A. R., Franx, A., Bruinse, H. W., Numans, M. E., Grobbee, D. E., & Bots, M. L. (2009). Cardiovascular risk factor assessment after pre-eclampsia in primary care. *BioMed Central Family Practice, 10*(77). doi: 10.1186/1471-2296-10/77
- Polit, D. F., & Beck, C. T. (2008). *Nursing research: Generating and assessing evidence for nursing practice*. St. Louis: Lippincott, Williams, and Wilkins
- Ray, J. G., Vermeulen, M. J., Schull, M. J., & Redelmeier, D. A. (2005). Cardiovascular health after maternal placental syndromes (CHAMPS): Population-based retrospective cohort study. *Lancet, 366*, 1797-1803. doi: 10.1016/S0140-6736(05)67726-4
- Redding, C. A., Rossi, J. S., Rossi, S. R., Velicer, W. F., & Prochaska, J. O. (2000). Health behavior models (Special issue). *The International Electronic Journal of Health Education, 3*, 180-193. Retrieved from <http://www.iejhe.siu.edu>
- Rich-Edwards, J. W. (2012). The predictive pregnancy: What complicated pregnancies tell us about mother's future cardiovascular risk. *Circulation, 125*, 1336-1338. doi: 10.1161/CIRCULATIONAHA.112.093872
- Roberts, J. M. & Catov, J. M. (2012). Pregnancy is a screening test for later life cardiovascular disease: Now what? *Women's Health Issues, 22*(2), e123-e128. doi:10.1016/j.whi.2012.01.001

- Roberts, J. M., & Hubel, C. A. (2010). Pregnancy: A screening test for later life cardiovascular disease. *Women's Health Issues, 20*(5), 304-307. doi: 10.1016/j.whi.2010.05.004
- Robbins, C. L., Dietz, P. M., Bombard, J., & Valderrama, A. L. (2011). Gestational hypertension: Cardiovascular disease risk marker. *American Journal of Obstetrics & Gynecology, 204*, 336e1-9. doi: 10.1016/j.ajog.2010.11.005
- Rodie, V. A., Freeman, D. J., Sattar, N., & Greer, I. A., (2004). Pre-eclampsia and cardiovascular disease: Metabolic syndrome of pregnancy. *Atherosclerosis, 175*, 189-202. doi: 10.1016/j.atherosclerosis.2004.01.038
- Roes, E. M., Sieben, R., Raijmakers, M. T. M., Peters, W. H. M., & Steegers, E. A. P. (2005). Severe preeclampsia is associated with a positive family history of hypertension and hypercholesterolemia (2005). *Hypertension in Pregnancy, 24*, 259-271. doi: 10.1080/10641950500281076
- Romundstad, P. R., Magnussen, E., B., Smith, G. D., & Vatten, L. J. (2010). Hypertension in pregnancy and later cardiovascular risk: Common antecedents? *Circulation, 122*, 579-584. doi: 10.1161/CIRCULATIONAHA.110.943407
- Samwiil, L., Mercer, C., Jarrett, P., & O'Malley, S. (2004). Blood pressure and urinalysis are often omitted in women who have suffered pre-eclampsia at their six-week postnatal check. *BJOG: An International Journal of Obstetrics and Gynecology, 111*, 623-625. doi: 10.1111/j.1471-0528.2004.00136.x
- Schwarzer, R. & Renner, B. (2000). Social-cognitive predictors of health behaviors: Action self-efficacy and coping self-efficacy. *Health Psychology, 19*. 487-495. doi: 10.1037//0278-6133.19.5.487
- Schwarzer, R. (2011). Health behavior change. In H. S. Friedman (Ed.), *Oxford handbook of health psychology* (pp. 591-611). New York: Oxford University Press
- Silva, L. M., Coolman, M., Steegers, E. A. P., Jaddoe, V. W. V., Moll, H. A., Hofman, A.,... Raat, H. (2008). Low socioeconomic status is a risk factor for preeclampsia: The generation R study. *Journal of Hypertension, 26*, 1200-1208. doi:10.1097/HJH.0b013e3282fcc36e

- Smith, G. K., Walker, M. C., Liu, A., Wen, S. W., Swansburg, M., Ramshaw, H.,... Hladunewich, M., (2009). A history of preeclampsia identifies women who have underlying cardiovascular risk factors. *American Journal of Obstetrics & Gynecology*, 200(1), 58.e1-58.e8. doi: 10.1016/j.ajog.2008.06.035
- Strecher, V. J., DeVellis, B. M., Becker, M. H., & Rosenstock, I.M. (1986). The role of self-efficacy in achieving health behavior change. *Health Education Quarterly*, 13(1), 73-92. doi: 10.1177/109019818601300108
- Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics*. Boston, MA: Pearson
- Thanavaro, J. L., Thanavaro, S., & Delicath, T. (2008). Coronary heart disease knowledge tool for women. *Journal of the American Academy of Nurse Practitioners*, 22(2), 62-69. doi:10.1111/j.1745-7599.2009.00476.x
- Thanavaro, J. L., Thanavaro, S., & Delicath, T. (2010). Health promotion behaviors in women with chest pains. *Heart & Lung*, 39(5), 394-403. doi: 10.1016/j.hrtlng.2009.10.016
- United States Department of Health and Human Services. (2010). *Healthy People 2020 framework*. Retrieved from <http://www.healthypeople.gov/2020/consortium/HP2020Framework.pdf>
- Valdés, G., Quezada, F., Marchant, E., von Schultendorff, A., Morán, S., Padilla, O., & Martínez, A. (2008). Association of remote hypertension in pregnancy with coronary artery disease: A case control study. *Hypertension*, 53, 733-738. doi: 10.1161/HYPERTENSIONAHA.108.12068
- Valdiviezo, C., Garovic, V. D., & Ouyang, P. (2012). Preeclampsia and hypertensive disease in pregnancy: Their contributions to cardiovascular risk. *Clinical Cardiology*, 35(3), 160-165. doi:10.1002/clc.21965
- van Pampus, M. (2005). Long-term follow-up after pre-eclampsia/HELLP syndrome. *International Congress Series*, 1279, 273-277. doi: 10.1016/j.ics.2005.001.008
- Wen, S. W., Xie, R. H., Tan, H., Walker, M. C., Smith, G. N., & Retnakaran, R. (2012). Preeclampsia and gestational diabetes mellitus: Pre-conception origins? *Medical Hypotheses*, 79(1), 120-125. doi: 10.1016/j.mehy.2012.04.019

- Wikström, A. K., Haglund, B., Olovsson, M., Lindeberg, S. N. (2005). The risk of maternal ischemic heart disease after gestational hypertensive disease. *BJOG: An International Journal of Obstetrics and Gynecology*, *12*, 1486-1491. doi: 10.1111/j.1471-0528.2005.00733.x
- Wolf, M., Hubel, C. A., Lam, C., Sampson, M., Ecker, J. L., Ness, R. B.,... Thadhani, R. (2004). Preeclampsia and future cardiovascular disease: Potential role of altered angiogenesis and insulin resistance. *The Journal of Clinical Endocrinology & Metabolism*, *89*(12), 6239-6243. doi: 10.1210/jc.2004-0548
- World Health Organization (2009). *Cardiovascular diseases*. (Fact Sheet No. 317). Retrieved from <http://www.who.int/mediacentre/factsheets/fs317/en/index.html>
- Yinon, Y., Kingdom, J. C. P., Odutayo, A., Moineddin, R., Drewol, S., Lai, V.,... Hladunewich, M. A. (2010). Vascular dysfunction in women with a history of preeclampsia and intrauterine growth restriction: Insights into future vascular risk. *Circulation*, *122*, 1846-1853. doi: 10.1161/CIRCULATIONAHA.110.948455
- Yoder, S. R., Thornburg, L. L., & Bisognano, J. D. (2009). Hypertension in pregnancy and women of child-bearing age. *The American Journal of Medicine*, *122*, 890-895. doi:10.1016/j.amjmed.2009.03.036
- Young, B., Hacker, M. R., & Rana, S. (2012). Physician's knowledge of future vascular disease in women with preeclampsia. *Hypertension in Pregnancy*, *31*, 50-58. doi:10.3109/10641955.2010.544955

APPENDIX A
IRB APPROVAL LETTER



Institutional Review Board for Human Use

Form 4: IRB Approval Form
Identification and Certification of Research
Projects Involving Human Subjects

UAB's Institutional Review Boards for Human Use (IRBs) have an approved Federalwide Assurance with the Office for Human Research Protections (OHRP). The Assurance number is FWA00005960 and it expires on August 29, 2016. The UAB IRBs are also in compliance with 21 CFR Parts 50 and 56.

Principal Investigator: SPRATLING, PATSY

Co-Investigator(s):

Protocol Number: **X111223008**

Protocol Title: *CVD Knowledge and Risk Perception Among Women with Recent Preeclampsia: Interventional Education in Disease Management and Prevention*

The IRB reviewed and approved the above named project on 2-10-12. The review was conducted in accordance with UAB's Assurance of Compliance approved by the Department of Health and Human Services. This Project will be subject to Annual continuing review as provided in that Assurance.

This project received EXPEDITED review.

IRB Approval Date: 2-10-12

Date IRB Approval Issued: 2-10-12

Marilyn Doss, M.A.
Vice Chair of the Institutional Review
Board for Human Use (IRB)

Investigators please note:

The IRB approved consent form used in the study must contain the IRB approval date and expiration date.

IRB approval is given for one year unless otherwise noted. For projects subject to annual review research activities may not continue past the one year anniversary of the IRB approval date.

Any modifications in the study methodology, protocol and/or consent form must be submitted for review and approval to the IRB prior to implementation.

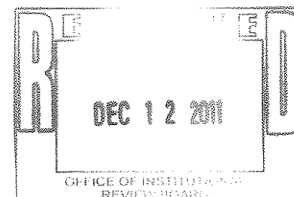
Adverse Events and/or unanticipated risks to subjects or others at UAB or other participating institutions must be reported promptly to the IRB.

470 Administration Building
701 20th Street South
205.934.3789
Fax 205.934.1301
irb@uab.edu

The University of
Alabama at Birmingham
Mailing Address:
AB 470
1530 3RD AVE S
BIRMINGHAM AL 35294-0104

APPENDIX B
LETTER OF SUPPORT

THE UNIVERSITY OF MISSISSIPPI MEDICAL CENTER
2500 North State Street
JACKSON, MISSISSIPPI 39216-4505



School of Medicine
Department of Obstetrics and Gynecology
Division of Maternal-Fetal Medicine

Area Code 601
984-5358

December 6, 2011

The University of Alabama at Birmingham
IRB
1530 3rd Avenue South
Birmingham, Alabama 35294-1150

Re: Research Project with Patsy Spratling, RN MSN
Doctoral Student at University of Alabama at Birmingham

To Whom It May Concern:

I will be working with Ms. Patsy Spratling on her research project entitled "CVD Knowledge and Risk Perception Among Women with Recent Preeclampsia: Interventional Education in Disease Management and Prevention". This study is being submitted to the IRB at the University of Mississippi Medical Center for its approval. The doctoral student will be working with myself and other members of the clinical research staff of the University of Mississippi Dept OBGYN MFM Division to identify and collect patients for this endeavor. Once approved by the IRB at UMMC, Ms. Spratling will contact patients by telephone who have been identified as having just delivered with preeclampsia. Research candidates will have agreed to talk to Ms. Spratling before she calls them postpartum, using the telephone number the patient provides for this purpose. The data she collects will be turned over to her doctoral nursing program there in Birmingham. I agree to assist in all aspects of this endeavor.

Yours sincerely,

A handwritten signature in black ink, appearing to read "James N. Martin, Jr.", written over a horizontal line.

James N. Martin, Jr., M.D.
Professor, OBGYN
Director, Division of Maternal-Fetal Medicine
President, ACOG 2011-2012

**University of Mississippi Medical Center
Institutional Sanction**

From: James N. Martin <jnmartin@umc.edu>
To: patsy spratling <pspratli@bellsouth.net>
Cc: Laura K. Bufkin <lbufkin@umc.edu>
Sent: Tue, February 7, 2012 1:05:37 PM
Subject: FW: 2011-0278 Initial Application

Per the IRB chief, we can proceed without formal review and approval of the convened IRB. Laura is back half-time this week and full-time thereafter, ready to set this up to start as soon as possible. I assume that this will be satisfactory with the IRB. I will maintain this set of communications to verify the recommendation to proceed without formal IRB approval but institutional sanction in place. JNM

From: Nancy A Olson
Sent: Tuesday, February 07, 2012 9:03 AM
To: James N. Martin
Subject: RE: 2011-0278 Initial Application

Dr. Martin –

Since all you are doing is informing prospective participants about the availability of the research, obtaining their permission for Ms. Spratling to contact them and sending her the contact information, and you will destroy the information after it is sent, UMMC is not considered to be engaged in the research and the study does not require IRB review here. The easiest thing for you to do is to log into Ideate and withdraw the submission. That will close the loop within our system and ensure that you do not receive reminders for it. Thank you for your help sorting this out. Please let me know if you have questions or need additional information.

Nancy

Nancy A. Olson, J.D.

Director

Human Research Office/Institutional Review Boards

University of Mississippi Medical Center

APPENDIX C
STUDY RECRUITMENT MATERIALS

Recruitment Flyer

COLLABORATIVE RESEARCH STUDY: University of Mississippi Medical Center, Jackson and University of Alabama at Birmingham

Researcher: Patsy Spratling, MSN, RN, lives in the Jackson, MS area and is a doctoral student at the University of Alabama at Birmingham. She will be conducting a study at the University of Mississippi Medical Center, in the Department of Obstetrics and Gynecology, starting in February 2012.

Director: Dr. James N. Martin, Jr. is the Director of Maternal-Fetal Medicine at the Wiser Hospital for Women & Infants, University of Mississippi Medical Center in the Department of Obstetrics and Gynecology

Reason for the Study:

- HEART DISEASE is the LEADING CAUSE OF DEATH IN WOMEN.
- Women who experience High Blood Pressure while pregnant with preeclampsia may be at risk for developing Heart Disease later in life.
- A study of the knowledge and beliefs that women have about Heart Disease may help doctors and nurses better inform and communicate to women how they may prevent Heart Disease later in life.

Are You Eligible to Participate?

- ✓ Are you 19 years of age or older?
- ✓ Were you pregnant within the last 12 months?
- ✓ Did you have High Blood Pressure due to preeclampsia while you were pregnant?
- ✓ Do you speak/understand the English language?
- ✓ Are you willing to speak on the telephone for a one-time interview?

If you are interested in taking part in this study or would like more information, please call: 601-984-5377

Participants will be compensated for taking part in the study.



**CVD Knowledge and Risk Perception among Women with Recent Preeclampsia:
Interventional Education in Disease Management and Prevention
Protocol #: X111223008.**

Contact Information

To: University of Mississippi Medical Center Department of Obstetrics and Gynecology
Maternal Fetal Medicine Department Research Staff

From: Patsy M. Spratling

Re: Research Study” Cardiovascular Disease Knowledge and Risk Perception among
Women with Recent Preeclampsia: Interventional Education in Disease Management”.

I will be conducting the above titled study in the UMMC Department of Obstetrics and Gynecology Maternal-Fetal Medicine Division with women who have been diagnosed with preeclampsia, eclampsia, and/or HELLP syndrome within the past 12 months. During hospitalization and/or during postpartum follow-up clinic visits, please provide the contact information sheet, which follows, to the above referenced patient population. The information sheet will inform patients that I will contact them within 2-4 weeks. Please ask the patient to provide their contact information on the contact information sheet and return the completed contact information sheet to me.
Thanks for your cooperation,

Patsy M. Spratling

**Cardiovascular Disease Knowledge and Risk Perception among Women with
Recent Preeclampsia: Interventional Education in Disease Management and
Prevention**

**Protocol #: X111223008
Patient Information Sheet**

Purpose: You are being invited to be in a research study titled: “CVD Knowledge and Risk Perception among Women with Recent Preeclampsia: Interventional Education in Disease Management and Prevention”; Protocol #: X111223008. You are being asked to participate in the study because you experienced high blood pressure while you were pregnant. Women, who experience high blood pressure due to preeclampsia while they are pregnant, may be at risk for heart disease later in life. We are doing this study to learn how women’s knowledge of heart disease affects their beliefs for developing heart disease. We also want to learn how to best inform women about heart disease.

This is a dissertation research study and is a required element for completing the Doctor of Philosophy (PhD) Degree in Nursing at University of Alabama at Birmingham (UAB). Patsy Spratling, MSN, RN lives in the Jackson, Mississippi area, however she is enrolled in the PhD nursing program at UAB; Patsy will be the principle investigator conducting this study.

Eligibility: You are eligible to participate in this study if you are: (1) at least 19 years of age; (2) have been pregnant within the last 12 months; (3) had a diagnosis of preeclampsia, eclampsia, and/or HELLP syndrome during the most recent pregnancy; (4) are able to read and speak English; and (5) are willing to participate in the study.

Research Procedures: If you enter the study, you will be interviewed over the telephone. Questions that you will be asked will help us learn about women’s understanding of heart disease. If you agree to participate, information that may help you to reduce your risk for developing heart disease will be provided. It will take about 30-45 minutes to answer questions that you will be asked.

Confidentiality: Information that you provide during the telephone interview will be kept private to the extent allowed by law. However, research information that identifies you may be shared with the UMMC and UAB Institutional Review Boards (IRB) and others who are responsible for ensuring compliance with laws and regulations related to research, such as the Office for Human Research Protections (OHRP). The findings of this study may be published for purposes of expanding knowledge. However, your identity will not be given out.

Risks and Discomforts: There is the potential risk of loss of confidentiality. However, I am the principle investigator (PI) in this study and I will conduct the telephone interviews in a private location; all efforts will be made to keep your information confidential. Your name will not be used on any questionnaires; only an identification number. In addition, all hard copies of completed study questionnaires will be kept in a locked cabinet at my office. All electronic data will be stored on an Ironkey encrypted thumb drive that only I have access to.

Benefits: Participants may or may not receive a direct benefit from being in this research study, but participants will receive information about ways to decrease risk for developing cardiovascular disease. This research will provide information that may help others in the future.

Alternatives: Your participation in this study is completely voluntary. By agreeing to participate, you are giving consent to participate in the study.

Refusal or Withdrawal without Penalty: Whether or not you take part in this study is your choice. There will be no penalty if you decide not to be in the study. If you decide not to be in the study, you will not lose any benefits you are otherwise owed. You are free to withdraw from this research study at any time or you may refuse to answer any questions. Your choice to leave the study will not affect your relationship with UMMC or UAB.

Cost of Participation: There will be no cost to you from taking part in this study.

Payment for Participation in Research: You will receive a \$5.00 gift card by mail for your participation in this study.

Questions:

- If you have questions about this study please call Patsy Spratling at 601-622-1130 or Dr. James Martin at 601-984-5327.
- If you have questions about your rights as a research participant, or concerns or complaints about the research, you may contact:
 - **UAB-** the Office of the Institutional Review Board for Human Use (OIRB) at the University of Alabama at Birmingham (UAB) at (205) 934-3789 or 1-800-822-8816. If calling the toll -free number, press the option for “all other calls” or for an operator/attendant and ask for extension 4-3789. Regular hours for the Office of the IRB are 8:00 a.m. to 5:00 p.m. CT, Monday through Friday. You may also call this number in the event the research staff cannot be reached or you wish to talk to someone else.
 - **UMMC-** the Chairman of the University of Mississippi Medical Center’s Institutional Review Board, 2500 North State Street, Jackson, Mississippi 39216; telephone, 601 984-2815; facsimile, 601 984-2961. The Institutional Review Board is a group of people not involved with this study who have reviewed the study to protect your rights.

**Cardiovascular Disease Knowledge and Risk Perception among Women with
Recent Preeclampsia: Interventional Education in Disease Management and
Prevention**

Protocol #: X111223008

Patient Contact Information:

Please provide your name and the best telephone number. Patsy Spratling will contact you within 2-4 weeks of your hospital discharge or clinic visit.

Thank you so much for helping with this important research,
Sincerely,

Patsy Spratling

Patient's Name: _____

Best telephone number to call: _____

APPENDIX D

PERMISSION TO USE RESEARH INSTRUMENTS

Re: CHD Knowledge tool

Saturday, February 20, 2010 9:15 AM

"Joanne Thanavaro" <jthanava@slu.edu>

[Add sender to Contacts](#)**From:**

"patsy spratling" <pspratli@bellsouth.net>

To:**Message contains attachments**

1 File (26KB)

[Microsoft Word - CHDK Tool With Answers.pdf](#)

Hello Patsy,

I'm so glad to hear that you are interested in using my tool and I am giving you permission to do so. I've attached a copy with answers. I am quite interested in hearing how your research turns out, so please let me know about your findings. Best of luck with your research.

On Fri, Feb 12, 2010 at 8:44 PM, patsy spratling <pspratli@bellsouth.net> wrote:

Dear Dr. Thanavaro,

My name is Patsy Spratling and I am currently a student in the PhD nursing program at the University of Alabama at Birmingham; my research interest is cardiovascular disease in women. I have read your article, "Coronary Heart Disease Knowledge Tool for Women" and I am very interested in the tool that was described in your article because women's knowledge of CHD is also of interest to me and this is an area that will be addressed in my study. Will you please allow me to use the CHD knowledge tool in my study? If I may use this tool, please forward a copy of the tool to me along with your permission to use it. Any assistance that you give to me will be greatly appreciated.

Sincerely,

Patsy Spratling

pspratli@bellsouth.net

or

mssprat@uab.edu

Dr. Joni Thanavaro

DNP, APRN, ACNP-BC, ANP-BC, DCC

Associate Professor

Coordinator, Adult Nurse Practitioner Program

St. Louis University

School of Nursing

3525 Caroline Street

St. Louis, Mo. 63104

314-977-8993

From: Ralf Schwarzer [ralf.schwarzer@fu-berlin.de]
 Sent: Sunday, November 13, 2011 9:58 AM
 To: Patsy M Spratling; health@zedat.fu-berlin.de
 Subject: Re: Permission to use research instruments in dissertation study

certainly,

also look for more recent papers under www.hapa-model.de

<http://www.hapa-model.de/>>I also attach some

At 16:15 13.11.2011, Patsy M Spratling wrote:

Dear Dr. Schwarzer, <?xml:namespace prefix = o ns = "urn:schemas-microsoft-com:office:office" />

My name is Patsy Spratling and I am doctoral nursing student at University of Alabama at Birmingham. In my proposed dissertation study, I will be examining cardiovascular disease (CVD) risk perception and perceived self-efficacy among women who recently experienced hypertensive disorders during pregnancy. I have read your article: Social-cognitive predictors of health behavior: Action self- efficacy and coping self-efficacy (Schwarzer & Renner, 2000). The information captured by the instruments reported in this article and the variables that will be examined in my proposed study are closely aligned.

In my dissertation study, may I use the CVD Risk Perception instrument and the Perceived Self-Efficacy instrument which were reported in Social-cognitive predictors of health behavior: Action self-efficacy and coping self-efficacy (Schwarzer & Remer, 2000)? Any assistance that you give to me will be greatly appreciated.

Sincerely,
 Patsy M. Spratling

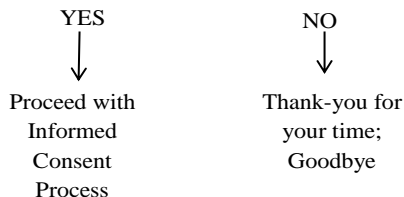
Prof. Dr. Ralf Schwarzer, Freie Universität Berlin, Psychology
 Habelschwerdter Allee 45, 14195 Berlin, Germany
 Email: ralf.schwarzer@fu-berlin.de
 Web: <http://www.RalfSchwarzer.de/><<http://www.ralfschwarzer.de/>>

APPENDIX E
TELEPHONE INTERVIEW SCRIPT

Telephone Interview Script

Hello, am I speaking with (potential participant's name)?
 My name is Patsy Spratling and I am a doctoral nursing student at University of Alabama at Birmingham. While you were in the hospital, a member of the research team informed you that I would call. I am conducting a research as part of my doctoral dissertation and I would like to talk with you about the study. Is this a convenient time?

Next Action will be based on Potential Participant's Response



Informed Consent:

Research Purpose: You are being invited to be in a research study titled: "Cardiovascular Disease Knowledge and Risk Perception among Women with Recent Preeclampsia: Interventional Education in Disease Management and Prevention"; Protocol #: X111223008. You are being asked to participate in the study because you experienced high blood pressure, due to preeclampsia, while you were pregnant. Women, who experience high blood pressure due to preeclampsia, while they are pregnant, may be at risk for heart disease later in life. We are doing this study to learn how women's knowledge of heart disease affects their beliefs for developing heart disease. We also want to learn how to best inform women about heart disease.

Research Procedures: If you enter the study, you will be interviewed over the telephone. Questions that you will be asked will help us learn about women's understanding of heart disease. If you agree to participate, information that may help you to reduce your risk for developing heart disease will be provided. It will take about 30-45 minutes to answer questions that you will be asked.

Confidentiality: Information that you provide during the telephone interview will be kept private to the extent allowed by law. However, research information that identifies you may be shared with the UMMC and UAB Institutional Review Boards (IRB) and others who are responsible for ensuring compliance with laws and regulations related to research, such as the Office for Human Research Protections (OHRP). The findings of this study may be published for purposes of expanding knowledge. However, your identity will not be given out.

Risks and Discomforts: There is the potential risk of loss of confidentiality. However, I am the principle investigator (PI) in this study and I will conduct the telephone interviews in a private location; all efforts will be made to keep your information confidential. Your name will not be used on any questionnaires; only an identification number. In addition, all hard copies of completed study questionnaires will be kept in a locked cabinet at my office. All electronic data will be stored on an Ironkey encrypted thumb drive that only I have access to.

Benefits: Participants may or may not receive a direct benefit from being in this research study, but participants will receive information about ways to decrease risk for developing cardiovascular disease. This research will provide information that may help others in the future.

Alternatives: Your participation in this study is completely voluntary; therefore you have the option to participate or not to participate.

Refusal or Withdrawal without Penalty: Whether or not you take part in this study is your choice. There will be no penalty if you decide not to be in the study. If you decide not to be in the study, you will not lose any benefits you are otherwise owed. You are free to withdraw from this research study at any time or you may refuse to answer any questions. Your choice to leave the study will not affect your relationship with UMMC or UAB.

Cost of Participation: There will be no cost to you from taking part in this study.

Payment for Participation in Research: You will receive a \$5.00 gift card by mail for your participation in this study.

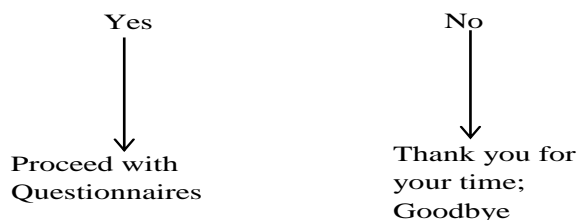
Questions:

- If you have questions about this study please call Patsy Spratling at 601-622-1130 or Dr. James Martin at 601-984-5327.

If you have questions about your rights as a research participant, or concerns or complaints about the research, you may contact:

- **UAB-** the Office of the Institutional Review Board for Human Use (OIRB) at the University of Alabama at Birmingham (UAB) at (205) 934-3789 or 1-800-822-8816. If calling the toll -free number, press the option for “all other calls” or for an operator/attendant and ask for extension 4-3789. Regular hours for the Office of the IRB are 8:00 a.m. to 5:00 p.m. CT, Monday through Friday. You may also call this number in the event the research staff cannot be reached or you wish to talk to someone else.
- **UMMC-** the Chairman of the University of Mississippi Medical Center’s Institutional Review Board, 2500 North State Street, Jackson, Mississippi 39216; telephone, 601 984-2815; facsimile, 601 984-2961. The Institutional Review Board is a group of people not involved with this study who have reviewed the study to protect your rights.

Question to Potential Participant: Are you willing to participate in this study?
Next Action will be based on Potential Participant’s Response



APPENDIX F
STUDY QUESTIONNAIRES

Demographic Questionnaire

1. What is your age?
 - 1) 19-24
 - 2) 25-29
 - 3) 30-34
 - 4) 35-39
 - 5) > 40
2. What race do you identify with?
 - 1) White/Caucasian
 - 2) Black/African-American
 - 3) Hispanic
3. How many children have you given birth to, including your last pregnancy?
 - 1) 1
 - 2) 2
 - 3) 3
 - 4) 4
 - 5) 5
 - 6) > 5
4. Did you have more than one child with any of your deliveries?
 - 1) Yes
 - 2) No
5. Did you have preeclampsia, eclampsia, and/or HELLP syndrome with previous pregnancies?
 - 1) Yes
 - 2) No
 - 3) Don't Know
 - 4) N/A; This Was The Index Pregnancy
6. What is your level of education?
 - 1) Less than High school
 - 2) High school graduate/GED
 - 3) Some College
 - 4) College Graduate
7. What is your annual income?
 - 1) $\leq 10,000$
 - 2) 10,001-20,000
 - 3) 20,001-30,000
 - 4) 30,001-40,000
 - 5) > 40,001
 - 6) Declined to Answer
8. What is your Marital Status?
 - 1) Single- Never Married
 - 2) Married
 - 3) Married, but Separated
 - 4) Living with Significant Other
 - 5) Divorced
 - 6) Widow

Modified Coronary Heart Disease Knowledge Tool for Women

Directions: Please respond to each statement as True or False

Question	True	False
1. Heart disease, related to heart artery blockages, develops slowly over many years and can easily go undetected.		
2. Obesity may cause heart disease		
3. Symptoms of heart pain or heart attack may include: chest pain; chest tightness; and unusual fatigue		
4. Women are more likely to get heart disease after menopause than before menopause.		
5. African American women are more likely than White women to die from a heart attack or stroke.		
6. High cholesterol levels may cause heart artery blockages.		
7. Symptoms of heart pain or heart attack may include: neck, shoulder, arm, or back pain, and dizziness.		
8. Once women are diagnosed or identified as having heart disease, they are more likely than men to become seriously ill or die.		
9. Stress may cause heart disease.		
10. There is no evidence that hormone therapy or hormone replacement prevents heart disease.		
11. High blood pressure may cause heart disease.		
12. Some forms of heart disease may result in stroke.		
13. A high fat diet may cause clogged heart arteries.		
14. Moderate alcohol use (1-2 drinks per day) may prevent heart disease.		
15. Smoking may cause heart artery blockages.		
16. Low levels of some female hormones may increase heart artery blockages in women.		
17. African American women are more likely to have heart disease than White women.		
18. Reducing dietary cholesterol may prevent clogged heart arteries.		
19. Routine exercise may prevent heart disease.		
20. Symptoms of heart pain or heart attack may include: shortness of breath; sweating; and nausea.		
21. Reducing dietary red meat may prevent heart artery blockages.		
22. A family history of heart disease, from clogged heart arteries, may increase your risk of getting heart disease.		
23. Diabetes may increase the chance of having a heart attack.		
24. Heredity is a risk factor of heart disease related to clogged heart arteries that cannot be changed.		
25. Heart disease and stroke are the leading cause of health care problems and death in women.		

CVD Risk Perception Scale

Note: CVD Risk Perception was measured both Pre and Post Education Intervention

Item Stem: If you compare yourself to other women your age, what do you estimate the likelihood of experiencing:

Heart disease:

1 2 3 4 5 6 7

High Blood Pressure:

1 2 3 4 5 6 7

Stroke:

1 2 3 4 5 6 7

Note: CVD Risk perception was measured on a 7-point Likert scale where:

1 = Much below average

2

3

4 = Average

5

6

7 = Much above average

All responses recoded to original scale values (-3 to +3) for statistical analyses

APPENDIX G

TELEPHONE EDUCATION INTERVENTION SCRIPT

Education Intervention Script

(Flesh-Kincaid Grade Level: 7.5)

Script: At the beginning of the study, I asked you several true or false questions; each question was true. I will go over those questions again so that you may better understand about heart disease and ways to prevent it. First, I would like to share a few facts about heart disease in women.

Education Intervention:

- ✓ Women are more likely to develop heart disease after menopause than before menopause.
 - Low levels of some female hormone may increase heart artery blockages in women; however there is no evidence that hormone therapy or hormone replacement prevents heart disease.
- ✓ Once women are diagnosed or identified as having heart disease, they are more likely than men to become seriously ill or die.
- ✓ Heart disease and stroke are the leading cause of health care problems and death in women.
- ✓ Heredity is a risk factor of heart disease, related to clogged/blocked heart arteries, which cannot be changed.
 - Having a family history of heart disease due to clogged heart arteries may increase your risk of developing heart disease.
- ✓ African American/Black women are more likely to have heart disease than White women. Also, African American/Black women are more likely than White women to die from a heart attack or stroke.
- ✓ Some forms of heart disease may result in stroke.

Script: Now, I will share some information about different elements that may increase your risks for developing heart disease. However, each of these elements can be controlled. By controlling these elements, you may reduce your risks for developing heart disease.

Education Intervention:

- ✓ Moderate alcohol use (1-2 drinks per day) may prevent or decrease risks for developing heart disease.
- ✓ High blood pressure may cause heart disease and because you had high blood pressure due to preeclampsia when you were pregnant, you may be at risk for developing heart disease.
- ✓ If heart disease is related to heart artery blockages, it develops slowly over many years and can easily go undetected. There are several things that may be done to reduce your risks of developing heart artery blockages.
 - High cholesterol levels and eating a high fat diet may cause clogged or blocked heart arteries.
 - Reducing cholesterol and red meat in your diet may prevent clogged/blocked heart arteries.
 - Obesity may cause heart disease, but
 - Routine exercise may help with weight control and may prevent heart disease.
 - It is recommended that you are physically active for at least 30 minutes (10 minutes at a time) on most days of the week
 - You encouraged balancing your calories by eating the foods you like, but eat less and avoid oversized portions.
 - Other suggestions are to: (1) make half your plate fruits and vegetables; (2) make at least half of the grains that you eat whole grains; (3) switch to fat-free or low fat (1%) milk; and (4) drink water instead of sugary drinks.
 - Smoking may cause heart artery blockages.
 - Do not smoke or use other types of tobacco products.
 - Stress may cause heart disease; and
 - Diabetes may increase the chance of having a heart attack.

Script: I have shared information on several elements related to heart disease in women. Before we finish, I would like to share with you symptoms of heart pain or heart attack. In addition, I want to encourage you to have your blood pressure and cholesterol levels checked by your healthcare provider on a regular basis.

Education Intervention:

- ✓ Symptoms of heart pain or heart attack may include: chest pain; chest tightness; unusual fatigue; shortness of breath; sweating; and nausea.

APPENDIX H

GUIDELINES FOR CARDIOVASCULAR DISEASE PREVENTION IN WOMEN
MAIL-OUT BROCHURE POST STUDY

Know your Numbers:**Blood Pressure**

- ✓ Less than 120/80 mm Hg

Total Cholesterol

- ✓ Less than 200 mg/dL

LDL Cholesterol

- ✓ Less than 100 mg/dL

HDL Cholesterol

- ✓ Greater than 50 mg/dL

Triglycerides

- ✓ Less than 150 mg/dL

Non-HDL Cholesterol

- ✓ Less than 130 mg/dL

To Attain these Numbers:

- ✓ Control Weight
- ✓ Increase Physical Activity
- ✓ Alcohol Moderation
- ✓ Sodium Restriction
- ✓ Increase Intake of Fruits
- ✓ Increase Intake of Vegetables
- ✓ Increase Intake of Low-Fat Dairy Products

Remember to:

- ✓ Have your Blood Pressure, Cholesterol and Lipid Levels checked by a healthcare provider on a regular basis.
- ✓ If you are prescribed Blood Pressure or Cholesterol Medication: Take your Medication as Prescribed.

Thank-You for Your Participation
Patsy M. Spratling, MSN, RN

Guidelines Adapted from "Effectiveness-Based Guidelines for the Prevention of Cardiovascular Disease in Women-2011 Update: A Guideline from the American Heart Association," by Mosca et al. 2011. *Circulation: Journal of the American Heart Association*, 123, pp. 1252-1254.

Guidelines for Cardiovascular Disease Prevention in Women



Lifestyle Habits

Cigarette Smoking:

Do not to smoke.

Avoid environmental tobacco smoke.

Physical Activity:

Engage in moderate exercise at least 150 minutes per week or vigorous exercise for 75 minutes per week.

Engage in muscle-strengthening activities that involve all major muscle groups on at least 2 or more days per week.

Weight Maintenance/Reduction:

Maintain or lose weight through an appropriate balance of physical activity and caloric intake.

Engage in at least 60 to 90 min of moderate-intensity physical activity (e.g., brisk walking) on most, and preferably all, days of the week.

Dietary Intake:

Consume a diet rich in fruits and vegetables.

Choose whole-grain, high-fiber foods.

Limit intake of saturated fat, cholesterol, alcohol, sodium, and sugar.

Avoid *trans*-fatty acids.

Consume fish, especially oily fish, at least twice a week.

Note: If you become pregnant- avoid eating fish with the potential for the highest level of mercury contamination (e.g., shark, swordfish, king mackerel, or tile fish)

Dietary Guidelines

Nutrient	Servings	Serving Size
Fruits and vegetables	≥ 4 ½ cups per day	1 cup raw leafy vegetable, 1/2 cup cut-up raw or cooked vegetable, 1/2 cup vegetable juice; 1 medium fruit, 1/4 cup dried fruit, 1/2 cup fresh, frozen, or canned fruit, 1/2 cup fruit juice
Fish	2 times per week	3 ½ ounces cooked (preferably oily types of fish, such as salmon)
Fiber	30 grams per day (1.1 grams/ 10 grams carbohydrate)	Bran cereal, berries, avocado, etc.
Whole Grains	3 per day	1 slice bread, 1 oz dry cereal, 1/2 cup cooked rice, pasta, or cereal (all whole-grain products)
Sugar	5 times per week (no more than 450 calories from sugar sweetened beverages)	1 tablespoon sugar, 1 tablespoon jelly or jam, 1/2 cup sorbet, 1 cup lemonade
Nuts, legumes, and seeds	No more than 4 times per week	1/3 cup or 1 1/2 oz nuts (avoid macadamia nuts and salted nuts), 2 tablespoons peanut butter, 2 tablespoon or 1/2 oz seeds, 1/2 cup cooked legumes (dry beans and peas)
Saturated fat	Less than 7%/total energy intake	Found in fried foods, fat on meat or chicken skin, packaged desserts, butter, cheese, sour Cream, etc.
Cholesterol	Less than 150 mg per day	Found in animal meats, organ meats, eggs, etc.
Alcohol	No more than 1 per day	4 oz wine, 12 oz beer, 1.5 oz of 80-proof spirits, or 1 oz of 100-proof spirits
Sodium	No more than 1500 mg per day	
<i>Trans</i> -fatty acids	None	None