ENHANCING TOBACCO ABSTINENCE FOLLOWING HOSPITALIZATION

by

Donna D. Caruthers

BSN, University of Pittsburgh, 1978

MSN, University of Pittsburgh, 1982

Submitted to the Graduate Faculty of the

School of Nursing in partial fulfillment

of the requirements for the degree of

Doctor of Philosophy

University of Pittsburgh

2005

UNIVERSITY OF PITTSBURGH

FACULTY OF NURSING

This dissertation was presented

by

Donna D. Caruthers

It was defended on

April 12th, 2005

and approved by

Susan Albrecht, PhD, RN, Associate Professor

Kenneth Perkins, PhD, Professor

Co-Dissertation Director Susan Sereika, PhD, Associate Professor

Dissertation Director Jacqueline Dunbar-Jacob, PhD, RN, Professor

ENHANCING TOBACCO ABSTINENCE FOLLOWING HOSPITALIZATION

Donna D. Caruthers, PhD

University of Pittsburgh, 2005

Tobacco use continues to be the leading cause of morbidity and mortality in the United States. Public Health Service sponsored clinical guidelines support smoking cessation interventions at every clinical encounter with a smoking patient. The primary aim of this research protocol proposed to examine the efficacy of a 12-week nurse-delivered relapse management intervention designed with conceptual underpinnings from Self-efficacy Theory to enhance smoking abstinence of hospitalized smokers following their hospital discharge. A randomized, controlled two-group design with an intent-to-treat approach was used. The sample consisted of 80 consenting smokers prospectively recruited during hospitalization. Subjects were randomly assigned by equal allocation to a special intervention group (SI) or an enhanced usual only group (UC). All subjects received enhanced usual care. Participants assigned to the intervention group received 8 telephone intervention sessions with a nurse over 11 weeks after discharge. Intervention was directed towards enhancing self-efficacy to maintain tobacco abstinence. Follow-up visits occurred 12 and 24 weeks following hospital discharge. Data collection included smoking point prevalence with validation by exhaled carbon monoxide. At 12 weeks, 20% (n = 8) UC and 40% (16) SI subjects were abstinent ($_{LR}\chi 2 = 4.87$, df = 1, p = .014). At 24 weeks, 15% (n = 6) UC and 42% (n = 16) SI subjects were abstinent ($_{LR}\chi 2 = 7.69$, df = 1, p = .004). There were significant differences between treatment assignments, particularly when confounding variables for current employment and greater lengths of hospital stay were

controlled in the analyses. Self-efficacy with the Relapse Situation Efficacy Questionnaire was predictive of 12-week smoking status. Treatment adherence was significantly related to smoking behavior in the treatment group. The two groups did not differ in smoking lapse or with self-efficacy over time. Recruitment sites did differ with respect to smoking status, but only at 12-weeks after discharge. There were no significant cohort differences. Future research is needed to improve tobacco abstinence following hospitalization and to examine treatment adherence with an emphasis on strategies for improvement of treatment adherence with hospitalized smokers.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

PREFACE

I would like to acknowledge my dissertation committee, Jacqueline Dunbar-Jacob, PhD, RN, FAAN, Susan Sereika, PhD, Kenneth Perkins, PhD and Susan Albrecht, PhD, RN for their invaluable direction and assistance with this research. Their mentorship was important to the completion of my studies and this project.

I would like to express my gratitude and acknowledgement to the National Institutes of Nursing Research for the predoctoral fellowship award funding granted to conduct this research. In addition, I am grateful to the Pennsylvania Nursing Foundation and the Eta Chapter of Sigma Theta Tau for their generous financial support that assisted this project.

I would like to thank the participants who set out to change their smoking behavior as part of this study. I appreciate the time and effort they provided. This project could not have come to fruition without them.

The nurses and healthcare providers at the University of Pittsburgh Medical Center and Jefferson Regional Hospital provided important recruitment assistance to this project. I am truly thankful for your recruitment efforts and promotion of this project. To the nurses on 4D and 5D at UPMC Presbyterian, this project began and was nurtured along the way by your very caring staff of professionals. I am indebted to your willingness and persistence to assist hospitalized smokers change their behaviors.

The staff of the Managing Medications Project at the School of Nursing were instrumental in assisting my efforts in completing the last phase of this project. I would like to thank Dr. Erlen who provided ongoing guidance and support for my doctoral student and dissertation activities. I would not have made this defense date without her and Michelle Meyers. I have many "thanks" to give to Irene Petrovich for her support, assistance, and more.

To my family and friends, you deserve the largest gratitude for your help, cheers, and support of my efforts to conduct this research project. To my father, Donald Dvorsky, thank you for the many lessons, support, and love. From the beginning to the end, you have been an invaluable support mechanism to this most amazing project in my career. Bill Caruthers, through tough and easy times you have stood by my decision to pursue this career path and provided every means of support I requested and you are due my sincerest gratitude.

Finally, this research is dedicated to three very special individuals. First, this project is dedicated to my mother who was my first mentor in nursing and life. Finally, this project is dedicated to my children, Kathy and Bill, who are my present and future. Their support and sacrifice to assist my efforts in completing this project were provided unselfishly and full of love.

1. CHAPTER ONE

1.1. STATEMENT OF THE PROBLEM

1.1.1. Introduction

Entering the 21st century, tobacco exposure remains the leading preventable cause of death, accounting for 18% of all United States [U.S.] deaths and 10% of the deaths around the world (Center for Disease Control and Prevention, 1993a, 1997c, 1999c; McGinnis & Foege, 1993; Mokdad, Marks, Stroup, & Gerberding, 2004; World Bank, 1999). Furthermore, tobacco exposure in the U.S. is associated with the prevalence of a myriad of health disorders across age groups (Center for Disease Control and Prevention, 1999a; Pauwels & Rabe, 2004). Costs related to tobacco exposure and consumption include escalating individual and societal monetary expenditures, as well as a loss in quality of life (Bartecchi, MacKenzie, & Schrier, 1994; Cohen & Barton, 1998; Hodgson, 1992; MacKenzie, Bartecchi, & Schrier, 1994; Maxwell & Hirdes, 1993). Annually, health-related economic losses for tobacco related illnesses total $157 billion (Center for Disease Control and Prevention, 2002).

The declining trend of tobacco consumption prevalence has stalled in recent years in the U.S., along with a global trend towards an increase in tobacco dependence (Chollat-Traquet, 1992; Corrao, Guindon, Cokkinides, & Sharma, 2000; McCann, 2000; World Bank, 1999). In the U.S., 22.1% of the adult population consumes cigarettes, the leading commercial product source of tobacco (Center for Disease Control and Prevention, 2004d). Furthermore, of 32 million adults who received healthcare coverage through Medicaid programs in 2000, more than 36% were

smokers (Center for Disease Control and Prevention, 2004c). Although effective interventions exist to alter the physical assault associated with tobacco use, interventions have not resulted in a dramatic change in tobacco exposure due in part to the limited availability of these interventions (Ad Hoc Working Group on Treatment of Tobacco, 2001). Most individuals dependent upon tobacco require several attempts at self-help oriented abstinence efforts with 70 to 90 percent relapsing in their quest to stop their tobacco dependency (Center for Disease Control and Prevention, 1993b). Data from 2002 indicated 41% of current smokers attempted to quit smoking for at least one day within a 12-month period (Center for Disease Control and Prevention, 2004a). Additionally, data reported 50% of ever-smokers in the U.S. were now former smokers, which is the highest level of former smokers and supports the need for readily available cessation strategies (Center for Disease Control and Prevention, 2004a). In August of 2002, a national action plan to combat smoking and increase tobacco abstinence was suggested by the Subcommittee on Cessation of the Interagency Committee on Smoking and Health (ICSH) (Fiore, Croyle, Curry, Cutler, Davis, Gordon, Healton, Koh, Orleans, Richling, Satcher, Seffrin, Williams, Williams, Keller, & Baker, 2004). This plan had 10 recommendations including the promotion of evidenced-based cessation interventions.

There are essentially two types of tobacco exposure, passive and active. Passive exposure to tobacco products in the environment is an unintentional exposure for the affected individual. Approximately 35% of all children in the U.S. are exposed to environmental tobacco smoke [ETS] (Klerman, 2004), such as the passive exposure a child receives form from the side stream smoke of a parent's cigarette. Those passively exposed to tobacco products are at risk for the development of medical disorders, such as recurrent respiratory infections, cancer, and exacerbations of asthma (Center for Disease Control and Prevention, 2000a; Wahlgren, Hovell,

Meltzer, & Meltzer, 2000). Another example of passive exposure is the effect of tobacco consumption by pregnant mothers on their unborn babies. Nicotine from tobacco can cross the placenta, placing the child at risk for premature delivery or spontaneous abortion, third trimester intrauterine growth retardation, and sudden infant death syndrome following delivery (Brown, 1996; Lieberman, Gremy, Lang, & Cohen, 1994; Pollack, 2001).

Active tobacco exposure occurs when an individual consumes tobacco by smoking (e.g., cigarettes, cigars, pipes) or orally rubbing (e.g., snuff) or chewing (e.g., chewing tobacco) the product. An important benefit of abstinence for tobacco consumers (active exposure) includes decreased risk for the development of fatal diseases, such as cardiovascular disease, cancer, and lung disease (Mokdad et al., 2004; U.S. Department of Health and Human Services, 1981, 1982, 1983, 1984, 1985, 1991). For example, patients who continue smoking tobacco following a percutaneous cardiovascular revascularization are at greater risk for a subsequent myocardial infarction and/or death than nonsmokers and smokers who abstain following the procedure (Hasdai, Garratt, Grill, Lerman, & Holmes, 1997). The Lung Health Study, a multi-center smoking cessation trial, demonstrated that the course of declining lung function in middle-aged smokers could be altered to parallel the normal lung function decline in nonsmokers (Kanner, 1996). Apart from the benefits in decreasing risk for disease and premature death, abstaining smokers treated with surgery or medical wound management experience outcome benefits, which include fewer complications, improved wound and bone healing, and decreased medical expenditures (Battaglia, Di Mario, Piccoli, Vianello, Farinati, & Naccarato, 1987; Glassman, Anagnost, Parker, Burke, Johnson, & Dimar, 2000; Grossi, Zambon, Machtei, Schifferle, Andreana, Genco, Cummins, & Harrap, 1997; Hollinger, Schmitt, Hwang, Soleymani, & Buck, 1999; Lavernia, Sierra, & Gomez-Marin, 1999).

Tobacco use, specifically cigarette smoking, has added hidden medical risks besides increasing risk for the development of fatal chronic disorders and incurring increased economic burden on individuals and society. Evidence in the literature has associated smoking with poor adherence to prescribed medical treatment (Atkins, Mion, Mendelson, Palmer, Slomka, & Franko, 1997; Dew, Roth, Thompson, Kormos, & Griffith, 1996; Glynn, Buring, Manson, LaMotte, & Hennekens, 1994; Toljamo & Hentinen, 2001; Vaur, Vaisse, Genes, Elkik, Legrand, & Poggi, 1999; Weir, Maibach, Bakris, Black, Chawla, Messerli, Neutel, & Weber, 2000). Therefore, the smoking patient may compound his/her medical status by not only increasing health risks for disease development, but also impeding the efficacy of prescribed medical treatments. In an effort to further cloud this situation, case reports document that patients who abstain from tobacco, without medical supervision, as they are receiving medical treatment for other disorders (e.g., cancer) have experienced increased poor adherence to prescribed medical treatment (Gritz, Schacherer, Koehly, Nielsen, & Abemayor, 1999b; Moadel, Lederberg, & Ostroff, 1999). However, due to gaps in the literature, it is not clear what impact supervised tobacco abstinence can have on medical treatment adherence for other health disorders in a population of smokers. Given that current smokers are more likely to be poor adherers, healthcare professionals need to question when or if smokers, receiving tobacco dependency treatment, improve their adherence to prescribed medical treatment.

Tobacco control efforts have included community, individual, and policy interventions aimed to eliminate or substantially reduce tobacco exposure (Stillman, Hartman, Graubard, Gilpin, Murray, & Gibson, 2003). Rationale for such activity is based upon the consequences of exposure and health related benefits of tobacco reduction and/or abstinence for all individuals exposed to tobacco. Of the various clinical guidelines for tobacco dependency treatment, two

clinical guidelines for health care providers have been published by the U.S.D.H.H.S.. These guidelines promote evidenced-based treatment interventions for tobacco dependency, identification of target populations, and goals for future research (American Psychiatric Association, 1996; Fiore, Bailey, Cohen, & et al., 1996, 2000; U.S. Department of Health and Human Services, 1986; U.S. Preventive Services Task Force, 1996).

The identification of hospitalized smokers as a special interest population of tobacco consumers in the 1996 clinical guideline coincided with a national effort to require smoke-free environments in all U.S. hospitals (Beemer, 1993; Fiore & Jorenby, 1992; Goldstein, Westbrook, Howell, & Fischer, 1992; Holmes, Mateczun, & Pentzien, 1991). Over the last decade, a medical/surgical hospitalization for a tobacco dependent patient has been considered a "window of opportunity" to introduce tobacco abstinence (Emmons & Goldstein, 1992). Furthermore, this situation has been termed a "teachable moment" for intervention messages and activities aimed to motivate a smoker towards tobacco abstinence. Rigotti, et al. (2000) found patients with biologically confirmed abstinence during hospitalization were four times more likely to remain abstinent from tobacco after discharge; however, barriers with adherence to smoke-free policies in hospital settings existed (Goldstein et al., 1992) and patients continue to struggle with adherence to smoking abstinence during hospitalization (Rigotti, Arnsten, McKool, Wood-Reid, Pasternak, & Singer, 2000). Although findings continue to support this teachable moment as a unique opportunity for tobacco abstinence, hospitalization alone in these smoke-free environments is not enough to bring about maintained abstinence during and following a hospital admission for all smokers.

1.1.2. Significance of the study

Hospital-based tobacco dependency intervention has the potential of reaching smokers diagnosed with various comorbid medical and psychological disorders (France, Glasgow, & Marcus, 2001; Halpern, Schmier, Ward, & Klesges, 2000; Hennrikus, Lando, McCarty, Klevan, Holtan, Huebsch, Jestus, Pentel, Pine, Sullivan, Swenson, & Vessey, 2005; Narsavage & Idemoto, 2003; Quist-Paulsen & Gallefoss, 2003; Rigotti, Munafo, Murphy, & Stead, 2003; Simon, Carmody, Hudes, Snyder, & Murray, 2003; Sivarajan Froelicher, Miller, Christopherson, Martin, Parker, Amonetti, Lin, Sohn, Benowitz, Taylor, & Bacchetti, 2004; Taylor, Houston-Miller, Killen, & DeBusk, 1990; Wewers, Bowen, Stanislaw, & Desimone, 1994). Maintained tobacco abstinence has the potential to decrease the progression of currently diagnosed tobacco related disorders in this population, as well as decrease the risk for additional related diseases. Over a decade ago, the benefit of tobacco cessation for hospitalized smokers was recognized as having the greatest potential of preventing the development of tobacco related comorbid disorders and associated costs to life (Emmons & Goldstein, 1992). Unfortunately, high rates of relapse to smoking occur within the first few months of an abstinence attempt across various types of tobacco dependency interventions (France et al., 2001; Halpern et al., 2000; Matheny & Weatherman, 1998; Narsavage & Idemoto, 2003; Ratner, Johnson, Richardson, Bottorff, Moffat, Mackay, Fofonoff, Kingsbury, Miller, & Budz, 2004; Rigotti, Arnsten, McKool, Wood-Reid, Pasternak, & Singer, 1997; Taylor et al., 1990; Taylor, Miller, Herman, Smith, Sobel, Fisher, & DeBusk, 1996; Wewers et al., 1994; Wewers, Jenkins, & Mignery, 1997). Brief (< 20 minutes) counseling during hospitalization without further intervention has not been effective for smoking intervention in this population (Rigotti et al., 2003). Findings reported in the literature pertaining to hospitalized tobacco consumers, however, suggest significant long-term changes in tobacco

dependence can be accomplished with hospital-based programs to introduce abstinence followed by aggressive relapse prevention following discharge. Frequent follow-up efforts (Miller, Smith, DeBusk, Sobel, & Taylor, 1997b; Quist-Paulsen & Gallefoss, 2003; Sivarajan Froelicher et al., 2004; Stevens, Glasgow, Hollis, Lichtenstein, & Vogt, 1993; Stevens, Glasgow, Hollis, & Mount, 2000; Taylor et al., 1996) and pharmacological options have also enhanced abstinence rates in this population (DeBusk, Miller, Superko, Dennis, Thomas, Lew, Berger, Heller, Rompf, & Gee, 1994; Miller et al., 1997b). The latter option, pharmacological treatment is dependent upon a patient's medical condition, but non-drug programs can be available to most regardless of medical status.

For those smokers using a hospital admission as an opportunity to also obtain tobacco abstinence, prevention of relapse following discharge is necessary to successfully carry a smoke-free lifestyle forward. Nurses are the most likely healthcare professionals to initiate a non-drug tobacco abstinence intervention program. Therefore, the purpose of this study was to examine the efficacy of a 12-week cessation promotion and relapse maintenance program initiated prior to hospital discharge to a population of hospitalized smokers.

1.1.3. Specific aims

1.1.3.1. Aim 1: The primary aim was to examine the efficacy of a 12-week nurse-delivered telephone abstinence promotion and relapse management intervention designed to enhance self-efficacy and smoking abstinence for smokers desiring to abstain following hospital discharge as measured by self-reports of smoking behavior validated by carbon monoxide [CO].

Hypothesis 1a: The group of hospitalized smokers randomly assigned to a 12-week abstinence promotion and relapse management intervention following discharge was hypothesized to have a greater number of participants with smoking abstinence (smoking point prevalence verified by CO) 12 weeks following discharge as compared to subjects who were assigned to only enhanced usual care.

Hypothesis 1b: The group of hospitalized smokers randomly assigned to a 12-week abstinence promotion and relapse management intervention following discharge was hypothesized to have a greater number of participants with smoking abstinence (smoking point prevalence verified by CO) 24 weeks following discharge as compared to subjects who were assigned to only enhanced usual care.

1.1.3.2. Aim 2: A two part secondary aim of the study was to examine relationships between smoking point prevalence and a) selected baseline covariates (e. g. self-efficacy, relapse situation efficacy, perceived treatment efficacy, social support for tobacco abstinence, and affective states, in particular depressive symptoms) and b) treatment adherence. The first part of this secondary aim examined the relationship between the outcome of smoking point prevalence and conceptually driven variables from Self-efficacy Theory. The following hypotheses 2a – 2e apply to the first part of this secondary aim. The second secondary aim was to examine treatment related variables, such as time to smoking lapse, the outcome of smoking point prevalence, treatment adherence, and self-efficacy. Hypotheses 2f – 2i apply to the second part of this aim.

Hypothesis 2a(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived self-efficacy as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES].

Hypothesis 2a(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived self-efficacy as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES].

Hypothesis 2b(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for relapse situation efficacy as measured by the Relapse Situation Efficacy Questionnaire [RSEQ].

Hypothesis 2b(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for relapse situation efficacy as measured by the Relapse Situation Efficacy Questionnaire [RSEQ].

Hypothesis 2c(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for outcome expectancy as measured by the Perceived Therapeutic Efficacy Scale for Relapse Maintenance [PTES-RM].

Hypothesis 2c(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived treatment efficacy as measured by the Perceived Therapeutic Efficacy Scale for Relapse Maintenance [PTES-RM].

Hypothesis 2d(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived social support for tobacco abstinence.

Hypothesis 2d(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived social support for tobacco abstinence.

Hypothesis 2e(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by an inverse relationship with baseline scores for affective states, specifically depressive symptoms as measured by the depression/dejection subscale on the Profile of Mood States [POMS].

Hypothesis 2e(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by an inverse relationship with baseline scores for affective states, specifically depressive symptoms as measured by the depression/dejection subscale on the Profile of Mood States [POMS].

Hypothesis 2f: The time to the first smoking lapse was hypothesized to be longer for subjects who were assigned the 12-week abstinence promotion and relapse management intervention as compared to subjects who were assigned to only enhanced usual care.

Hypothesis 2g(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with treatment adherence rates.

Hypothesis 2g(2): Hypothesis 2g(1): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with treatment adherence rates.

Hypothesis 2h: Treatment adherence was hypothesized to have a positive relationship with baseline (T_0) perceived self-efficacy as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES].

Hypothesis 2i: Subjects in the treatment group were hypothesized to have an increase in self-efficacy, as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES], from baseline (T_0) to follow-up measurements at T_1 and T_2.

1.1.4. Definition of terms

1.1.4.1. Tobacco abstinence (from smoked products). The process of tobacco smoking requires the ignition of a cigar, pipe, or cigarette containing tobacco by a flame. Once lit, stoking by inhalation and puffing are required. It is during the combustion of the tobacco that nicotine, carbon monoxide, and other substances are emitted and inhaled by the individuals smoking the tobacco product (Thomas, 1997). The National Health Interview Survey has used the following definitions of smokers (Center for Disease Control and Prevention, 2004a). The standard definition of an "ever smoker" has been defined as an individual who has smoked 100 or more cigarettes in their lifetime. Current smokers have been defined as individuals reporting themselves to be smoking within the time-period of an interview. Former smokers have been defined as individuals that are presently not smoking or abstinent within the time period of an interview or questionnaire (Center for Disease Control and Prevention, 2004a; Nelson, Emont, Brackbill, Cameron, Peddicord, & Fiore, 1994). Therefore, smoking behaviors can be defined by historical use, current consumption, or abstinence. Popular measures of abstinence from tobacco for a tobacco use treatment program include 7-day point prevalence of smoking abstinence (abstinence requires self-reported no tobacco use for a 7 day period prior to the follow-up visit) and continuous abstinence (abstinence is required at all follow-up visits following tobacco dependence treatment) (Shipley, Rosen, & Williams, 1982; Sivarajan Froelicher et al., 2004; Smith, Reilly, Houston Miller, DeBusk, & Taylor, 2002).

For the purposes of this study, tobacco abstinence following hospital discharge was defined by 7-day point prevalence. This variable was operationalized with a self-reported measure of tobacco abstinence and biological validation with expired carbon monoxide measurements. Abstinence was defined as no tobacco use in the form of cigarettes, cigars, or

pipes for 7 days prior to the follow-up visit and an exhaled carbon monoxide reading less than or equal to 8 parts per million [ppm]. Follow-up intake questionnaires will also document recent passive exposure to tobacco, as well as medical disorders and treatments that may impact exhaled carbon monoxide results (Kharitonov & Barnes, 2001).

For recruitment purposes, an eligible current smoker for this project was defined as a patient who smoked tobacco within 30 days of the current hospital admission. This definition of current smoking status is consistent with definitions of current smoking for previous intervention studies with hospitalized smokers (Dornelas, Sampson, Gray, Waters, & Thompson, 2000; Miller et al., 1997b; Taylor et al., 1996). The rationale of using this definition over 7-day point prevalence is that patients admitted for elective procedures may have been asked by their physicians to refrain from tobacco consumption prior to elective surgical procedures. Therefore, they could have experienced greater than 7 days of abstinence at baseline.

Abstinent smokers beyond baseline were defined as self-reporting abstinence from tobacco with validation by exhaled carbon monoxide testing less than or equal to 8 ppm. An intake questionnaire assessed for passive exposure to tobacco, as well as medical disorders and treatments that potentially impacted exhaled carbon monoxide results (Kharitonov & Barnes, 2001). Therefore, patients were assessed for inflammatory lung disorders and the use of inhaled or oral steroid medications.

1.1.4.2. Lapse of tobacco abstinence. A lapse of tobacco abstinence was defined as a puff (or more) on a cigarette following an attempt of tobacco abstinence, regardless of the length of the attempted abstinence. A lapse can occur at any time following the initiation of abstaining from tobacco (smoking). With respect to the primary aim of this study, the time to a first lapse was assessed by self-report. A lapse was considered as an abstinence violations effect [AVE]

(Shiffman, Balabanis, Paty, Engberg, Gwaltney, Liu, Gnys, Hickcox, & Paton, 2000; Shiffman, Hickcox, Paty, Gnys, Kassel, & Richards, 1997). Therefore, a first lapse had to follow the planned date for a quit day of a smoking abstinence effort to be considered the initial AVE. During the administration of the intervention for relapse management, lapses were defined as at least one puff on a cigarette. Self-reported lapses were also documented at follow-up visits. At least six consecutive days of lapsing were required before a definition of a relapse applied.

1.1.4.3. Relapse of tobacco abstinence. Relapse to smoking was defined as self-reported smoking one cigarette for at least 6 consecutive days or a CO reading of greater than 8 ppm (Shiffman et al., 2000). The seventh day of consecutive days of smoking one cigarette was defined as the first day of the relapse. The previous 6 days of smoking a cigarette were considered smoking lapse days.

1.1.4.4. Intervention adherence. For this study, intervention adherence was defined as behavior pertaining to the participation and completion of relapse maintenance activities. Operationally, this type of adherence behavior was defined by a mean summary score, which included scores for completion of intervention homework and participation with telephone intervention session activities. Summary adherence scores were defined for each of these areas by the following formula.

$$\textit{Intervention component adherence} = \frac{\textit{amount completed}}{\textit{total available for completion}} \; X\,100$$

A mean score for these two adherence components was calculated by totaling the component scores and dividing by two. For example, if a subject completed all elements for their assigned homework, the homework adherence score was a summary total of the 8 homework scores (each completed homework counted as a score of 1), divided by 8 possible homework assignments, which was then multiplied by 100. In this case, the adherence for homework was 100% ([8/8] * 100). This approach was also used with the completion of weekly telephone intervention sessions. To continue this example, if the mean summary score for the completion of the sessions was 90% and the mean summary score for homework was 85%, the mean total intervention adherence score was 87.5% ([85% + 90%]/2).

1.1.4.5. Self-efficacy. Individual perception or self-appraisals of confidence to perform a specified behavior defines this term of self-efficacy (Bandura, 1982, 1997). For the purpose of this study, self-efficacy related to relapse management for tobacco abstinence following hospital discharge was operationalized by a summary score for the "Tobacco Abstinence Self-Efficacy Scale" [TASES]. This tool was developed by the investigator to examine general and situational self-efficacy to maintain tobacco abstinence.

1.1.4.6. Perceived self-efficacy for coping. Self-efficacy pertaining to situations for relapse of tobacco use is of interest when promoting abstinence from tobacco. This type of self-efficacy pertains to confidence in maintaining abstinence in the face of specific situational factors, such as negative affect, positive affect, restrictive situations, idle time/boredom, social/food situations, low arousal, and craving (Catley, O'Connell, & Shiffman, 2000; Gwaltney, Shiffman, Norman, Paty, Kassel, Gnys, Hickcox, Waters, & Balabanis, 2001; Shiffman et al., 2000). In other words, the confidence of maintaining abstinence in specific situations provided a measure of an individual's confidence in coping with situations that pose a high-risk threat to abstinence effort.

This type of situational self-efficacy for abstinence effects was operationalized with individual items scaled from 1 (Not confident) to 4 (Extremely confident) on a modified version of the Relapse Situation Efficacy Questionnaire, [RSEQ] (Gwaltney et al., 2001). All subjects were provided with a ranking of their situational self-efficacy for relapse risk prior to hospital discharge. Information pertaining to the hierarchy of responses was used to direct intervention activity for the special intervention group receiving phone calls and enhanced usual care.

1.1.4.7. Outcome expectancy. For the purpose of this investigation, outcome expectancy was defined as an individual's belief or perception of their confidence in the efficacy of their prescribed tobacco dependency treatment ("Stay Quit Study" or "SQS") in lowering their risk for the smoking related comorbidity of heart disease. The Perceived Therapeutic Efficacy Questionnaire for Relapse Management [PTES - RM] is a modified version of the Perceived Therapeutic Efficacy Questionnaire used by adherence research studies at the University of Pittsburgh School of Nursing Center for Research of Chronic Disorders (CRCD). This 10-item tool rated a subject's treatment confidence responses between 0 and 10.

1.1.4.8. Perceived social support. The perception of support or resources available from others during the process of tobacco abstinence has previously demonstrated a relationship with tobacco abstinence efforts (Chandola, Head, & Bartley, 2004; Cohen, Kamarck, & Mermelstein, 1983; Helgason, Tomson, Lund, Galanti, Ahnve, & Gilljam, 2004; Mermelstein, Cohen, Lichtenstein, Baer, & Kamarck, 1986). Furthermore, social support is considered a source of influence for self-efficacy with respect to verbal persuasion (Bandura, 1997). This study variable was operationalized with an individual intake question pertaining to the perception of support for tobacco abstinence from a subject's significant other.

1.1.4.9. Nicotine dependence. The addiction to nicotine been described as producing drug tolerance, physical dependence, and satisfying or enjoyable effects (Benowitz, 1998b; U.S. Department of Health and Human Services, 1988), which requires the user to maintain a chronic consistent intake of nicotine. Nicotine dependence was operationalized by the Fagerstrom Test for Nicotine Dependence [FTND]. This questionnaire was developed as a paper-pencil tool to assess nicotine dependence (Fagerstrom & Schneider, 1989; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991; Pomerleau, Majchrzak, & Pomerleau, 1989). The FTND is a modified version of the Fagerstrom Tolerance Questionnaire [FTQ] (Radzius, Moolchan, Henningfield, Heishman, & Gallo, 2001).

1.1.4.10. Nicotine withdrawal. As tobacco users abstain from their tobacco products, symptoms may be experienced indicating the lack of nicotine intake in the body. These symptoms which have been defined by various sources include: 1) depressed mood or dysphoria; 2) irritability/frustration/anger; 3) anxiety/restlessness; 4) increased appetite/hunger; 5) decreased heart rate; 6) difficulty concentrating/impaired cognitive function; 7) insomnia/sleep disturbance; 8) craving; and 10) somatic complaints of headaches, gastrointestinal disturbances, and dizziness (Hughes & Hatsukami, 1986; Hughes, Gust, Skoog, Keenan, & Fenwick, 1991; Snyder, Davis, & Henningfield, 1989). Nicotine withdrawal was operationalized in this study by use of the Nicotine Withdrawal Form developed by Drs. Hughes and Hatsukami, which contained 12 items with Likert scaling from 0 - 4 (Hughes & Hatsukami, 1986). With respect to Bandura's Self Efficacy Theory and for the purpose of this study, nicotine withdrawal symptoms were considered as a potential physiological influence of self-efficacy for tobacco abstinence (Bandura, 1997).

1.1.4.11. Affective states. Emotional or affective states represent reactions, which may be conditioned responses to antecedents. These affective states may be negatively (e.g., anxiety or sorrow) or positively (e.g., feelings of happiness) charged emotional reactions. Bandura (1997) identified affective states as a potential source individuals draw upon in the process of developing perceptions of self-efficacy. In addition to these conditioned responses to stimuli, the dependence upon nicotine can have an effect upon the mood lability, which leads to increases in feelings of stress and depressive symptoms in tobacco dependent individuals (Hall, Munoz, Reus, & Sees, 1993; Parrott, 2004; U.S. Department of Health and Human Services, 1988). The instrument entitled, "Profile of Mood States [POMS]," was used to operationalize affective states for this project. The POMS has 65 items with Likert scaling from 0 - 4. Specifically, the subscale pertaining to depression and dejection was used to measure depressive symptoms.

1.1.5. Summary.

This study used a randomized controlled design with an intent-to-treat for the primary aim. Intervention subjects received the 12-week relapse maintenance intervention and the control groups received enhanced usual care. Bandura's Self-efficacy Theory provided the guiding conceptual framework for the intervention strategies and assessment procedures (Bandura, 1997). Baseline smoking status was validated with carbon monoxide testing. Follow-up point-prevalence measures were conducted 12 weeks (at the end of treatment) and 24 weeks following discharge. Since treatment adherence may provide insightful information to this relapse maintenance process following hospital discharge, the intent-to-treat approach was not used with regard to research questions pertaining to treatment adherence in the secondary aim.

2. CHAPTER TWO

2.1. LITERATURE REVIEW

2.1.1. Tobacco: A historical perspective

In 1492, Christopher Columbus provided the first documentation of European observations of tobacco consumption by native inhabitants in the American continents and islands (Columbus, 1990). The tobacco plant was taken to the European continent and greeted with mixed reviews over the next two centuries (Baudry, 1988; James I King of England (1566-1625), 1900; Kiernan, 1991; Monardes, 1967; Philaretes, 1936). Monardes, a 15th century physician, believed tobacco to be a medicinal herb of unlimited use (Goodman, 1993; Kiernan, 1991; Monardes, 1967). King James I of England was the first monarch to publicly condemn tobacco smoking and recognized the negative impact it had on the smoker and those passively exposed (James I King of England (1566-1625), 1900). In addition, King James I was the first to institute taxation on tobacco as a means of tobacco control by a governing body (James I King of England (1566-1625), 1900).

Revolutionary changes in the late 1800's greatly impacted tobacco production and consumption for the next century, which included: 1) introduction of tobacco in a cigarette form, 2) development of equipment for the mass production of cigarettes, 3) new tobacco curing techniques, 4) invention of safety matches, and 5) the formation of the American Tobacco Company under the direction of James Duke (Goodman, 1993; Kiernan, 1991; National Institutes of Health, 1997; Tilley, 1948; U.S. Department of Health and Human Services, 1988).

Several historical events in the early to mid 20th century contributed to the increase in cigarette tobacco consumption, such as cigarette advertising, World War I, and World War II (National Institutes of Health, 1997). The peak of cigarette consumption by males occurred following World War I. For women, the greatest peak of consumption, thus far, occurred just prior to World War II (U.S. Department of Health and Human Services, 1985).

Research efforts during the late 1800's through the mid 1900's provided identification of nicotine and the actions of chemical transmitters upon the nervous system. Drs. Langley and Dickinson studied the effect of nicotine upon neurophysiology (Langley, 1889). Their 1889 findings of frog neuron stimulation with nicotine provided the foundation for future studies of neurotransmitters and addictive substances. Chouppe (1888) and Dorsey (1936) each studied the effects of chemicals as neuron blocking antagonists to nicotine stimulation. This research provided a foundation for pharmaceutical preparations engineered to block nicotine uptake at neurotransmitter sites (Chouppe, 1888; Dorsey, 1936). Case study reports, during the early to mid 1900's, provided descriptive information regarding tobacco consumption behaviors, such as smoking. These case study findings provided the foundation for theories and assumptions regarding those individuals that indulge in tobacco consumption (Barnett, 1955; Bergler, 1946; Brill, 1922; Finnegan, Larson, & Haag, 1945; Freedman, 1948; Jacobson, 1943; Johnston, 1942; MacArthur, 1958).

In 1938, Dr. Pearl suggested a link existed between smoking tobacco and the development of cancer (Pearl, 1938). However, the science and medical communities did not seriously consider Dr. Pearl's suggestion until the 1950's. At that time, Drs. Hammond and Horn released their findings of a large cohort study in the U.S., which linked cancer to smoking (Hammond, 1954). At the same time in the United Kingdom, Drs. Dole and Hill reported similar

findings on a smaller cohort sample (Doll & Hill, 1954; Hill, 1956). The findings by these men led a flurry of research activity to confirm or disprove their findings. Eventually, these studies and those that followed led to the U.S. Surgeon General's Report in 1964, establishing tobacco smoking as a cause of lung cancer (U.S. Department of Health, 1964). Since that time, research has further substantiated the health consequences of smoking and benefits of tobacco cessation (U.S. Department of Health and Human Services, 1981, 1982, 1983, 1984, 1985, 1988, 1989, 1990, 2000). The 1988 U.S. Surgeon General's Report focused on the role of nicotine as the key psychopharmacological addictive substance in tobacco products (U.S. Department of Health and Human Services, 1988).

2.1.2. The psychopharmacological connection to tobacco dependency

The primary addictive substance common to all forms of tobacco products is nicotine (U.S. Department of Health and Human Services, 1988). This plant alkaloid, soluble in water and lipids, is further described as a tertiary amine containing both pyridine and pyrrolidine rings (Benowitz, 1998b; Clarke, 1993; U.S. Department of Health and Human Services, 1988). In addition to nicotine, other alkaloids (e.g., nornicotine, anabasine, myosmine, nicotyrine, and anatabineare) are contained in tobacco, some with similar but less effective properties of action than nicotine (U.S. Department of Health and Human Services, 1988). An unidentified compound may also contribute to this addiction process by way of decreasing the availability of monoamineoxidase [MAO] B, which breaks down released dopamine (Fowler, Volkow, Wang, Pappas, Logan, MacGregor, Alexoff, Shea, Schlyer, Wolf, Warner, Zezulkova, & Cilento, 1996; Fowler, Volkow, Wang, Pappas, Logan, MacGregor, Alexoff, Wolf, Warner, Cilento, & Zezulkova, 1998). Nicotine itself has not been implicated in MAO B activity (Fowler, Volkow, Logan, Pappas, King, MacGregor, Shea, Garza, & Gatley, 1998; Fowler, Wang, Volkow,

Franceschi, Logan, Pappas, Shea, MacGregor, & Garza, 1999). Current literature supports nicotine's stimulation of dopamine release in the mesolimbic system as key to the process of addiction (Gamberino & Gold, 1999; Pomerleau, 1992). With over 4000 chemicals in a cigarette, there is potential that this form of tobacco administration provides a combination of chemicals that enhance nicotine's addictive role (Gamberino & Gold, 1999; U.S. Department of Health and Human Services, 1988).

Nicotine's dual soluble nature facilitates transportation, absorption, and metabolism in humans and animal models. Nicotine can be absorbed through the mucosa in the mouth and nose, capillary-alveolar membranes, and topically on the skin (U.S. Department of Health and Human Services, 1988). In tobacco smoke, nicotine is carried on tar droplets to terminal airways and alveoli of the lungs (Benowitz, 1998b; U.S. Department of Health and Human Services, 1988). Speed and dose of nicotine's absorption in the body is controlled by tobacco's pH level and delivery method (Henningfield, Cohen, & Pickworth, 1993; Henningfield & Keenan, 1993). For example, chewing tobacco has a high alkaline pH of 8, which enhances nicotine absorption in the oral or buccal cavity. There is higher oral absorption of nicotine from cigars and pipes due to the pH of the tobacco, which is also more alkaline and similar to chewing tobacco. Furthermore, nicotine delivery by cigarettes is not absorbed in the buccal cavity due to the ionization of nicotine at a lower pH of 5.5 (Gori, Benowitz, & Lynch, 1986; Henningfield et al., 1993). The large capillary-alveolar membrane surface area found in the lungs enhances the rapid delivery of nicotine with cigarette smoking. With pulmonary inhalation of nicotine, arterial loading accelerates the delivery of nicotine to the brain due to the bypass of the venous system

and right side of the heart. Inhalation delivery of nicotine to the brain can occur in 10 to 19 seconds, but drops off dramatically as nicotine is delivered to the peripheral body cells (Benowitz, 1998b).

The liver is the primary site of nicotine metabolism. Nicotine has a short half-life of approximately 2 hours. As nicotine is metabolized, it is converted into metabolites, such as cotinine, which has a half-life of 22 hours, and nicotine n-oxide. The function or roles of nicotine's metabolites are also under study for potential reinforcement of the effects initiated by nicotine administration (Crooks & Dwoskin, 1997). Excretion of nicotine primarily occurs through the kidneys, but the amount excreted is dependent upon urinary pH and flow (U.S. Department of Health and Human Services, 1988).

Although tobacco dependency is a disorder impacting the entire body, the brain is a central target for the initiation of destructive and addictive activities. Cellular and animal studies have provided much of the evidence supporting nicotine's neurotransmitter activity on the central and peripheral nervous system. Studies to date suggest that nicotine is an agonist stimulating presynaptic and postsynaptic nicotine receptors (Goldstein, 1994; Lena, Changeux, & Mulle, 1993; Rosecrans & Karan, 1993). Research of neuronal receptors indicate nicotine binds with nicotinic acetylcholine receptors [nAChRs] and has a particularly high specificity for binding at beta [β] 2 subunits, one of at least 16 known nAChRs subunits (Watkins, Koob, & Markou, 2000). Picciotto, et al. (1998) reported that engineered mice lacking this β2 subunit would not self-administer nicotine. This subunit is considered critical in the reinforcement of nicotine (Picciotto, Zoli, Lena, Marubio, Merlo-Pich, Fuxe, & Chageux, 1998). Evidence also suggests that combinations of nAchRs subunits containing the β2 subunit are located through out the mesolimbic dopamine system (Wada, Wada, Boulter, Deneris, Heinemann, Patrick, & Swanson,

1989; Zoli, Lena, Picciotto, & Changeux, 1998). Therefore, β2 subunits are considered critical in the release of dopamine by nicotine, which is central to the drug reinforcing properties of nicotine (Benowitz, 1998b; Watkins et al., 2000). Watkins, et al. (2000) have suggested that the ventral tegmental area [VTA], prefrontal cortex, amygdala, septal area, and nucleus accumbens are among the structures containing subunits with combinations of β2 subunits and alpha [α] subunits. Acetylcholine released by nicotine stimulation of receptors may contribute the additional release of dopamine in structures, such as the VTA and substantia nigra (Watkins et al., 2000).

Dopamine and acetylcholine are not the only substances released in response to nicotine activation of nAChRs receptors. Nicotine stimulation of receptors is also implicated in the release of other substances. These substances include norepinephrine, epinephrine, β-endorphins (opioid peptides), hormones (e.g., growth hormones, adrenocorticotropic hormone [ACTH], vasopressin), serotonin, glutamate, and gamma-aminobutyric acid [GABA]. Therefore, nicotine activates five neurotransmitter classes, which include: 1) amino acids (e.g., glutamate, GABA), 2) monoamine catecholamines (e.g., dopamine, epinephrine, norepinephrine), 3) monoamine indolamine (e.g., seratonin), 4) acetylcholine, and 5) neuropeptides (e.g., β endorphins) (Pinel, 1997). Among the amino acids, glutamate release has excitatory properties, where as GABA has inhibitory properties. Both of these amino acid neurotransmitters may play a role in the drug reinforcement of nicotine (Watkins et al., 2000). Sympathetic nervous system stimulation by epinephrine is responsible for alterations in cardiac function, such as elevated heart rate and blood pressure (Benowitz, 1992, 1994, 1998b; Clarke, 1993). Although seratonin release is activated by nicotine, limited research is available to distinguish a reinforcement role with nicotine (Watkins et al., 2000). However, seratonin may have an impact with regard to mood

alteration (Benowitz, 1992, 1994). Further research in this area of neurotransmitter activity is necessary to understand the role of nicotine with these neurotransmitters and hormones in the process of relapse to tobacco dependency and potential interventions to prevent relapse.

Information in the 1988 U.S. Surgeon General's Report compared nicotine with other illicit substances, such as heroin and cocaine (U.S. Department of Health and Human Services, 1988). Nicotine, as with these other drugs of dependence, centrally induces 1) psychoactive effects of drug discrimination, 2) reinforcement for continued use, and 3) controlled or compulsive administration (Benowitz, 1998b; Henningfield et al., 1993; U.S. Department of Health and Human Services, 1988). Specific behavior patterns were also identified, which included: self-dosing regardless of associated harm, drug cravings, predictable pattern of personal use, and the process of relapse following abstinence (Benowitz, 1992; U.S. Department of Health and Human Services, 1988). One notable difference between other abused psychoactive drugs and nicotine is drug intoxication. Although drug intoxication can be observed with psychoactive drugs, such as alcohol and opiates, it is rarely observed with tobacco dependency (Clarke, 1993).

As with other substances of abuse, the following are also associated with nicotine addiction: tolerance, physical dependence, and satisfying or enjoyable effects (Benowitz, 1998b; U.S. Department of Health and Human Services, 1988). Nicotine produces physical tolerance as evidenced by the diminished response to each dose over a 24-hour period. From a neural receptor level, tolerance occurs due to the process of neural receptor site stimulation, followed by periods of desensitization before receptors are available for the process to repeat. Furthermore, this process of desensitization and receptor blocking is considered key in the development or activation of additional nicotinic receptor sites in the brain (Benowitz, 1992, 1994, 1998b).

Although nicotine has a peak and trough effect with regard to serum nicotine levels, the overall effect of nicotine rises from the initial morning dose and then plateaus till smokers are abstinent during the hours of sleep (Henningfield et al., 1993). However, there is documented variability in human tolerance responses (Keeley, Pirwitz, Landau, Lange, Hillis, Foerster, Conrad, & Willard, 1996; Perkins, 1995; Pomerleau, Pomerleau, & Marks, 2000). Evidence suggests there is acute tolerance to nicotine with cardiovascular responses, but greater variability of tolerance in behavioral responses (Perkins, 1995; Pomerleau et al., 2000).

Physical drug dependence can also be marked with the observation of withdrawal symptoms following a period of abstinence, which can include: 1) depressed mood or dysphoria; 2) irritability/frustration/anger; 3) anxiety; restlessness; 4) increased appetite/hunger; 5) decreased heart rate; 6) difficulty concentrating/impaired cognitive function; 7) insomnia/sleep disturbance; 8) craving; and 10) somatic complaints of headaches, gastrointestinal disturbances, and dizziness (Hughes & Hatsukami, 1986; Hughes et al., 1991; Snyder et al., 1989). According to the Diagnostic and Statistical Manual (4^{th} edition), nicotine withdrawal, as well as nicotine dependence are recognized substance dependence disorders by the American Psychiatric Association (American Psychiatric Association, 2001). Withdrawal symptoms reach their peak between 1 to 3 weeks following abstinence from nicotine. Furthermore, observation of any of these symptoms of nicotine withdrawal can occur within 24 hours of tobacco abstinence. However, personal variation with

withdrawal symptoms does exist, with reports of initial symptoms within hours after abstinence and lasting from 2 weeks to several months (American Psychiatric Association, 1994; Benowitz, 1992; Hughes & Hatsukami, 1986; Snyder et al., 1989).

Studies have identified relationships between behavior and the process of tobacco dependence, which includes the development of tolerance, drug discrimination, withdrawal symptoms, and relapse following tobacco abstinence (Henningfield & Woodson, 1989; U.S. Department of Health and Human Services, 1988). Although situational factors have been identified as a source of variability between cigarette smokers, they also provide sets of conditioned events prompting individual tobacco consumption behavior (Perkins, 1995; Shiffman, Gnys, Richards, Paty, Hickcox, & Kassel, 1996a). The frequency of these conditioning events is important both within and between tobacco dependent subjects (Perkins, Epstein, & Jennings, 1991).

A complex orchestration of the cellular biological activity noted previously with psychological processes provides the postulated framework for tobacco dependency. From the psychological side of the framework, behavioral cues, such as situational events, enhance the reinforcement of tobacco use and nicotine addiction. The biopsychological interaction may account for the initiation of relapse due to behavioral cueing established during the addiction process. This framework embraces the interaction of psychological, biological, and environmental processes (e.g., social pressures and economics) (Clarke, 1993; Henningfield et al., 1993; Stollerman, 1993). However, more

research is needed to understand the interaction of these constructs to prevent or reverse the psychopharmacological remodeling in the CNS associated with tobacco addiction, as well as alter the process to support nicotine abstinence.

2.1.3. Genetics and tobacco dependency

Genetic predispositions are under investigation as they relate to tobacco initiation, addiction, abstinence, and protective factors related to tobacco use. This area of research has potential to provide more information on inherited variability with regard to tobacco experimentation and dependence, as well as gene therapy for prevention and treatment of tobacco dependency (Perkins, 1995). Genetic predisposition with regard to dopamine activity (e.g., release, reuptake, and inhibition) has provided information regarding a dopamine transporter gene polymorphism (SLC6A3) and receptors (DRD2)(David, Niaura, Papandonatos, Shadel, Burkholder, Britt, Day, Stumpff, Hutchison, Murphy, Johnstone, Griffiths, & Walton, 2003). Studies suggest that individuals with SLC6A3-9 genotypes are less likely to seek smoking as an external reward and experience longer periods of sustained abstinence if they do smoke (David et al., 2003; Lerman, Caporaso, Audrain, Main, Bowman, Lockshin, Boyd, & Shields, 1999; Sabol, Nelson, Fisher, Gunzerath, Brody, Hu, Sirota, Marcus, Greenberg, Lucas, Benjamin, Murphy, & Hamer, 1999).

Defective CYP2A6 alleles have been related to an alteration in nicotine metabolism to cotinine (Tyndale & Sellers, 2001). Results from studies of this genetic defect suggest that males with defective CYP2A6 alleles smoke fewer cigarettes and are less likely to be tobacco dependent. In addition, CYP2A6 without defect has been associated with the activation of tobacco related carcinogen activity. Therefore, defects in this allele appear to provide a protective factor towards the activation tobacco related carcinogens (Tyndale & Sellers, 2001). However, this area of research is limited by technology, and must be kept in mind as discoveries are made. For example, difficulty with previous allele genotyping has led to the discovery of new

technology in mapping and naming CYP2A6 alleles. These newer techniques have provided additional information regarding the activity of this allele in European populations (Zabetian, Gelernter, & Cubells, 2000).

The use of genetic research techniques is also providing an opportunity to look at individual personality factors as they relate to smoking. For example, mediated relationships between neuroticism and smoking have been studied with regard to the presence or absence of 5-HTTLPR S genotypes (Lerman, Caporaso, Audrain, Main, Boyd, & Shields, 2000). Results by Lerman et al., (2000) suggest individuals with 5-HTTLPR S not L (l/l) genotype were positively associated. These findings encourage further examination of individual factors with tobacco dependence.

Research in this area is new and expanding with various limitations. The focus of current research is narrow with concentrated effort directed to specific alleles for tobacco use, dependence, and metabolism of nicotine. Nicotine and tobacco investigators have called on this research area to encompass a systematic approach across manifestations of smoking that will provide a comprehensive accounting of genetic information as it relates to tobacco initiation, dependence, treatment, and risk for smoking related pathology (Pomerleau & Kardia, 1999; Swan, 1999).

2.1.4. Types of tobacco products

The dried leaves of the tobacco plant (e.g., Nicotiana tabacum) are used in combination with other plants and substances in the manufacturing of cigars, cigarettes, pipe tobacco, snuff, and chewing tobacco (U.S. Department of Health and Human Services, 1988). All of these products

provide delivery of nicotine. Cigarettes, pipes, and cigars are typically ignited and stoked (puffing) for product consumption. Snuff and chewing tobacco are rubbed or chewed in the mouth.

2.1.5. Prevalence of tobacco consumption by adults

2.1.5.1. Cigarettes. Although the Surgeon General's Report in 1964 initiated a decline in U.S. tobacco consumption, the prevalence of cigarette smoking has stalled to a slow decline across genders. Progress towards tobacco abstinence and has yet to reach the targets set forth by the Healthy People 2000 campaign (Center for Disease Control and Prevention, 1999f; U.S. Department of Health and Human Services, 1991, 2000). Cigarettes are the most popular form of tobacco purchased in the U.S. The global consumption of cigarettes in 1997 was estimated at 5.3 trillion cigarettes (Barnum, 1994). The average percentage of adults in the U.S. consuming cigarettes was 22% (Center for Disease Control and Prevention, 1999b, 2004d). At least 29% of men and 24% of women smoke cigarettes. On average, Black males (30.1%) smoke more than White (29%), Hispanic (29.2%), and Asian/Pacific Islander (24.1%) males. With regard to females, White females (25.9%) smoke more than Black (22.2%), Hispanic (17.3%), Asian/Pacific Islander (9%) females. However, the percentage smokers within Native American (American Indian & Alaskan Native) male (40.9%) and female (40%) natives is greater than all other ethnicities (Center for Disease Control and Prevention, 1999b, 2004b).

The average smokers, regardless of gender, have between 9 to 11 years of education, range in age between 18 to 44 years of age, and report incomes below poverty status. It is important to note that smoking prevalence by age categories are essentially equal for those in the 18 - 24 year age group as compared to the age group of 24 to 44 years in 1997. Tables presented by the CDC prior to 1997 reported the most prevalent percentage of smokers are in the 25 to 44

year old age category (Center for Disease Control and Prevention, 1999b). Current findings represent a significant change in the age trend and raise concern for the young adult population with regard to their exposure to tobacco, exposure of those around them, and future impact on health, quality of life, life span, and related economics. With regard to females, the age bracket encompassing 18 to 44 years represents the childbearing and raising years. Therefore, smoking prevalence in this age group raises concerns for tobacco exposure and associated mortality and morbidity risks to a fetus during pregnancy and to children.

2.1.5.2. Other tobacco products. Although cigarettes are the most widely used tobacco product, other tobacco products provide avenues to tobacco dependence, such as cigars, pipes, and chewing tobacco. These other products can reach serum nicotine levels near 15ng/ml, but take a longer period of time to reach the serum level than cigarettes. The individual consumption pattern determines the doses received (Benowitz, Porchet, Sheiner, & Jacob, 1988). Cigar smoking among adults was surveyed in 1998 according to "ever cigar smoking" and "past month cigar smoking." For men, over 64% acknowledged smoking at least a few puffs of a cigar, while 10% noted smoking a cigar within 30 days of the survey. Fewer women (16%) compared to men ever puffed a cigar, and even fewer (1%) admitted to smoking a cigar within 30 days of being surveyed (Center for Disease Control and Prevention, 1999e). However, these rates represent an increase in the use of cigars over earlier decades and raise concern for the impact this use of cigars will have on the future prevalence of head, neck and oral cancers.

Surveillance of smokeless tobacco was undertaken in 1991 by the CDC from a representative sample of adults 18 years or older. Findings, at that time, suggested adult men (5.6%) were more likely to use smokeless tobacco products (e.g., snuff, chewing tobacco) than women (0.6%) (Center for Disease Control and Prevention, 1993c). Younger adults between the

ages of 18 to 24 years of age (4.2%) had the highest consumption rate of smokeless tobacco. A comparison of smokeless tobacco use based upon ethnicity indicated American Indian/Native Alaskan populations had the highest rate (5.4%) of consumption as compared to White (3.1%), Black (2.3%), and Asian/Pacific Islander (0.7%) populations in the U.S. This CDC report also noted that 25% of the smokeless tobacco users also smoked cigarettes. The overall prevalence of smokeless tobacco use in 1991 was three times greater than reported consumption in 1972 (Center for Disease Control and Prevention, 1993c).

2.1.6. Prevalence of tobacco consumption by adolescents

New smokers are initiated into tobacco dependence at various ages, but daily consumption of tobacco typically starts during the adolescent years with 3000 youngsters added to the smoking population everyday (McGinnis & Foege, 1993). In little more than 10 years, the smoking prevalence among adolescents has increased (Center for Disease Control and Prevention, 1999b). According to CDC surveillance data from 1997, 42.7% of surveyed U.S. high school students used cigarettes, cigars, and smokeless tobacco. More than 70% of students reported at least one puff or more on a cigarette, while over 36% considered themselves as current smokers of at least 1 day of tobacco product use within 30 days of the survey. Almost 17% of the students reported frequent use of at least 20 cigarettes within 30 days of being surveyed. Approximately 20% of male and female White students were frequent users of cigarettes. Among males, frequent cigarette use was nearly 11% for Hispanics and 7.2% for Blacks. Prevalence of frequent cigarette use was less among female Hispanics (8.1%) and Black (4.3%). The overall use of smokeless tobacco among adolescents was 9.3%. Cigar use was 22% and existed primarily among males (Center for Disease Control and Prevention, 1998). One key to controlling tobacco exposure is the elimination of tobacco initiation and consumption by adolescents (Fiore et al., 2000).

2.1.7. Consequences of tobacco consumption and exposure

2.1.7.1. Mortality. Annually, at least 430,000 deaths in the U. S. are attributed to tobacco exposure (Center for Disease Control and Prevention, 1997c). With regard to the global mortality attributed to tobacco, the World Health Organization estimated that over 4 million individuals died in 1998 as a result of a tobacco-related illness (World Health Organization, 1999). Furthermore, death due to tobacco is anticipated to increase to 10 million by 2030 if current trends in smoking prevalence are not thwarted (World Health Organization, 1999). With most tobacco consumers living in developing countries, tobacco related deaths are projected to escalate in these countries from current estimates of 50% to 70% in 20 years (World Bank, 1999). Follow-up data findings on mortality from the Lung Health Study indicate that smoking cessation has had a dramatic impact with less mortality and morbidity among special intervention subjects who quit smoking as compared to the usual care subjects who continued to smoke (Anthonisen, Skeans, Wise, Manfreda, Kanner, & Connett, 2005).

2.1.7.2. Physiological alterations. Tobacco can have numerous biopsychological effects for individuals actively or passively exposed. With over 4000 chemicals in a cigarette, the effects imparted do not necessarily occur from one chemical, but from the combination of several (U.S. Department of Health and Human Services, 1988). Although nicotine has the primary addictive role in tobacco dependency, it is also responsible for the development of changes resulting in fatal and comorbid disorders. As noted earlier, nicotine activates the release of many neurotransmitters. Cardiovascular stimulation results from the release of catecholamines by nicotine with marked increases in heart rate, cardiac contractility, vascular constriction, serum free fatty acids, and decreases in arterial elastic recoil (Benowitz, 1988a, 1988b; Benowitz et al., 1988; Stefanadis, Tsiamis, Vlachopoulos, Stratos, Toutouzas, Pitsavos, Marakas, Boudoulas, &

Toutouzas, 1997). This increase in fatty acids has been suggested as the cause for increased serum low-density lipids in smokers. Platelet aggregation has also been suggested to result from nicotine stimulation (Chiang, Castleden, & Leahy, 1992). In addition, heart rate variability is decreased, which has been proposed to precipitate sudden cardiac death (Levin, Levin, & Nagoshi, 1992; Yotsukura, Koide, Fujii, Tomono, Katayama, Ando, Suzuki, & Ishikawa, 1998). The repeated activation of these processes contributes to the pathogenesis for cardiovascular disease.

Additional chemicals also participate in this process. Soluble gases, such as carbon monoxide and nitrous oxide are produced with cigarette smoking. The absorption of carbon monoxide interferes with the oxygen transport, which shifts the carboxyhemoglobin curve (U.S. Department of Health and Human Services, 1986). Although the body produces carbon monoxide on a cellular level, the amount entering the blood stream from cigarette smoking is typically greater than what the body normally produces (Pinel, 1997). The effects from smoking, as noted with nicotine and carbon monoxide, contribute to myocardial infarctions, stroke, peripheral vascular disease, hypertension, and sudden death (Benowitz, 1998a; U.S. Department of Health and Human Services, 1983).

Pulmonary remodeling can occur from the repeated use of tobacco (American Thoracic Society, 1996). Cigarette smoking impairs mucociliary function, decreases elastic recoil of the alveoli (leading to air trapping and increased forced vital capacity (FVC)), increases mucus secretion, and accelerates age related decreases in expiratory volume (Beck, Doyle, & Schachter, 1981; Lams, Sousa, Rees, & Lee, 1998; Swan, Roby, Hodgkin, Mittman, Peters, & Jacobo, 1994; U.S. Department of Health and Human Services, 1984). These changes reflect the results of inflammation, ulceration, fibrosis, increase in inflammatory cells, and the inhibition of $alpha_1$-

antiprotease (Hance, Basset, Saumon, Danel, Valeyre, Battesti, Chretien, & Georges, 1986; U.S. Department of Health and Human Services, 1982, 1984, 1985, 1989). Tumor development due to the exposure to carcinogens further impairs pulmonary function (U.S. Department of Health and Human Services, 1982). These changes result in various pulmonary disorders, such as lung cancer, chronic obstructive pulmonary disease [COPD], pulmonary fibrosis, asthma, and respiratory infection (American Thoracic Society, 1996; Le Souef, 2000; Sethi & Rochester, 2000).

2.1.7.3. Morbidity. Premature mortality accounts for one facet impacting those individuals consuming or exposed to tobacco. The four leading fatal chronic disorders, which include cancer (e.g., lung, bladder, cervix), heart disease, stroke, and COPD, are attributed to tobacco (Center for Disease Control and Prevention, 1997c). In the U.S., fatal chronic disorders are associated with 70% of all deaths (Center for Disease Control and Prevention, 1999a). Although these disorders progressively lead to death, afflicted individuals typically encounter decreases in their quality of life, years of productivity, and increases in monetary burdens (U.S. Department of Health and Human Services, 1981, 1990). The consequence of tobacco exposure also includes nonfatal diseases, such as cataract development and periodontal disease. Recovery from illness is also impacted by consumption of tobacco, such as alterations in bone and tissue healing (Christen, Manson, Seddon, Glynn, Buring, Rosner, & Hennekens, 1992; Cuff, McQuade, Scheidt, Sutherland, & Van Dyke, 1989; Mosely, Finseth, & Goody, 1978).

Some evidence has emerged in the last decade linking smoking as a risk factor for the development of autoimmune oriented chronic disorders. Smoking has been independently linked to the reoccurrence of clinical symptoms and endoscopic evidence in individuals diagnosed with

Crohn's Disease, a type of inflammatory bowel disease. In addition, smokers diagnosed with Crohn's are more likely to require surgery for their bowel disorder than nonsmokers (Cottone, Rosselli, Orlando, Oliva, Puleo, Cappello, Traina, Tonelli, & Pagliaro, 1994).

Disease risk, complications, and clinical symptoms for various arthritis disorders, the most prevalent cause of chronic disability in the U.S. (Center for Disease Control and Prevention, 1994), have been associated with smoking (Jonsson, Thorsteinsson, & Valdimarsson, 1998; Lavernia et al., 1999; Saag, Cerhan, Kolluri, Ohashi, Hunninghake, & Schwartz, 1997). Results from animal studies suggest that nicotine may decrease fluid extravasion within joints (Maio, Dallman, Benowitz, Bashbaum, & Levine, 1993). This alteration may be related to the pathogenesis of arthritis manifested by inflammatory processes of the joints. For example, heavy smoking is associated with the development of rheumatoid arthritis in patients lacking a familial tendency (Hutchinson, Shepstone, Moots, Lear, & Lynch, 2001).

Not all chronic disorders or illnesses related to tobacco are prematurely fatal, but do impact upon daily function and quality of life. For example, evidence suggests individuals with a smoking history of 20 cigarettes per day have a 2-fold risk for the development of cataracts over those smoking less than 20 cigarettes per day and than nonsmokers (Christen et al., 1992). Dental disorders, such as tooth loss and

periodontal disease are related to the use of tobacco in various forms, such as cigarettes, cigars, and chewing tobacco (Albandar, Streckfus, Adesanya, & Winn, 2000; Bergstrom, Eliasson, & Dock, 2000; Cuff et al., 1989).

There is a risk for concomitant chemical dependency and tobacco use. Furthermore, tobacco use may often precede the use of other substances of abuse (Henningfield, Clayton, & Pollin, 1990). Previous surveys of alcohol dependent subjects found at least 80% were dependent

upon cigarettes (Center for Disease Control and Prevention, 1997a). With regard to symptoms of dependence, both users of cigarettes and cocaine had a greater frequency of dependency feelings than those using alcohol or marijuana (Center for Disease Control and Prevention, 1995b). There is also risk for disease associated with concomitant use of substances, such as the risk for head and neck cancer among those individuals who drink alcohol and smoke (Vander Ark, DiNardo, & Oliver, 1997). There is increased risk for reoccurrence of cancer, if patients continue smoking and drinking following treatment (Christensen, Moran, Ehlers, Raichle, Karnell, & Funk, 1999). Evidence suggests that patients with a current history for both alcohol and tobacco dependence are more likely to have a greater impairment of general and mental health than patients hospitalized without these co-dependencies (Patten, Schneekloth, Morse, Herrick, Offord, Wolter, Williams, & Hurt, 2001).

Tobacco smoking is also prevalent among patients diagnosed with comorbid psychiatric disorders, in addition to chemical dependencies (Glassman, 1993). For patients diagnosed with schizophrenia, smoking impacts the effectiveness of prescribed medications and decreases the magnitude of some symptoms of schizophrenia due to the dopamine released by nicotine stimulation of receptors in the brain. Nicotine increases the metabolism of treatment medications, which impacts the effectiveness of prescribed medications (Lyon, 1999). As with schizophrenia, similarities in neurotransmitter pathways may provide the underlying explanation for the positive association between smoking and depression, as well depressive symptoms (Covey, Glassman, & Stetner, 1998; Lerman, Caporaso, Main, Audrain, Boyd, Bowman, & Shields, 1998; Quattrocki, Baird, & Yurgelun-Todd, 2000). Smoking has been associated with increased negative affect and depression (Hall et al., 1993). Research evidence suggests some smokers may have a genetic

predisposition for depressive symptoms due to alterations in dopamine transmission, which may be related to the use of smoking to alleviate feelings of negative mood or affect (Lerman et al., 1998).

Finally, the impact of tobacco exposure has been linked to complications, increased costs for treatment, and recovery from illnesses requiring medical or surgical intervention, such as microvascular surgery, plastic surgery, and joint replacements (Craig & Rees, 1985; Grossi et al., 1997; Haverstock & Mandracchia, 1998; Hollinger et al., 1999; Kwiatkowski, Hanley, & Ramp, 1996; Lavernia et al., 1999; Mosely et al., 1978). Both nicotine and carbon monoxide have been identified from tobacco use as potential culprits resulting in impaired bone and tissue healing (Lovich & Arnold, 1994). Findings from animal studies have been used to further evaluate the effect of nicotine upon healing from intervention on tissue and/or bone with evidence linking nicotine to a delay in tissue healing and potential impact upon biomechanical properties of bone (Hollinger et al., 1999; Lovich & Arnold, 1994; Silcox, Daftari, Boden, Schimandle, Hutton, & Whitesides, 1995).

2.1.7.4. Cost. There is substantial cost incurred for the health care of tobacco exposure related illnesses. In 1993, tobacco related illnesses, specifically smoking, carried a collective price tag of $72.7 billion for all associated expenditures across the country in all 50 states and the District of Columbia (Miller, Zhang, Rice, & Max, 1998). The 1993 total of state Medicaid expenditures for smoking related illnesses was $12.9 billion. Approximately 21.7% was used for hospital expenditures (Miller, Zhang, Novotny, Rice, & Max, 1998). Smokers incur higher medical costs than their nonsmoking counterparts. Male smokers require $9,000 more for medical costs over their lifetime than nonsmoking males. Female smokers require $10,000 more than their nonsmoking counterparts. This cost is in addition to the decreased life span and years of

productive life lost as a result of smoking (Hodgson, 1992). More recent estimates have estimated that tobacco incurs a cost $157 billion in healthcare expenditures (Center for Disease Control and Prevention, 2002).

The cost of tobacco exposure entails more than monetary payments for health care. Individuals impacted by the associated illnesses often have altered lifestyles due to diminished functional capacity for activities of daily living from personal hygiene to occupational pursuit. Years of lost productivity are also felt by society with a loss of adults under the age of 65 years in the work force. Such losses to the population at large impact the work force available to contribute to a nation's economy (Center for Disease Control and Prevention, 1993a, 1995a).

2.1.8. Benefits of tobacco exposure elimination

Considering the extent to which tobacco exposure increases the risk for premature death, comorbid health disorders, and poor treatment responses, there is a need for effective interventions to prevent the consequences of tobacco use (Samet, 1992; U.S. Department of Health and Human Services, 1989, 1990). There are noted benefits to decreasing these risks, such as a decrease lung cancer risk over 10 years for former cigarette smokers. As noted previously, reoccurrence of head and neck cancers (e.g., larynx, esophagus, mouth) increase if cigarette smoking and alcohol consumption continue, which suggests a need for these patients to abstain from both substances (Christensen et al., 1999). Risk for other smoking associated cancers (e.g., pancreas, bladder) also decrease with abstinence (U.S. Department of Health and Human Services, 1990).

Cardiovascular disease has been a long-standing and leading cause of death in the U.S. However, smoking abstinence has been noted to decrease the risk for coronary artery disease [CHD] by 50% following 1 year of abstinence. Although the risk for CHD continues to decrease

with abstinence, 15 years of abstinence are required to achieve the same risk as a nonsmoker (Lightwood & Glantz, 1997; U.S. Department of Health and Human Services, 1990). Risk for peripheral vascular disease [PVD] and cerebrovascular disease, particularly strokes, also diminish if abstinence from tobacco exposure can be maintained (Kawachi, Colditz, Stampfer, Willett, Manson, Rosner, Speizer, & Hennekens, 1993; U.S. Department of Health and Human Services, 1990). With regard to PVD, healing following current ulcerative and surgical events are impaired if patients continue to smoke. Abstinence of tobacco use decreases the effect of nicotine and carbon monoxide on wound healing (Hollinger et al., 1999; Lind, Kramhoft, & Bodtker, 1991; U.S. Department of Health and Human Services, 1988).

Tobacco abstinence efforts can dramatically alter the risk for COPD and other pulmonary related disorders. For smokers, one year of abstinence actually provides a "boost" in forced expiratory volumes measured in one second [FEV_1]. FEV_1 lung function measures in former smokers begin to parallel decline of nonsmokers, if abstinence is maintained following the initial year of abstinence (Burchfiel, Marcus, Curb, Maclean, Vollmer, Johnson, Fong, Rodriguez, Masaki, & Buist, 1995; Kanner, 1996). For the asthma patient living in an environment of tobacco exposure, abstinence by the tobacco consumer would decrease asthma exacerbations and risk for infection. Risks for influenza and pneumonia both decline with smoking abstinence and decreased tobacco exposure (U.S. Department of Health and Human Services, 1990).

Complications for surgery and hospitalization are also decreased if tobacco consumers can abstain for short and extended intervals. For example, surgical patients who smoke the morning of surgery are at greater risk for ST segment depression of cardiac function during anesthesia than nonsmokers, exsmokers, and smokers who abstained from smoking the morning of surgery (Woehlck, Connolly, Cinquegrani, Dunning, & Hoffmann, 1999). In addition,

abstinence from smoking prior to surgery also decreases the risk for pulmonary complications associated with coronary artery bypass grafting [CABG] (Warner, Warner, Offord, Schroeder, Maxson, & Scanlon, 1999; Warner, Divertie, & Tinker, 1984). As noted previously, endogenous nitrous oxide is a soluble gas neurotransmitter, which may have a bronchodilator effect when released in the airways (Pinel, 1997; Robbins, Millatmal, Lassi, Rennard, & Daughton, 1997). Evidence suggests that endogenous production of nitrous oxide [NO] may be decreased in smokers and requires at least 6 months of smoking abstinence for endogenous NO levels to increase to that of a nonsmoker (Hill, Ruggeroli, Pohorecki, Alonso, & Robbins, 1995).

2.1.9. Tobacco dependency interventions

The associated risk and effects of tobacco exposure, as well as the benefits of tobacco abstinence are well established (U.S. Department of Health and Human Services, 1988, 1989, 1990, 2000). Various tobacco control interventions are necessary to obtain the benefits of decreased or eliminated tobacco exposure. There are three general targets for tobacco control interventions, which include changes in policies, community awareness, and individual use (Emmons, Kawachi, & Barclay, 1997). Policy and community interventions have the potential for impacting tobacco exposure across a large population in a relatively short period of time. An example of a policy intervention includes "no smoking" policies for public areas and businesses. Taxation of tobacco is another example of policy instituted to impact consumption. Community interventions have included community awareness programs to educate populations of the risks associated with tobacco exposure and institute wide spread interventions to decrease exposure, such as the "Great American Smoke-out," which encourages all smokers to stop smoking for at least one day across the country each year (Center for Disease Control and Prevention, 1997b).

Tobacco abstinence interventions are aimed to impact tobacco dependency behavior. Since the release of the first U.S. Surgeon General's report, the decline in tobacco use has primarily been associated with self-help oriented efforts undertaken by tobacco consumers to quit smoking cigarettes or using other forms of tobacco products. However, this commonly used approach by patients has also been associated with relatively high rates of relapse with only 8 to 25% attaining abstinence (Center for Disease Control and Prevention, 1992). Some patients turn to more intensive therapies (e.g., group interventions, attendance at clinics, medications) or in some cases alternative approaches (e.g., herbal remedies, hypnosis, acupuncture) to change their tobacco use behavior (Center for Disease Control and Prevention, 1992; Curry, 1993; Schwartz, 1992). Health professionals are especially encouraged to promote treatment for tobacco dependency with their patients (Fiore et al., 1996, 2000).

According to the most recent guidelines for the treatment of tobacco use, adequate assessment and promotion of tobacco abstinence should be an integral part of regular clinical care by health professionals (Fiore et al., 2000). The "5 A's" (ask, advise, assess, assist, arrange) were designed as reminders or guides for steps health professionals should take to promote tobacco abstinence with patients and their families. Although promotion of abstinence is a first step, additional interventions aimed to change and maintain new behaviors of abstinence are also necessary to assist patients beyond promotion of the concept (Fiore et al., 2000; Manley, Epps, & Glynn, 1992). Therefore, the currently published clinical practice guidelines offer evidence-based intervention information aimed to assist patients with initiation and maintenance of tobacco abstinence. Treatment methods follow along three different arms, which include pharmacological, non-pharmacological alternative treatments, and cognitive-behavioral interventions.

2.1.9.1. Theories, frameworks, and models. Tobacco dependency treatment has been framed within various theories, concepts, and models. Within the medical setting, one approach has been a disease model of tobacco dependency, which considers the biological addiction and associated manifestations as a disease process. Treatment based upon this model may lean towards medically oriented interventions, such as pharmacotherapies. However, there are limitations with this approach to tobacco dependency. First, findings in the literature suggest pharmacotherapies alone provide limited success. More than 50% of individuals using nicotine replacement medication with minimal supportive interventions relapse to a tobacco consuming behavior within the first year of treatment (Fiore et al., 2000; Silagy, Mant, Fowler, & Lancaster, 2000). The second limitation pertains to the patient's perception of their tobacco dependency as an illness within the disease model framework. For example, treatment effects may be negative if this model approach reinforces perceptions that an individual lacks any personal control over the tobacco behavior (Bandura, 1997; Beck, Wright, Newman, & Liese, 1993; Elder, Ayala, & Harris, 1999).

Other frameworks look beyond the disease process orientation to the behavior of tobacco use. These theories or models are directed to changing behavior with respect to tobacco consumption and include the Health Belief Model (Conrad, Campbell, Edington, Faust, & Vilnius, 1996; Manfredi, Lacey, Warnecke, & Petraitis, 1998; Schmitz, Spiga, Rhoades, Fuentes, & Grabowski, 1999), Cognitive Processing (Elder et al., 1999), Behavioral Modification (Antonuccio, Boutilier, Ward, Morrill, & Graybar, 1992; Rigotti, McKool, & Shiffman, 1994), Theory of Planned Behavior (Norman, Conner, & Bell, 1999), Social Cognitive Theory (Social Cognitive Learning Theory) (Langlois, Petosa, & Hallam, 1999), and Self-efficacy Theory (Condiotte & Lichtenstein, 1981; de Vries & Backbier, 1994; McIntyre, Lichtenstein, &

Mermelstein, 1983; Shiffman et al., 2000). The Transtheoretical Model of Change, also cited in the tobacco dependency literature, has been described as an "eclectic model" for health behavior change (Abrams, Herzog, Emmons, & Linnan, 2000; Perz, DiClemente, & Carbonari, 1996; Prochaska, 2000; Prochaska, DiClemente, Velicer, Ginpil, & Norcross, 1985; Prochaska & Velicer, 1997; Ruggiero, Tsoh, Everett, Fava, & Guise, 2000). Intervention strategies related to these frameworks incorporate concepts such as motivation for behavioral change, self-beliefs, conditioning, barriers, problem-solving, coping strategies, and social support. In addition, some frameworks also include the physiological or somatic aspects related to tobacco dependency, such as substance withdrawal symptoms and affective states (Bandura, 1997; Elder et al., 1999; Prochaska & Velicer, 1997; Schwartz, 1992; Wewers & Ahijevych, 1996; Wewers, Ahijevych, & Sarna, 1998).

An example of a framework incorporating both the disease model and behavioral concepts is the biopsychosocial framework, which considers tobacco dependence as a chronic health disorder. Physiological, behavioral, and environmental factors represent a triad of key constructs for this framework. Furthermore, this framework supports a psychopharmacological treatment, which is a comprehensive approach incorporating pharmacotherapy, counseling, and behavioral therapies (Fiore et al., 2000). In otherwise healthy patients, this combination therapy approach has been suggested as one with the greatest potential with respect to a treatment response for tobacco dependence. However, this may also be the limitation. For those individuals with comorbid disorders and presenting manifestations of those disorders may not be candidates for this approach, particularly if their current medications and treatment have adverse reactions with pharmacotherapies used for tobacco dependence (Groudine & Morley, 1996; Hughes, 1993; Villarreal, Hong, & Omens, 1999).

2.1.9.2. Pharmacological treatment. Currently supported approaches in pharmacotherapy include nicotine medications and sustained release bupropion, which have received Food and Drug Administration [FDA] approval for the treatment of tobacco dependency (Ferry, 1999; Fiore et al., 2000). However, evidence suggests that clonidine (noradrenergic agonist) and nortriptyline (noradrenergic tricyclic antidepressant) have demonstrated evidence of efficacy for the treatment of tobacco dependence, but lack FDA approval for such treatment. These medications also have evidence of risk for substantially more side effects than nicotine replacement and bupropion (Covey, Sullivan, Johnston, Glassman, Robinson, & Adams, 2000; Hall, Reus, Munoz, Sees, Humfleet, Hartz, Frederick, & Triffleman, 1998; Tsoh, Humfleet, Munoz, Reus, Hartz, & Hall, 2000).

Other medications have also been studied for treatment efficacy of tobacco dependence, which include mecamylamine (nicotine antagonist), lobeline (cross tolerance with nicotine), anxiolytics (e.g., buspirone - seroternergic agonist), amitriptyline hydrochloride (tricyclic antidepressant inhibits seratonin and norepinephrine uptake), and fluoxetine hydrochloride (inhibits seratonin reuptake) (Ferry, 1999). Unfortunately, most of these drugs have not provided evidence of efficacy, particularly when used alone for tobacco dependency (Hughes, Stead, & Lancaster, 2000; Stead & Hughes, 2000). A 5-week combination therapy with mecamylamine and nicotine patch suggested promising results with end of treatment (37.5 % versus 12.5%) and 1-year abstinence rates (37.5% versus 4.2%) for the combined treatment significantly higher than the placebo group (Rose, Behm, Westman, Levin, Stein, & Ripka, 1994). Although fluoxetine has not provided evidence of efficacy across smokers, researchers suggest further research of

fluoxetine in populations of depressed cigarette smokers may be warranted (Blondal, Gudmundsson, Tomasson, Jonsdottir, Hilmarsdottir, Kristjansson, Nilsson, & Bjornsdottir, 1999; Dalack, Glassman, Rivelli, Covey, & Stetner, 1995).

Four types of nicotine medications are available in the U.S. These products include gum (polacrilex), transdermal-delivery devices (nicotine patch), nasal spray, and oral inhaler. Nicotine replacement medications provide three avenues of support during efforts to abstain from tobacco, which include: reduction of withdrawal symptoms, decreased reinforcement from nicotine in tobacco, and activation of desired effects of nicotine, such as affective/mood and cognitive changes. Although these nicotine delivery systems offset effects of complete abstinence from nicotine, the delivered doses are usually less than the amount delivered by cigarette smoking. Therefore, recommended therapy includes both nicotine medication and behavioral or counseling therapy. Nicotine medications provide the opportunity for patients to engage in behavioral methods of coping with urges and situational factors with assistance in abating withdrawal symptoms and tolerance (Henningfield, 1995).

Nicotine gum is dispensed over-the-counter [OTC] in either 2 mg or 4mg doses. In addition, flavored products are also available, such as orange and mint flavoring. Two types of transdermal nicotine systems are available for OTC purchase, which differ by dose/hours applied topically. The 24-hour delivery system is available in a three doses of nicotine (21mg, 14mg, 7mg) with a recommended stepped dosing approach over 8 weeks of therapy. The 16-hour system contains 15mg of nicotine and is also recommended for use over 8 weeks. Dosing can be individualized with OTC or prescribed transdermal preparations. Both the nicotine gum and patch provide a slow and stable release of nicotine over time with daily delivery less than what is usually delivered by cigarettes smoking. Refer to Table 1 and 2 for information pertaining to the

delivery method, abstinence effects, pharmacodynamics, and pharmacokinetics. The transdermal systems require approximately three days before reaching maximum dose effects (Fiore et al., 2000; Henningfield, 1995).

Nicotine spray and inhalers deliver nicotine to the nasal mucosa and oral mucosa, respectively. These nicotine medications are rapidly delivered and absorbed. Inhaler dosing is dependent upon inhalations, i.e. which there are an estimated 80 inhalations per 4mg cartridge (Anonymous, 2001; Fiore et al., 2000; Silagy et al., 2000). Abstinence rates also vary with the delivery system and dose of nicotine replacement therapies (Table 1). The estimated abstinence rates are greater with the inhaler and spray than the patch and gum delivery systems (Bohadana, Nilsson, Rasmussen, & Martinet, 2000; Bolliger, Zellweger, Danielsson, van Biljon, Robidou, Westin, Perruchoud, & Sawe, 2000; Hjalmarson, Franzon, Westin, & Wiklund, 1994; Hjalmarson, Nilsson, Sjostrom, & Wiklund, 1997; Schneider, Olmstead, Nilsson, Mody, Franzon, & Doan, 1996). However, treatment adherence may be impacted by the required frequent dosing administrations and unpleasant sensations experienced on the mucosa of the nose and mouth.

Nicotine replacement products have been used with subjects diagnosed with comorbid disorders and/or hospitalized. In hospitalized samples, intervention including the option of using nicotine replacement yielded higher abstinence rates as compared to usual care groups (Miller et al., 1997b; Taylor et al., 1996). However, additional reports in the literature suggest the use of nicotine replacement products in hospital settings is less than 10% as per subject self-report and pharmacy records (Emmons, Goldstein, Roberts, Cargill, Sherman, Millman, Brown, & Abrams, 2000; Rigotti, Arnsten, McKool, Wood-Reid, Singer, & Pasternak, 1999). Three studies have demonstrated the safety of nicotine replacement use for patients diagnosed or recovering from

illnesses, such as coronary artery disease (Joseph, Norman, Ferry, Prochazka, Westman, Steele, Sherman, Cleveland, Antonnucio, Hartman, & McGovern, 1996; Mahamarian, Moye, Nasser, Nagueh, Bloom, Benowitz, Verani, Byrd, & Pratt, 1997; Working group for the study of transdermal nicotine in patients with coronary artery disease, 1994). A recent case study noted the occurrence of hypotension during surgery and suggested a possible interaction between the use of vasopressin and the patient's nicotine replacement patch (Groudine & Morley, 1996). Concerns for drug interactions could be a barrier for prescription of nicotine replacement during hospitalization. However, further research is needed to determine why these medications are not prescribed or used during hospitalization.

Table 1 Nicotine replacement - dose and estimated abstinence effects.

Drug	Dose	Estimated Abstinence
Nicotine Gum	2 mg - 4mg	24% (2mg)
Nicotine Patch	21, 14, 7 mg (24 hr.)	18%
	15 mg (16 hr.)	
Nicotine Inhaler	4 mg/cartridge	23%
Nicotine Spray	1 mg (.5mg/nostril)	31%

Note: Adapted from (Fiore et al., 2000; Silagy et al., 2000)

Minimal adverse reactions, side effects, and drug interactions have been noted in the pharmacology literature regarding nicotine replacement products. Topical reactions to patches and ulcer formation from gum preparations, as well as headaches, represent the vast majority of complaints with these products. Nicotine gum use may also result in gastrointestinal complaints (e.g., nausea, vomiting). These products may be questioned and contraindicated for use with patients: 1) status post an acute myocardial infarction (1-4 weeks), 2) experiencing life threatening arrhythmias, 3) severe or progressing angina pectoris, and 4) pregnancy. Although not contraindicated, patients with the following disorders and/or conditions should be well supervised while treated with nicotine replacement products: vascular disorders, chronic obstructive pulmonary disease (using theophylline), coronary artery disease, gastrointestinal ulcers, renal/hepatic disease, diabetes, and severe hypertension. In addition, due to drug interactions, patients using nicotine replacement products and taking one of the following medications need to be monitored for drug potentiation: acetaminophen, adrenergic antagonists (e.g., prazosin, labtalol), furosemide, imipramine, insulin, oxaepam, pentazocine, propranolol, theophylline, and caffeine. However, adrenergic agonists (e.g., isoproterenol, phenylephrine) may need to be increased when administered with nicotine replacement products (Anonymous, 2001).

Bupropion hydrochloride is an FDA approved drug treatment of tobacco dependency under the trade name of Zyban. It is primarily used for the treatment of major depression under the trade name of Wellbutrin. With regard to the treatment of depression, the anticipated action encompasses blocking the reuptake of seratonin, norepinephrine, and dopamine. The mechanism of bupropion's action in treating tobacco dependency is not known. The standard dose for tobacco dependency treatment requires a loading dose of 150mg of sustained release bupropion

once a day for approximately 3 days. This dose is followed by twice a day dosing of 150mg sustained release tablets for the prescribed treatment interval (e.g., approximately 2 to 3 months). Unlike the nicotine replacement products, patients are prescribed this medication for approximately 1 to 2 weeks before they are scheduled to quit using tobacco products. Please refer to Table 2 for pharmacokinetics (Anonymous, 2001).

Table 2 Pharmacokinetics

	Pharmacokinetics		
Medication	Metabolism	Excretion	Half-life
Nicotine Replacement	Liver, Lungs, Renal	Renal (20%*) Breast Milk	1-2 hrs.
Bupropion, Oral	Liver	Renal	14 hrs.
Clonidine			
Oral	Liver	Renal (70%*)	12-16 hrs.
Transdermal	Liver	Renal (70%*)	12-16 hrs.
Nortriptyline, Oral	Liver	Renal Breast Milk	18-28 hrs.
Fluoxetine, Oral	Liver	Renal (12%*)	2-7 days

Note: Adapted from Mosby GENRx, 2001*(Anonymous, 2001)*

Due to the effect of bupropion for tobacco dependency treatment, a new treatment avenue has been discovered for nicotine dependent patients. A recent study compared bupropion to nicotine replacement medications, as well as a treatment arm receiving both bupropion and nicotine replacement (Jorenby, Leischow, Nides, Rennard, Johnston, Hughes, Smith, Muramoto, Daughton, Doan, Fiore, & Baker, 1999). At 12 months, Jorenby, et al. (1999) reported abstinent rates of 35.5 % for the combination therapy group, 30.3% for bupropion alone, 16.4% for nicotine patch treatment, and 15.6% for the placebo group. Additional findings suggest bupropion may also cost less than nicotine replacement medications (Nielsen & Fiore, 2000). Finally, a recent study has examined the use of bupropion in a population of smokers with COPD (Tashkin, Kanner, Bailey, Buist, Anderson, Nides, Gonzales, Dozier, Patel, & Jamerson, 2001). This study is the first to report findings in population with a comorbid disorder. Tashkin, et al., (2001) reported significantly higher continuous abstinence rates for patients receiving sustained release bupropion at all time points (4-7 weeks, 28% vs. 16%; 4 - 12 weeks, 18% vs. 10%; and 4 - 26 weeks, 16% vs. 9%). Although significantly higher for the bupropion group, Tashkin, et al. (2001) could not replicate the findings for bupropion (alone) found by Jorenby, et al., (1999). To date, results have not been reported regarding the use of bupropion with a sample of hospitalized smokers.

There are associated side effects with the use of bupropion, which include central nervous system (CNS) (e.g., seizures, headache, agitation, confusion), cardiovascular system (e.g., hypertension, hypotension, tachycardia), EENT (eye, ear, nose, and throat) (e.g., blurred vision, auditory disturbances), gastrointestinal system (e.g., nausea, vomiting, dry mouth, increased appetite, constipation), genitourinary system (e.g., impotence, frequency, retention) and integumentary system (e.g., rash, pruritis). This drug is contraindicated in cases of

hypersensitivity, seizure disorders, and eating disorders. Precautions and monitoring are also necessary for those patients with renal and hepatic disease, a recent MI, cranial trauma, pregnancy, or lactation (Anonymous, 2001).

Various medication interactions occur with the use of bupropion, which include increased side effects if taken with alcohol, cimetidine, levodopa, and phenytoin. Bupropion potentiates the effects of the following drugs and may require their doses to be altered: antihistamines, barbiturates, benziodiazepines, CNS depressants, MAO inhibitors, and phenothiazines. Health professionals must also be aware that bupropion may affect laboratory results, such as liver function tests, blood glucose, alkaline phosphatase, and false urinary catecholamines (Anonymous, 2001).

2.1.9.3. Non-pharmacological alternative treatment. In addition to the above pharmacological interventions, tobacco dependent individuals have sought treatment in other forms, such as hypnosis, acupuncture, and herbal remedies. Currently however, these treatments lack support from evidence-based research studies. Not only are few randomized trials available for review of these treatments, available findings report a lack of significance when compared to placebo controls (Lambe, Osier, & Franks, 1986; Waite & Clough, 1998; White, Resch, & Ernst, 1998, 1999; Yiming, Changxin, Ung, Lei, & Kean, 2000). With regard to herbal remedies, there is a great need for more information pertaining to interaction effects between herbal remedies and prescribed therapies. These facts are not well known by the lay population and further research is needed to investigate additional interactions with medications and concurrent health disorders.

2.1.9.4. Cognitive-behavioral treatment. As previously noted, various theoretical frameworks have been used with regard to cognitive-behavioral treatment of substance abuse and specifically tobacco dependency. These frameworks include and are not limited to Behavior Modification,

Social Learning Theory, Self-Efficacy Theory, Health Belief Model, Theory of Planned Behavior, and the Transtheoretical Model of Change. Cognitive-behavioral interventions have been initiated through self-help manuals, individual counseling, and group approaches (Fiore et al., 2000; Schwartz, 1992). In addition, proactive telephone counseling, as an intervention delivery mechanism, has been significantly effective (Reid, Pipe, & Dafoe, 1999; Stead & Lancaster, 2001; Zhu, Tedeschi, Anderson, Rosbrook, Byrd, Johnson, & Gutierrez-Terrell, 2000). Although various frameworks have been used with regard to tobacco intervention research, studies rarely use one specific type of cognitive-behavioral strategy. Instead, there is often a cluster of interventions used, such as problem-solving, social support, stress reduction, and counseling session support strategies (Fiore et al., 2000).

Behavioral modification approaches consider the antecedents and consequences of tobacco use behavior. A conceptual organization of behavioral modification for tobacco dependency treatment begins with the identification of: 1) the problem in behavioral terms, 2) measurable target outcome behavior (e.g., abstinence achieved and maintained for 12 months), and 3) antecedents and consequences of tobacco use. In order to obtain a change in tobacco use behavior, additional steps are necessary and include: setting objectives, implementing various strategies for behavior change (e.g., self-monitoring, counter-conditioning), and evaluating progress (Watson & Tharp, 1997).

Scheduled reduction consumption strategies provide an innovative cognitive-behavioral approach to smoking abstinence. Studies engaging this strategy found greater abstinence rates for participants assigned to scheduled reduction as compared to other treatment assignments (Cinciripini, Lapitsky, Seay, Wallfisch, Kitchens, & Van Vunakis, 1995; Cinciripini, Lapitsky, Wallfisch, Mace, Nezami, & Van Vunakis, 1994; Cinciripini, Wetter, & McClure, 1997). This

type of counter-conditioning uses scheduled smoking time points with progressive increases in time intervals between events. These events are scheduled at times when an individual would likely not smoke and time periods when they would likely smoke are newly designated as scheduled non-smoking time periods. As noted above, the studies using this type of strategy also used a set of other cognitive-behavioral strategies across study groups. The results of these studies provide an interesting strategy for future research and clinical consideration.

Aversive techniques have also been cited in the literature with cognitive-behavioral methods. However, supportive evidence is mixed and current use is rare (Fiore et al., 2000; Hajek & Stead, 2000). This type of intervention focuses on aversive stimuli to assist the process of conditioning towards abstinence from tobacco. Treatments have been categorized into three types, which include electric shock, cigarette smoke, and imaginal stimuli (Colletti, Payne, & Rizzo, 1987; Schwartz, 1992). Among the more noted therapies are the rapid smoking techniques, which have been cited with effective outcomes (Colletti et al., 1987; Fiore et al., 2000; Schwartz, 1992). However, reproducibility of earlier results has been questioned and results in the literature have often been based upon self-reported smoking status for follow-up measures (Colletti et al., 1987). In addition, these interventions have limitations with regard to their impact on the function and response of the cardiopulmonary system during the procedure. Therefore, these procedures are not recommended for patients with severe medical disorders. Due to the physical side effects, poor adherence to using this treatment may be encountered (Colletti et al., 1987). Other aversive interventions include rapid puffing, breath holding, and exposure to stale tobacco odors when used tobacco butts are kept in a jar with water (Schwartz, 1992).

In addition to behavioral approaches, psychotherapeutic approaches have been used within a cognitive treatment model for tobacco dependency to either reduce consumption of tobacco or achieve abstinence by targeting emotional reactions and self-defeating behavior (Beck et al., 1993). Over the course of this type of therapy, maladaptive beliefs and faulty cognitive thoughts are altered. Individuals dependent upon tobacco are helped to identify the emotions and problems that lead to their tobacco consuming behavior. For example, personal beliefs regarding one self, coupled with emotions and addictive beliefs leads to addictive behavior seen with tobacco dependency and other substances of abuse. Once these underlying beliefs or reasons for tobacco use are identified, strategies can be developed and practiced to reduce the intensity and frequency of urges for tobacco use, as well as develop a plan for control. Within this framework, it is important to define cravings and urges. Cravings are the internal yearn for tobacco, which may lead to urges for tobacco. Although these terms have been used interchangeably, an urge refers to the process of acting upon the craving. According to the cognitive treatment model, craving alone does not necessarily lead to tobacco consumption. Urges are the combination of internal beliefs and cravings that may result in the action of tobacco consumption (Beck et al., 1993).

2.1.10. Influencing factors of tobacco abstinence and relapse

Several influencing factors of tobacco abstinence and relapse have been identified in the literature, which include sociodemographic factors (e.g., age, gender, ethnicity, income, education, and social support) (Freund, D'Agostino, Belanger, Kannel, & Stokes, 1992; Kabat & Wynder, 1987; Mermelstein et al., 1986), tobacco-use related factors (e.g., nicotine dependence, abstinence violation, nicotine withdrawal) (Hill, Schoenbach, Kleinbaum, Strecher, Orleans, Gebski, & Kaplan, 1994; Rohren, Croghan, Hurt, Offord, Marusic, & McClain, 1994; Westman,

Behm, Simel, & Rose, 1997), and personal factors (e.g., weight concerns, depression, mood, self-efficacy, motivation, diagnosis of tobacco related disease) (Freund et al., 1992; Haaga, 1990; Rohren et al., 1994). In addition, combinations of these factors have been reported as significant predictors of early smoking relapse (within the first 4 weeks of quitting), such as the pairing of depressed mood and tobacco craving (Swan, Ward, & Jack, 1996), however, further clarity of their role in impacting abstinence and relapse is needed.

2.1.10.1. Sociodemographic factors. Evidence implicating sociodemographic factors as predictors of smoking abstinence suggest older smokers were more likely to quit smoking than younger smokers (Fortmann & Killen, 1995; McWhorter, Boyd, & Mattson, 1990; Murray, Gerald, Lindgren, Connett, Rand, & Anthonisen, 2000; Ockene, Kristeller, Pbert, Hebert, Luippold, Goldberg, Landon, & Kalan, 1994). Murray et al. (2000) noted that older subjects who did not associate smoking with emotional coping were more likely to be abstinent 5 years following randomization in the Lung Health Study. If the analysis is limited to the identification of age as a predictor for tobacco use status, the function of age as a predictor is not clear. Further analysis is needed to clarify whether abstinence in older subjects is a function other variables, such as aging, functional capacity, and/or the impact of tobacco use upon quality of life (health status). However, McWhorter et al. (1990) reported age as an independent predictor of tobacco abstinence among subjects followed in the NHANES I study. In addition, younger ages were predictive of smoking relapse in this sample population. In a retrospective study of tobacco intervention of elderly subjects (N = 613), predictors for tobacco abstinence at six months included hospitalization at the time of intervention counseling, nonsmoking significant other, and greater motivation to quit smoking (Dale, Olsen, Patten, Schroeder, Croghan, Hurt, Offord, & Wolter, 1997). In a study of smoking abstinence following coronary revascularization, subjects

who were older and diagnosed with unstable angina were less likely to continue smoking (Hasdai, Garratt, Grill, Mathew, Lerman, Gau, & Holmes, 1998). Therefore, evidence from these retrospective studies suggests that the combination older smokers and presence of tobacco related medical disorders, which are more often noted in older smokers was associated with smoking abstinence. Wewers et al. (1994) noted hospitalized subjects with illnesses unrelated to tobacco use were less likely to abstain from tobacco following hospitalization than subjects diagnosed with cardiovascular and oncological problems related to tobacco use. Therefore, literature findings suggest that age may not be a consistent independent predictor of tobacco abstinence, particularly if subjects are diagnosed with tobacco related medical disorders or experience hospitalization at the time of they attempt to quit smoking.

Ethnicity has been identified as a possible influencing factor for tobacco abstinence. McWhorter et al. (1990) reported that White subjects were more likely to quit smoking among the sample population followed as part of the NHANES I study. However, the consideration of additional factors may be necessary for the interpretation of findings when studies report ethnic differences in tobacco abstinence and relapse. In poor urban settings, Black males were reported to have the highest smoking rate and the lowest smoking quit rates (Hyman, Simons-Morton, Dunn, & Ho, 1996). In both urban and rural areas, low income was suggested as a factor for a lack of access to preventive services for Black smokers (Hueston & Hubbard, 2000). Ethnic differences have been noted with regard to the number of cigarettes smoked versus cotinine findings among females (Ahijevych & Gillespie, 1997; Ahijevych & Parsley, 1999). Ahijevych and Parsley (1999) noted differences for women who smoked menthol cigarettes. These female smokers, predominantly Black, had larger puff volumes and higher cotinine levels (Ahijevych & Parsley, 1999). These authors suggested that Black females who smoke menthol cigarettes might

not be accurately assessed for tobacco dependence if volume of cigarettes smoked is used as the determinant for level of nicotine dependence. In light of the information presented, ethnicity alone might not contribute as an independent influencing factor in tobacco use, abstinence, and relapse. Therefore, additional factors need to be examined for clarification.

Gender differences have been suggested in various studies of smoking abstinence. Results from the Lung Health Study suggested females were less likely to engage in tobacco abstinence, but gender was not a predictor of relapse across the sample (Nides, Rakos, Gonzales, Murray, Tashkin, Bjornson-Benson, Lindgren, & Connett, 1995). However, as the study advanced to the 36th month of follow-up, females were more likely than males to relapse. The engagement of females in quitting smoking was not different than males in the National Cancer Institute's Community Intervention Trial for Smoking Cessation (COMMIT), however, females were more at risk for relapse within the first ten days of quitting than males (Royce, Corbett, Sorensen, & Ockene, 1997).

Concern for weight gain, a personal influencing factor, is another variable paired with gender differences in the literature as it relates to tobacco abstinence (Perkins, 2001; Perkins, Levine, Marcus, & Shiffman, 1997). More than twice as many females anticipate weight gain with smoking abstinence efforts than males (Pirie, Murray, & Luepker, 1991). However, the impact of weight gain upon abstinence efforts and relapse is inconsistent (Nides, Rand, Dolce, Murray, O'Hara, Voelker, & Connett, 1994; Pirie, McBride, Hellerstedt, Jeffery, Hatsukami, Allen, & Lando, 1992). Findings from the Lung Health Study suggested that weight gain contributed to relapse (Nides et al., 1994). Interventions aimed to augment weight gain have not significantly impacted endpoint measures of tobacco abstinence and relapse, but nicotine replacement and bupropion treatments may delay the onset of weight gain (Holm & Spencer,

2000; Jorenby, Hatsukami, Smith, Fiore, Allen, Jensen, & Baker, 1996; Nides et al., 1994; Perkins, 2001). Weight gain presents a potential barrier to tobacco abstinence efforts in females and cognitive behavioral interventions aimed to defuse weight concerns may hold promise with respect to female concerns for weight gain associated with tobacco abstinence (Perkins, 2001).

Social support is another influencing variable of tobacco abstinence efforts (Murray, Johnston, Dolce, Lee, & O'Hara, 1995; Roski, Schmid, & Lando, 1996). In addition, gender differences have also been related to social support and tobacco abstinence (Bjornson, Rand, Connett, Lindgren, Nides, Pope, Buist, Hoppe-Ryan, & P, 1995; Rice, Templin, Fox, Jarosz, Mullin, Seiggreen, & Lepczyk, 1996). Evidence suggests supportive significant others, particularly marital partners impacts initial and long-term abstinence rates (Murray et al., 1995; Rice et al., 1996). Furthermore, this evidence suggests that tobacco abstinence efforts for males might specifically benefit from social support. In addition to the general population of smokers, social support has been identified as a potential influencing factor of abstinence for pregnant adult and adolescent females (Albrecht, Payne, Stone, & Reynolds, 1998; Lindqvist & Aberg, 2001; McBride, Curry, Grothaus, Nelson, Lando, & Pirie, 1998). Social support measures have included consideration of marital status, living and/or working with smoker(s), and the type of support received by the subject attempting to abstain from tobacco use (Collins, Emont, & Zywiak, 1990; Murray et al., 1995; Rice et al., 1996; Roski et al., 1996).

2.1.10.2. Tobacco-use factors. Nicotine dependence, withdrawal symptoms, and abstinence violation have been examined as predictors of tobacco abstinence and relapse (Hill et al., 1994; Rohren et al., 1994; Westman et al., 1997). The Fagerstrom Tolerance Questionnaire (FTQ), a paper-pencil measure of nicotine dependence, and modified versions of the FTQ have been used to examine the relationship of nicotine dependence to abstinence and relapse (Killen, Fortmann,

Kraemer, Varady, & Newman, 1992; Rohren et al., 1994). This tool was found to correlate with biological validation measures of smoking status (e.g., carbon monoxide and cotinine) and predicted smoking abstinence with non-pharmacological tobacco abstinence treatment (Fagerstrom & Schneider, 1989). However, in studies with pharmacological preparations, such as nicotine replacement (e.g., gum), nicotine dependence by paper-pencil assessment has not predicted relapse or time to relapse (Gilbert, Crauthers, Mooney, McClernon, & Jensen, 1999).

Withdrawal symptoms, including craving, have been associated with early relapse (Killen et al., 1992; Swan et al., 1996). Nearly 50% of subjects (N = 289) subjects enrolled in a nicotine patch trial self-reported "craving" as the reason for their smoking relapse (Norregaard, Tonnesen, & Petersen, 1993). Evidence from a study of withdrawal profiles suggests the occurrence of late withdrawal symptom patterns might also be associated with relapse (Piasecki, Fiore, & Baker, 1998; Piasecki, Niaura, Shadel, Abrams, Goldstein, Fiore, & Baker, 2000). In addition, the occurrence of negative affect paralleled the occurrence of withdrawal symptoms. Among a sample of subjects diagnosed with head and neck cancer, relapse was more likely for those who experienced greater levels of craving and anxiety (Gritz et al., 1999b). For hospitalized smokers (N = 650), the occurrence of withdrawal symptoms and tobacco craving while hospitalized was predictive of violating hospital smoke-free policies (Rigotti et al., 2000). In a study of hospitalized patients recovering from a coronary artery bypass graft (N = 87), four independent predictors of 12-month abstinence included less than 3 previous cessation attempts, abstinence 1 week prior to surgery, motivation to abstain, and lack of difficulty in maintaining abstinence while hospitalized (Rigotti et al., 1994).

Abstinence violation has been a consistent factor associated with relapse (Kenford, Fiore, Jorenby, Smith, Wetter, & Baker, 1994). The use of tobacco within a short time frame of a "quit day" has been predictive of relapse (Garvey, Bliss, Hitchcock, Heinold, & Rosner, 1992; Nides et al., 1995). This type of lapse or abstinence violation near a "quit day" has been predictive of relapse with sample populations of self-quitters, as well as with subjects participating with nicotine replacement intervention trials (Garvey et al., 1992; Nides et al., 1995). An examination of predictors from two nicotine replacement studies reported quit date abstinence and low nicotine dependence (as measured with the FTQ) as significant predictors of smoking abstinence 6 months following initiation of tobacco abstinence (Westman et al., 1997).

As with the previously noted influencing factors, combinations of tobacco related factors with personal factors of depression, mood, and self-efficacy have been associated with abstinence and relapse. For example, the combination of low self-efficacy, depression, and the occurrence of withdrawal symptoms has been associated with relapse (Scholte & Breteler, 1997). With respect to the examination of other combinations, study results suggest females with severe premenstrual symptoms might be at risk for severe nicotine withdrawal symptoms if they initiate a tobacco abstinence attempt during the luteal phase of the menstrual cycle (Allen, Hatsukami, Christianson, & Brown, 2000; DeBon, Klesges, & Klesges, 1995; O'Hara, Portser, & Anderson, 1989; Perkins, Levine, Marcus, Shiffman, D'Amico, Miller, Keins, Ashcom, & Broge, 2000).

2.1.10.3. Personal factors. Since weight concerns and tobacco related disorders were reviewed in conjunction with sociodemographic factors, this section will focus upon mood/depression, motivation, and self-efficacy as predictors of tobacco abstinence. Although research results have identified the presence of negative mood, depression, low motivation, low self-efficacy as predictors for relapse, other studies have considered these variables in combination to predict

relapse (Bolman & de Vries, 1998; de Vries & Backbier, 1994; Hall, Munoz, Reus, Sees, Duncan, Humfleet, & Hartz, 1996; Kinnunen, Doherty, Militello, & Garvey, 1996; Shiffman, Hickcox, Paty, Gnys, Kassel, & Richards, 1996b). For example, study results of a Finnish sample of smokers (N = 3,403) found an association between high depression scores on the Beck Depression Inventory (BDI) and low smoking cessation self-efficacy, particularly in males when adjusted for smoking consumption rate (Haukkala, Uutela, Vartiainen, McAlister, & Knekt, 2000). For female smokers in this sample, high depression was associated with motivation to quit smoking.

Depression has emerged as a variable with an impact upon abstinence and relapse, as well as a tobacco-use related chronic disorder with increased risk of emerging as a result of the process of quitting tobacco use (Hall et al., 1993). Subjects with depressive symptoms at the time of an abstinence attempt also report more severe nicotine withdrawal symptoms as compared to subjects lacking depressive symptoms. In a study investigating the use of clonidine for tobacco abstinence treatment, the occurrence of major depression was a significant risk factor for tobacco abstinence treatment failure (Glassman, Covey, Dalack, Stetner, Rivelli, Fleiss, & Cooper, 1993). The risk for development of major depression following tobacco abstinence has been reported to increase as a function of previous depressive episodes (Covey, Glassman, & Stetner, 1997).

Tobacco abstinence may precipitate increased negative mood, which may be related to the withdrawal process. Study results indicate that treatment with nicotine replacement medication led to a decrease in negative mood and fatigue, as measured by the Profile of Mood States inventory (POMS) (Gentry, Hammersley, Hale, Nuwer, & Meliska, 2000). Negative mood

prior to a lapse in abstinence has been associated with difficulty in lapse recovery. However, negative mood experienced following a lapse in abstinence has been associated with increased lapse recovery (Borland, 1992). Lapses monitored with real-time palm computer assessments were related by subjects to negative mood and smoking cues (Shiffman, Paty, Gnys, Kassel, & Hickcox, 1996c).

Motivation towards abstinence has been associated with tobacco abstinence attempts and abstinence assessed upon follow-up (Hill et al., 1994; Rigotti et al., 1994). Intention to quit tobacco use or motivation has been associated with smokers diagnosed with tobacco related disorders (Ho, 1998). With respect to smokers screened in medical clinics or acute health care institutions, intention or motivation to quit tobacco use has been predictive of later abstinence (Richmond, Kehoe, & Webster, 1993; Rigotti et al., 1994). Smokers identified at work site screening, particularly labor-oriented positions, have been less motivated towards tobacco abstinence than management oriented employees (Abrams & Biener, 1992).

Self-efficacy has been identified as a moderate and consistent predictor of tobacco abstinence and relapse (Bolman & de Vries, 1998; Condiotte & Lichtenstein, 1981; Gulliver, Hughes, Solomon, & Dey, 1995; Gwaltney et al., 2001; Karanci, 1992; Yates & Thain, 1985). Karnaci (1992) reported in a study of 174 smokers that high self-efficacy was related to habit situations while low self-efficacy was more often associated with affective situations. With respect to motivation, high self-efficacy was associated with intrinsic motivation and later stages change associated with the Transtheoretical Model of Change. Low self-efficacy was associated with smokers categorized as precontemplators or contemplators (Bolman & de Vries, 1998). In an early descriptive study of smokers (N = 45) diagnosed with pulmonary disease, high self-efficacy was predictive of short-term abstinence at 1 and 3 months following the initial interview

(Devins & Edwards, 1988). Condiotte and Lichtenstein (1981) provided early evidence that low self-efficacy related to specific situations was associated with relapse and the situation responsible for the relapsed.

More recent studies have focused upon looking at self-efficacy over the course of a tobacco dependency intervention (Haaga & Stewart, 1992; Shiffman et al., 2000; Shiffman et al., 1997; Spanier, Shiffman, Maurer, Reynolds, & Quick, 1996). For example, Gulliver, et al. (1995) found that as self-efficacy declined over the course of a tobacco abstinence intervention, subjects were more likely to relapse to tobacco use. The self-efficacy scale incorporated an assessment of situational factors relevant to self-efficacy in smoking abstinence. Dr. Shiffman has completed and contributed to several studies examining the concept of self-efficacy as it relates to self-efficacy (Gwaltney et al., 2001; Shiffman et al., 2000; Shiffman et al., 1996b; Spanier et al., 1996). Shiffman, et al. (1997) reported the impact of smoking lapses resulted in increased negative affect and diminished self-efficacy. Furthermore, self-efficacy assessed on a daily basis predicted lapses on following days and more likely to predict relapse than baseline self-efficacy, which agrees with findings by Gulliver, et al. (1995) (Shiffman et al., 2000). However, smokers who experience relapse are more likely to rebound back to abstinence if they experienced higher self-efficacy following the relapse event (Spanier, et al., 1996). Research results of self-efficacy with lapses suggested moderate self-efficacy following a lapse was predictive of maintained abstinence longer than, but those subjects with low self-efficacy following a lapse (Haaga & Stewart, 1992). Haaga and Stewart (1992) suggested that these findings were consistent with Self-efficacy theory and behavior change. Self-efficacy of moderate level following a lapse is more likely to assist a smoker to maintain abstinence as compared to smokers with high or low self-efficacy following a lapse.

2.1.11. Treatment adherence

Limited information is available in the literature with regard to treatment adherence specific to tobacco dependency (Burke & Dunbar-Jacob, 1995; Cooper, Klesges, Debon, Zbikowski, Johnson, & Clemens, 2005; Kamarck & Lichtenstein, 1988; Killen, Fortmann, Davis, & Varady, 1997). Treatment adherence to both pharmacological and cognitive-behavioral interventions are sparingly reported in the literature (Swan, Valdes, Ring, Khroyan, Jack, Ton, Curry, & McAfee, 2004). Although tobacco dependence treatment studies use biological assays (e.g., cotinine, exhaled carbon monoxide, thiocyanate) to confirm self-reported smoking status, these methods are not defining adherence to the treatment, but clarifying self-reported behavior.

Findings from the treatment adherence literature suggests that predictors of adherence may include cognitive-motivational factors, affective states, and previous adherence to the behavior of interest (Dunbar-Jacob, Schlenk, Burke, & Matthews, 1998a). There are predictive factors for tobacco abstinence that parallel these findings regarding predictors of adherence. For example, affective states (particularly negative mood states), previous lapses or abstinence violation, low self-efficacy, and motivation have been associated with relapse in tobacco dependency programs (Hall et al., 1993; Shiffman et al., 2000; Shiffman et al., 1996b; Swan et al., 1996). Kamarck (1988) reported a significant relationship between program adherence and use of coping strategies to abstinence outcome, illustrating the importance of monitoring adherence to tobacco treatment.

A wide range of theories and models have been used in the research of treatment adherence, which are similar to those used in other health behavior research (Dunbar-Jacob et al., 1998a; Dunbar-Jacob, Schlenk, & Caruthers, 2002b; Elder et al., 1999). With regard to tobacco dependence, the Social Cognitive Theory has been proposed as a framework for adherence

intervention specific to nicotine dependence treatment (Abrams, Borrelli, Shadel, King, Bock, & Niaura, 1998). A self-regulatory method with strategies to enhance self-efficacy outlined the foundation for this proposed approach. Furthermore, this framework and intervention closely parallels relapse prevention efforts within the framework of the Relapse Prevention Model (Marlatt, 1979) and Self-Efficacy Theory (Bandura, 1997).

Measurement of treatment adherence depends to some degree upon the treatment behavior under observation. With regard to medication treatment adherence, various methods have been used, which include direct observation, biological assays, self-report, diaries, clinic pill counts, electronic devices, and pharmacy records (Dunbar-Jacob, Sereika, Rohay, & Burke, 1998b). Direct observation of drug ingestion provides one of the most reliable observations of medication adherence, however, there are limitations pertaining to feasibility and cost of implementing this method across studies. Biological markers have been used to validate drug ingestion. Reliability of this measurement is hampered by adequate information of drug pharmacodynamics. In addition, individual characteristics may also impact results, such as individual metabolism rates.

Within the tobacco treatment literature, cotinine measures have been employed to monitor adherence to nicotine replacement. However, nicotine metabolism rates can impact this measurement. As noted earlier, there is evidence of genetic differences in nicotine metabolism. Pill count and self-report measure have also been used in various studies, but these measures are limited by memory of drug taking events. In addition, the phrasing of the adherence questions for self-report may influence a subject's response (Dunbar-Jacob et al., 1998b). Self-report with a diary format was used to provide patterns of nicotine gum use for the treatment of tobacco

dependence (Killen, Fortmann, Newman, & Varady, 1990). However, patterns of medication use or adherence were not disclosed. In a study of adherence with nicotine patch treatment, a dispensing log of nicotine patches supplied to subjects and a count of returned used patches was used to determine adherence to nicotine patch administration (Alterman, Gariti, Cook, & Cnaan, 1999). Adherence to prescribed patch therapy was approximately 50%.

Electronic measurement of medication use is an indirect measurement that often provides a lower estimate of adherence than self-report measures (Dunbar-Jacob et al., 1998b). Electronic event monitors, such as the electronic Drug Exposure Monitor (eDEM) manufactured by AARDEX Corporation, contain microchips in a medication bottle cap (AARDEX, 1998). The opening and closing of the cap on the bottle actuates the time stamping of the assumed medication administration. There have been no tobacco treatment studies to date with published reports of the use of this device for medication treatment adherence. However, the Lung Health Study used canister weights and an inhaler chronolog to time stamp the administration of an inhaled pulmonary medication provided to subjects in this tobacco dependence treatment study. A limitation noted upon analysis of the data was dose dumping prior to clinic follow-up visits (Rand, Nides, Cowles, Wise, & Connett, 1995; Simmons, Nides, Rand, Wise, & Tashkin, 1996; Tashkin, Rand, Nides, Simmons, Wise, Coulson, Li, & Gong, 1991). An additional limitation with the eDEM includes the disruption of patient use of personal medication organizers. Due to the technology available with the eDEM, observations of medication administration patterns are enhanced with opportunities to explore the activities underlying observed nonadherence patterns (Rand et al., 1995; Simmons et al., 1996; Tashkin et al., 1991).

However, dissemination of this type of adherence monitoring has been limited in literature. There is a need to track and describe this type of treatment adherence with both pharmacological and cognitive-behavioral tobacco abstinence interventions. Enhancing our understanding of adherence to tobacco dependency treatment may assist in the development of interventions that will not only improve treatment adherence, but also improve outcomes in abstinence.

2.1.12. Relationship of tobacco dependence treatment and medical treatment adherence

Evidence suggests current smokers are at risk for poor adherence to prescribed medication treatment. For example, current smokers had poor adherence to prescribed medication administration of aspirin for the preventive treatment of myocardial infarctions (Glynn et al., 1994). Adherence rates for current smokers ranged from 10.3% and 14.6% in the placebo and active drug treatment groups. In a study of hypertensive patients receiving medication, the 40% of current smokers were most likely to change or stop taking their medication without physician advice (Weir et al., 2000). A smaller percentage of current smoking patients (29%) were adherent to their medication regimen. Furthermore, current smokers were likely to be categorized as non-adherent due to forgetfulness in medication administration. A study using electronic event medication monitors was used to assess adherence to the antihypertensive medication Trandolapril and found current smoking status to be the first independent predictor of overall medication adherence with an odds ratio of 1.65 ($p = .0001$) (Vaur et al., 1999). In addition to medication adherence, current smokers have been associated to poor adherence with glycemic control (Toljamo & Hentinen, 2001), medical treatment associated with post-heart transplantation (Dew et al., 1996), and self-extubation from ventilatory support (Atkins et al.,

1997). Although current smoking is a risk for poor treatment adherence, it is not clear when or if smokers abstaining from tobacco obtain improvement in their medical treatment adherence within a short or extended time frame from initiating tobacco abstinence.

In addition, there are gaps in the literature specific to the impact of tobacco dependence treatment upon medical treatment adherence for comorbid disorders. For example, a recent case study noted concern for poor treatment adherence to oncological treatments and associated development of psychiatric comorbidity in response to unsupervised nicotine withdrawal (Moadel et al., 1999). In this case, medication and support by the health professionals assisted in the alleviation of the adherence problems identified. Gritz et al. (1999) also suggested the need for medical supervision of patients abstaining from tobacco and experiencing nicotine withdrawal symptoms as these subjects were receiving care for head and neck cancer. The sparse availability of adherence information with regard to tobacco dependence treatment and limited concern for potential interactions of unsupervised nicotine abstinence in comorbid populations emphasizes the absence of descriptive adherence information of hospitalized tobacco dependent populations diagnosed with comorbid disorders and managing complex treatment regimens. Future research is needed to fill these informational gaps pertaining to adherence of tobacco dependent individuals. Building upon the descriptive information towards the development of interventions promoting treatment adherence and abstinence may provide a key towards decreasing relapse.

2.1.13. Hospitalization and tobacco dependence intervention

Hospitalization for medical-surgical procedures has been viewed as an opportunity for the introduction of tobacco abstinence and related intervention. Although the setting may be consistent across studies in the literature, there is variation in the interventions, procedures,

subjects, and outcomes of abstinence. In comparing the intervention of these studies, differences were apparent with regard to the inclusion of follow-up intervention contact and/or relapse prevention interventions, as well as intervention intensity. With regard to follow-up and relapse prevention, studies of hospitalized smokers can be categorized in the following manner: 1) interventions solely provided during hospitalization (Joseph, Nichol, & Anderson, 1993; Pederson, Wanklin, & Lefcoe, 1991), 2) interventions provided during hospitalization with less than 4 follow-up calls (Rigotti et al., 1997), and 3) interventions provided during hospitalization with a relapse prevention component offered following hospital discharge and/or 4 or more intervention follow-up sessions (DeBusk et al., 1994; Dornelas et al., 2000; Froelicher, Li, Mahrer-Imhof, Christopherson, & Stewart, 2004a; Griebel, Wewers, & Baker, 1998; Johnson, Budz, Mackay, & Miller, 1999; Miller et al., 1997b; Molyneux, Lewis, Leivers, Anderton, Antoniak, Brackenridge, Nilsson, McNeill, West, Moxham, & Britton, 2003; Neighbor, Stoop, & Ellsworth, 1994; Polednak, 2000; Ratner et al., 2004; Rigotti et al., 1997; Rigotti, Singer, Mulley, & Thibault, 1991; Simon, Solkowitz, Carmody, & Browner, 1997; Stevens et al., 1993; Stevens et al., 2000; Taylor et al., 1990; Taylor et al., 1996; Wewers et al., 1994; Wewers et al., 1997). Please refer to Table 3 for abstinence rates for nine of the studies cited above.

Follow-up contact may be critical with achieved abstinence rates for this population. As the average length of hospital admissions decrease, fewer opportunities are available to interact with hospitalized smokers before they are discharged to their familiar home environment, which may encourage relapse to smoking. Studies with fewer than 4 follow-up contacts did not have significant differences between their treatment and usual care groups (Joseph et al., 1993;

Pederson et al., 1991; Rigotti et al., 1997). Table 4 provides a comparison of intervention components and frequency information regarding telephone follow-up counseling calls for six studies.

Table 3 Abstinence rates for nine studies of hospitalized smokers

		Treatment group %(n)	Usual care group %(n)	Treatment group %(n)	Usual care group %(n)
Authors	n	6-month	Follow-up	12- month	Follow-up
Taylor et al., 1990	166	-	-	61% (84) [c]	32% (82)
DeBusk et al., 1994	685	-	-	70% (292)[a]	53% (293)
Taylor et al., 1996	628	40% (315) [c]	26% (313)	31% (315) [b]	21% (313)
Rigotti et al., 1997	615	15% (325)	37% (325)	-	-
Simon et al., 1997	324	22% (143)	14% (131)	15% (157) [a]	8% (142)
Johnson et al., 1999	102	46% (52)	31% (50)	-	-
Dornelas et al., 2000	100	67% (54) [a]	43% (46)	55% (54) [a]	34% (46)
Froelicher et al., 2004	177	52% (65)	41% (52)	48% (58)	42% (52)
Ratner et al., 2004	237	31% (29)	20% (22)	27% (22)	26% (23)

[a] $p = .05$, [b] $p = .006$, [c] $p = .001$

Table 4 Comparison of intervention components across studies of hospitalized smokers

	Taylor 1990	Taylor 1996	Rigotti 1997	Simon 1997	Johnson 1999	Dornelas 2000
Conceptual Framework	SLT[a], NA[b]	SLT[a], NA[b], RPM[c]	TTM[d]	SLT[a], TTM[d]	TTM[d]	TTM[d]
Type of Interventionist	Nurse	Nurse	Research Assistant	Public Health Coordinator	Nurse	Psychologist
No. of hospital sessions	1	1	1	1	2	1
No. of weekly calls in 1st month	2 - 3	2 - 3	1 - 2	3	4	2
No. of calls after 1st month	4	1	? 1	2	2	5
Used manual & media tape	Both	Both	Manual	Video	Video	No
Prescribed Rx	Yes	Yes	No	Yes	No	No

[a] SLT Social Learning Theory, [b] Nicotine Addiction, [c] Relapse Model
[d] Transtheoretical model of change

Each of the studies compared in Table 4 used a conceptual framework to direct the study and intervention methods. Several studies used the Transtheoretical Model of Change (TTM). Johnson et al. (1999) acknowledge the importance of the role of self-efficacy in the TTM. Three of the studies used Social Learning Theory (SLT) within the conceptual framework. Both of the intervention studies by Taylor and colleagues (1990; 1996) incorporated a nicotine addiction model and the later study also included the relapse prevention model. In the earlier study by Taylor et al. (1990), self-efficacy was assessed for situational efficacy pertaining to relapse. The nurse interventionist used this information to initiate counseling procedures with subjects and results from this study were significant with a moderate to large effect size for an intervention study. Contents of the intervention may have also been an important determinant of the success of the intervention. Of the studies listed on Table 4, those studies that acknowledged providing coping strategies were the only studies with significant abstinence rates for the treatment group (Dornelas, et al. 2000; Simon et al., 1997; Taylor et al., 1990; Taylor et al., 1996). Most of these studies also provided a manuals, audiotapes, and videotapes as part of the instructional process, which is noted in Table 4. In addition, usual care activities typically included promotional messages of abstinence, brief counseling, and information (e.g., manuals, brochures) (Dornelas, et al. 2000; Johnson et al., 1999; Rigotti et al., 1997; Simon et al., 1997; Taylor et al., 1990; Taylor et al., 1996).

Intervention strategies ranged from minimal to intensive and with or without nicotine replacement options. Although nicotine gum was available and offered to patients conducted by Taylor et al. (1990), only 5 subjects opted for its use and only 3 eventually were abstinent by study end. Those with intensive interventions and follow-up sessions, as well as options for nicotine replacement, had significantly higher quit rates at 12-month follow-up visits than groups

or studies with minimal interventions and no nicotine replacement options (DeBusk et al., 1994; Lewis, Piasecki, Fiore, Anderson, & Baker, 1998; Miller et al., 1997b; Simon et al., 1997; Stevens et al., 1993; Taylor et al., 1990; Taylor et al., 1996).

Finally, authors noted differences in abstinence rates with respect to admitting diagnoses and the existence of tobacco related illnesses (Lewis et al., 1998; Wewers et al., 1994). For example, Lewis et al. (1998), reported a significant difference towards tobacco abstinence for patients diagnosed with respiratory related disorders as compared to other diagnoses ($p = .00001$). Wewers, et al., (1994) reported similar findings with respect to a comparison of general medical/surgical, cardiovascular, and oncology patients. Abstinence rates were greater in the cardiovascular and oncology groups.

Although studies have demonstrated a relationship between self-efficacy and tobacco abstinence (Gwaltney et al., 2001; Shiffman et al., 2000; Shiffman et al., 1996b), few studies for this tobacco dependent population targeted intervention efforts to enhance related self-efficacy. The study reported by Dornelas et al., (2000) did incorporate self-efficacy within the conceptual framework for the research and noted low self-efficacy as a predictor of relapse. Johnson et al., (1999), incorporated the use of self-efficacy measures, but did not find differences in self-efficacy between treatment and usual care groups. Subjects lost to follow-up may have impacted available data pertaining to self-efficacy in this latter noted study, meanwhile illustrating the loss of power with missing information from follow-up sessions.

In summary, these studies demonstrated consistent findings that higher intensity programs with adequate follow-up and provision of counseling for coping strategies for relapse prevention could obtain greater abstinence at 12 months than interventions studies providing minimal or brief assistance. However, these studies did not provide adequate information to

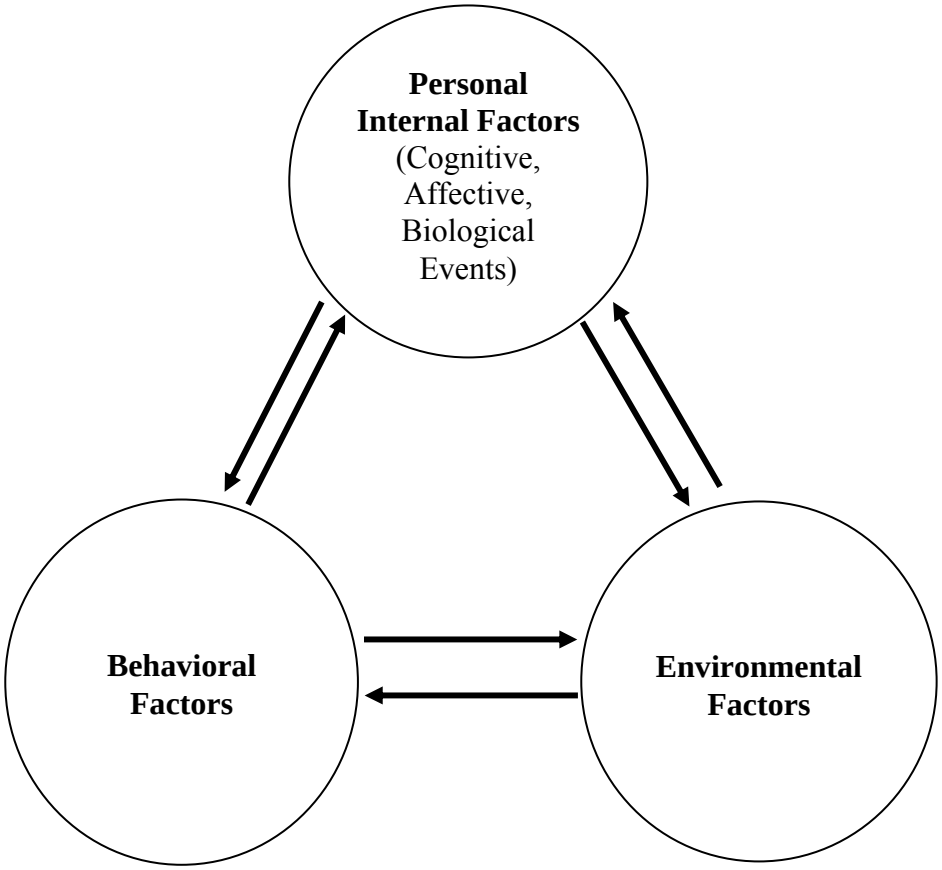
determine if a low intensity in-hospital program and high intensity relapse prevention program can obtain long-term abstinence. Unfortunately, most of these studies did not adequately describe their follow-up intervention activities. Although some authors provided the length of a telephone follow-up session, this information did not provide disclosure of the content or adherence to the follow-up intervention protocol by project staff. Therefore, there remains a need to further investigate conceptually driven interventions that focus particularly upon enhancing self-efficacy, such as the initial study conducted by Taylor and colleagues (1990). Intervention efforts aimed to enhance self-efficacy have the potential to strengthen abstinence efforts and offer necessary personalizing of the intervention for the hospitalized smoker. Finally, interventions need to examine the importance of adequate follow-up counseling to prevent relapse following an abstinence attempt motivated by a hospital admission.

2.1.14. Conceptual framework

Social Cognitive Theory (previously Social Cognitive Learning Theory) has been used as a framework to promote smoking abstinence (Hovell, Jones, & Adams, 2001; Martin, Froelicher, & Miller, 2000; Osler & Jespersen, 1993) in part because it provides constructs pertinent to the adoption or change in a behavior (Bandura, 1997). In order to change a behavior, an individual must self-examine and learn pertinent information and skills, as well as related personal beliefs (Bandura, 1989). These activities of acquisition are necessary to control cognitive processes, emotional states, and action specific to the behavior in question. Although the process can be applied to a particular behavior, the information, skills, and beliefs are general in scope and applicable for use across situations and events for one behavior, as well as with new behaviors. Furthermore, the successful process of adopting a new behavior requires hardiness to sustain the behavior and flexibility towards unanticipated or adverse events (Bandura, 1997).

The underlying framework and assumption of the Social Cognitive Theory rests on a triad of classes of determinants with reciprocal causation to each other (see figure 1). These constructs include personal, behavioral, and environmental factors (Bandura, 1986). Therefore, an alteration or change impacting one of these factors will eventually impact the two remaining factors. In order to make a lasting behavioral change, one must consider making an impact on at least the personal and environmental factors. For example, by taking tobacco products out of the environment, it does not guarantee that tobacco abstinence behavior will ensue from this intervention. However, by intervening on the environment and aspects of the individual, such as coping strategies to cues and cravings for tobacco, the impact on the behavior should be greater than intervention on the environment alone.

The mechanism for this change in behavior requires conscious effort on the part of the individual. Furthermore, this change in behavior is mediated by cognitive processing with coinciding successful performance (Bandura, 1977). As part of this cognitive processing, personal perceptions and beliefs are important mediators. Therefore, a key mediating construct of this theory required for personal agency (action) is that of self-efficacy, which governs self-beliefs (cognitions) of confidence in achieving a change in behavior, as well as the confidence in taking action (Bandura, 1997).



(Adapted from: Bandura, A. (1997). *Self-efficacy: The exercise of control*.

(pp. 5-6).New York, NY: W.H. Freeman Company.)

Figure 1 Determinants in triadic reciprocal causation

2.1.14.1. Self-efficacy theory. Bandura (1997) noted that Self-Efficacy Theory is an important component within the complex structure of the Social Cognitive Theory because of its bearing upon motivation, action, and cognitive acquisition. In addition, Self-Efficacy Theory has been defined as a belief system with structure, function and effect on human agency for change, which

encompasses self-regulation of action, cognitive processes, motivation, and somatic states (Bandura, 1997). Self-efficacy theory has been used as a conceptual framework in the treatment of addictive behaviors, such as tobacco dependency and will provide the framework for this research (Bandura, 1997; Dornelas et al., 2000; Gwaltney et al., 2001; Shiffman et al., 2000; Shiffman et al., 1996b).

In order to modify and maintain a health behavior within this conceptual framework of self-efficacy, previous and proposed efforts have used intervention approaches that incorporated self-regulation (Abrams et al., 1998; Berg, Dunbar-Jacob, & Sereika, 1997). Bandura (1997) suggests that in addition to perceived self-regulation, beliefs of required performance of a treatment and recovery from lapses or relapses in behavior are necessary to initiate and maintain a change in behavior. Therefore, perceptions to act on the behavior impact motivation and initiation in the process while perceptions of self-regulation and lapse/relapse recovery impact ongoing maintenance of the newly acquired behavior.

Realizing that changing behavior, such as tobacco use, requires maintaining or enhancing self-efficacy, it is important to understand the sources an individual will draw upon in defining their perceived self-efficacy. According to Bandura (1977, 1997), there are four informational sources for self-efficacy: performance or mastery experiences, vicarious experience, verbal persuasion, and physiological and/or affective states. Acknowledgement of these sources is not enough. The information provided by these sources requires active cognitive processing and reflective thought (Bandura, 1997).

2.1.14.2. Source of self-efficacy: Mastery experience. Performance or mastery experience has a robust impact upon the development of self-efficacy. Successes and failures help to define self-efficacy in this information source (Bandura, 1977, 1997). In the case of tobacco dependence

treatment, success with abstinence in the face of cues for relapse provides positive information of ability and supports confidence of the ability to abstain. Lapses and relapse to smoking, however, may be considered as a failure in the ability to abstain, which may undermine self-efficacy for abstinence.

2.1.14.3. Source of self-efficacy: Vicarious experience. Distinct and salient observations relevant to the behavior of interest are used for comparison. According to Bandura (1997), modeled attainments of others provide an individual with vivid observations for evaluative diagnostics of oneself. In addition, modeling provides an example for learning. For the tobacco user considering or attempting abstinence, observations of others like themselves provides information through modeling, particularly observations of individuals successful in their coping with relapse promoting situations. These observations provide a pattern of predictable situations and the results of the control or lack of control used by the model observed.

2.1.14.4. Source of self-efficacy: Verbal persuasion. Social influence through verbal persuasion has potential to enhance self-efficacy. One type of verbal persuasion can be provided in evaluative feedback. However, the framing of the feedback information is critical to the degree self-efficacy is enhanced or diluted. Feedback stressing personal attributes to the situation may provide better results than attributions of improvement. In addition, the type of feedback offered over the course of changing behavior may need to change. Although positive feedback regarding the effort expended can have a positive impact on motivation during the beginning stages of changing a behavior, feedback accentuating that their progress demonstrates their ability is necessary in later stages of changing the behavior (Bandura, 1997).

2.1.14.5. Source of self-efficacy: Physiological and affective states. The fourth informational source individuals use to develop their self-beliefs comes from their own internal state of affairs, which encompasses physiological and affective (emotional) states. These particular states provide feedback pertaining to level of function and reaction to stressors. In particular, biopsychological feedback can serve as an indication of coping responses, which also becomes incorporated in self-efficacy beliefs. These indicators may arise from overt physical observations, such as vigor or fatigue, or physiological changes associated with autonomic system activation (Bandura, 1997). For tobacco dependent individuals, nicotine withdrawal symptoms may provide a source of physiological information detrimental to their perceived efficacy to maintain tobacco abstinence, particularly for individuals with minimal coping skills. Another example is that of negative emotional states. Negative mood may have detrimental influence upon perceptions of efficacy. Furthermore, the amount of awareness to these psychobiological states and their resulting influence upon self-efficacy may be individual and context specific.

In addition to influencing self-efficacy, psychological and biological activity may be influenced by self-efficacy (Bandura, 1992). According to Bandura (1992), biological effects (neuroendocrine, catecholamine, and opioid function) can be initiated through the course of coping with stressors, which is dependent upon an individual's perceived self-efficacy in the presence of a stressor. Considering the triad of personal, environmental, and behavioral factors, a stressor encountered from the environment will impact the behavioral and personal factors. The presence of low or high self-efficacy mediates perceptions of the environmental stressor and reactions to the stressor by the remaining factors. In the case of psychological stressors, reactions

(e.g., anxiety) are dependent upon perceptions of coping self-efficacy (Bandura, Cioffi, Taylor, & Brouillard, 1988; Bandura, Taylor, Williams, Mefford, & Barchas, 1985). In a laboratory study by Bandura, et al. (1985), catecholamine activity to a stressor was augmented through guided mastery intervention aimed at increasing perceived coping efficacy. In a second study, Bandura, et al. (1988) examined the opioid activity to pain and found coping self-efficacy impacted the opioid response. These findings support evidence for the mediation of coping self-efficacy to stressor.

2.1.14.6. Outcome expectancies. In addition to self-efficacy expectancies or beliefs, outcome expectancies are necessary for motivation and ongoing action toward behavior change. Bandura (1997) further suggests that these anticipated outcomes take one to three forms, which include physical, social, and self-evaluative. Within each of these, there are positive and negative expectations. Therefore, these forms of outcome expectancy provide judgment criteria for an individual to use in the assessment of the consequences to the action taken. It must be clear that outcome expectancies reflect anticipated outcome resulting from the behavior of interest and that behavior is not the outcome (Bandura, 1997). A tobacco dependent patient engaged in self-regulation strategies to maintain abstinence may have outcome expectancies related to health function, such as improved lung or cardiovascular health. The gauge they may use as an indicator could include increased stamina with activity or lack of exertional dyspnea. Maintaining abstinence would be seen as a behavioral goal, but not the personal outcome of interest or outcome expectancy.

2.1.14.7. Self-regulation. The predictive nature of self-efficacy with regard to tobacco intervention efforts is useful in developing a strategy of self-regulation for tobacco dependent individuals interested or motivated to change this behavior to one of tobacco abstinence

(Gulliver et al., 1995; Haaga & Stewart, 1992; Karanci, 1992). Perceived self-efficacy influences behavior change, such as tobacco dependence, through mediation of four processes, which include cognitive, motivational, affective, and selective processes (Bandura, 1997). Cognitive processes are important in the evaluation of beliefs and action, as well as the development to strategies through problem solving. Motivational processes require a cognitive representation to drive the goals, anticipated outcomes, and causal attributions. Affective processes can also regulate behavior through perceptions of self-efficacy to cope and control, as well as self-evaluative perceptions of actions taken. Finally, selection processes encompass an individual's environment and what choices or decisions they make about that environment to regulate their behavior (Bandura, 1997).

There are four functions of self-regulation, which include self-monitoring, goal setting (proximal), strategy development, and internalized motivation (Bandura, 1997). Self-regulation for tobacco dependence treatment requires self-monitoring of behavior associated with tobacco use and abstinence, which becomes important in self-evaluation of ability and effort. Challenges are determined through goal setting, but the goals must be proximal in nature. Therefore, goals that provide a challenge need to be short range in expectation rather than long range, such as years. In addition, goals must be flexible and adaptable for the individual, but maintain some structure that can be self-evaluated. Bandura (1997) suggests that goals are mediated by self-influence in the form of self-evaluation reaction of the effort, self-efficacy of ability to reach the goal, and re-evaluation of perceptions when the goal is met. Developing a plan to change a behavior requires the integration of an assessment of the monitored behavior and perceived efficacy to identify vulnerabilities, which can provide direction of needed coping and problem

solving skills. Mastery and modeling can also be used at this time to identify strategies to use and reflective evaluation of strategies used. Finally, internalized motivation is necessary to maintain self-regulatory effort. The concert of these activities working together becomes evident with respect to motivation and the need for goals and feedback to maintain motivation to continue (Bandura, 1997).

2.1.15. Summary

Although the literature pertaining to nicotine addiction and associated treatment is vast, there are particular gaps in the literature regarding intervention research with Self-Efficacy Theory conceptual underpinnings targeting hospitalized smokers, tobacco dependence treatment adherence, and the impact of tobacco dependence treatment upon medical treatment adherence in comorbid populations of smokers. Limited studies have incorporated Self-Efficacy Theory for tobacco intervention. Even fewer studies have used Self-Efficacy Theory with a comorbid population of smokers. DeBusk et al. (1994) used Self-Efficacy Theory to drive an intervention effort for patients rehabilitating from a recent myocardial infarction. The interventions were directed towards changing cardiovascular behavioral risk factors. Therefore, tobacco dependence intervention was directed to 252 subjects of the entire sample of 585 subjects. However, biologically validated findings from the final follow-up visit were encouraging with a 70% abstinence rate for subjects offered the tobacco dependence intervention as compared to 53% assigned usual care ($p = .03$).

Dornelas et al. (2000) used the Transtheoretical Model of Change with the integration of Self-Efficacy Theory as the conceptual framework for a hospital based tobacco intervention provided to patients diagnosed with a myocardial infarction ($N = 100$). By the 12-month follow-up visit after treatment, 55% ($n = 54$) of subjects assigned the protocol intervention were

abstinent as compared to 34 % (n = 46) in usual care (p = .05). Self-efficacy was an independent predictor of abstinence. Furthermore, 93% of the subjects with low self-efficacy and assigned usual care had relapsed by the 12-month follow-up visit. Only 50% of the intervention subjects with low self-efficacy had relapsed by the same time point. This study was limited by the lack of biological validation of the smoking status at follow-up time points.

A third study explored motivational determinants of tobacco abstinence in a hospitalized cardiac population (Bolman & de Vries, 1998). An intervention for tobacco dependence was not offered. The Attitude-Social Influence Efficacy Model, as well as concepts from the Transtheoretical Model of Change provided the conceptual framework for this study. Self-efficacy was incorporated as part of the first model. Boman and deVries (1998) reported that externally motivated subjects for tobacco abstinence had fewer positive attitudes and social support, as well as low self-efficacy expectations.

These three studies explored questions pertaining to tobacco abstinence, motivation, and self-efficacy in populations of hospitalized smokers. Of interest are the commonalities of their findings, which included the association of low self-efficacy associated with positive smoking status or relapse. In addition, the association of low self-efficacy and motivation in relation to relapse provides consistent evidence with perceived self-efficacy and motivational processes. The research noted above and other research using smokers diagnosed with cardiovascular disorders support the need and success for tobacco dependence intervention with this population of smokers. However, available literature with controlled tobacco intervention trials across smoking hospital populations is limited. Studies that have provided intervention to this vast population have reported disappointing findings of high relapse rates following hospitalization (Rigotti et al., 1997; Wewers et al., 1997). Although self-efficacy has been a concept measured

for predictive associations to smoking outcome, hospital-based interventions do not actively incorporate an approach aimed to enhance self-efficacy within the intervention framework. Therefore, research is needed to investigate the effectiveness of self-efficacy enhancing strategies for relapse prevention across diagnostic groups of hospitalized patients.

Although nicotine replacement treatment is highly recommended with patients diagnosed with various disorders, including cardiovascular problems, the administration of these medications by physicians remains low (Rigotti et al., 1999). There is a need to explore these barriers in treatment. However, there are patients who may not be eligible candidates for such therapy. As noted previously, nicotine replacement has been associated with a case finding of hypotension during surgery (Groudine & Morley, 1996). More information is needed on the use of nicotine replacement therapies for surgical candidates. Due to comorbidities and concomitant use of various medications, bupropion may not be an appropriate choice for some hospitalized patients. Due to the time required for the loading doses, bupropion may not be efficacious for short hospital admissions. Finally, patients may not opt for medication treatment. Therefore, cognitive behavioral therapies need to be actively explored with hospitalized smokers, particularly relapse prevention interventions aimed for an extended period following hospital discharge when they at greatest risk for relapse.

In light of this gap in the literature, this research study proposes to investigate the efficacy of a tobacco relapse maintenance intervention for hospitalized smokers with varying diagnoses and comorbidities. Figure 2 provides a graphic representation of the Self-Efficacy Theory conceptual framework that will guide this project. An assessment of relapse vulnerability

related to coping self-efficacy will be used to target a relapse factor of greatest risk for each subject. The relapse maintenance interventions will use this information as a starting point in this process of self-regulation of tobacco abstinence behavior.

Treatment adherence to the cognitive behavioral intervention component of this study will be monitored. Few intervention studies have reported cognitive behavioral tobacco treatment adherence (Kamarck & Lichtenstein, 1988). There are no studies of hospitalized smokers in the literature that have provided information pertaining to this type of treatment adherence and relevance to treatment outcome. Therefore, this study proposes to describe tobacco dependence treatment adherence in a population of hospitalized smokers. This information may further assist in efforts to understand barriers and reinforcement variables for tobacco dependence treatment in this population of smokers.

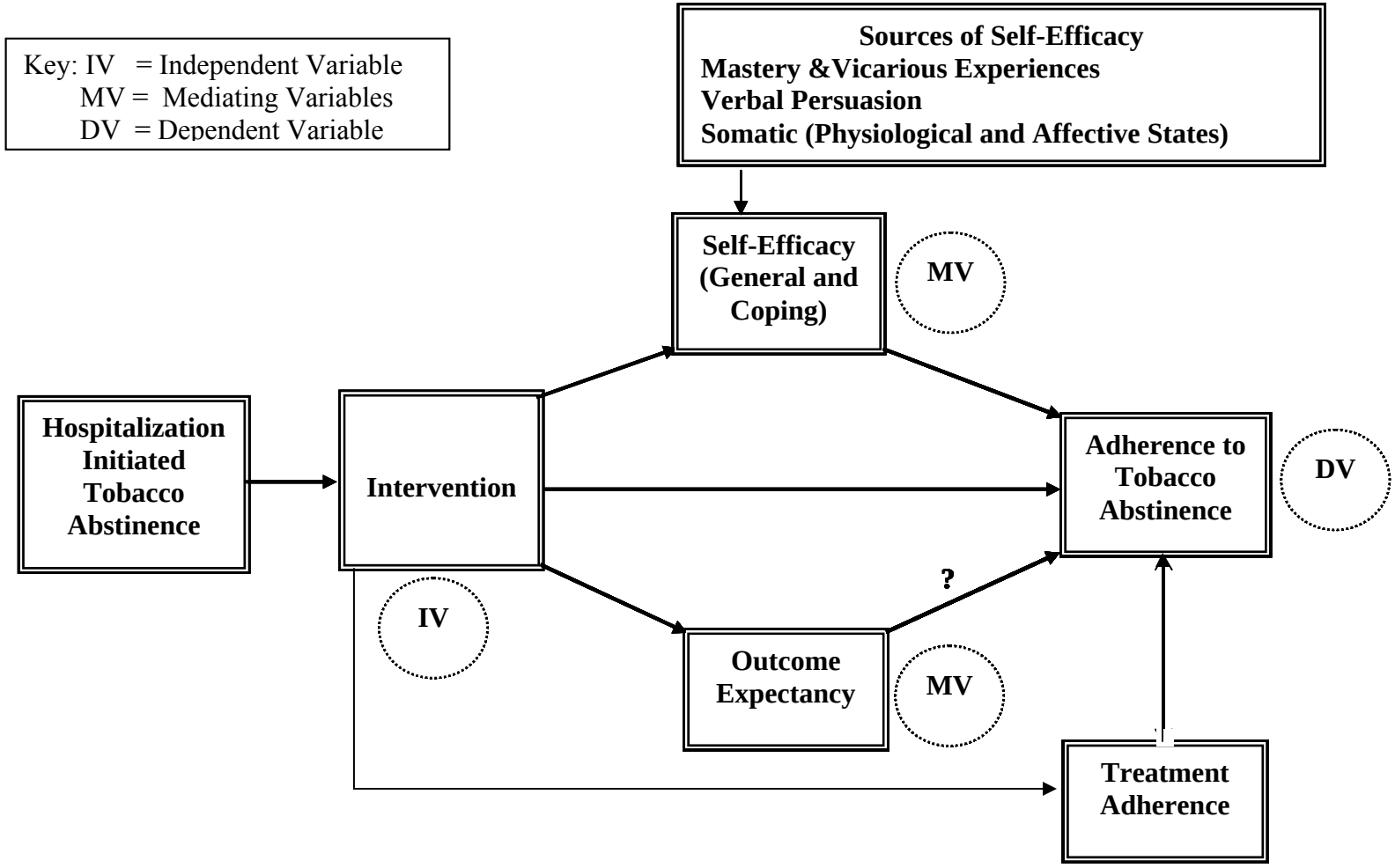


Figure 2 Conceptual Framework

3. CHAPTER THREE

3.1. PRELIMINARY RESEARCH

3.1.1. Introduction

A large proportion of current smokers (70%) have self-reported the desire to quit smoking at the time of their annual physicals, but less than 50% acknowledge receipt of specific direction for smoking cessation by healthcare professionals (Goldstein, Niaura, Willey-Lessne, DePue, Eaton, Rakowski, & Dube, 1997). Abstinence rates between 8 - 25% have been achieved with self-help smoking cessation tactics among the general public (Center for Disease Control and Prevention, 1992). Intensive cessation programs that combined behavioral therapy with nicotine replacement treatment have yielded four fold increases in abstinence rates over the usual cessation practices by smokers (Kanner, 1996).

Furthermore, prevalence and detailed descriptive information pertaining to this population of smokers remains limited. For example, few studies of hospitalized smokers have described the comorbid health status of hospitalized smokers (Miller et al., 1997b; Rigotti et al., 1997; Stevens et al., 1993; Taylor et al., 1990; Wewers et al., 1994). In addition, few studies have reported the prevalence of tobacco use among hospitalized patients (Rigotti et al., 1997). As reported by Rigotti, et al. (1997) the prevalence of hospitalized smokers in that study greatly exceeded the national average of 24%. Limited available prevalence information and potential for wide variation impacts the forecasting required to implement hospital-based smoking intervention.

Finally, high relapse rates continue to occur for this smoking population following hospital discharge. High relapse rates coupled with a lack of motivation to re-initiate smoking abstinence may plague smokers following their discharge from the smoke-free confines of the hospital (Miller et al., 1997b; Rigotti et al., 1997; Wewers et al., 1994). This information, as well as the relationship of additional variables, may be pertinent to the development and clinical implementation of evidence-based interventions specific to this smoking population of hospitalized smokers.

In light of limited available data pertaining to the tobacco use, cessation efforts, and health status of hospitalized smokers, as well as assessments of usual care interventions offered to this population, this descriptive study was undertaken. The purpose was to assess tobacco and health related characteristics of hospitalized smokers, and the hospital based smoking interventions they received that may contribute to the design of a hospital-based smoking cessation intervention.

The Transtheoretical Model of Change [TMC] was selected as the guiding conceptual framework for this study with regard to assessment of readiness or motivation to quit smoking (Prochaska, 1995). The originators of this model suggest behavior change, such as tobacco use, occurs for an individual through the interaction of the processes, stages, and levels of change, which are the core concepts of the model (Prochaska, 1995). Proponents of this model suggest the assessment of an individual's "stage of change" is an indication of motivation to change health behavior (Orleans, Kristeller, & Gritz, 1993). Therefore, in this study, stage of change was used to operationalize motivation for abstinence from smoking.

3.1.2. Preliminary research aims

The primary aim of this study was to describe tobacco and health related characteristics of a hospitalized patient population with current smoking histories. A second aim proposed to describe the smoking cessation counseling intervention provided to this population of smokers. The following research questions were used to further direct the activities of this project.

1. What are the tobacco consumption characteristics of hospitalized smokers admitted to medical-surgical patient care units at a metropolitan university health center?
2. What is the number and type of chronic comorbid conditions among hospitalized smokers?
3. Are there differences in the smoking stage of change assessed during the hospital admission and following discharge?
4. What smoking cessation interventions do hospitalized smokers receive before discharge?

Finally, an exploratory analysis examined whether characteristics of tobacco dependency, socioeconomic factors, and self-efficacy towards smoking abstinence were associated with motivation, modification, abstinence attempts, and smoking behavior following hospital discharge.

3.1.3. Methods

3.1.3.1. Participants. The subjects for this study were recruited from a convenience sample of patients admitted to patient care units at a metropolitan university medical center. Across these patient care units, 1,334 patients completed a recruitment flyer designed to identify hospitalized smokers interested in study participation. Similar to Pennsylvania and national averages, the proportion of smokers identified by recruitment in this setting was 24% (n = 314) of those

screened for this study (Center for Disease Control and Prevention, 1997c, 1999d). An adult smoker was defined as an individual 18 years or older who smoked at least one cigarette within 30 days of their hospital admission. Of the 314 identified hospitalized smokers, 118 (38%) consented to participate with this project and comprised 9% of the total hospital population screened. Inclusion into the study required participants to be: 1) adults between the ages of 18-70 years, 2) admitted to 1 of 8 designated hospital patient care units, and 3) to have a current smoking history as defined above. Exclusion criteria included the following: 1) recruitment directly from intensive care, transplant, or oncology units; 2) diagnosis of terminal cancer; 3) patients under evaluation or awaiting organ transplantation; 4) diagnosed at the time of recruitment with a recent cerebral vascular disorder; 5) senile dementia; 6) Alzheimer disease; 7) smoking abstinence for greater than 1 month; 8) non-English speaking patients; and 9) lack of a home telephone and/or mailing address.

3.1.3.2. Procedures. This approved descriptive study for human subjects recruited hospitalized smokers admitted to 8 medical/surgical units at a large metropolitan university health center over 16 months (2/1999 - 5/2000). Identified and consenting subjects were recruited to complete two surveys. A baseline survey was completed during the subject's hospital stay while the follow-up survey was carried out after discharge. Following consent procedures, subjects were randomized to one of three follow-up survey groups. These follow-up groups were examined for the process of calling participants following hospitalization for follow-up status on smoking. The information from this process was intended to assist in the development of procedures for contacting participants in a clinical trial testing telephone relapse prevention interventions provided after hospitalization. In order to ensure that each follow-up survey group approximated the regional recruitment site and cross sectional representation of post-discharge tobacco use

related activities, randomization with minimization was used to balance the groups by gender and race. Therefore, group assignment determined when the follow-up contact was initiated at 1, 4, or 12 weeks following hospital discharge.

3.1.3.3. Measures. Seven survey instruments were used to assess subjects' tobacco related characteristics. The baseline survey included six measures listed below and required 45 - 60 minutes to complete. A 5-10 minute follow-up interview was conducted by telephone following discharge.

3.1.3.4. CRCD Sociodemographic Questionnaire [SDM]. A modified version of the 24-item sociodemographic questionnaire developed by the Center for Research of Chronic Disorders [CRCD] at the University of Pittsburgh School of Nursing was used to collect demographic information that may impact an individual's health status, such as age, race, gender, education, marital status, health insurance, religiosity, employment, and income. Modifications included the addition of data collection areas completed by the investigator of the subject's chief complaint, diagnosis, physician ordered treatment, past medical and surgical histories.

3.1.3.5. CRCD Comorbidity Index [COM]. A 76-item comorbidity survey developed by the CRCD at the University of Pittsburgh School of Nursing was used to measure comorbid risks of the hospitalized smoker and was completed by interview with study personnel or independently by the subject.

3.1.3.6. General Health Survey [GHS]. A 19-item questionnaire was used to supplement the SDM and COM for health related information pertinent to a hospitalized smoker's health and environmental exposure history. Questions for this instrument were also adapted from those used as part of the Lung Health Study.

3.1.3.7. Tobacco Use Questionnaire [TUQ]. Tobacco consumption, smoking behavior, and tobacco related health status were collected by interview with a modified version of items used by the Lung Health Study. Previous studies note these factors may be predictive of smoking relapse (Bjornson et al., 1995; Stapleton, Russell, Feyerabend, Wiseman, Gustavsson, Sawe, & Wiseman, 1995). A short form of this modified instrument was used for the follow-up interview.

3.1.3.8. Fagerstrom Tolerance Questionnaire [FTQ]. Severity of nicotine dependence was measured with the 8-item FTQ (U.S. Department of Health and Human Services, 1986). This scale was imbedded in the TUQ noted above. Cronbach's alpha for the FTQ in American populations has been reported as .47 and has acceptable test-retest reliability regardless of data collection method (e.g., telephone interview, paper/pencil self-report) (Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994).

3.1.3.9. Stages of Change Questionnaire [SOC]. Due to the use of the Transtheoretical Model for Change as a conceptual framework for this study with regard to motivation towards smoking abstinence, an algorithm assessing the stages of change was used to assess motivation for health behavior change at baseline and follow-up interviews. Various brief methods have been used to measure SOC, such as an algorithm and ladder concept (Biener & Abrams, 1991). Assessment of SOC has been noted in the literature to provide a consistent assessment regarding the stage of readiness for health risk behavior change in various smoking populations (Pallonen, Leskinen, Prochaska, Willey, Kaariainen, & Salonen, 1994; Simon et al., 1997; Wewers et al., 1994).

3.1.3.10. Smoking Intervention Questionnaire [SIQ]. The SIQ was a 4-item survey tool used to assess the subject's recollection of information and counseling provided before hospital discharge by healthcare professionals at the recruitment site. This assessment of healthcare practice was not conducted during hospitalization so that usual practices would not be inadvertently changed during the course of the study.

3.1.4. Data analysis.

3.1.4.1. Descriptive statistics. A preliminary analysis of the data with descriptive statistical techniques was completed as a prerequisite to further inferential testing of this study's proposed research questions. This analysis included by means of scatter plots, histograms, box-plots, and stem-and-leaf plots as a graphical description of the sample population and collected data. SPSS version 8 was used to conduct the statistical assessments for both continuous and categorical variables.

Summary statistics were completed for each continuous variable, which included measures of central tendency (mean, median, mode) and variation or dispersion (standard deviation, variance, range, semi-quartile range, skewness, kurtosis). A significance level of .05 was used for the descriptive analyses and exploratory analytic techniques. Univariate sample distributions were generated to describe the characteristics of the hospitalized smokers enrolled in this study. Pearson correlation coefficient was used to assess bivariate relationships of continuous variables. In addition to analyzing the overall sample, subset analysis was completed by gender and race characteristics.

Discrete variables were also analyzed with nonparametric techniques, such as Fisher's Exact Test, Pearson chi-square test for independence, and phi-coefficients. Frequency distributions, range, and mode were generated to describe categorical and dichotomous variables.

3.1.4.2. Data screening procedures. Data for this study were collected with paper-pencil instruments and required prescreening for data inconsistencies, recording errors, and missing entries. All instruments were prepared with Teleform software (version 6) prior to data collection. Following scanning, database entries were systematically entered in to Paradox tables (version 9). Data dictionaries were developed for data editing and analysis.

A preliminary analysis was conducted with descriptive, univariate, bivariate, multivariate, and regression statistics to assess: 1) sample distributions, 2) existence of strong interrelationships between variables (multicollinearity), 3) outliers, 4) patterns of missing data, and 5) assumption violations for normality, linearity, homogeneity of variance, and independence. In addition to the above statistical techniques, graphical measures were used to assess statistical assumptions, sampling distribution characteristics, for univariate and multivariate outliers, influencing cases, and assumption violations. Analysis for the research questions required use of SPSS version 8 software to conduct the following statistical procedures: Pearson χ^2 test for independence, Student t-test, and logistic binary regression.

3.1.4.3. Sample characteristics. Subjects were on average middle age (M = 45 years, SD = 11.5; n = 96) with at least 13 years (SD = 2.5, n = 96) of education and reported having two children (SD = 1.4, n = 72) (Table 5). With regard to relationships, 32% (n = 37) were married and 17% (n = 20) divorced. Approximately 33% of the subjects reported growing up in a large or small urban city. As noted previously with regard to the participants, the sample was predominantly male (60%; n = 70) and white (78% n = 92), 17% (n = 20) black, 5% (n = 6) other, which was consistent with local racial distributions. Sixty-eight percent of the subjects (n = 118) identified a religious affiliation and of those, 46% (n = 54) noted that spirituality or their religious beliefs were somewhat important to their life.

The employment and income status of subjects was split between those currently employed (35%, n = 41) with an average salary that ranged from $20,000 to $29,000 and those who reported disability impacting employment (31%, n = 36). One percent of the subjects reported being unemployed. Most (70%, n = 82) of the subjects, reported having some type of insurance coverage for healthcare needs (e.g., private, Medicare, Medicaid).

The leading chief complaints documented for hospitalization included chest pain (15%, n = 18) and automobile accidents (14%, n = 17). Shortness of breath (10%, n = 12) was the third most common complaint requiring hospitalization. Cardiovascular related disorders (31%, n = 36) were the leading documented diagnoses pertaining to the hospitalization of these subjects.

Table 5 Sociodemographic and socioeconomic characteristics (n = 118)

	Baseline
Males, % (n)	60 (72)
Education, Mean (± SD) (Years)	13 ± 2.5, 96
Ethnicity	
White	78 (92)
Black	17 (20)
Other	5 (6)
Age, Mean (± SD) (Years)	45 ± 11.5, 96
Employment, % (n)	
Employed	35 (41)
Unemployed	1 (1)
Disabled	31 (37)
Retired, Not Working	6 (7)
Other	7 (8)
Refused to Disclose Status	20 (24)
Insurance, % (n)	70 (82)

3.1.5. Results

3.1.5.1. Tobacco consumption characteristics. At baseline, subjects (refer to Table 6) were smoking at least one pack of cigarettes per day (M = 21, SD = 11.8, n = 104) and reported initiating their daily tobacco consumption in their adolescence (M = 16yrs., SD = 5.7, n = 103). Subjects reported a significant effort to decrease their cigarette consumption following discharge (follow-up cigarettes/day M = 12, SD = 11.2, n = 82) (t_{73} = 5.57, p = .000). The Marlboro brand of cigarettes, a tobacco brand high in nicotine content, was the preferred brand for consumption by approximately 30% of the subjects.

According to the average FTQ score (M = 6, SD = 1.9, n = 105), subjects were nicotine dependent, but most were not reporting high dependence scores, such as 9 or 10. At least 50% (n = 59) of the subjects reported stress as a leading primary trigger to consume cigarettes. Over 50% of the subjects indicated they found the first cigarette of the day to be the most satisfying and began smoking within 30 minutes of rising from bed in the morning.

Sixty-two percent (n = 73) of the subjects reported making at least one attempt to quit smoking for 24 hours in the last year. Of those subjects, 24% (n = 28) had attempted smoking cessation at least 4 times. Most (49%, n = 57) subjects were the only smoker in their household. Approximately 70% (n = 82) of the subjects reported their spouse or significant other would like them to give up smoking. Sixty-seven percent of the subjects desired to quit smoking, but more than half (55%, n = 65) thought they would be successful. Furthermore, only 18% (n = 21) of the participating subjects previously used organized smoking cessation programs/materials and 32% (n = 37) used nicotine replacement products to aid cessation. Most subjects considered a "cold turkey"

cessation approach or use of pharmaceuticals as their preferred method to initiate abstinence. Finally, 59% (n = 69) of the subjects believed they would gain weight with a smoking cessation attempt.

Table 6 Tobacco characteristics

Tobacco characteristics	Baseline
Cigarettes/day, Mean ± SD, (n)	21 ± 11.8, (104)
Age of Smoking Initiation, Mean ± SD, (n)	16 ± 5.7, (103)
FTQ score, Mean ± SD, (n)	6 ± 1.9, (105)
Confidence for Cessation, % (n)	55 (65)
Significant Other Support for Cessation, % (n)	70 (82)
Motivated to Quit Smoking, % (n)	67 (79)
Previous 24 hour Quit Attempt, % (n)	62 (73)
Anticipate Weight Gain with Cessation, % (n)	59 (69)
Participation With Cessation Programs, % (n)	18 (21)
Previous Use of Nicotine Replacement, % (n)	32 (38)
Cessation Treatment Preference	
"Cold Turkey Abstinence," % (n)	27 (32)
Behavior Modification Interventions, % (n)	11 (13)
Pharmaceuticals, % (n)	28 (33)
Tobacco Control Legislation, % (n)	1 (18)
Surgery, % (n)	1 (1)
Did Not Know, % (n)	20 (24)
Did Not Respond, % (n)	12 (14)

At baseline, 67% (n = 79) of the subjects reported a desire to quit smoking, but only 21 took action to change their smoking behavior during hospitalization. When contacted at follow-up, nearly half (n = 10) of these subjects relapsed to smoking after their cessation attempt associated with their hospital admission. Of the 118 subjects recruited at baseline, 84 subjects were available for follow-up contact. Deaths, change in residence, and wrong addresses/telephone numbers accounted for the decrease in subjects available for follow-up. More than half of the subjects (54%; n = 45) contacted at follow-up reported an attempt toward cessation following discharge, but only 12 subjects who considered a change in behavior during admission reported successful smoking abstinence after discharge.

3.1.5.2. Number and type of chronic comorbid conditions. Of the 118 subjects at baseline, 97 completed the self-report questionnaire for comorbid disorders. The 10 leading comorbid conditions reported by subjects included headache (39%, n = 37), hypertension (37%; n = 36), sudden weakness of limbs (32%, n = 31), depression (31%, n = 30), rheumatic diseases (31%, n = 30), sudden numbness (28%, n = 27), coronary artery disease [CAD] (26%, n = 25), anxiety (26%, n = 25), myocardial infarction (24%, n = 23), and irregular heart rhythms (22%, n = 21). In addition, 19% (n = 18) reported digestive ulcers and 16% (n = 15) skin disorders. Approximately 84% of the subjects diagnosed with hypertension were also prescribed antihypertensive medications. Over 42% (n = 40) of the subjects reported they had either an old or new fracture of at least one bone while 23% (n = 22) reported at least two fractures. Chart reviews indicated that on average, subjects had 3 (SD = 2.6) documented comorbid conditions in addition to their admitting hospital diagnosis. Furthermore, chart reviews indicated hypertension (25%, n = 24) as the leading reported past medical problem. With regard to surgical history, 27% (n = 26) of the subjects had at least one previously documented surgical procedure.

Subjects were also asked to report symptoms experienced during their hospitalization (baseline). The nine of the most commonly reported symptoms included fatigue (51%, n = 49), shortness of breath (47%, n = 47), back problems (43%, n = 43), joint complaints (38%, n = 37), generalized pain (37%, n = 36), limb weakness (34%, n = 33), sleeping problems (34%, n = 33), nausea (30%, n = 29), and itching (30%, n = 29). In addition to the symptoms noted above, 28% (n = 27) reported experiencing loss of appetite, night sweats, vision problems, and/or weight gain. Nicotine withdrawal symptoms were not measured in this study. Therefore, it is unclear whether there is overlap between symptoms reported for their hospitalization and nicotine withdrawal. Additional symptoms noted by subjects included chest pain, urinary symptoms, abdominal pain, weight loss, vomiting, diarrhea, balance problems/dizziness, skin rash, constipation, and fevers.

When asked to report on respiratory disorders experienced by family members, 18% (n = 17) indicated they had children with asthma and 12 % (n = 11) noted a history of asthma for their mothers. Other respiratory illnesses were not reported with the frequency of these two categories for asthma. Over 56% (n = 54) reported their fathers were cigarette smokers and 31% (n = 30) had mothers that smoked. Thirty-nine percent of the subjects reported that their siblings smoked cigarettes.

3.1.5.3. Baseline and follow-up SOC. Seventy-one percent (n = 84) of the subjects completed their follow-up interview and 29% (n = 34) were completely unavailable for follow-up. Discharges to locations other than home (e.g., rehabilitation facilities, nursing homes, homes of extended family) and death accounted for some of the difficulties in successfully contacting subjects following hospitalization. Baseline findings were not statistically different between those subjects contacted for follow-up versus those unavailable for contact.

An analysis was conducted to examine relationships between the baseline and follow-up SOC status (n = 84) for the subjects successfully contacted for follow-up. Without consideration for time, the SOC status for these subjects did not remain the same following discharge and did not move in a forward direction of change. There was a significant change in status from baseline to follow-up ($\chi^2 = 27.8$, df = 9, n = 84, p = .001) (Refer to Table 7).

Of interest in the movement in SOC status, 50% (n = 6) of the subjects categorized at the precontemplation stage for baseline SOC measure moved forward to another SOC category after discharge. Most baseline contemplators (46%, n = 13) did not change their SOC status, but at least 43% (n = 12) did move forward in their follow-up SOC status while 11% (n = 3) regressed toward to precontemplation. Of interest, none of the subjects categorized in a preparation stage moved completely backward to precontemplation. Again, most subjects in a preparation stage did not change status upon follow-up (43%, n = 10), 35% (n = 8) regressed, and 22% (n = 5) had forward progression to the action stage. Unlike those in the baseline preparation stage, 19% (n = 4) of the subjects categorized in the action status at baseline regressed to the precontemplation stage. Subjects at baseline action stage were similar to those at precontemplation stage with nearly half remaining at their baseline stage (48%, n = 10) and 52% (n = 11) changing status, but in this case a backward movement away from active cessation.

Table 7 Baseline versus follow-up SOC status

	Follow-up				
Baseline	1	2	3	4	Total
1. Precontemplation	6 (7.1%)	4 (4.8%)	1 (1.2%)	1 (1.2%)	12 (14.3%)
2. Contemplation	3 (3.6%)	13 (15.5%)	6 (7.1%)	6 (7.1%)	28 (33.3%)
3. Preparation	0 (0.0%)	8 (9.5%)	10 (11.9%)	5 (6.0%)	23 (27.4%)
4. Action	4 (4.8%)	5 (6%)	2 (2.4%)	10(11.9%)	21 (25.0%)
Total	13(15.5%)	30 (35.7%)	19 (22.6%)	22(26.2%)	84 (100%)

3.1.5.4. Hospital-based cessation interventions. Approximately 50% (n = 42) of the subjects reached for follow-up received one cessation message from a healthcare professional. Only 9% (n = 8) of these subjects contacted at follow-up received pre-discharge cessation intervention beyond the efforts of a professional cessation message. Nicotine replacement therapy was the most frequently offered assistance for these eight subjects. Other cessation interventions received by these subjects (n = 8) included flyers for cessation programs, a smoking assessment, anti-anxiety drugs, or resources to contact regarding cessation after discharge. No subjects were offered relapse prevention interventions during or following hospitalization.

3.1.5.5. Predictors of smoking behavior. Logistic regression with backward stepwise entry with SPSS software (version 8) was initially used to examine potential predictor variables associated with four different outcome variables, which included: (1) motivation towards smoking abstinence during hospitalization, (2) post-hospitalization attempts at smoking abstinence, (3)

post-hospitalization modification of smoking behavior, and (4) sustained smoking abstinence. Following assessment with backward stepwise entry, tests of the full models with the predictor variables were completed for each of the four analyses. Self-efficacy and years of formal education were associated with motivation/desire towards smoking abstinence during hospitalization ($\chi^2 = 22.0$, df = 2, n = 94, p= .001; $R^2_{\text{Cox Snell}} = .21$) (Table 8). As noted in Table 9, only self-efficacy was associated with post hospitalization attempt at smoking abstinence ($\chi^2 = 10.7$, df = 1, n = 73, p= .001; $R^2_{\text{Cox Snell}} = .14$). Presence of a significant other and employment were associated with modification of smoking behavior following hospitalization ($\chi^2 = 16.9$, df = 2, n = 71, p= .001; $R^2_{\text{Cox Snell}} = .21$) (Table 10). Table 11 provides the analysis of tobacco abstinence following discharge as the outcome and older age of initiation to daily smoking, self-efficacy, presence of a significant other, and employment as predictor variables ($\chi^2 = 43.5$, df = 4, n = 92, p= .001; $R^2_{\text{Cox Snell}} = .38$).

Table 8 Logistic regression analysis for motivation towards abstinence

Variables	b	Wald Test	Odds Ratio	95% Confidence Intervals	
				Lower	Upper
Self-Efficacy	2.05	13.19	7.76	2.57	23.44
Education (Years)	0.32	3.89	1.38	1.00	1.89
Constant	-4.01	3.71			

Table 9 Logistic regression analysis for cessation attempt post hospitalization

Variables	B	Wald Test	Odds Ratio	95% Confidence Intervals	
				Lower	Upper
Self-Efficacy	1.67	9.86	5.30	1.87	15.03
Constant	-0.81	3.64			

Table 10 Logistic regression analysis for smoking modification

Variables	b	Wald Test	Odds Ratio	95% Confidence Intervals	
				Lower	Upper
Significant Other	1.81	7.93	6.11	1.73	21.53
Employment	-1.94	7.89	0.44	0.04	0.56
Constant	-0.51	5.22			

Table 11 Logistic regression analysis for follow-up smoking abstinence

Variables	b	Wald Test	Odds Ratio	Confidence Intervals	
				Lower	Upper
Employment	-3.35	10.33	0.04	0.01	0.28
Self-Efficacy	2.39	10.76	10.89	1.62	73.13
Smoking Initiation Age	0.21	9.81	1.23	1.08	1.35
Significant Other	2.71	6.04	15.00	2.88	78.18
Constant	-7.29	15.41			

3.1.6. Summary

As noted previously in the recruitment information, 24% of those patients screened for the study self-reported a positive smoking status, which is comparable to the national average in the general population (Center for Disease Control and Prevention, 1997c), but unlike the findings reported by Rigotti, et al., (1997) of a hospitalized sample, which consisted of a larger percentage (10%) of smokers. Orleans, Kristeller, Gritz, (1993) noted that the prevalence of smoking is not known for hospitalized smokers and is estimated to be similar to the national average. Some limitations were noted with the recruitment procedures used for this project. Since this study did not offer an intervention, some smokers may not have identified themselves with a positive smoking history. In addition, short hospital stays may have impacted the screening process. Although the admission forms for the hospital recruitment site required a specification for smoking status, staff did not routinely use this hospital assessment form to identify eligible patients to receive study flyers. As noted by Fiore, Jorenby, Schensky, Smith, Bauer, and Baker (1995), failure to identify patients with a positive current smoking status has been a reoccurring problem within the delivery of healthcare, in spite of the mounting evidence of the deadly relationship of tobacco with illness and premature death. Systematic procedures, such as chart or file prompts identifying smokers and considering smoking status as a vital sign for assessment, have been suggested as reminders for healthcare professionals to follow through with intervention messages and cessation assistance during hospitalization (Adams, 1995; Robinson, Laurent, & Little, 1995). However, compliance of healthcare professionals with these practices may require further intervention.

Baseline TUQ results suggested subjects were nicotine dependent, consumed at least a pack of cigarettes per day, and had a long smoking history. These findings are similar to other studies of hospitalized smokers (Miller, Johnson, Mackay, & Budz, 1997a; Rigotti et al., 1997; Rigotti et al., 1994). As noted by other studies, these hospitalized smokers were interested in changing their smoking behavior (Glasgow, Stevens, Vogt, Mullooly, & Lichtenstein, 1991; Goldstein et al., 1997; Rigotti et al., 1997). Furthermore, these subjects were interested in changing their behavior regardless of the lack of available interventions from healthcare professionals in the hospital setting.

Baseline stage of change information indicated that over 80% of the subjects were interested in changing their smoking behavior, but there was both forward and backward movement on this continuum of motivation to quit smoking following hospital discharge. Research findings for SOC indicate most smokers, across the general population, are categorized in either the precontemplation or contemplations stages (Prochaska & Velicer, 1997). Findings from this study differed from those reports and found hospitalized smokers were more likely to be categorized in either the contemplation (33.3%) or preparation (23%) stages. More research is needed in this area of motivation with regard to this population of smokers and associated interventions aimed to improve and promote motivation for cessation.

Hospitalization has been considered as a "teachable moment" and "window of opportunity" for cessation from tobacco (Emmons & Goldstein, 1992; Orleans et al., 1993). The findings from this study would suggest hospitalization was perceived by patients as an opportunity to consider a change in smoking behavior; however, as noted by this study and others, the relapse from abstinence after discharge and regression with regard to motivation for smoking abstinence suggest more research with relapse prevention/maintenance interventions

beyond discharge is needed to move smokers in a forward progression towards maintained abstinence (Johnson et al., 1999; Miller et al., 1997b; Simon et al., 1997; Stevens et al., 1993). The no smoking policy for hospitals may provide extrinsic motivation and a favorable setting to initiate abstinence behavior, but additional interventions are necessary to carry the behavior forward and sustain the effort following discharge (Neighbor et al., 1994).

Follow-up information suggested that many subjects reported a significant attempt to decrease cigarette consumption as a means to change or modify their smoking behavior. This finding has been found by other studies with hospitalized patient samples (Wewers & Gonyon, 1989). Although more than half of the subjects at follow-up reported making an attempt to quit smoking after discharge and received a cessation message from a healthcare professional, most were not able to sustain their effort of abstinence after hospital discharge. Few smokers acknowledged receiving smoking cessation intervention assistance from healthcare professionals (other than a cessation message) during their hospitalization. Since most of the smokers did not receive any cessation assistance beyond a professional message to quit smoking, they may have lacked the knowledge and resources to meet the challenge to quit smoking.

The evidence of benefits due to tobacco abstinence have been documented in not only healthy samples, but in those subjects diagnosed with chronic conditions related to their smoking behavior (U.S. Department of Health and Human Services, 1990). In addition, continued smoking has been associated with greater use of medical treatments than individuals with no smoking exposure, (Hodgson, 1992; Sarna, Brown, Lillington, Wewers, & Brecht, 2000). With regard to age, elderly smoking patients can also reap the benefits from smoking abstinence and should not be limited in receiving smoking intervention messages or cessation assistance. Evidence suggests that elderly smokers have higher cessation rates than younger smokers

(Wewers et al., 1998). Therefore, hospitalized patients, regardless of age or medical history, and society would benefit from increased smoking abstinence in this population of smokers. However, studies reveal that the provision messages for abstinence and cessation assistance is currently lacking in healthcare delivery systems, such as hospital settings (Fiore et al., 2000; Goldstein, Hellier, Fitzgerald, Stegall, & FIshcher, 1987; Neighbor et al., 1994; Padula, 1992; Sarna et al., 2000).

In this sample of hospitalized smokers, self-efficacy was a key variable associated with motivation toward quitting, attempts to quit, and actual smoking cessation. Self-efficacy for cessation has been noted in the literature as a predictor for abstinence upon follow-up (de Vries, Mudde, Dijkstra, & Willemsen, 1998; Dornelas et al., 2000; Vernon, Crane, Prochazka, Fairclough, & MacKenzie, 1999) Future intervention studies need to consider the implication of self-efficacy in the design and implementation of cessation and relapse maintenance interventions in this population (Bandura, 1997). In addition, self-change to quit smoking and modify smoking behavior following discharge were associated with the presence of a significant other and employment in this population. More intervention research is needed to incorporate supportive social networks for hospitalized smokers once they are discharged and associated monitoring of social support. If employment or income status is an indication of smoking abstinence, mechanisms are needed to provide intervention to those smokers with limited financial resources, who have the potential of impacting state healthcare funds if their smoking behavior continues.

Smokers from this sample reported symptoms consistent with not only their admitting diagnosis and comorbid conditions, but also nicotine withdrawal (e.g., fatigue, sleep disorders, and gastrointestinal disturbance). It is unclear whether there was an overlap of symptoms. If an

overlap existed, this presents a challenge to the patient and healthcare professionals in sorting these symptoms so that appropriate therapy can be provided. Recent case reports regarding the implications of nicotine withdrawal in patients receiving medical treatment suggested the assessment and intervention for nicotine withdrawal is not a routine consideration in hospitalized smokers (Moadel et al., 1999; Zabaneh, Ejaz, & Christiansen, 1994). Furthermore, concern has been raised regarding the impact of nicotine withdrawal on the adherence to medical treatment (Moadel et al., 1999). More research is needed regarding this potential interaction and effects upon outcomes to medical treatment.

One must be reminded that these results do not reflect representation of the total number of hospitalized smokers, only those willing to participate. The results are limited for generalizing beyond this sample. Future studies of hospitalized smokers need to consider measuring tobacco, comorbidity characteristics, and nicotine withdrawal symptoms, as well as smoking prevalence across gender and race. More information is needed regarding the cessation barriers and challenges that impact this particular smoking population.

Finally, hospitalized smokers in this sample perceived their hospital admission as an opportunity for change, but they lacked skills and assistance to meet this challenge of changing their smoking behavior. In order to prevent further mortality and morbidity by eliminating tobacco exposure, the healthcare community needs to address interventions that would alter this course of continuing relapse following hospital discharge by means of improved preparation of healthcare professionals (e.g., physicians and nurses) to assess and assist their patients in smoking cessation interventions. Future research is needed to address available and optimal smoking intervention with relapse prevention for hospital settings, particularly with the current trend for shorter hospital stays. The identified variables associated with changes in smoking

behavior need to be incorporated in future research of this population and considered in the development of smoking intervention programs targeting the hospitalized smoker. Routine consistent implementation, beyond current trends, of smoking assessments and cessation interventions in healthcare delivery settings, such as hospitals, have the potential to impact individuals and society by decreasing the prevalence of smoking related disorders, associated costs required to treat these disorders, and years of lost potential productivity.

4. CHAPTER FOUR

4.1. METHODS

4.1.1. Research design

The conceptual framework of Self-Efficacy Theory drove the intervention focus of the primary aim, which was consistent with the findings in preliminary work that self-efficacy is a strong predictor of nonsmoking status or tobacco abstinence. This intervention study used a randomized controlled two-group design. Study variables were measured at three different time points, which included Baseline (T_0), 12 weeks following discharge (T_1), and 24 weeks following discharge (T_2). The focus of the primary aim was to examine the efficacy of the intervention on smoking status at follow-up. Two secondary aims were used to test variable relationships. The first secondary aim examined the predictive relationship of conceptual variables to predict smoking status at T_1 and T_2. The second secondary aim examined relationships between treatment related variables (lapse, treatment adherence, smoking behavior, and self-efficacy), as well as within subject testing of self-efficacy over time.

4.1.2. Setting

Subjects were recruited from a sample of adult patients hospitalized on medical or surgical units within two hospitals associated with an academic medical center and one suburban hospital. Each of these hospitals provided medical/surgical treatments to patients. Intensive Care/Critical Care patient units were not used as recruitment locations within these facilities. These locations

provided initial access to patients with positive smoking histories as defined by this project. Consent for study participation, baseline measures, and tobacco abstinence promotion were conducted prior to hospital discharge.

4.1.3. Population

It was anticipated that a sample of 150 subjects would be recruited for participation from tertiary medical/surgical patient care units. Prior to the implementation of the preliminary study, the administrative personnel from hospital site A reported that the combined weekly census for hospital site A ranged from 250 - 300 patients. Approximately 25% of the patients identified in the preliminary study had a positive current smoking status. The U.S. and Pennsylvania average smoking rates range from 24 to 25%. Therefore, this project anticipated a similar representation of smokers within the sample of hospitalized patients. Each week, the estimated number of eligible hospitalized smokers for study participation ranged from 60 to 72 patients. Recruitment results from the preliminary study suggested that of all patients screened for that study, 9% agreed to participate, which suggested that recruitment projections for this study expected four subjects enrolled each week over 22 weeks. In the preliminary study, the data suggested that the 9% of subjects who agreed to participate were similar to the participants recruited for this study with respect to admission for a tobacco related health problem, age, marital status, employment, health insurance, education, and cigarette consumption. More males participated in the previous study.

4.1.3.1. Human subject selection criteria. Subject selection was guided by the following inclusion and exclusion criteria. Inclusion criteria for this project required participants to be admitted to a medical or surgical patient care unit and a current smoker. Patients were required to be 18 years or older, of either gender, with no exclusion by diagnosis unless admitted for

transplantation or terminal condition with death imminent. A current smoker for recruitment purposes was defined as a patient who had smoked one cigarette within 30 days of the current hospital admission. This definition of smoking for study inclusion was consistent with the definition of current smoking for previous intervention studies (Dornelas et al., 2000; Miller et al., 1997b; Taylor et al., 1996), and the preliminary study presented in chapter 3. Patients could have been asked by their physician to refrain from smoking prior to their hospital admission, particularly for surgical admissions. Approximately 40-60 % of smokers relapse beyond one month of a smoking cessation attempt (Fortmann & Killen, 1995). Therefore, smokers abstaining just prior to and during a hospital admission are at risk for relapse following discharge.

Exclusion criteria for this project included the following: 1) diagnosis of cancer in a terminal state, 2) patients under evaluation for organ transplantation or awaiting transplantation, 3) cerebral vascular disorders, 4) senile dementia, 5) Alzheimer disease, 6) abstinence from smoking greater than one month, 7) non-English speaking patients, 8) lack of a home telephone, 9) lack of a mailing address, 10) lack of any ability to participate with self-care activities, and 11) transfer to a rehabilitation hospital or nursing home following hospital admission. These exclusion criteria eliminated patients who were either too ill to participate at the time of recruitment, lacked cognitive function to participate with intervention activities, or lacked home facilities necessary for study participation (e.g., telephone, home address).

4.1.3.2. Gender, ethnic minority, and admitting diagnosis. Recruitment efforts attempted to sample equally of males and females with minority sampling representative of the local statistics. In addition, sampling also controlled for admitting diagnoses with respect to relationship to whether related to tobacco consumption. At the time this study was proposed (2001), 1992 and 2000 data on race/minority were used. According to the 1992 estimated distributions of

Allegheny County residents by race/minority, Whites comprised 87% and Blacks 12% of the surrounding population total (Anonymous, 1996; U.S. Census Bureau, 2002). This study and the preliminary study had a greater sampling of Blacks/African Americans than the Allegheny census estimates. Estimates from the 2000 census reported 85% Whites, 12% Blacks/African American, 3% other. Gender for Allegheny County was estimated by the 2000 census to have 53% females and 47% males (U.S. Census Bureau, 2002). The sampling of females was higher than the estimates for the Allegheny census by 7%. The current study enrolled 48 women (60%) and 32 (40%) men. The preliminary study did obtain similar sampling by race/minority to the county estimates, but did not obtain similar sampling by gender, which enrolled a greater percentage of men. Cardiovascular admitting diagnoses were the predominant type of diagnoses encountered with the preliminary study, however, this pilot study anticipated enrolling across tobacco related and non-related diagnoses.

4.1.3.3. Minority - adolescents. Adolescent patients admitted to primarily adult hospital facilities represent a minority within the admitted hospital population. However, age status should not preclude smoking patients of this age group from participation in this project. Adolescent smokers younger than 18 years were not encountered with recruitment for the preliminary study. Participants were required to be at least 18 years of age or older.

4.1.3.4. Estimated sample size. *S*everal intervention studies have been conducted with hospitalized smokers. Many of these studies have been underpowered. Taylor et al. (1990) reported the results of a smoking cessation program for hospitalized smokers following a myocardial infarction. The sample consisted of 166 subjects randomized to either treatment or usual care. Twelve months following randomization, abstinence rates were significantly different between the two randomized groups ($\chi 2$ [1, n = 166] = 14.04, p = .0001, effect size (w) = .29).

This study had one of the largest reported effect sizes for smoking cessation in a hospitalized population, but this effect has not been replicated in a sample of hospitalized smokers with varied diagnoses 12 months following randomization or the end of study treatment. Table 12 provides an overview of four studies that reported abstinence rates less than 6 months following randomization or the end of tobacco abstinence treatment with samples of smokers recruited during hospitalization. Although two of these studies had significant differences in smoking abstinence between study groups, none of them had effect sizes (w) greater than .18.

Table 12 Short-term (< 6 months) abstinence results for hospitalized smokers

Author/Year	n	$\chi 2$	df	p - value	Effect Size w
Wewers, et al. (1994)	80	1.39	1	.24	.13
Taylor, et al. (1996)	628	21.15	1	<.001	.18
Rigotti, et al. (1997)	635	8.73	1	.003	.12
Griebel, et al. (1998)	28	.24	1	.62	.09

Table 13 provides an overview of tobacco abstinence findings 6 months following hospital discharge or the end of tobacco abstinence treatment. Most studies had low effect sizes less than .16. However, Dornelas, et al. (2000) had an effect size (w) of .27 for greater tobacco abstinence at 6 months. Similar to Taylor, et al. (1990), the sample was comprised of subjects with cardiovascular disorders.

Table 13 Six-month abstinence results for hospitalized smokers

Author/Year	n	$\chi 2$	df	p - value	Effect Size w
Taylor, et al. (1996)	628	7.70	1	.006	.11
Rigotti, et al. (1997)	615	.08	1	.78	.01
Simon, et al. (1997)	274	3.41	1	.06	.11
Johnson, et al. (1999)	102	2.50	1	.11	.16
Dornelas, et al. (2000)	100	5.42	1	.02	.23

Table 14 provides the results of 12-month measures of tobacco abstinence between study groups for five different studies of hospitalized smokers. All but one of these studies had statistically significant findings. Effect sizes (w) ranged from .07 to .29. Studies by Taylor, et al. (1990) and Dornelas, et al. (2000) both studied subjects with cardiovascular disorders and had effect sizes greater than .23, but less than .3.

Table 14 Twelve-month abstinence rates for hospitalized smokers

Author/Year	n	$\chi 2$	df	P - value	Effect Size w
Taylor, et al. (1990)	166	14.04	1	.0002	.29
Taylor, et al. (1996)	628	8.25	1	.004	.12
Miller, et al. (1997)	1942	8.14	2	.02	.07
Simon, et al. (1997)	299	4.10	1	.04	.12
Dornelas, et al. (2000)	100	5.84	1	.02	.24

Power versus sample size curve in Figure 3 provides a graphic illustration of power (.2 - 1.0), sample sizes (50 to 800), and effect sizes (w = .1, .15, .2, .25, and .3) for a $\chi 2$ analysis with an $\alpha = .10$ (one tailed hypothesis). This test of power analysis does not take into consideration multiple testing. The effect sizes for the studies previously noted in Table 11 are plotted on this curve to graphically illustrate their effect sizes and sample sizes in relationship to the analysis for power and sample size for this study.

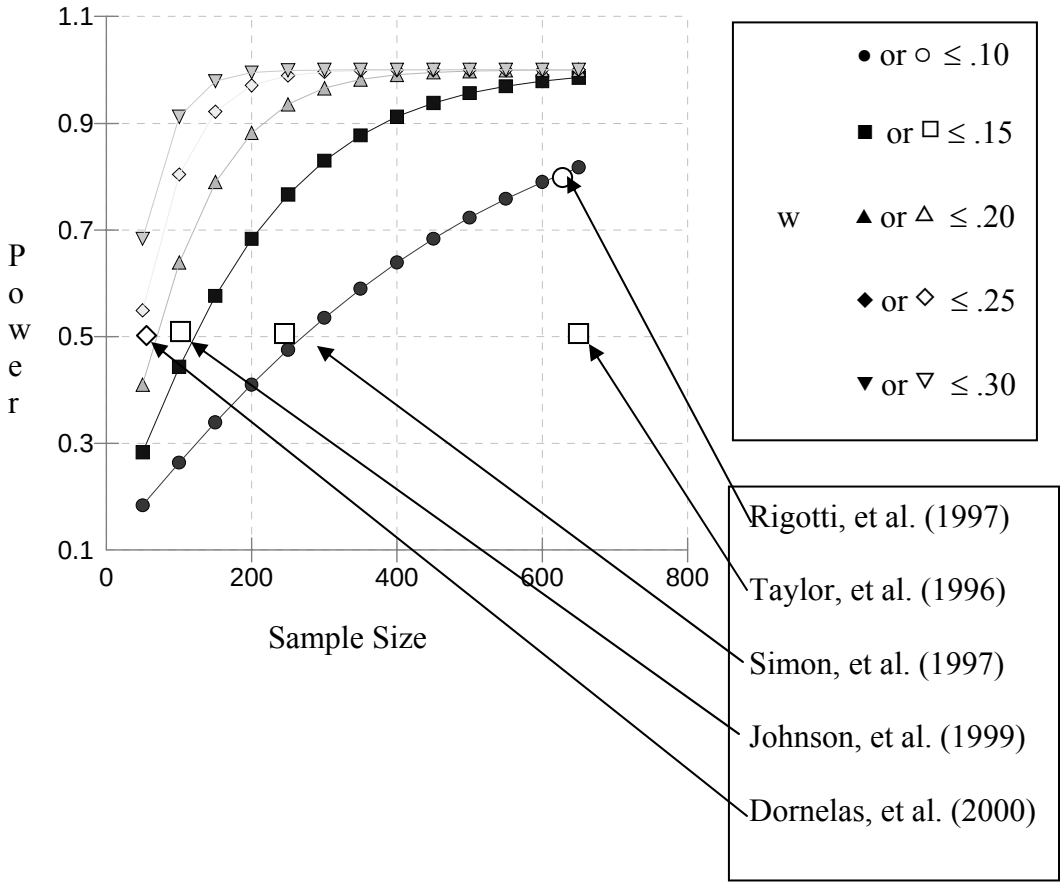


Figure 3 Power curve vs. sample size by effect size (w) with df = 1, alpha = .1, $\chi 2$ test

The preliminary study results presented in chapter 3 had a sample size of 118 subjects. Of all subjects, only 22 self-reported a non-smoking status at the follow-up interview after hospital discharge, which represented 18.6% of the total population of study subjects. Approximately 80% of the subjects were smoking by the follow-up interview. This rate of tobacco abstinence following hospital discharge was greater than the usual care abstinence rates reported for the general smoking population (Fiore, Novotny, Pierce, Giovino, Hatziandreu, Newcomb, Surawicz, & Davis, 1990). However, the percentage of subjects abstinent upon follow-up in the preliminary study was similar to the usual care rate of abstinence found in a sample of hospitalized subjects reported by Miller, et al. (1997). The results of the preliminary study were consistent with expectations of abstinence with minimal to no professional healthcare intervention effort in a hospitalized sample. Therefore, an enhanced usual care group for this currently proposed study will likely have an abstinence rate greater or equal to approximately 19% within the first six months following hospital discharge.

Power analysis, effect size, and sample size were considered after reviewing the above findings. Sample size versus effect size for a $\chi 2$ analysis is reflected in Figure 4, which was produced with PASS 2000 software. Sample sizes for this curve range from 69 to 785 for a $\chi 2$ with 1degree of freedom (df). The effect sizes range from .1 to .3, a power = .8, α = .05 and .10 respectively. Since the hypothesis for efficacy of the intervention considered a one-tailed effect, both .05 and .10 were used in this power and sample size analysis. Table 15 numerically represents this information. If an effect size of .3 were obtained for the intervention treatment over the enhanced usual care groups, a sample size of 70 (35 per group) would have been a reasonable sample size to recruit within the scheduled 22 weeks for recruitment, however, due to

anticipated attrition of subjects following hospital discharge, an additional 18 subjects were projected as necessary in addition to the total sample to ensure a sample size by the T_2 (24 weeks following hospital discharge) follow-up period.

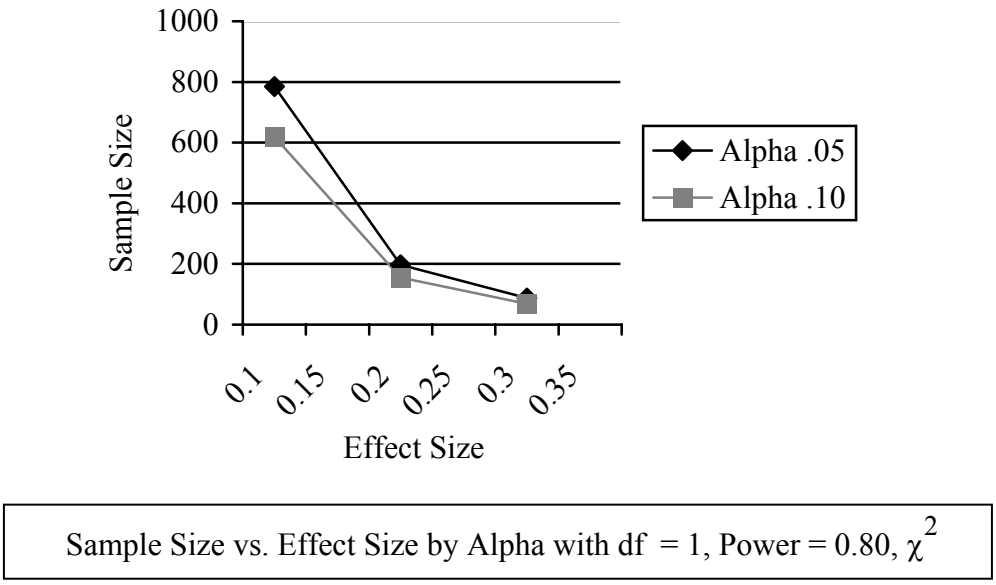


Figure 4 Sample size versus effect size curve

Table 15 Results of a Chi-square power analysis

Power	Sample size	Effect size w	$\chi 2$	df	α
.80	785	.10	7.85	1	.05
.80	619	.10	6.19	1	.10
.80	197	.20	7.88	1	.05
.80	155	.20	6.20	1	.10
.80	88	.30	7.92	1	.05
.80	69	.30	6.21	1	.10

4.1.3.5. Feasible sample size. The sample sizes estimated for an effect size of .1 and .2 range between 619 and 155, although one must consider if this is a feasible sample size to obtain from a hospital location for a clinical study within a 22-week recruitment window. As previously noted the preliminary study to this project recruited 118 hospitalized smokers from approximately 6 to 8 nursing units in a metropolitan university-based hospital. Of 1,322 patients screened, 24% (314) of the responders were current cigarette smokers and only 38% of these individuals were willing to participate in a survey regarding smoking or 9% of the total screened. This recruitment effort required a larger screening of patients to obtain a 9% yield for recruitment or 155 subjects. A sample size of 150 was established for this project, designed to be a pilot for future behavioral intervention studies. If the intervention approach was novel and provided an effect size similar to Taylor's et al (1990), a smaller sample size of 66 to 88 subjects would provide a feasible sample size for this preliminary study. With respect to intervention, follow-up, and scheduling contacts, a sample of 150 subjects was estimated to require a minimum of 1200 separate contacts.

4.1.3.6. Proposed sample size. Given the information previously provided, an estimated sample size of 150 subjects was initially established. The study by Miller et al. (1997) reported a retention rate of 86%, suggesting a retention rate of 75% as conservative. A sample size of 150 was anticipated to require more assets and recruitment time. Therefore, it was estimated that approximately 130 subjects would complete the study.

In summary, a projected sample size of 150 smokers enrolled during hospitalization was anticipated to preliminarily examine the efficacy of a 12-week tobacco abstinence and relapse management program initiated at the time of hospital discharge. In addition to examining the

efficacy of this nurse provided intervention, relationships were examined between smoking abstinence and intervention treatment adherence, as well as with covariates (e.g., perceived self-efficacy, perceived treatment efficacy, and relapse situation efficacy).

4.1.4. Sampling and assignment procedures

Randomization of subjects to treatment assignment was used to control for unknown variables that may introduce bias to the outcome results (Freidman, Furberg, & De Mets, 1996). Specifically, adaptive randomization by minimization was used, which attempts to prevent imbalances from baseline characteristics that may affect results. Freidman et al. (1996) indicate that this method of randomization is incorporated in clinical trials. Their example was that of trials regarding cancer. Furthermore, Friedman et al. (1996) report that the strength of this method of randomization provides unbiased estimates of the treatment effect and protects the overall marginal balance. Power may also be slightly enhanced if the stratification does not include all possible covariates (Freidman, Furberg, & De Mets, 1996).

This study planned to recruit equally by gender and proportionally by white and black subjects from the participating hospital patient care units in order to reflect proportions of the surrounding county statistics. In addition, the preliminary study had difficulty obtaining participation of female subjects. Therefore, to maintain balance between genders was necessary if an adequate number of females were not included. Fortunately, this study did not have difficulty recruiting female participants. Ethnic status was closely monitored during recruitment. Subjects were randomly assigned to intervention groups by means of a baseline adaptive randomization procedure that adjusted the random assignment to maintain balance of the groups for minority and gender sampling, as well as tobacco related diagnosis categories for the current admission (Friedman, Furberg, & De Mets, 1996). Therefore, study groups had an equal number

(ratio 1:1) assigned for treatment while controlling for an equal number by gender, ethnicity, and tobacco related diagnosis. In order to enhance recruitment by gender, ethnicity, tobacco related diagnosis, and age, at least one nurse located on each of the patient care units was asked to assist the recruitment efforts of this study by participating as a liaison between the patient care unit and the research project to ensure that patients had an opportunity to obtain information regarding the study and ask questions of the study investigator. This request for a liaison was used in the preliminary study, which assisted the identification of eligible subjects for recruitment.

This process of adaptive randomization attempted to provide a balance of the treatment assignment with respect to potential sociodemographic (e.g., gender, ethnicity) and tobacco related admission (e.g. 3 categories) characteristics capable of introducing accidental bias of the results if not controlled. The randomization procedures used a computer-generated assignment to eliminate predictability of this procedure. This computer software program for randomization was developed with the assistance of support staff from the Center for Research in Chronic Disorders. Gender contained two categories male and female. Ethnicity contained three categories (e.g., White, Black, Other). Within the surrounding county, individuals of White or Black ethnicity comprised 99% of the population. All other ethnic minorities comprise the remaining 1% of the population. Tobacco related diagnosis was divided into three categories for the purposes of randomization (e.g., tobacco related diagnosis and comorbidities, a non-tobacco related diagnosis with tobacco related comorbidities, and a non-tobacco related admission and no tobacco related comorbidities.

4.1.5. Procedures

4.1.5.1. Recruitment procedures. Subjects were prospectively recruited following hospital admission, but prior to discharge. Patients received a flyer from the unit nurse for this study upon admission to their patient care unit. An overview of the project's aims, population of interest, and procedures were provided to patient care unit staff (e.g., nurses, physicians, respiratory therapists, etc.) as part of an inservice regarding the project. As noted earlier, effort was made to request a nurse liaison from the patient care units to assist with the identification of eligible subjects for recruitment. This practice was successful in one of the hospitals used for recruitment. In both of the other sites, cardiopulmonary rehabilitation staff were required to see all smoking patients, therefore, nurse liaisons were not available for the project at those sites. Recruitment flyers were provided to all patient care units and particularly to nursing staff on a daily basis at hospital "A". Recruitment materials were provided to the cardiopulmonary therapists at the other two hospitals. In most cases, the healthcare providers provided the recruitment flyers to their patients and returned completed flyers to the study investigator. Patients were also able to contact the study directly by calling a number located on the bottom of the recruitment flyer to request information or a visit by study investigator. In addition, patients interested in study participation were permitted to request hospital staff to contact the study personnel by telephone/pager for an informational visit in the early phases of this project.

Due to changes in the implementation of the Health Insurance Portability and Accountability Act (HIPAA) within the first year of the study, recruitment flyers required alteration to eliminate the disclosure of confidential patient information in an unprotected or secure manner. New recruitment brochures (See Appendix B) were designed in a tri-fold form. The brochure described the study and contact procedures. A form on the inside of the brochure

required the patient to complete the information, close the form, and seal it with the provided adhesive tag. The brochure instructed patients to provide the sealed brochure to their nurse. The staff initiated a telephone call to the investigator when they received a completed brochure from a patient to instruct the investigator that a completed brochure was available for pick-up. No additional information was disclosed to the investigator on the telephone. The investigator did not have any information regarding a patient until the brochure was opened by the investigator.

In addition to the brochure, a "consent to approach" was available to hospital staff to use in order to disclose information regarding the project. This form was used in only one recruitment situation. Referring staff and patients preferred the brochure to read about the project and indicate study interest. Due to the changes required to protect patient information useful recruitment information regarding the sample was not available.

Patients interested in study information were presented with an overview and explanation of this study by the investigator and/or a recruitment coordinator. A recruitment coordinator was used in the first year of the study. After thoroughly reviewing the study consent form, only those patients agreeable to participation signed a consent form approved by the Institutional Review Board at the University of Pittsburgh. This consent form was thoroughly reviewed with each prospective subject. If still agreeable to study participation, patients were asked to initial and sign a study consent form prior to discharge and active study participation.

4.1.5.2. Study procedures. Following consent, baseline data collection was completed. Randomization to group assignment was then carried out. All subjects received usual care smoking cessation activities provided by the institution where they were admitted and enhanced interventions from the study to promote tobacco abstinence. Based upon the results of the preliminary study, messages promoting tobacco abstinence by healthcare providers were the

most frequent usual care interventions provided to hospitalized smokers. Only 9% of the subjects in the preliminary study received interventions beyond an abstinence promotion message. Therefore, this study incorporated an enhanced usual care format by providing each subject with a promotional message for tobacco abstinence and a manual for tobacco dependency treatment published by the Center for Disease Control and additional references from the Arizona Smokers Helpline (Refer to Appendix A). The clinical guidelines for smoking intervention, published by the Public Health Service, promotes a minimal intervention of at least three minutes of counseling for tobacco dependent patients (Fiore et al., 2000).

4.1.5.3. Enhanced usual care group. Subjects in this study received minimal intervention provided by healthcare providers within the hospital and an enhanced usual care intervention from the study. An enhanced usual care program was provided to assure that each participant started with the same smoking cessation information at the time of hospitalization. The practice guidelines regarding the promotion of smoking cessation by healthcare providers promotes the implementation of smoking cessation assistance with counseling (Fiore et al., 1996). The enhanced usual care consisted of a health promotional message from study personnel to abstain from tobacco due to associated health risks. In addition, these subjects received the study manual for their personal support and direction towards tobacco abstinence along with a profile of relapse risk based upon scores from the RSEQ. The manual consisted of the "You Can Quit" booklet available on the web from the Center for Disease Control and Prevention (Center for Disease Control and Prevention, 2000b). Additional informational sheets were used with permission from the Arizona Helpline (See Appendix B). Study specific informational sheets were prepared regarding strategies and relapse strategy categories (See Appendix A). Important points on each page of the "You Can Quit" booklet were highlighted with printed labels (See

Appendix A). The booklets were prepared with blank sheets of paper to accommodate notes and journal entries by participants. The nurse investigator provided an overview of the content in the manual with each subject prior to discharge, as well as a review of nicotine addiction, the relapse risk profile, and associated interventions. Subjects assigned to only the "enhanced usual care" did not receive any additional interventions for tobacco dependency from study personnel following their hospital discharge. Furthermore, subjects assigned to the enhanced usual care group completed baseline measures prior to hospital discharge, which was standard with all study subjects. Follow-up measures were completed at 12 weeks and 24 weeks following discharge. Subjects were called to schedule their follow-up appointments at 10 to 11 weeks post discharge for the first follow-up session (T_1). Scheduling calls for the final follow-up visit T_2 (24 weeks following discharge) occurred between the 22nd and 23rd week.

4.1.5.4. Tobacco abstinence and relapse maintenance intervention-Intervention group. Subjects assigned the study intervention received usual care, enhanced usual care interventions, and the relapse maintenance intervention following hospital discharge. The relapse maintenance intervention used Self-efficacy Theory as the conceptual framework. The goal of this approach was to enhance the perceived self-efficacy of these subjects with regard to their tobacco abstinence and relapse maintenance efforts. Intervention activities began with an initial session prior to hospital discharge. Efforts were made to deliver this session within 24 hours prior to discharge.

The initial intervention session was comprised of three parts, which included: 1) assessment of self-knowledge, 2) modeling, and 3) goal setting. The initial task addressed by the interventionist with the subject was an assessment of situational efficacy for tobacco relapse. Essential to the process of enhancing self-efficacy was the assessment of self-knowledge of how

to manage risk situations for relapse and nicotine withdrawal symptoms (Bandura, 1997). This process was necessary before goals and modeling strategies were planned. As part of this session, the scores from the RSEQ (completed for the baseline assessment) were used as a starting point for the intervention group subjects in preventing relapse (Gwaltney et al., 2001). Based upon findings by Gwaltney, et al. (2001), the specific relapse situation receiving the lowest confidence score with this instrument best predicted subsequent relapse to tobacco use. In addition, the authors suggested using the lowest confidence situation as a starting point to thwart relapse. Subjects were assessed as to how they would cope with these relapse factors (negative affect, positive affect, restrictive situations, idle time, social/food situations, low arousal, and craving) and nicotine withdrawal symptoms.

Modeling assisted with the development of planned coping strategies for identified high-risk relapse situations. Verbal modeling by the interventionist began this process of modeling within the intervention sessions. Subjects were requested to document this information in the journal pages provided in the back of the study manual. These journal entries were anticipated to assist the participant with a written reminder of the intervention discussion. Subjects were asked to maintain this journal of the sessions for the first 12 weeks after hospital discharge. In addition, the journals were then available for the subject to document self-monitoring of their tobacco abstinence in order to evaluate their mastery effort at subsequent intervention sessions with their nurse interventionist. The interventionist and subject agreed upon a planned goal to guide the subject until the 2nd intervention session, as well as the time of day and telephone number the interventionist should use for the next intervention session.

The second intervention session was the first telephone intervention delivered to the subject and occurred the day following discharge. All subsequent interventions were delivered by telephone by the interventionist to the subject. The interventionist reviewed the goal planned from the last intervention session. This procedure was done at all subsequent intervention sessions. Subject was asked to review any lapses or success with their tobacco abstinence since hospital discharge. Lapses were defined with subjects as one puff of a cigarette. For interventions focusing upon lapses that occurred in the proceeding interval between interventions, there were four components addressed in the intervention. These included the following: 1) assessment of the situation related to the lapse and those with out lapses, 2) re-labeling of the modeling components that impact maintaining abstinence, 3) verbal persuasion for reinforcement, and 4) goal setting for the next interval between interventions. With guidance by the interventionist, the subject completed an assessment of their planned strategies modeled in the last intervention session. A format of "what worked and what did not," as well as "how well" did planned strategies work was used following the disclosure of their tobacco abstinence behavior. If subjects lapsed, the interventionist re-labeled the situation that led to the lapse and modeled planned coping strategies for use over the next week. The subject and interventionist determined the goal for the next interval between intervention sessions and briefly reviewed the re-labeled coping model. Finally, the interventionist used verbal persuasion to strengthen the subject's self-efficacy beliefs, which encouraged and acknowledged the subject's ability to abstain from tobacco. The session ended after the next telephone session was scheduled.

When success in maintaining abstinence was encountered by the subject, the intervention focused upon the following components: 1) verbal reinforcement of ability to abstain in high-risk situations, 2) comparison of the experience with the model provided in the last intervention with

experience since last intervention, and 3) goal setting for the next interval between interventions. In the case of successful abstinence since hospital discharge, the interventionist provided verbal reinforcement of their mastery of the abstinence goal set the day before. An assessment or review was discussed regarding how the participant coped with risk situations for relapse. The experience was compared with the modeling discussed at the previous intervention. With guidance by the interventionist, the subject selected a goal for the next interval between intervention sessions and coping modeling for relapse situations was reviewed. The session ended after the next intervention session was scheduled for the next week.

Subsequent sessions had a format as outlined for session 2. The path the intervention call took depended upon whether the participant experienced a lapse or they were successful with tobacco abstinence. One-week intervals occurred between sessions 2 through 6. Two-week intervals occurred between sessions 6 through 8. Intervention session 9 was the final intervention session and occurred 3 weeks following session 8, which coincided with 11 weeks following hospital discharge. Subjects were reminded at session 8 that scheduling for the first follow-up visit (T_1) would be done at session 9. At session 9, subjects were asked to schedule the day and time for their follow-up visit. The final review session provided an opportunity for the subject to evaluate his/her efforts over the past 11 weeks of the intervention activities and discuss strategies (e.g., vicarious experience and modeling) for continued relapse maintenance efforts for their tobacco dependence. Please refer to Figure 5 for a graphic depiction of the sequence of intervention sessions.

4.1.5.5. Intervention fidelity. Telephone interventions were recorded in order to monitor the adherence of the nurse interventionist to the treatment protocol. There was only one nurse interventionist for this study. Contingent upon agreement by the subject, intervention sessions

were recorded by the nurse. Only the dialog by the nurse was recorded. Participants were referred to by first name to maintain confidentiality. Approximately 15% of each type of intervention session was reviewed to assess intervention fidelity. These tapes were reviewed by the interventionist and a third party. Figure 5 provides a graphical illustration of the intervention outline. This structure was maintained in the intervention calls reviewed. There was consistency of the information requested regarding lapses and the reinforcement provided at the end of the intervention session.

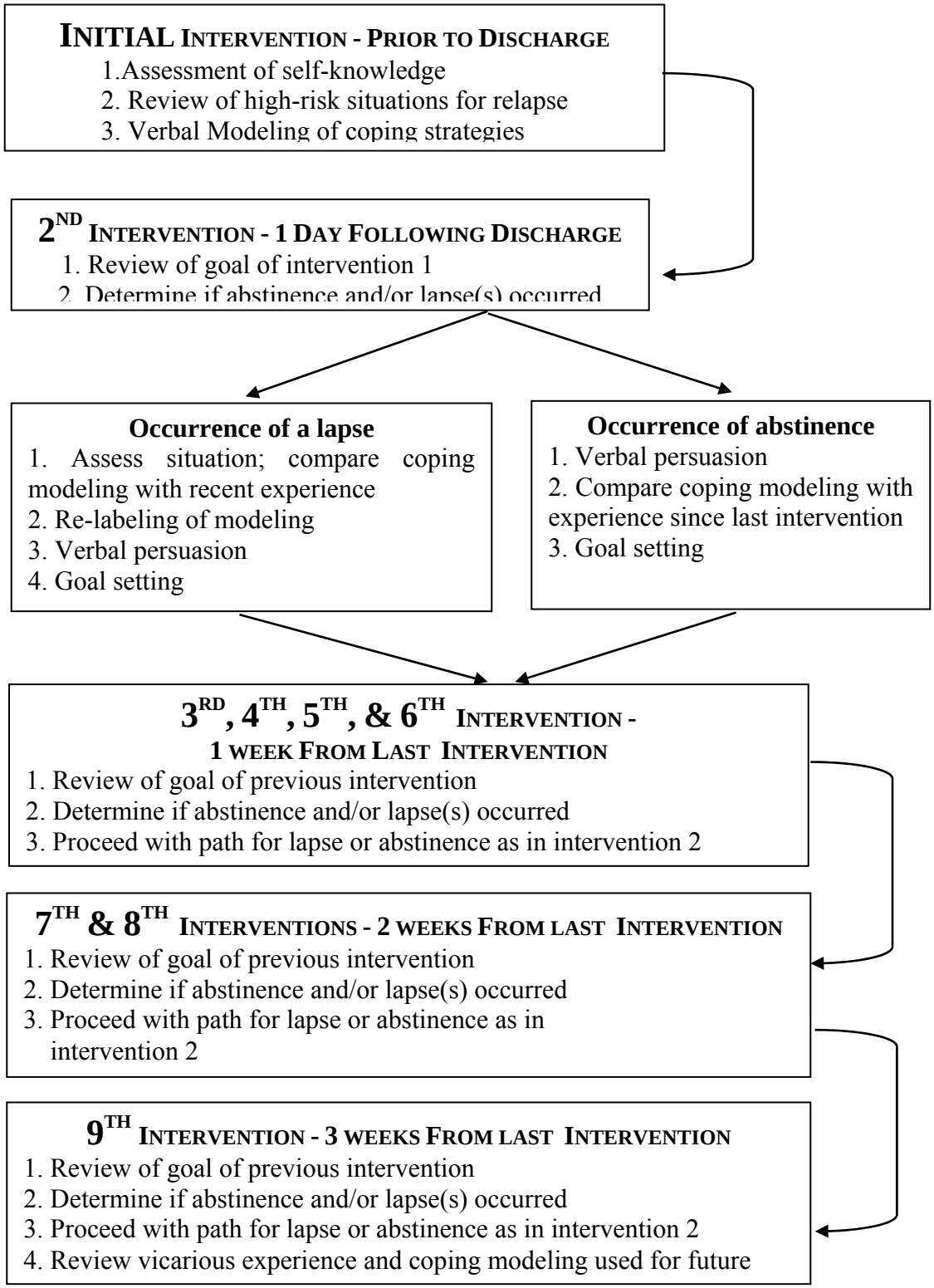


Figure 5 Graphic depiction of the intervention process

4.1.5.6. Baseline and follow-up visits. All subjects, regardless of study group assignment, were anticipated to participate with baseline and follow-up activities. Baseline measurements were completed during hospitalization (T_0). Follow-up measurements were completed at 12 weeks (T_1) and 24 weeks (T_2) post hospital discharge. Arrangements were made to meet with subjects for follow-up visits at the University of Pittsburgh School of Nursing. If the subject could not be transported, arrangements were made for a visit near or in the subject's home.

4.1.5.7. Baseline activities. Consenting subjects completed subject identification information and baseline measures. In addition to obtaining self-reported smoking status with carbon monoxide validation, the following baseline measures were completed by the participant unless they were physically incapable of completing the forms (e.g. broken arm) or were fatigued: 1) CRCD Sociodemographic Questionnaire, 2) Tobacco Consumption Questionnaire, 3) Fagerstrom Test for Nicotine Dependence, 4) Profile of Mood States inventory, 5) Hughes-Hatsukami Tobacco Withdrawal form, 6) Tobacco Abstinence Self-efficacy Scale, 7) Perceived Treatment Efficacy Scale for Relapse Management, 8) CRCD Comorbidity Questionnaire. The principal investigator completed an intake questionnaire regarding home address and phone numbers with the participant by interview. At this time, subjects received their study contact information and study participation packet, which included their tobacco abstinence manual. After obtaining baseline measures, study personnel completed a review of chart data pertaining to the subject's hospital diagnosis, medical/surgical treatment, and past medical/surgical history.

4.1.5.8. Follow-up visit T1. Subjects assigned to enhanced usual care only were contacted by telephone 10 to 11 weeks following hospital discharge to schedule their first follow-up visit (FUP) (T_1) with the investigator. As previously noted, scheduling of subjects assigned to the intervention with enhanced usual care will occur at their 9^{th} intervention session. All subjects

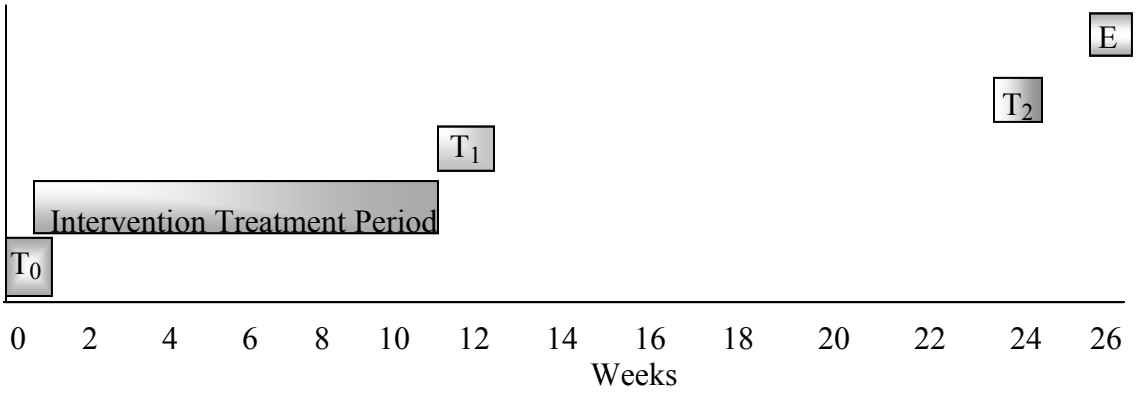
will be asked to provide a self-report of their smoking status as defined in chapter 1. At least one exhaled carbon monoxide reading was collected from subjects prior to the end of their follow-up visit. The questionnaires completed at baseline were re-administered at this time. In addition, subjects were asked to complete a questionnaire regarding smoking cessation interventions they received from hospital personnel since the time of their hospitalization 12 weeks prior to the first follow-up visit.

4.1.5.9. Follow-up visit T2. Between 22 and 23 weeks following hospital discharge, all subjects were contacted to schedule their second and final follow-up visit (T_2) with the investigator. Activities at these visits were identical to those completed at T_1.

4.1.5.10. Study exit. All subjects were sent a thank you card for their participation in the project. Subjects were reminded that the study tobacco abstinence manual contained information pertaining to smoking cessation and relapse maintenance resources. These resources included telephone numbers and web sites for programs, hotlines, and informational services.

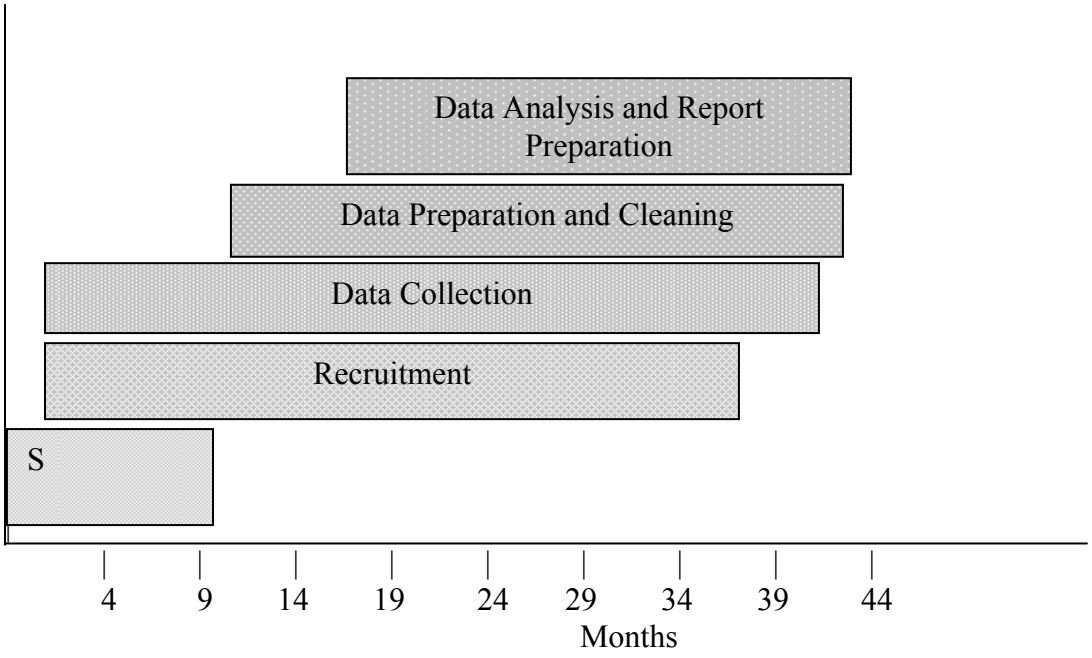
4.1.5.11. Time frame. The total time any one subject participated in this study was 26 weeks beyond hospital discharge. Figure 4 provides a graphic representation of the activities of this project for an individual subject assigned to the intervention group. The initial start-up phase required the following activities: 1) prepare and print study materials, 2) preparation of the database, and 3) obtain telephone access for the data collection phase. Data collection began with the enrollment of the first subject and continued until the last patient enrolled exited the study. The time-period for data collection required a 38 months and approximately two weeks (July 2002 – September 2004). Data preparation and downloading began with the active enrollment of subjects. Data cleaning began following preparation of database tables and downloading baseline measures. Baseline data analysis began shortly after the enrollment of the last subject into the

study, which included analysis of baseline subject characteristics. Further data analysis was completed as data collection and data cleaning were completed for T_1 and T_2. Report generation followed the analysis of results. Figure 7 provides an overview of study activity for this proposed time frame.



(E - Study Exit)

Figure 6 Study activity time frame for an intervention group subject



(S = Administrative start-up)

Figure 7 Overview of study activity time frame

4.1.5.12. Risk/benefit ratio. There was minimal risk associated with participation in this project. Subjects could become fatigued while completing the survey questions and they were monitored for complaints of fatigue and distress. Interviews for intervention and data collection were monitored and provided breaks according to requests by subjects. Written recording assistance was provided with form completion if subjects were fatigued. Participants were permitted to dictate answers to study personnel. There was potential benefit for subjects by participating in this project. There was potential that they would develop an increased awareness of their own smoking behavior regardless of treatment assignment. Subjects also had the potential to change their smoking behavior. Future research and clinical practice will benefit by dissemination of results regarding the use of this cognitive behavioral approach to tobacco abstinence promotion and relapse management following hospital discharge.

4.1.5.13. Confidentiality. Confidentiality of subjects' names and personal information was protected by the following procedures. All data files (paper and computer diskettes) and code linking sheets were maintained in a locked file cabinet with restricted access to only the investigator. Computer files and reports used coded participant identification numbers to provide protection of patient identities. Future publications of study findings will not identify study participants. Subjects were instructed and assured that their responses to questions during the interviews, telephone dialogs, and follow-up sessions would be kept strictly confidential.

4.1.5.14. Costs and payments. The subjects, third party payers, hospitals where patients were seen for study recruitment, and the University of Pittsburgh did not incur charges for the conduct of this research project. A National Institute of Nursing Research predoctoral fellowship award (F31 NR07343) provided funding for salary support of the Principal Investigator and limited supplies. The Pennsylvania Nurses Foundation (Refer to Appendix B) and the Eta Chapter of

Sigma Theta Tau (Refer to Appendix B) provided funding to assist with the psychometric evaluation of the TASES and PTES. Subjects were not monetarily compensated for participating in this project, but did receive $10.00 stipends by the investigator to cover parking expenses incurred for attendance to follow-up visits at T_1 and T_2.

4.1.6. Measures

The primary dependent variable of interest for this study was smoking status. For purposes of this study, self-reported 7-day point prevalence of smoking status and biological measurement of smoking by exhaled carbon monoxide [CO] were used to define the smoking status of subjects at all follow-up visits. Together, these measures provided greater sensitivity in determining the smoking status of subjects. Subjects who reported abstinence for a seven-day period prior to the follow-up period and had an exhaled CO ≤ 8 ppm were defined as abstinent from inhaled tobacco. Subjects who report self-reported abstinence for seven days prior to a follow-up visit, but had CO > 8 ppm were defined as a smoking. However, low CO readings within 2 ppm were explored further to assess if the participant had an active pulmonary inflammatory process (e.g. exacerbation of asthma or chronic bronchitis) or acknowledged exposure to higher than normal concentrations of CO (e.g., occupational exposures). Subjects who reported current smoking in the seven days preceding the follow-up visit, but had CO ≤ 8 ppm were considered as current tobacco smokers. When subjects reported they were currently smoking and had CO > 8 ppm were categorized as currently smoking.

A carbon monoxide monitor is a gas detection sensor for expired alveolar carbon monoxide. The MicroCO™ carbon monoxide monitor was used for this project to measure for exhaled carbon monoxide. This device has a reported sensitivity of detecting carbon monoxide at 1 part per million (ppm) and a sensing range of 0 - 500 ppm. This CO sensor has a cross

sensitivity for hydrogen of < 3% and can operate in a temperature range from 0 to 40 degrees Celsius. The minimum life of the CO lithium sensor battery is 12 months and sensor drift of 2% per month. Recalibration is required monthly. A three-digit display appears on the top surface of the device to display CO in ppm. The MicroCO™ weighs 160 gm, has dimensions of 170 x 60 x 26 mm, and operates at atmospheric pressure (± 10%). The power source is a 9-volt battery, which provides 30 hours of operating time. Disposable cardboard mouthpieces that fit over the sensor were used for each subject (Micro Medical Limited, 1998).

Exhaled carbon monoxide measurements are efficient low cost biological markers for smoked tobacco consumption. The usefulness of this CO measure as a reliable biomarker is limited by a short window of 2-3 hours or 4-8 hours depending upon activity status (Coburn, Forster, & Kane, 1965). Both sensitivity and specificity of exhaled carbon monoxide range near 90% (Jarvis, Tunstall-Pedoe, Feyerabend, Vesey, & Saloojee, 1987). An acceptable CO range indicative of no smoking exposure in a general population ranges inclusively between 8 - 10 ppm (Cummings & Richard, 1988). This study used 8 ppm as the cutoff point. Subjects with readings greater than 8 ppm were considered as smoking regardless of self-reported smoking status.

At baseline and follow-up visits, medical information relevant to respiratory disorders and treatment, as well as environmental CO exposure, were recorded on the recording form for the CO reading. The information was not used as part of the algorithm to determine smoking status. As noted in a recent review of exhaled biomarkers, endogenous CO readings can be elevated by the presence of infectious processes (Kharitonov & Barnes, 2001). The administration of inhaled corticosteroids can decrease endogenous CO production. Therefore,

information pertinent to confounding factors of exhaled CO was measured for future analysis regarding the interpretation of this measure in comorbid populations. This information has not been documented regarding this smoking population.

In addition to smoking status, the aims of this study proposed to examine other relevant variables to tobacco abstinence and relapse. The study examined variables related to the Self-efficacy Theory, the conceptual framework of this study. Therefore, the study examined variables that operationalized perceived self-efficacy and outcome expectancy. Measures of self-efficacy included the Tobacco Abstinence Self-efficacy Scale and the Relapse Situation Efficacy Questionnaire. The Perceived Treatment Efficacy Scale for Relapse Management was used to measure outcome expectancy.

Treatment adherence was another concept of interest for examination by this study. As a measure of intervention effectiveness, the relationship between tobacco abstinence and adherence to the study intervention were examined. Adherence was defined as the homework completion. Presumably, intervention should only be effective in those who followed it.

4.1.6.1. Tobacco Abstinence Self-Efficacy Scale (TASES). The TASES was used to measure a general index of self-efficacy as it relates to tobacco abstinence. Items from this general measure of self-efficacy were scored 10 to 100, inclusively. A summation of the item scores provided a general score of self-efficacy for tobacco abstinence. The TASES contained 45 items. There were five subscales. Items 1 – 10 measured the perceived self-efficacy to control urges. The second subscale measured confidence in resisting urges over time after hospitalization (items 11 – 16). Items 17 – 20 measured confidence in resisting a puff of a cigarette with respect to hospital discharge and in the event of lapsing. Confidence in resisting a puff of a cigarette in particular situations was included in items 21 – 41. Finally, items 42 – 45 measured confidence

in resisting smoking during hospitalization. Psychometric properties of this tool were examined following the end data collection for the study. The TASES had an adequate test of internal consistency (Cronbach alpha = .99, n = 80). Item removal did not reveal any change in the internal consistency. Test-retest conducted between 39 of the subjects in the control group from baseline to T_1 revealed a test-retest reliability coefficient of .79. There was no significant change over time with respect to the first and second measurement of the TASES ($t = -1.4$, $df = 38$, $p = .170$).

A factor analysis was conducted to examine the structure of the TASES. The determinant was $9.955e^{-38}$, which raised concern for multicollinearity. The Kasier-Meyer-Olkin measure of sampling adequacy was .895 and indicated the factor analysis was appropriate for a correlation matrix from the TASES. Bartlett's test of sphericity suggested the correlation matrix was not an identity matrix (Approximate $\chi 2 = 5381.81$, $df = 990$, $p = .0001$). Five factors were extracted by principal component analysis. Varimax rotation was used to ease interpretation of factors. Loading was predominately on the first factor. The rotated component matrix revealed the five factors were similar to the five subscales specified in the development of the TASES. These five subscales include self-efficacy in control of urges and resisting smoking urges: in situations, over time, for a puff of cigarette, and during hospitalization. Therefore, the factor analysis supported the structure of the TASES and its five subscales as originally designed.

The Relapse Situation Efficacy Questionnaire [RSEQ] was used to test for convergent validity. Some similarities were expected between the two scales due to the measurement of situations of risk for tobacco relapse. However, the TASES attempted to measure the level of self-efficacy required to abstain from tobacco during and following hospitalization. Figure 8 graphically presents the correlations and p-values for the convergent validity testing between the

TASES and the RSEQ. In addition, the relationship of the TASES with other measures from the conceptual model, the POMS and PTES, were conducted. Spearman correlations were computed due to concern for normal distributions, particularly with the POMS and PTES. This figure illustrates that significant relationships existed between the baseline scores for all the covariate pairings except between the POMS and PTES. The RSEQ and TASES were highly correlated (r_s = .81, p = .0001). Moderate correlations were noted between the TASES and PTES (r_s = .64, p = .0001), as well as the RSEQ and the PTES (r_s = .49, p = .0001). Smaller inverse correlations noted between the TASES and the POMS (r_s = -.20, p = .01), as well as the RSEQ and the POMS (r_s = -.15, p = .05). Correlations between the TASES and the RSEQ were expected to be high due to the questions contained within each tool. Correlations between the TASES and the POMS were not expected to be as high, but a correlation in a negative direction was expected since affect is a source of self-efficacy. The correlation between the PTES and the TASES was exploratory for future validity referencing.

The TASES was examined for discriminant validity. The Fagerstrom Test for Nicotine Dependence [FTND] was not a concept depicted in this study's conceptual framework. Therefore, no relationship was anticipated between the FTND and the TASES. The Pearson Product Correlation between the two variables was equal to -.123 (n = 80), which was not significant (p = .278), which supported the discriminant expectation these two variables would not be related.

These findings provided an initial psychometric review of the TASES tool, which supported the reliability and stability of the tool in measurement over time. The construct validity of the tool was also supported with convergent and discriminant validity specific with

what it converged and with what it was unrelated. The factor analysis suggested the existence of some redundancy within the structure of the TASES. Further testing is warranted, as well as further examination of variable redundancy.

4.1.6.2. Relapse Situation Efficacy Questionnaire. A index of situational self-efficacy for abstinence effects was measured by the Relapse Situation Efficacy Questionnaire RSEQ (Gwaltney et al., 2001). There are 43 items scaled from 1 (Not confident) to 4 (Extremely confident) on the modified version of RSEQ. Gwaltney, et al., (2001) noted the RSEQ measures 7 factors, which include negative affect, positive affect, restrictive situations, idle time, social-food, low arousal, and craving. This tool is reported to have adequate internal consistency for the previously listed seven factors (Cronbach α's = .77 - .91) and the total factor of the RSEQ (Cronbach α = .96). The RSEQ has a low test-retest correlation of .52 (Gwaltney et al., 2001). Test-retest for the TASES was higher. Furthermore, Gwaltney, et al., (2001) noted the predictive association between the lowest scored abstinence self-efficacy (ASE) factor prior to treatment and a subsequent lapse. Therefore, this study used the baseline findings to rank relapse risk factors that could lead to a lapse and possible relapse for all subjects at baseline. The intervention used this relapse risk factor profile as a "starting point" with regard to potential relapse situations and associated coping strategies. By working on this vulnerable area, the subject was provided assistance with the situations posing the greatest risk for tobacco lapse and relapse.

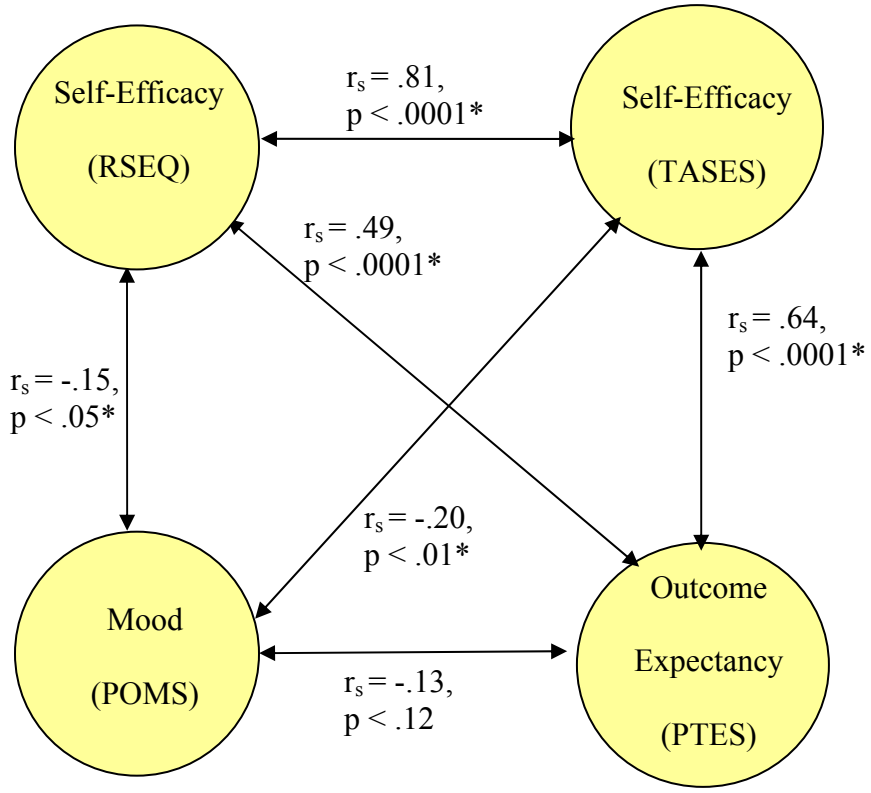


Figure 8 Spearman correlations between covariates

4.1.6.3. Perceived Therapeutic Efficacy Scale for Smoking Abstinence Treatment. Outcome expectancy is a concept found within Self-efficacy Theory, which is thought to be a mediating variable when individuals are attempting to change behavior, such as tobacco use (Bandura, 1997). The field of adherence research has embraced this concept through the measurement of perceived treatment efficacy (Dunbar-Jacob, Sereika, Burke, Kwoh, Rosella, McCall, Locke, Holmes, Bondi, Canty, & Starz, 1993; Schlenk, 2001). Therefore, this study used a modified version of the Perceived Therapeutic Efficacy Scale (Burke, Dunbar-Jacob, Sereika, & Ewart, 2003; Dunbar-Jacob et al., 1993; Schlenk, 2001). This scale contains 10 items scored from 0 to 10 inclusively. The adherence research team at the University of Pittsburgh School of Nursing CRCD has used this outcome expectancy scale in the study of subjects diagnosed with rheumatoid arthritis, cardiac disease, osteoarthritis, and fibromyalgia to examine medication, exercise, and dietary interventions (Burke et al., 2003; Dunbar-Jacob et al., 1993; Schlenk, 2001). Low scores are suggestive of low perceived therapeutic efficacy while high scores reflect higher perceptions of therapeutic efficacy. Internal consistency for these measures has ranged between an $\alpha = .87$ to an $\alpha = .96$ (Dunbar-Jacob et al., 1993; Schlenk, 2001). Test-retest scores were between $r = .61$ to $r = .90$. Higher test-retest scores occurred with repeat measures 16 weeks apart or less (Dunbar-Jacob et al., 1993; Schlenk, 2001).

Psychometric properties for this version of the PTES were examined following the end data collection for this study. The PTES had a high test of internal consistency (Cronbach alpha = .98, n = 80). Item removal did not reveal any change in the internal consistency. Test-retest was conducted between 17 of the subjects in the control group from baseline to T_1. Findings revealed a test-retest reliability coefficient of .54, which supports this tool as a state measure of

outcome expectancy. Subjects were tested 12 weeks beyond baseline in a small sample. There was no significant change over time with respect to the first and second measurement of the PTES ($t = -.226$, $df = 16$, $p = .824$).

A factor analysis was conducted to examine the validity of the PTES. The determinant was $2.243e^{-08}$, which raised concern for some multicollinearity. The Kasier-Meyer-Olkin measure of sampling adequacy was .911, which indicated the factor analysis was appropriate for this correlation matrix from the PTES. Bartlett's test of sphericity suggested the correlation matrix was not an identity matrix (Approximate $\chi 2 = 1318.03$, $df = 45$, $p = .0001$). The PTES had a one-factor solution extracted by varimax rotation method, which was anticipated.

4.1.6.4. Intervention adherence. Intervention adherence to the tobacco abstinence and relapse maintenance intervention pertains to the rate of participation and completion of intervention activities. Intervention homework and participation with telephone intervention session activities each provided a percent of the intervention completed as calculated by the following:

$$\textit{Intervention component adherence} = \frac{\textit{amount completed}}{\textit{total available for completion}} \quad X\ 100$$

These two components of the intervention were totaled and divided by 2 and multiplied by 100, which represented the rate of overall intervention adherence.

4.1.6.5. Potential influencing variables – Sources of self-efficacy. As noted in chapter 2, other variables may influence tobacco abstinence and relapse, such as sociodemographic variables (e.g., gender, ethnicity, education, income, social support), personal variables (e.g., mood, development of tobacco related disorders), and tobacco related variables (e.g., nicotine dependence, nicotine withdrawal, initial abstinence violation following a quit attempt). Bandura (1997) suggests sources of self-efficacy have the potential to impact changing behavior, such as

tobacco dependency. The four sources of self-efficacy include mastery experience, vicarious experience, verbal persuasion, and physiological/affective states (Bandura, 1997). There is some overlap between the influencing factors of abstinence noted above and sources of self-efficacy. For example, social support is a sociodemographic influencing variable and a potential source of self-efficacy through verbal persuasion and modeling (e.g., tobacco using friends). Personal variables of mood or affect and tobacco related variables (e.g. nicotine dependence and nicotine withdrawal) are also potential sources of self-efficacy as physiological or affective states. Therefore, the following instruments described below were used to measure variables for their potential influence on tobacco abstinence and relapse.

4.1.6.6. Sociodemographic Questionnaire. The University of Pittsburgh School of Nursing Center for Research in Chronic Disorders [CRCD] developed this 27-item paper-pencil questionnaire to collect demographic information by self-report. Items included in this questionnaire are those that may impact upon an individual's health status (e.g., age, race, gender, education, marital status). For the purpose of this study, additional questions were used to document the subject's reason for hospital admission, diagnosis, medical/surgical history, and length of stay on a study-generated form.

4.1.6.7. Tobacco Consumption Questionnaire [TCQ]. This questionnaire was adapted from the tobacco use questionnaires used by the Lung Health Study for the preliminary study. The questionnaire targets tobacco use, abstinence attempts, nicotine dependence, smoking behavior patterns, motivation for abstinence attempts, and tobacco related health status. These forms are available in the public domain. This instrument was completed by the participant, unless the participant was fatigued or physically incapable of writing, then it was completed by interview. The Fagerstrom Test for Nicotine Dependence [FTND] was included within this instrument and

will be discussed later. This intake questionnaire was modified for this study to incorporate the documentation of support. A modified version of the baseline instrument was used for follow-up visits with additional questions targeting information pertaining to smoking intervention received during the hospital admission in addition to study provided information and intervention. In addition, this follow-up visit version had items pertaining to initial lapses that occurred following hospital discharge.

4.1.6.8. Fagerstrom Test for Nicotine Dependence [FTND]. The FTND is a 6-item instrument used to measure nicotine dependence. Versions of the earlier version of this FTND, the Fagerstrom Tolerance Questionnaire, have been used by studies of nicotine dependence (Bobo, Lando, Walker, & McIlvain, 1996; Campbell, Prescott, & Tjeder-Burton, 1996; Hjalmarson et al., 1994). Scores for the both the FTND and the FTQ have Likert scaled items and items requiring dichotomous responses. The maximum score for the FTND is 10. The FTND differs from the FTQ on the cigarette consumption assessment (e.g., more categories), time from waking to first cigarette in the morning, questions pertaining to inhaling, and nicotine content/yield.

Psychometric results have not yielded high measures of internal consistency for this trait measure of nicotine dependence. The FTND has been reported with an Cronbach $\alpha = .64$ (Pomerleau et al., 1994). The FTQ version has acceptable test-retest reliability coefficients ranging between .78 and .88, inclusively (Pomerleau et al., 1994). This questionnaire has correlated well with carbon monoxide testing level (Becona & Garcia, 1995). The correlation of cotinine and the FTQ in a sample of adolescent smokers suggested the FTQ significantly correlated with cotinine ($r = .40$, $p , > .01$) (Prokhorov, De Moor, Pallonen, Hudmon, Koehly, & Hu, 2000). Concern has been raised that this tool has additional limitations with respect to

smoking populations with low consumption rates (< 10 cigarettes per week) (Shiffman, 1993). The FTQ and FTND continue to be popular tools for the assessment of nicotine dependence.

4.1.6.9. Tobacco Withdrawal Form (Hughes and Hatsukami). Symptoms of tobacco withdrawal were measured with the Tobacco Withdrawal Form developed by Drs. Hughes and Hatsukami (1986). This scale has 12 items with Likert scaling from 0 - 4. Intra-rater reliability coefficients for this nicotine withdrawal scale have been reported to range between .40 and .62 (p = .05). In addition, criterion validity was conducted by comparing the Tobacco Withdrawal form with the DSMIII symptoms for nicotine withdrawal and the Profile of Mood States [POMS]. Results of these comparisons were consistent and provided support for the validity of the Tobacco Withdrawal form (Hughes & Hatsukami, 1986).

4.1.6.10. Perceived social support. The overall measure of social support for tobacco abstinence was a summary score of the level of confidence in a significant other's support of the participant to stop smoking and the number of supportive individuals the participant identified to use to assist their tobacco abstinence efforts. Scores could range from one to six, inclusively. Social support has the potential to be a source of self-efficacy with respect to verbal persuasion (Bandura, 1997). In light of literature findings and data from the preliminary study suggesting social support as an influence of tobacco abstinence and relapse prevention, these social support questions were incorporated into the TCQ.

4.1.6.11. Profile of Mood States. The profile of mood states, otherwise known as the POMS, has 65 items and 6 subscales, which include: 1) tension/anxiety, 2) depression/dejection, 3) anger/hostility, 4) vigor/activity, 5) fatigue/inertia, and 6) confusion/bewilderment (McNair, et al. 1992). These items are Likert scaled from 0 to 4. According to a factor analysis reported by Mc Nair, et al. (1992), the structure of the POMS was supported. Internal consistency of each

subscale has been reported as ≥ .90 (McNair, et al., 1992; Snively, et al. 2000). According to the Educational and Industrial Testing Service, this instrument is appropriate for adult subjects starting at 18 years of age. Normative data has been compiled on outpatient and college populations. This instrument uses 65 adjectives aimed to describe a feeling or mood the subject may be experiencing. Subjects can indicate their responses using a 5-point Likert scale. According to the testing service, this instrument may require 3 to 5 minutes for completion. However, with the inpatient population of this project, more time is anticipated for instrument completion due to the variability in subject's medical conditions at the time of hospitalization. The POMS has been used in tobacco treatment studies with regard to measurements for mood, affect, and withdrawal symptoms (Cinciripini, Lapitsky, Seay, Wallfisch, Meyer, & van Vunakis, 1995; Craig, Parrott, & Coomber, 1992; Gentry et al., 2000; Gritz, Carmack, de Moor, Coscarelli, Schacherer, Meyers, & Abemayor, 1999a; Patten, Martin, Calfas, Brown, & Schroeder, 2000; Snively, Ahijevych, Bernhard, & Wewers, 2000). This instrument is not in the public domain and requires purchase.

4.1.6.12. CRCD Comorbidity Questionnaire. The CRCD Comorbidity Questionnaire was used to identify comorbid disorders of the study subjects. Information was obtained by subject completion of the questionnaire or by interview if subjects complained of fatigue or were unable to write during their hospital admission. The baseline form of this instrument contains 76-items and is currently used in various studies at the University of Pittsburgh, School of Nursing. A modified version with fewer items was used for follow-up visits.

4.1.6.13. Project specific tools. Two data intake tools were developed specific to the project. The first tool was used to obtain information pertaining to: current health treatment (e.g., current medications), other health risk behaviors, and alternative therapy use (e.g., vitamins, herbs, acupuncture). A second project tool was used to obtain information pertinent in order to make telephone calls and mailing study thank you cards at the end of the study.

4.1.7. Data management

4.1.7.1. Data screening procedures. Data for this study were collected with paper pencil instruments and the CO monitor. The following procedures assisted the investigator in controlling for data inconsistencies, recording errors, and missing entries in the database. Prior to data collection, all questionnaires were prepared in a Teleform format (Version 6). Therefore, all questionnaires were in a scannable format for database entry into Paradox (Version 9) tables with Teleform software (Version 6). Data dictionaries were developed for data editing and analysis. Each instrument was stored in its own subdirectory prior to data analysis.

The questionnaire data was checked for inconsistencies at least four times before analysis. First, questionnaires were visually checked before computer scanning for missing data and inconsistent data entry following item coding for CRCD Sociodemographic Questionnaire, TCQ, chart review, and CRCD Comorbidity Questionnaire. If inconsistencies and/or missing data points were noted, effort was made to contact the subject for clarification before scanning the questionnaire. If missing data could not be collected from the subject after numerous attempts, the data were coded as missing on the forms and Teleform scanning commenced. At the time of Teleform scanning, data were visually checked in the database for inconsistencies and missing entries not coded previously as missing. If these types of problems were found, verification was not completed until the problems were resolved, which required verification of

chart data with the database to confirm accurate entries and resolve missing entries that occurred with scanning. A screening of the data with statistical software packages provided the fourth check for data inconsistencies and missing values.

4.1.7.2. Preliminary data analysis. Preliminary analyses were conducted to assess: 1) sample distributions, 2) outliers, 3) patterns of missing data, 4) multicollinearity, and 5) assumption violations of normality, and independence. Preliminary analysis plan included 1) measures of central tendency, 2) frequency distributions, 3) contingency tables, 4) Pearson and Spearman correlations, and 5) t-tests of independence. Contingency tables were particularly useful to examine assumptions necessary for the use of the Pearson chi-square, such as frequencies in cells. For continuous/interval data, linear regression was also used to examine distributions, normality, and outliers. To further assess statistical assumptions and sampling distributions, graphical measures were used, which included histograms, scatter plots, box-plots, stem-and-leaf plots, residual plots, probability plots, and time-sequence plots. The information provided by these tests and plots provided univariate and multivariate diagnostic assessment of the data for outliers, influencing cases, and assumption violations. Tests were conducted with and without potential outliers to determine impact upon the data. The need for corrective measures was also examined, such as variable transformation for interval data.

4.1.7.3. Descriptive statistics. A preliminary analysis of the data with descriptive statistical techniques was a prerequisite to further inferential testing of this study's proposed research questions. Sample characteristics and data were either discrete (nominal and dichotomous) or continuous in nature with regard to measurement scales. Scatter plots, histograms, box-plots, and

stem-and-leaf plots were the measures of choice to present the graphical description of the sample population and collected data. Version 12 of SPSS was used to conduct the statistical assessments for both continuous and categorical variables.

Summary statistics were completed with each continuous variable of interest with regard to measures of central tendency (mean, median, mode) and variation or dispersion (standard deviation, variance, range, semi-quartile range, skew ness, kurtosis). A significance level of .05 was used for the descriptive analyses and exploratory analytic techniques. Univariate sample distributions were generated to describe the characteristics of the hospitalized smokers enrolled in this study. Bivariate relationships of continuous variables were assessed with the Pearson correlation coefficient and the Spearman correlation coefficient. In addition to analyzing the overall sample, subset analysis was completed by treatment group assignment, gender, race, and baseline categorical characteristics.

Discrete variables were also analyzed in a similar manner as the continuous variables (alpha = .05), but with the use of nonparametric techniques, such as Fisher's Exact Test, Pearson chi-square test, and phi-coefficients. Frequency distributions, ranges, and modes were generated to assess categorical and dichotomous variables. Finally, an assessment of these variables was conducted by treatment group assignment, gender, race, and baseline categorical characteristics.

4.1.7.4. Missing data. Both SPSS version 12 and BMDP (AM and 8D) were used to assess for missing data patterns. The procedure used to check for missing data and resolve missing data were previously presented under “Data Screening Procedures.” Due to findings from the preliminary descriptive study, the occurrence of missing data was a likely possibility because of present trends of short hospital stays. In the preliminary descriptive study of hospitalized smokers, subjects were interviewed for information, but due to their medical status and level of

fatigue, the investigator was required to hold interviewing. Subjects requested to complete their survey on their own or have the interviewer return at a later point in time. Problems were encountered with both of these subject requests. Due to shorter hospital stays, discharge notices were given to subjects before they completed their surveys and without notice to the investigator that discharge was imminent. When possible, missing data was collected by telephone. If contact with the subject was not possible, the data were considered missing and the process of scanning and verification of collected data proceeded. In a separate problem of missing data, some trauma victims provided inaccurate addresses and telephone numbers to the investigator and the hospital. These subjects could not be reached to collect missing data or administer follow-up surveys. The intent-to-treat model in the following section addresses missing data, however, baseline data collection did not warrant concerns for missing data. Outcome data were also available on all subjects except those lost to follow-up. The intent-to-treat model was used with respect smoking status. Similar to previous studies in this population, lost to follow-up participants were considered to be currently smoking for follow-up visits (Taylor et al., 1996; Dornelas et al., 2000; Johnson et al., 1999).

4.1.7.5. Handling of protocol deviations. This study used the "Intention to Treat" [ITT] model of analysis with respect to the primary aim and secondary aims. Specifically, all subjects were included in the primary aim analyses pertaining to their randomized treatment assignment, regardless of adherence to the study treatment (Friedman, Furberg, & De Mets, 1998). This analysis plan has been used and is recommended for clinical trials in smoking (Fiore, et. al., 2000). Furthermore, the aim of this analysis approach is to minimize the introduction of bias during statistical analysis and provide a conservative estimate of treatment efficacy, which is achieved by inclusion of all subjects in the analysis regardless of whether they complied with

study treatment (Lachin, 2000). Therefore, by maintaining study treatment assignment regardless of treatment compliance and study participation, introduction of bias of the outcome data is controlled. With respect to missing data, the last observation can be carried forward rather than imputing values for missing information (Lachin, 2000).

This study incorporated the examination of adherence measures due to the lack of descriptive information in the literature regarding adherence to tobacco abstinence relapse management interventions. By examining this phenomena as part of the secondary aims, information was obtained for use in future studies that may be used to decrease adherence barriers or enhance adherence as part of future interventions (Sereika & Davis, 2001). Therefore, the effort to use the deviation from the ITT analysis was not to report efficacy of the treatment as much as it is to provide a descriptive analysis of behavior to the treatment plan proposed for the intervention assignment group in this particular population of tobacco dependent individuals.

4.1.7.6. Data analysis. The primary outcome of interest is that of smoking status, specifically abstinence versus relapse, between the groups with regard to baseline (T_0), end of treatment (T_1), and exit measures (T_2) of smoking status.

Aim 1: The primary aim was to examine the efficacy of a 12-week nurse-delivered telephone abstinence promotion and relapse management intervention designed to enhance self-efficacy and smoking abstinence for smokers desiring to abstain following hospital discharge as measured by self-reports of smoking behavior validated by carbon monoxide.

Hypothesis 1a: The group of hospitalized smokers assigned to a 12-week abstinence promotion and relapse management intervention following discharge were hypothesized to have a greater number of participants with smoking abstinence (smoking point prevalence verified by CO) 12 weeks following discharge as compared to subjects who were assigned to only enhanced usual care.

Hypothesis 1b: The group of hospitalized smokers assigned to a 12-week abstinence promotion and relapse management intervention following discharge were hypothesized to have a greater number of participants with smoking abstinence (smoking point prevalence verified by CO) 24 weeks following discharge as compared to subjects who were assigned to only enhanced usual care.

4.1.7.7. Analysis of the primary aim. The level of significance for the primary aim was set at .05 and divided evenly across the two hypotheses. Previous studies have not resulted in negative outcomes for behavioral interventions for smoking abstinence. These studies have either been nonsignificant in their findings or resulted in significant positive findings for the treatment (DeBusk et al., 1994; Dornelas et al., 2000; Froelicher et al., 2004a; Griebel et al., 1998; Johnson et al., 1999; Miller et al., 1997b; Molyneux et al., 2003; Neighbor et al., 1994; Polednak, 2000; Ratner et al., 2004; Rigotti et al., 1997; Rigotti et al., 1991; Simon et al., 1997; Stevens et al., 1993; Stevens et al., 2000; Taylor et al., 1990; Taylor et al., 1996; Wewers et al., 1994; Wewers et al., 1997). Based upon these previous findings in the literature and typical use of one-sided tests in clinical trials for medical treatment testing (Hennekens & Buring, 1987; Overall, 1990), this study adopted a one-tailed test. The hypotheses were stated directionally. As pointed out by

Hennekens and Buring (1987), one-sided or two-sided tests when published should be clearly pointed out within the publication to allow the reader the ability to interpret the results based upon their needs.

Contingency tables. For the first two hypotheses associated with the primary aim, a contingency table analysis to describe the bivariate distribution of the data with odds ratios and confidence intervals, was generated with a one-tailed significance level of .05. The following assumptions were required for Pearson chi-square tests: 1) the sample of observations is a random sample and 2) each observation may only be classified in to exactly one row and one column (Conover, 1980). The Pearson chi-square does not require the assumption of a normal distribution. Furthermore, this test requires that cell frequencies for each of the represented categories be at least 1. In addition, cell frequencies for 80% of the categories are expected to be greater than 5. However, Conover (1980) suggested that this requirement for contingency table cells is conservative. Furthermore, he suggested cells as small as 1 would not likely "endanger" the validity of the test (Conover, 1980).

Logistic Regression. Analysis of smoking abstinence and covariates required use of logistic regression, as well as with the examination of treatment adherence to smoking abstinence in the secondary aims (Hosmer & Lemeshow, 2000; Liao, 1994). SPSS software was used to perform binary logistic regression analyses. This type of analysis was used for the primary aims and secondary aims (hypotheses 2a through 2e and 2g). The primary aim used backward stepwise regression to examine confounding variables from baseline data. Once significant confounding variables were identified, the variables were entered in the first step of the model prior to adding the treatment group assignment in the second step.

Subjects who dropped out from the study (e.g., do not attend follow-up visits) were categorized as smokers (Taylor et al., 1996; Dornelas et al., 2000; Johnson et al., 1999). Due to the dichotomous nature of this variable, it was necessary to use nonparametric statistical tests for the comparisons. Assumptions for the logistic regression include the following: 1) does not rely on distributional assumptions, 2) multicollinearity among the predictor variables might contribute to biased estimates and inflated standard errors, and 3) dependent variable is categorical (Hosmer & Lemeshow, 2000). In addition, a frequency of "zero" in a contingency table for the logistic regression is problematic. The univariate screening was used to identify the existence of this situation. Hosmer and Lemeshow (2000) suggest collapsing cells when this occurs or eliminate the category. The "zero" cell frequency presents problems in modeling the data, particularly when interaction terms are anticipated. The following items were generated in order to interpret the data with logistic regression techniques: odds ratio, variable coefficients, confidence intervals, log likelihood ratios, Wald statistics, classification tables, multicollinearity tables, the Hosmer and Lemeshow Goodnes of Fit test, and diagnostics. The Hosmer and Lemeshow Goodness of Fit test cannot be generated when the independent variable has limited variability as in the case of a dichotomous variable. This goodness of fit measure can be generated for multilevel categorical variables or continuous variables (Hosmer & Lemeshow, 2000). Syntax was developed to generate estimated probability, Cook's influence statistic, leverage, DfBeta, and residuals. Graphic representations of these diagnostics were used to look at the fit of the models generated by logistic regression.

The second aim of the study was divided in two parts for examination and discussion. The first secondary aim examined the relationship between conceptually driven variables from Self-efficacy Theory (e.g. self-efficacy, relapse situation efficacy, perceived treatment efficacy,

social support for tobacco abstinence, and depressive symptoms) to the outcome variable of smoking point prevalence. The selected alpha for this first secondary aim was .05, which was divided evenly across these ten analyses. Therefore, results were considered significant if the p – value for the $\chi_{LR}2$ statistic was $\leq .005$. The second secondary aim examined relationships between smoking point prevalence, treatment adherence, and self-efficacy as measured by the TASES.

Hypothesis 2a(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived self-efficacy as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES].

Hypothesis 2a(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived self-efficacy as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES].

Hypothesis 2b(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for relapse situation efficacy as measured by the Relapse Situation Efficacy Questionnaire [RSEQ].

Hypothesis 2b(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for relapse situation efficacy as measured by the Relapse Situation Efficacy Questionnaire [RSEQ].

Hypothesis 2c(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for outcome expectancy as measured by the Perceived Therapeutic Efficacy Scale for Relapse Maintenance [PTES-RM].

Hypothesis 2c(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived treatment efficacy as measured by the Perceived Therapeutic Efficacy Scale for Relapse Maintenance [PTES-RM].

Hypothesis 2d(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived social support for tobacco abstinence.

Hypothesis 2d(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived social support for tobacco abstinence.

Hypothesis 2e(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by an inverse relationship with baseline scores for affective states, specifically depressive symptoms as measured by the depression/dejection subscale on the Profile of Mood States [POMS].

Hypothesis 2e(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by an inverse relationship with baseline scores for affective states, specifically depressive symptoms as measured by the depression/dejection subscale on the Profile of Mood States [POMS].

Hypothesis 2f: The time to the first smoking lapse was hypothesized to be longer for subjects who were assigned the 12-week abstinence promotion and relapse management intervention as compared to subjects who were assigned to only enhanced usual care.

Hypothesis 2g(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with treatment adherence rates.

Hypothesis 2g(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with treatment adherence rates.

Hypothesis 2h: Treatment adherence was hypothesized to have a positive relationship with baseline (T_0) perceived self-efficacy as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES].

Hypothesis 2i: Subjects in the treatment group were hypothesized to have an increase in self-efficacy, as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES], from baseline (T_0) to follow-up measurements at T_1 and T_2.

Alpha for this second secondary aim was .05, which was divided evenly across the five tested hypotheses. Therefore, test statistics were required to have p – values ≤ .01. Analysis of smoking abstinence and covariates required use of logistic regression, as well as with the examination of treatment adherence to smoking abstinence (Hosmer & Lemeshow, 2000; Liao, 1994). Logistic regression analysis was discussed with hypotheses for the primary aim. The analysis plan for these hypotheses requires univariate logistic regression models.

An exploratory aim was added to the study due to the lengthy period of recruitment (3 years) and use of multiple hospital sites to enroll participants (three hospital sites). Since these tests were exploratory, two-sided (alpha < .05) logistic regression analyses were used to examine whether these variables had a relationship with smoking behavior when measured at the follow-up visits and upon the treatment assignment. A final analysis examined whether age had a relationship to smoking status outcome and/or impacted the effect of the treatment.

4.1.7.8. Survival analysis. Hypothesis 2f considered time to relapse between the intervention and enhanced usual care groups. The variable of interest for this hypothesis was time to first lapse, which was an interval variable. An initial lapse was defined as the first puff of cigarette

following quitting smoking during hospitalization. For this reason, survival analysis was used to examine the estimated time to the initial lapse of smoking (Le, 1997). According to Afifi and Clark (1996), survival analysis is used to describe an event of interest with respect to a given time frame. Therefore and more precisely, this analysis was describing the potential events prior to smoking relapse from the start of treatment or baseline (T_0) until study exit (T_2). In addition, since some subjects may be lost to follow-up, the chosen method of analysis must allow for right and interval censoring of cases. The Kaplan-Meier method does permit case censoring (Le, 1997). In addition, other measures, such as the Gehan-Wilcoxon Kaplan-Meier analysis may be used to examine sensitivity or lapse. SPSS version 12 software packages was used to conduct this analysis and provided a survival table, including time, cumulative survival and standard error, cumulative events, and number remaining, mean and median survival time, with standard error and confidence interval. Survival, hazard, log survival, one minus survival plots were also used to interpret this analysis. Prior to conducting the analysis, data from subjects was screened to identify whether there were variables to impact this analysis towards bias with the treatment. This type of difference would suggest that there were differences in the censored cases and bias the survival analysis results (Afifi & Clark, 1996).

Subjects who were abstinent from smoking at each follow-up visit were censored in this analysis. One subject died between baseline and T_1. This subject was censored in the analysis. In addition, subjects who reported abstinence, but had CO levels greater than 8ppm were considered as having experienced a lapse. The midpoint between a participant's last recorded abstinent observation and the follow-up visit was selected for the day of lapse (Daughton, Fortmann, Glover, Hatsukami, Heatley, Lichtenstein, Repsher, Millatmal, Killen, Nowak, Ullrich, Patil, & Rennard, 1999; Froelicher et al., 2004a). One subject reported tobacco

abstinence at T_1, but had a CO reading greater than 8 ppm. Day of lapse assigned to this subject was the 42nd day from baseline. Another subject had a similar observation at T_2 and was assigned a lapse event of the 126th day from baseline. Due to the intent to treat analysis, subjects lost to follow-up were considered as having lapsed. Therefore, they were assigned a lapse event of the 42nd day following T_0 since they were lost to follow-up by T_1.

Similar to the treatment of other study variables, treatment adherence to the intervention for the treatment group was described. In addition to measures of central tendency, correlations, t-tests, and nonparametric counterparts were used to examine patterns of adherence based upon sample characteristics. The analysis of intervention treatment adherence and perceived self-efficacy used Spearman correlation coefficients.

4.1.7.9. General linear mixed model. The final hypothesis within the secondary aims required an analysis with a General Linear Mixed Model with repeated measures. Perceived self-efficacy as measured by the TASES occurred at three distinct time points (T_0, T_1, & T_2) in the study and was the within-subject variable of interest in this analysis. The treatment group assignment was the between-subject variable of interest and had two categories (intervention group versus enhanced only usual care group). The model of interest includes an interaction of the two variables and the main effect of perceived self-efficacy. This analysis will examine whether self-efficacy changed over time, as well as by treatment group (Neter, Kutner, Nachtsheim, & Wasserman, 1996). The preliminary data analysis will provide univariate and multivariate information of these variables with respect to meeting the assumptions of independence, normality, homogeneity of variance and covariance for this analysis (Neter et al., 1996).

5. CHAPTER FIVE

5.1. RESULTS

5.1.1. Introduction

The primary purpose of this study was to examine the effectiveness of a nurse delivered tobacco abstinence promotion - relapse management program. Hospitalized patients with recent smoking histories were recruited for this project and introduced to the program during their hospital admission. For the special intervention group [SI], intervention extended for 11 weeks beyond hospital discharge in the form of nurse-delivered telephone calls aimed to promote the participant's tobacco abstinence and prevent relapse.

One hundred and six hospitalized smokers were referred and contacted to participate in this project (Refer to Figure 9). Eighty-four (79.2%) participants consented to screening. Only 80 (95.2%) individuals participated beyond screening and completed baseline materials. Of those who were initially screened and did not continue with the project, three were discharged before further participation could be carried out and one individual was transferred to another facility. Of all individuals contacted for participation, 59 (56%) were female and 47 (44%) were male. With respect to race, 82 (77%) self-reported being White, 23 (22%) were Black, and 1 (1%) was Other. Of those who did not participate with the project, 11 (42%) were female and 15 (58%) were male. The racial description of the nonparticipating patients included 19 (73%) White and 7 (27%) Black. Other descriptors of the nonparticipating subjects were not available. Three hospital sites were used to recruit subjects. At hospital site "A," 39 subjects participated with the

study. Thirty-eight subjects participated from hospital "B," and three participated from hospital "C." Hospitals "A" (SI, n = 17) and "B" (SI, n = 20) were nearly evenly divided in the number of participants assigned to treatment groups. All three of the participants from hospital "C" were randomized to the SI group. Three stratifying factors were used to randomize study participants, which included gender, race, and tobacco related diagnosis/comorbidity. Recruitment site was not used as a pre-randomization stratification factor. Table 16 displays the joint distribution of the pre-randomization stratification factors by treatment assignments for the 80 individuals who participated in the study.

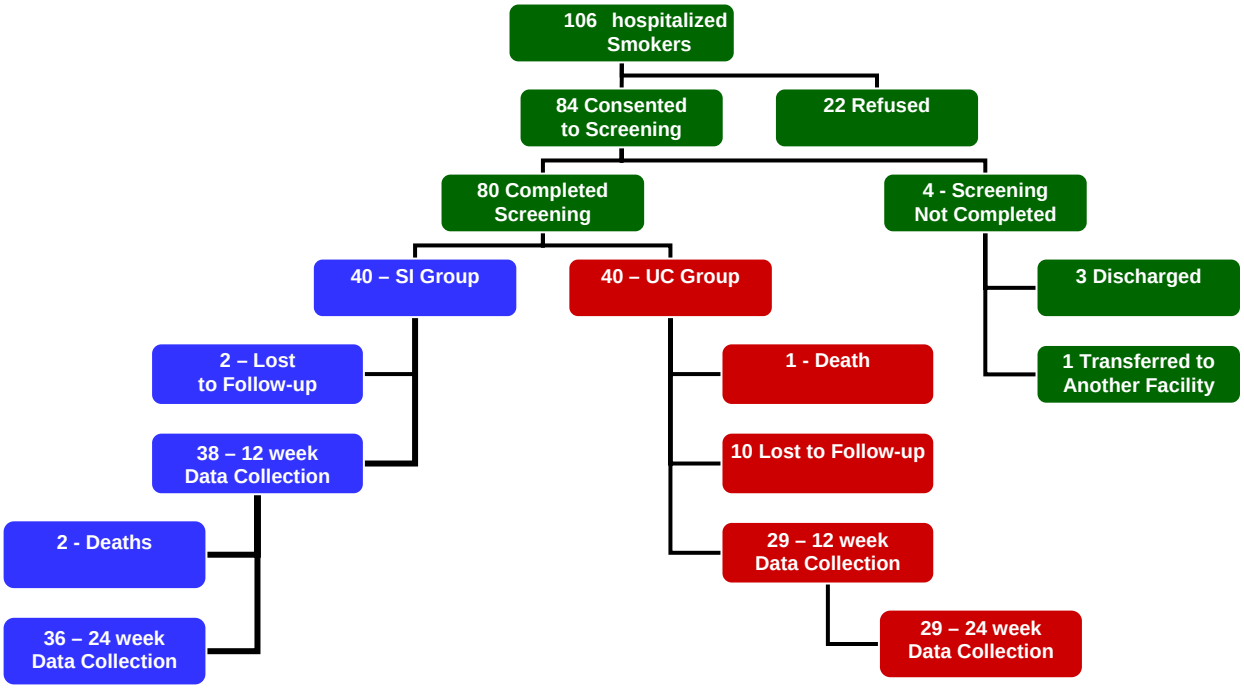


Figure 9 Diagram of participants from referral to final data collection

Table 16 Frequency of treatment groups by pre-randomization stratification factors

	Enhanced Usual Care		Special Intervention	
Stratification Factors	*f* (%)	n	*f* (%)	n
Gender				
Male	16 (40)	40	16 (40)	40
Female	24 (60)		24 (60)	
Ethnicity				
White	31 (78)	40	32 (80)	40
Non-white	9 (22)		8 (20)	
Comorbid Status				
1 [a]	7 (18)	40	6 (15)	40
2 [b]	4 (10)		7 (18)	
3 [c]	29 (72)		27 (67)	

[a] 1 = Non-tobacco related admission and non-tobacco related comorbidities
[b] 2 = Non-tobacco related admission, but tobacco related comorbidities
[c] 3 = Tobacco related admission and related comorbidities

5.1.2. Baseline characteristics

Forty subjects were randomly assigned to each treatment group. Complete baseline data were collected from the 80 participating subjects in this study. Tables 17 through 26 provide visual representation of the baseline characteristics of interest in this study. On average, participants were married, had children, and stated their income met their basic needs (Refer to Table 17). In addition, subjects were middle aged and had at least a high school education (Refer to Table 18). The treatment groups differed significantly with respect to: 1) current employment status, 2) having health insurance, and 3) whether surgery was received during the admission to the hospital ($p < .05$). Special intervention subjects were significantly more likely to be unemployed (63%, n = 25, $\chi^2 = 4.05$, $p = .05$), have health insurance (98%, n = 39, $\chi^2 = 3.91$, $p = .05$), and have received surgery during their admission (73%, n = 29, $\chi^2 = 4.27$, $p = .05$). In addition, the special intervention group ($M = 7.3$; $SD = 6.7$) had a significantly longer hospital stay than the usual care participants ($M = 4.88$, $SD = 2.91$) ($t = -2.10$, $p = .04$).

Tobacco consumption characteristics are displayed in Tables 19 through 24. Over 60% of the subjects in both treatment groups reported they were not smoking during their hospital admission. In addition, nearly 100% of the subjects expressed a desire to quit cigarette smoking. Seventy-eight percent or more of the subjects had previous 24-hour tobacco cessation attempts. Cardiopulmonary rehabilitation personnel did not see over 50% of the participants. Within all three hospitals used for recruitment, cardiopulmonary personnel provided limited smoking cessation information. Over 70% of the subjects did not have a history of using organized smoking cessation programs.

Tobacco consumption and nicotine dependence characteristics of the treatment groups are summarized in Tables 20 and 21. Study participants reported having smoked one pack of cigarettes per day for the 30 days prior to their hospitalization. Most subjects initiated their tobacco use when they were an older adolescent at approximately 18 to 19 years of age. Participants reported smoking a minimum of less than a half a pack of cigarettes on some days prior to their hospitalization and a maximum of 1½ packs of cigarettes per day. The mean CO reading for the Usual Care group was 6.42 (SD = 6.86) and the Special Intervention group was 7.16 (SD = 7.11). Two subjects in each group were not measured for baseline CO. Two participants had pulmonary infections, one participant was being monitored for tuberculosis, and the fourth participant had just received a nonfenestrated tracheotomy tube. The infections had the potential of contaminating the monitor for future use and the fourth patient could not exhale through her mouth at this time. On average, subjects reported experiencing at least six nicotine withdrawal symptoms during their hospital admission. Nicotine dependence, as measured by the Fagerstrom Test for Nicotine Dependence, ranged from 2 – 10 with a mean of 5.33 (SD=1.86) for enhanced usual care and 6.13 (SD=1.95) for special intervention participants. Fifty percent of the subjects lived at home with a smoker; however, six UC (15.0%) and nine SI (22.5%) subjects lived alone. On average, subjects in both groups reported a "significant other" in their life was supportive of their efforts to quit smoking (34 UC and 31 SI subjects). Over 90% of the subjects in each group reported they had a support person identified to assist them with quitting smoking following their hospital discharge. The score for perceived social support for tobacco abstinence did not differ between the groups (Refer to Table 20). The mean score for perceived social support across the two groups was 4.68 (SD=0.99).

Participants in both groups reported previous use of various cessation medications (Table 22). Less than a third of the patients in each group reported using nicotine gum; however, both groups reported greater previous use of the nicotine patch and bupropion. Among UC subjects, 40% (n = 16) reported previous experience with the nicotine patch and 12% (n = 5) reported previous use of bupropion. The use of the nicotine patch was greater, but not significantly so for the SI group. Fifty-three percent (n = 21) of the SI subjects reported previous use of the nicotine patch, however, 32% (n = 13) of SI subjects reported use of bupropion, which was significantly greater than the 12% (n = 5) of UC subjects who reported previous bupropion use ($\chi^2 = 4.59$, p= .04). Current use of cessation medications at the time of the baseline assessment is presented in Table 23. Few subjects used nicotine gum. Only one UC subject reported gum use. Ten percent (n = 4) of SI and 15% (n = 6) of UC subjects reported using the nicotine patch during hospitalization and after discharge. Only two subjects in each group used bupropion.

Table 17 Demographic and clinical characteristics by treatment assignment

Demographic Variables	Enhanced Usual Care		Intervention		χ^2 value, p-value
	f (%)	n	*f* (%)	n	
Marital Status					
Married/Attached	23 (58)	40	22 (56)	39	0.01, p = .95
Not Married	17 (42)		17 (44)		
Employed					
Yes	24 (60)	40	15 (38)	40	4.05, p =.05*
No	16 (40)		25 (63)		
Children					
Yes	37 (93)	40	32 (80)	40	2.64, p = .11
No	3 (7)		8 (20)		
Health Insurance					
Yes	34 (85)	40	39 (98)	40	3.91, p = .05*
No	6 (15)		1 (2)		
Income Meets Needs					
Yes	29 (76)	38	23 (59)	39	2.64, p = .11
No	9 (24)		16 (41)		
Received Surgery					
Yes	20 (50)	40	29 (73)	40	4.27, p = .04*
No	20 (50)		11 (27)		

*significant value

Table 18 Demographic variables by treatment assignment

Variables	Enhanced Usual Care	Special Intervention	t-test value, p-value
Age (Years)	M = 49.00 (SD = 11.10)	M = 52.68 (SD = 11.10)	-1.48, p = .15
n = 40/group	Mdn = 50.00	Mdn = 53.50	
	(Range = 24 – 74)	(Range = 27 – 77)	
Years of education	M = 12.36 (SD 1.66)	M = 12.95 (SD = 1.89)	-1.46, p = .15
n = 39/group	Mdn = 12.00	Mdn = 12.00	
	(Range = 9 - 18)	(Range = 9 – 18)	
Length of Hospital	M = 4.88 (SD = 2.91)	M = 7.30 (SD = 6.70)	-2.10, p = .04*
Stay (Days)	Mdn = 4.00	Mdn = 5.00	
n = 40/group	(Range = 2 – 18)	(Range = 1 – 33)	

*significant value

Table 19 Tobacco use variables by treatment assignment

Tobacco-use variables	Enhanced Usual Care *f* (%)	n	Intervention *f* (%)	n	χ^2 value, p-value
Smoking during hospital admission					
Yes	15 (38)	40	14 (35)	40	0.05, p = .85
No	25 (62)		26 (65)		
Desire to quit					
Yes	40 (100)	40	39 (98)	40	1.01, p = .31
No	0		1 (2)		
Previous 24 hour quit attempt					
Yes	31 (78)	40	34 (85)	40	0.74, p =.39
No	9 (22)		6 (15)		
Seen by rehab					
Yes	17 (43)	40	19 (48)	40	0.20, p =.66
No	23 (57)		21 (52)		
Previously used cessation program					
Yes	11 (28)	40	12 (30)	40	0.06, p = .81
No	29 (72)		28 (70)		

Table 20 Tobacco-use history and abstinence social support by treatment assignment

Variables	Enhanced Usual Care n = 40	Special Intervention n = 40	t-test value, p-value
Cigarettes/day over last 30 days	M = 17.45 (SD = 8.74) Mdn = 17.0 (Range = 1 – 40)	M = 21.80 (SD = 15.83) Mdn = 20.00 (Range = 0 – 80)	-1.90, p = .07
Age initiated daily smoking (years)	M = 17.45 (SD = 5.48) Mdn = 17.00 (Range = 10 – 32)	M = 18.58 (SD = 9.33) Mdn = 17.50 (Range = 8 – 68)	-0.66, p = .52
Minimum cigarettes/day over last 60 days	M = 8.35 (SD = 9.31) Mdn = 5.00 (Range = 0- 40)	M = 9.05 (SD = 13.78) Mdn = 5.5 (Range = 0 – 70)	-0.27, p = .80
Maximum cigarettes/day over last 60 days	M = 26.08 (SD = 11.51) Mdn = 25.00 (Range = 5 – 60)	M = 30.03 (SD = 21.34) Mdn = 21.00 (Range = 6 -110)	-1.03, p = .31
Social support for tobacco abstinence	M = 4.75 (SD = 0.81) Mdn = 5.00 (Range = 3 – 6)	M = 4.63 (SD = 1.15) Mdn = 5.00 (Range = 1 – 6)	0.56, p = .58

Table 21 Baseline CO, Nicotine dependence and withdrawal by treatment assignment

Variables	Enhanced Usual Care	Special Intervention	t-test value, p-value
Carbon Monoxide (ppm)	M = 6.42 (SD = 6.86)	M = 7.16 (SD = 7.11)	-0.46, p =.65
n = 38/group	Mdn = 5.00	Mdn = 5.00	
	(Range = 2 – 32)	(Range = 2 – 42)	
Nicotine Withdrawal	M = 14.2 (SD = 9.96)	M = 15.38 (SD = 9.38)	-0.54, p = .59
Total Score	Mdn = 13.50	Mdn = 16.00	
n = 40/group	(Range = 0 – 36)	(Range = 0 – 36)	
Number of Withdrawal	M = 6.33 (SD = 3.64)	M = 6.98 (SD = 3.39)	-0.83, p = .42
Symptoms	Mdn = 6.50	Mdn = 8.00	
n = 40/group	(Range = 0 – 12)	(Range = 0 – 12)	
Fagerstrom Test of	M = 5.33 (SD = 1.86)	M = 6.13 (SD = 1.95)	-1.88, p = .07
Nicotine Dependence	Mdn = 5.50	Mdn = 6.00	
n = 40/group	(Range = 2 – 9)	(Range = 2 – 10)	

Table 22 History of cessation medication use by treatment assignment

Medication	Enhanced Usual Care [a] *f* (%)	Special Intervention [b] *f* (%)	χ^2 value, p-value
Nicotine gum			
Yes	9 (23)	12 (30)	0.58, p = .45
No	31 (77)	28 (70)	
Nicotine patch			
Yes	16 (40)	21 (53)	1.26, p = .27
No	24 (60)	19 (47)	
Bupropion			
Yes	5 (12)	13 (32)	4.59, p = .04*
No	35 (88)	27 (68)	

*significant value
[a] n = 40, [b] n = 40
Note: (100% of subjects in both groups did not have a history of using the nicotine inhaler or spray)

Table 23 Cessation medication use during hospitalization by treatment assignment

Current medication	Enhanced Usual Care [a] *f* (%)	Special Intervention [b] *f* (%)	χ^2 value, p-value
Nicotine gum			
Yes	1 (2)	0 (0)	1.01, p = .32
No	39 (98)	40 (100)	
Nicotine patch			
Yes	6 (15)	4 (10)	0.46, p = .50
No	34 (85)	36 (90)	
Bupropion			
Yes	2 (5)	2 (5)	0.00, p = 1.00
No	38 (95)	38 (95)	

[a] n = 40, [b] n = 40

Self-efficacy for tobacco abstinence control was measured with the Relapse Situational Efficacy Questionnaire [RSEQ] and the Tobacco Abstinence Self-efficacy Scale [TASES]. Data are presented in Tables 24 and 25 respectively. Treatment groups did not differ on the total scores and subscale scores. Subjects in the UC group had a mean score of 111.18 (SD = 28.71) on the RSEQ and 2,785.75 (SD = 1,030.16) on the TASES. SI subjects had had a mean score of 108.75 (SD = 29.47) on the RSEQ and 2,726.75 (SD = 1,110.97) on the TASES.

Participants were asked to identify their outcome expectancy of quitting smoking with respect to their heart health as measured with the Perceived Therapeutic Efficacy Scale for Tobacco Abstinence [PTES]. Again, treatment groups did not differ on scores for this instrument with mean scores of 74.00 for the SI group and 78.00 for the UC group. Data were skewed to the right for both groups. The mode for each group was 100. Ten subjects in each group had a total PTES score of 100, comprising 25% of each treatment group. The median score for the SI group was 84.00 and 85.00 for the UC group. Table 25 displays the results of the means for each treatment group.

Baseline results for the Profile of Mood States [POMS] are presented in Table 26. SI subjects had significantly higher total score (M = 132.35, SD = 32.41) and confusion/bewilderment subscale score (M = 17.95, SD = 4.66) compared to the UC group (t = -2.18, p= .04 and t= -3.65, p = .001, respectively). The mean total score for the UC group was 116.4 (SD = 33.09) and the mean confusion/bewilderment subscale score was 14.20 (SD = 4.53). The treatment groups did not differ with respect to mean scores on the other POMS subscale scores.

Table 24 Baseline Relapse Situational Efficacy scores by treatment assignment

RSEQ Total/ Sub-Scores	Enhanced Usual Care [a]	Special Intervention [b]	t-test value, p-value
RSEQ Total	M = 111.18 (SD = 28.71) (Range = 61 -172)	M = 108.75 (SD = 29.47) (Range = 51 – 167)	0.37 p = .72
Negative Affect Score	M = 17.20 (SD = 7.00) (Range = 8-32)	M = 16.85 (SD = 6.65) (Range = 8-32)	0.23 p = .82
Positive Affect Score	M = 18.23 (SD = 4.44) (Range = 9-24)	M = 16.60 (SD = 4.63) (Range = 6-24)	1.60 p = .12
Restrictive Score	M = 21.20 (SD = 4.79) (Range = 12 – 28)	M = 20.88 (SD = 5.41) (Range = 10 – 28)	0.28 p = .78
Idle Time Score	M = 11.95 (SD = 3.88) (Range = 5 – 20)	M = 12.15 (SD = 4.07) (Range = 5 – 20)	-0.23 p = .83
Social/Food Score	M = 21.58 (SD = 7.19) (Range = 10 – 36)	M = 22.10 (SD = 6.45) (Range = 12 – 35)	-0.34 p = .74
Arousal Score	M = 16.68 (SD = 4.49) (Range = 9 – 24)	M = 15.75 (SD = 4.17) (Range = 8 – 24)	0.96 p = .35
Craving Score	M = 4.35 (SD 1.75) (Range = 2 – 8)	M = 4.43 (SD = 1.73) (Range = 2 – 8)	-0.19 p = .85

[a] n = 40, [b] n = 40

Table 25 Outcome expectancy (PTES) and self-efficacy (TASES)

PTES, TASES Total & TASES Sub-Scores	Enhanced Usual Care [a]	Special Intervention [b]	t-test value, p-value
PTES Total	M = 77.65 (SD = 23.28) (Range = 29 – 100)	M = 74.65 (SD = 27.22) (Range = 0 – 100)	0.53, p = .60
TASES Total	M = 2,785.75 (SD = 1,030.16) (Range = 840 – 4,500)	M = 2,726.75 (SD = 1,110.97) (Range = 470 – 4,500)	0.25, p = .81
Controlling urges	M = 619.25 (SD = 236.51) (Range = 130 – 1,000)	M = 591.50 (SD = 243.74) (Range = 100 – 1,000)	0.52, p = .61
Resisting smoking over time	M = 360.50 (SD = 155.38) (Range = 60 – 600)	M = 365.50 (SD = 171.85) (Range = 60 – 600)	-0.14, p = .90
Resisting a puff of a cigarette	M = 216.75 (SD = 120.03) (Range = 40 – 100)	M = 219.75 (SD = 121.94) (Range = 40 – 100)	-0.11, p = .92
Resisting a puff in situations	M = 1,238.3 (SD = 537.0) (Range = 350 – 2,100)	M = 1,211.5 (SD = 575.7) (Range = 230 – 2,100)	0.22, p = .84
Resisting a puff in the hospital	M = 351.00 (SD 91.09) (Range = 80 – 400)	M = 338.50 (SD 105.84) (Range = 40 – 400)	-0.57, p = .58

[a] n = 40, [b] n = 40

Table 26 Profile of Mood State scores by treatment assignment

Total and Sub-Scores	Enhanced Usual Care [a]	Special Intervention [b]	t-test value, p- value
POMS Total	M = 116.4 (SD=33.09) (Range = 67 – 229)	M = 132.35 (SD=32.41) (Range = 62 – 216)	-2.18, p = .04*
Tension & Anxiety	M = 22.15 (SD=6.81) (Range = 11 – 43)	M = 24.93 (SD=6.85) (Range = 11 – 42)	-1.12, p = .08
Depression & Dejection	M = 26.5 (SD=10.59) (Range = 15 – 61)	M = 30.65 (SD=14.05) (Range = 15 – 70)	-1.49, p = .15
Anger & Hostility	M = 19.3 (SD=8.13) (Range = 12 – 46)	M = 21.63 (SD=8.91) (Range = 12 – 49	-1.22, p = .23
Vigor & Activity	M = 16.9 (SD=5.34) (Range = 8 – 28)	M = 19.13 (SD=8.13) (Range = 8 – 39)	-1.45, p = .16
Fatigue & Inertia	M = 17.35 (SD=6.83) (Range = 7 – 35)	M = 18.08 (SD=6.72) (Range = 7 – 35)	-0.48, p = .64
Confusion & Bewilderment	M = 14.20 (SD=4.53) (Range = 9 – 32)	M = 17.95 (SD=4.66) (Range = 9 – 27)	-3.65, p = .001*

*significant value
[a] n = 40, [b] n = 40

5.1.3. Results for the primary aim

The primary study aim was to examine the efficacy of a 12-week nurse-delivered telephone tobacco abstinence promotion and relapse management intervention designed to enhance self-efficacy and smoking abstinence for smokers desiring to abstain following hospital discharge. The dependent variable of interest was smoking point prevalence validated by carbon monoxide [CO], which was measured 12 (T_1) and 24 (T_2) weeks following discharge. Results will be presented by the pre-specified hypotheses.

Hypothesis 1a: The group of hospitalized smokers assigned to a 12-week abstinence promotion and relapse management intervention following discharge were hypothesized to have a greater number of participants with smoking abstinence 12 weeks following discharge (smoking point prevalence verified by CO) as compared to subjects who were assigned to only enhanced usual care condition.

At the time of the 12-week follow-up, 31 (80%) UC subjects were smoking and 8 (20%) were abstinent. For the SI group, 24 (60%) subjects were smoking and 16 (40%) were abstinent (refer to Figure 10). One subject in each group reported abstinence, but had CO readings greater than 8 ppm suggesting they had resumed smoking. The total sample (n = 80) was not available at the first follow-up. Twelve subjects were lost by the first follow-up visit. These subjects were included in the analysis and were treated as smoking. Ten subjects were lost to follow-up in the usual care group while two were lost to follow-up in the special intervention group. The analysis was based on a total of 79 subjects due to the death (unrelated to the study) of a subject in the UC group. Table 27 presents the unadjusted logistic regression coefficients for the first directional primary aim hypothesis. Treatment assignment was significantly related to the point prevalence smoking status at the 12-week follow-up visit ($\chi_{LR}^2 = 3.6$, df = 1, p= .03, n = 79). The

odds ratio for the treatment variable at 12-weeks was 2.58, which suggested tobacco abstinence was 2.58 times more likely to occur among special intervention participants than the usual care group. While the coefficient was reported in the appropriate direction as expected and the effect of treatment was statistically significant based on a one-sided hypothesis test, the fit of the model was questionable. As presented in Table 28, the model correctly classified only the smoking subjects. None of the abstinent participants were correctly classified, which further supported that the model was poor at predicting tobacco abstinence by treatment assignment at 12 weeks follow-up. Hosmer and Lemeshow goodness of fit statistic was not generated since there was limited variability with the dichotomous independent variable (Hosmer & Lemeshow, 2000). The Nagelkerke R^2 was .063. Logistic regression diagnostic plots also supported the lack of fit for this model.

Hypothesis 1b: The group of hospitalized smokers assigned to a 12-week abstinence promotion and relapse management intervention following discharge were hypothesized to have a greater number of participants with smoking abstinence 24 weeks following discharge (smoking point prevalence verified by CO) as compared to subjects who were assigned to only enhanced usual care condition.

At the time of the 24-week follow-up, 33 (85%) UC subjects were smoking and 6 (15%) were abstinent. For the SI group, 22 (58%) subjects were smoking and 16 (42%) were abstinent (refer to Figure 11). A logistic regression analysis was used to compare the treatment groups on point prevalence of smoking at the 24-week follow-up visit. Table 29 provides the coefficients and model statistics for this analysis. Again, the hypothesis was directional and significance level of .05 was used. Two additional subjects were lost due to death (unrelated to the study) between the 3 month and 6 month follow-up visits. Therefore, two subjects from the SI group and one

from the UC group were not available for the analysis. A total of 77 subjects were used for this analysis. The same 12 subjects who were lost to follow-up at 3 months were also lost to follow-up at this measurement point. One subject from the UC group reported abstinence at this time, but had a CO reading greater than 8 ppm and was represented as smoking in the analysis. Treatment assignment was significantly related to the point prevalence smoking status at the 24-week follow-up visit ($\chi_{LR}2 = 6.92$, df = 1, p = .005, n = 77). The Nagelkerke R^2 was .123. The odds ratio for the treatment variable at 24-weeks was 4.00, which suggested tobacco abstinence was four times more likely to occur among special intervention participants than the usual care group. Similar to the first hypothesis, this model only correctly classified smoking subjects and not abstinent subjects (refer to Table 30). Assessment of the model with logistic regression diagnostics included estimated probabilities (range = .15 - .42), Cook's influence statistics (range= .005 - .14), leverage statistics (range = 0.0256 - 0.0263), and the change in the values of regression coefficients (range=-.171 - .064). Plots of the latter three diagnostics with the estimated probabilities further indicated a poor fit of this model.

Additional logistic regression analyses were conducted to examine the impact of baseline subject characteristics that were significantly different between the treatment groups (current employment, length of hospital stay, health insurance, surgery during hospitalization, previous use of bupropion, total score on the POMS, and confusion/bewilderment subscale score). Based on backward stepwise logistic regression analyses using the likelihood method, only current employment and length of hospital stay were retained for covariate adjustment as possible confounders. An adjusted logistic regression model re-examined the effect of treatment assignment on smoking status at 3 months follow-up controlling for current employment and length of hospital stay. The full model was significant ($\chi_{LR}^2 = 21.64$, df = 3, p = .0001, n = 79).

The χ_{LR}^2 test statistic for the first block of the model was significant and suggested that employment (OR = 0.167) and longer hospital stay (OR = 1.222) were associated with smoking abstinence at 3 months follow-up (Refer to Table 31). When the treatment variable was added, the value of the χ^2 test statistic decreased for this second block of the model (relative to the unadjusted model), but remained significant ($\chi_{LR}^2 = 4.87$, df = 1, p = .014), suggesting an effect for the special intervention over enhanced usual care (Refer to Table 32). The odds ratio was 3.734 for the treatment assignment, which suggested the odds of tobacco abstinence was 3.73 times more likely to occur for participants in the special intervention group than the usual care group when employment and length of stay were controlled in the analysis. The 90% confidence interval was 1.345 to 10.363. The upper end of the confidence interval (10.363) from the odds ratio was the area of interest within the interval for this one-tailed test. Findings suggested that the fit of this model was an improvement over the model that did not control for confounding variables. The classification table also supported the improved fit of this expanded model (see Table 33). Prediction of smokers decreased, but the prediction of abstinent subjects increased. In addition, the overall sensitivity increased. The Nagelkerke R^2 also improved with this adjusted model. Logistic regression diagnostics also indicated an improvement in fit from the single independent variable model previously discussed.

A covariate-adjusted logistic regression model was also estimated for smoking point prevalence at 24-weeks follow-up. Based on stepwise logistic regression analyses using the likelihood method, only the baseline covariates of current employment and length of stay were significantly related to smoking point prevalence at 24-weeks follow-up. Tables 34 through 36 present the results of this second adjusted logistic regression analysis controlling for the covariates of current employment and length of stay before entering the treatment variables. The

model which included the baseline covariates and the effect for treatment was significant (χ_{LR}^2 = 21.82, df = 3, p = .0001, n = 77). Once again, employment and longer hospital stays were associated with smoking abstinence. The second block of the analysis, controlling for the covariates and entering the treatment variable, was significant (χ_{LR}^2 = 7.69, df = 1, p = .004, n = 77), an improvement over the unadjusted model with only the treatment effect. The odds ratio was 5.469 for the treatment assignment, which suggested the odds of tobacco abstinence was 5.47 times more likely to occur for participants in the special intervention group than the usual care group when employment and length of stay were controlled in the analysis. The 90% confidence interval was 1.878 to 15.931. As with the 12-week follow-up analysis, the sensitivity and specificity of the test improved with respect to overall prediction of smoking point prevalence and was further supported by logistic regression diagnostics. The Nagelkerke R^2 also improved with this adjusted model. Therefore, the adjusted logistic regression analyses were used to examine the two primary aim hypotheses. In both cases, these indicated a significant treatment effect.

Outliers were identified within both treatment groups for length of stay, POMS total, and POMS subscale scores. The same subjects were outliers for all of these measures. Sensitivity analyses were conducted of the primary and following secondary aim with these subjects removed from the database. Although there were changes in scores, the tests of the hypotheses continued to demonstrate significant findings. In addition, longer hospital admissions and current employment continued to be significant covariates to the treatment assignment with respect to smoking abstinence for the primary aim.

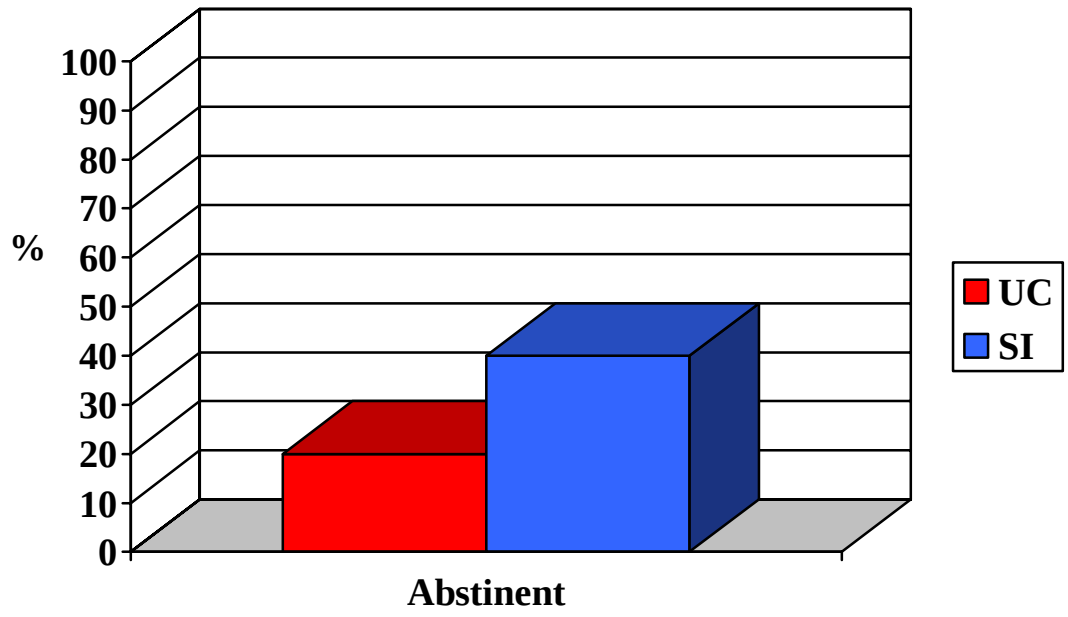


Figure 10 Smoking Status at 3 months by treatment assignment

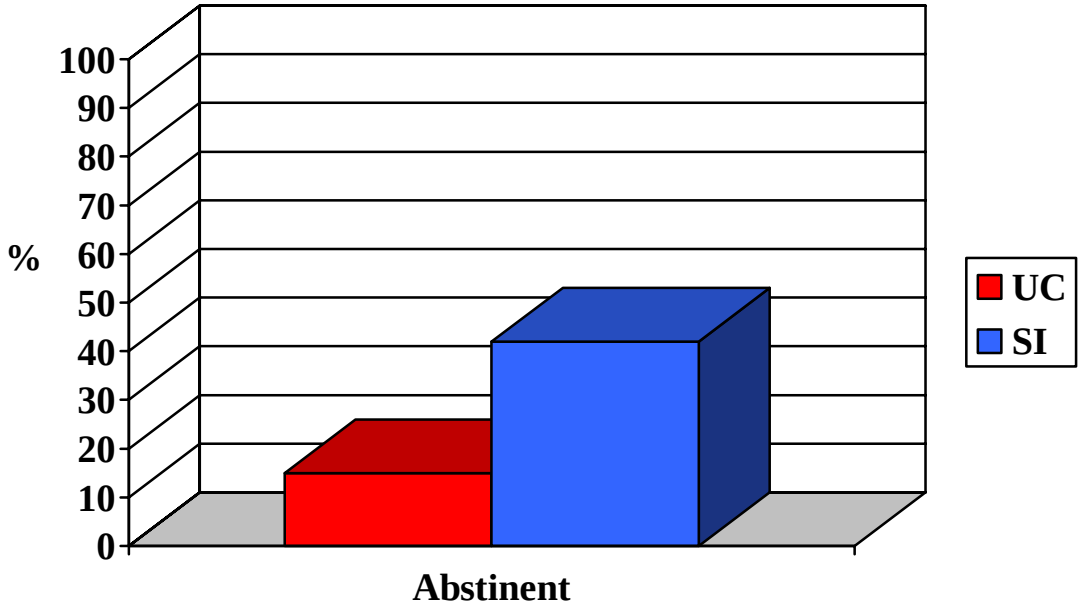


Figure 11 Smoking Status at 6 months by treatment assignment

Table 27 Logistic regression model for point prevalence tobacco abstinence at three months with treatment assignment

	b	SE(b)	Wald	Df	p*	Odds Ratio
Treatment group	0.949	0.511	3.446	1	.063	2.583
Constant	-1.355	0.397	11.667	1	.001	0.258

*two-tailed p-values

$\chi_{LR}2 = 3.6$, df = 1, p = .03*; -2 Log Likelihood = 93.42; Nagelkerke $R^2 = .063$.

Table 28 Classification of predictive model for tobacco abstinence point prevalence at 3-month follow-up

		Predicted	
		Smoking status	Percentage Correct
Observed	Smoking	Abstinent	
Smoking	55	0	100.0
Abstinent	24	0	0.0
Overall Percentage			69.6

Table 29 Logistic regression model for point prevalence tobacco abstinence at six months with treatment assignment

	b	SE(b)	Wald	df	p*	Odds Ratio
Treatment group	1.386	0.552	6.303	1	.012	4.000
Constant	-1.705	0.444	.14.754	1	.0001	0.182

*two-tailed p-values

$\chi_{LR}2 = 6.92$, df = 1, p = .005*; -2 Log Likelihood = 85.22; Nagelkerke $R^2 = .123$.

Table 30 Classification of predictive model for tobacco abstinence point prevalence at 6-month follow-up

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	55	0	100.0
Abstinent	22	0	0.0
Overall Percentage			71.4

Table 31 Logistic regression model for point prevalence at 3-month follow-up with baseline covariates

	b	SE(b)	Wald	df	p*	Odds Ratio
Current employment	-1.789	0.635	7.947	1	.005	0.167
Length of stay	0.201	0.071	8.021	1	.005	1.222
Constant	-1.331	0.482	7.637	1	.006	0.264

*two-tailed p-values

Block 1: $\chi_{LR}2 = 16.77$, df = 2, p = .001*; Block 1: -2 Log likelihood = 80.25
Nagelkerke $R^2 = .270$; $\chi_{HL}2$ of 1.889, df = 6, p = .930.

Table 32 Logistic regression model for point prevalence at 3-month follow-up with treatment assignment and baseline covariates

	b	SE(b)	Wald	df	p*	Odds Ratio
Current employment	-2.237	0.716	9.769	1	.002	0.107
Length of stay	0.198	0.075	6.896	1	.009	1.219
Treatment Group	1.317	0.621	4.507	1	.034	3.734
Constant	-1.842	0.575	10.249	1	.001	0.159

*two-tailed p-values

Block 2: $\chi_{LR}2 = 4.87$, df = 1, p = .014*; Model $\chi 2 = 21.64$, df = 3, p = .0001;
Block 2: -2 Log likelihood = 75.38; Nagelkerke $R^2 = .339$; $\chi_{HL}2$ of 5.180, df = 8, p = .738.

Table 33 Classification of predictive model point prevalence at 3-month follow-up with treatment assignment and baseline covariates

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	50	5	90.9
Abstinent	14	10	41.7
Overall Percentage			75.9

Table 34 Logistic regression model for point prevalence at 6-month follow-up with baseline covariates

	b	SE(b)	Wald	df	p*	Odds Ratio
Current employment	-1.454	0.632	5.297	1	.021	0.234
Length of stay	0.195	0.070	7.708	1	.005	1.216
Constant	-1.535	0.492	9.743	1	.002	0.215

*two-tailed p-values

Block 1: $\chi_{LR}2 = 14.13$, df = 2, p = .0006*; Block 1: -2 Log likelihood = 78.01
Nagelkerke $R^2 = .240$; $\chi_{HL}2$ of 6.247, df = 7, p = .511.

Table 35 Logistic regression model for point prevalence at 6-month follow-up with treatment assignment and baseline covariates

	b	SE(b)	Wald	df	p*	Odds Ratio
Current employment	-1.990	0.723	7.576	1	.006	0.137
Length of stay	0.194	0.077	6.322	1	.012	1.214
Treatment Group	1.699	0.650	6.834	1	.009	5.469
Constant	-2.248	0.624	12.996	1	.0003	0.106

*two-tailed p-values

Block 2: $\chi_{LR}2 = 7.69$, df = 1, p = .004*; Model $\chi 2 = 21.82$, df = 3, p = .0001*;
Block 2: -2 Log likelihood = 70.313; Nagelkerke $R^2 = .354$; $\chi_{HL}2$ of 8.165, df = 7, p = .318.

Table 36 Classification of predictive model point prevalence at 6-month follow-up with treatment assignment and baseline covariates

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	50	5	90.9
Abstinent	12	10	45.5
Overall Percentage			77.9

5.1.4. Results for the secondary aims

This first secondary aim examined the relationship between the outcome behavior of smoking point prevalence at T_1 and T_2 and conceptually driven variables from Self-efficacy Theory (refer to Figure 2) (i.e.., self-efficacy, relapse situation efficacy, perceived treatment efficacy, social support for tobacco abstinence, and affective states, in particular depressive symptoms). Univariate logistic regression was the method used to independently test each of these relationships, which are represented by hypotheses 2a – 2e. The overall level of significance for this first secondary aim was .05, which was divided evenly across these ten analyses (i.e., results were considered significant if the p–value for the χ_{LR}^2 statistic was $\leq .005$.

Hypothesis 2a(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived self-efficacy as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES].

Hypothesis 2a(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived self-efficacy as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES].

Table 37 presents the regression coefficient table and test statistic for the T_1 test of baseline self-efficacy measured by the TASES. The model was not statistically significant ($\chi_{LR}^2 = 6.72$, df = 1, p = .006, n = 79), suggesting that baseline perceived self-efficacy does not predict 12-week smoking point prevalence. The 99% confidence interval for the TASES coefficient was .999 – 1.001. Table 38 presents the classification of the dependent variable predicted by the self-efficacy variable measured by the TASES. Sensitivity and specificity for the overall prediction and abstinent status were low. The model predicted smoking status at 92.7%.

The T_2 examination of the baseline TASES total scores is presented in Table 39. As with the analysis at T_1, the model was not significant ($\chi_{LR}^2 = 4.937$, df = 1, p = .014, n = 77), suggesting that baseline perceived self-efficacy is not related to smoking point prevalence at 24-weeks follow-up. The 99% confidence interval for the TASES coefficient was .999 – 1.001. Table 40 displays the sensitivity and specificity of the model as a classification table. This model was unable to predict abstinent smokers, but correctly identified all smoking subjects. The overall rate of correct classification for the model was below 72%.

Hypothesis 2b(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for relapse situation efficacy as measured by the Relapse Situation Efficacy Questionnaire [RSEQ].

Hypothesis 2b(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for relapse situation efficacy as measured by the Relapse Situation Efficacy Questionnaire [RSEQ].

The model for baseline self-efficacy as measured by the RSEQ was significant for smoking point prevalence at T_1 ($\chi_{LR}^2 = 7.75$, df = 1, p = .003, n = 79). As reported in Table 41 the regression coefficient for the RSEQ was significant. The coefficient for the RSEQ was significant (90% CI = 1.001 – 1.050). Table 42 presents the sensitivity and specificity of the model to predict the outcome variable in a classification table reporting an overall rate of 74.7%. Specificity was 94.5% and 29.2% for smoking and abstinence prediction, respectively, for the 12-week smoking point prevalence. The model was significant, but the fit of the model was an improvement over the TASES ability to predict as a measure of self-efficacy at T_1. Logistic regression diagnostic plots also suggested questionable fit of this independent variable to the dependent variable.

The examination was completed with the T_2 smoking point prevalence and the RSEQ. The model was borderline significant with a $\chi_{LR}^2 = 6.997$, df = 1, p = .005 (n = 77). Table 43 presents the regression coefficient, Wald statistic, p-value for the Wald statistic, and odds ratio. The regression coefficient for the RSEQ was not significant (99% CI = .999 – 1.049), suggesting there was a lack of fit of the model. The classification table presented by Table 44 suggested the overall classification rate of 74.0% for this model. The model had difficulty predicting tobacco abstinence, but had a 94.5% rate at correctly predicting smoking subjects at 24-weeks following baseline. Logistic regression diagnostics and plots further supported a potential lack of fit of the model.

Hypothesis 2c(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for outcome expectancy as measured by the Perceived Therapeutic Efficacy Scale for Relapse Maintenance [PTES-RM].

Hypothesis 2c(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived treatment efficacy as measured by the Perceived Therapeutic Efficacy Scale for Relapse Maintenance [PTES-RM].

Based on a logistic regression analyses, no significant relationship between the PTES-RM and smoking status at T_1 was found ($\chi_{LR}^2 = 3.936$, df = 1, p = .024, n = 79) The PTES coefficient was not significant (99% CI = .991 – 1.054). (Refer to Table 45). Sensitivity and specificity of the first model presented in Table 46 suggested the model lacked adequate fit. Results were not significant for the second model that tested the relationship between PTES and T_2 smoking point prevalence ($\chi_{LR}^2 = 3.231$, df = 1, p = .037, n = 77) (Refer to Table 47). The

PTES coefficient at 24-weeks was not significant (99% CI = .989 – 1.053). This second model was also not predictive of abstinent subjects, but correctly predicted subjects who resumed smoking at a rate of 100%. The overall sensitivity of each model was similar. Hosmer and Lemeshow tests of "lack of fit" for each model were not significant (Model for T_1: $\chi_{HL}^2 = 4.689$, df = 6, and p = .585; Model for T_2: $\chi_{HL}^2 = 4.912$, df = 6, and p = .555). Significant results for these tests would have indicated lack of fit.

Hypothesis 2d(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived social support for tobacco abstinence.

Hypothesis 2d(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with baseline scores for perceived social support for tobacco abstinence.

Based on the likelihood ratio test statistic for the model, no significant relationship was found between baseline social support for tobacco abstinence and smoking point prevalence measured at T_1 ($\chi_{LR}^2 = 3.767$, df = 1, p = .374, n = 79) (Refer to Table 49). The coefficient for social support was not significant (99% CI = .586 – 2.075). The overall classification rate for the model with baseline social support as predictor was 69.6%, with all subjects who resumed smoking at 12 weeks being correctly classified and no abstinent subjects being correctly classified (Table 50). The second part of the hypothesis tested social support with the dependent variable measured at T_2. Similar to the findings at T_1, baseline social support for tobacco abstinence was not significantly associated with smoking point prevalence measured at T_2 ($\chi_{LR}^2 = 2.779$, df = 1, p = .299, n = 77). The social support coefficient with 24-week smoking status

was not significant (99% CI = .585 – 2.279). Tables 51 and 52 present the results of the model fitting and classification table for the model of social support and smoking point prevalence at T_2.

Hypothesis 2e(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by an inverse relationship with baseline scores for affective states, specifically depressive symptoms as measured by the depression/dejection subscale on the Profile of Mood States [POMS].

Hypothesis 2e(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by an inverse relationship with baseline scores for affective states, specifically depressive symptoms as measured by the depression/dejection subscale on the Profile of Mood States [POMS].

The baseline POMS subscale score for depression and dejection was examined as a predictor of smoking status at T_1 and T_2 (Refer to Tables 53 through 56). The logistic regression model for this covariate and the dependent variable at T_1 was not significant ($\chi_{LR}^2 = 0.033$, df = 1, p = .429, n = 79). Table 53 provides the test statistic and coefficient data. The coefficient for depressive symptoms was not significant (99% CI .947 – 1.048). The classification table (Refer to Table 54) of the predictive ability of this model suggested poor prediction for smoking abstinence. The depression and dejection subscale for the POMS was also not significantly predictive of smoking point prevalence at T_2 ($\chi_{LR}^2 = 0.010$, df = 1, p = .462, n = 77). The coefficient and classification data for this second logistic regression analysis are presented in Tables 55 through 56. The coefficient was not significant for depressive symptoms with smoking status at 24-weeks (99% CI = .944 – 1.055). Classification table demonstrates the poor predictive ability of this model.

Table 37 Logistic regression of baseline TASES as a predictor of 3-month smoking status

	b	SE(b)	Wald	df	P*	Odds Ratio
TASES–Self-efficacy	0.001	0.0003	6.047	1	.014	1.001
Constant	-2.651	0.815	10.589	1	.001	0.071

*two-tailed p-values

Block 1: $\chi_{LR}2$= 6.72, df = 1, p = .006; -2 Log likelihood = 90.30

Nagelkerke R^2 = .115; $\chi_{HL}2$ = 9.749, df = 8, and p = .283

Table 38 Classification of baseline TASES as a predictor of 3-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	51	4	92.7
Abstinent	19	5	20.8
Overall Percentage			70.9

Table 39 Logistic regression of baseline TASES as a predictor of 6-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
TASES–self-efficacy	0.001	0.0003	4.541	1	.033	1.001
Constant	-2.517	0.826	9.293	1	.002	0.081

*two-tailed p-values

Block 1: $\chi_{LR}2$= 4.937, df = 1, p = .014; -2 Log likelihood = 87.196

Nagelkerke R^2 = .089; $\chi_{HL}2$ = 12.135, df = 8, and p = .145

Table 40 Classification of baseline TASES as a predictor of 6-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	55	0	100.0
Abstinent	24	0	0.0
Overall Percentage			71.4

Table 41 Logistic regression of baseline RSEQ as a predictor of 3-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
RSEQ – self-efficacy	0.025	0.009	6.936	1	.008	1.025
Constant	-3.646	1.127	10.472	1	.001	0.026

*two-tailed p-values

Block 1: $\chi_{LR}2$= 7.75, df = 1, p = .0026*; -2 Log likelihood = 89.274

Nagelkerke R^2 = .132; $\chi_{HL}2$ = 9.63, df = 8, and p = .292

Table 42 Classification of baseline RSEQ as a predictor of 3-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	52	3	94.5
Abstinent	17	7	29.2
Overall Percentage			74.7

Table 43 Logistic regression of baseline RSEQ as a predictor of 6-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
RSEQ – self-efficacy	0.024	0.009	6.328	1	.012	1.024
Constant	-3.648	1.148	10.093	1	.001	0.026

*two-tailed p-values

Block 1: $\chi_{LR}2 = 6.997$, df = 1, p = .0049*; -2 Log likelihood = 85.136

Nagelkerke $R^2 = .124$; $\chi_{HL}2 = 7.569$, df = 7, and p = .372

Table 44 Classification of baseline RSEQ as a predictor of 6-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	52	3	94.5
Abstinent	17	5	22.7
Overall Percentage			74.0

Table 45 Logistic regression of baseline PTES as a predictor of 3-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
PTES	0.022	0.012	3.361	1	.067	1.022
Constant	-2.540	0.999	6.471	1	.011	0.079

*two-tailed p-values

Block 1: $\chi_{LR}2 = 3.936$, df = 1, p = .0236; -2 Log likelihood = 93.083

Nagelkerke $R^2 = .069$; $\chi_{HL}2 = 4.689$, df = 6, and p = .584

Table 46 Classification of baseline PTES as a predictor of 3-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	55	0	100.0
Abstinent	24	0	0.0
Overall Percentage			69.6

Table 47 Logistic regression of baseline PTES as a predictor of 6-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
PTES	0.020	0.012	2.781	1	.095	1.020
Constant	-2.503	1.018	6.043	1	.014	0.082

*two-tailed p-values

Block 1: $\chi_{LR}2 = 3.231$, df = 1, p = .037; -2 Log likelihood = 88.902

Nagelkerke $R^2 = .059$; $\chi_{HL}2 = 4.912$, df = 6, p = .555

Table 48 Classification of baseline PTES as a predictor of 6-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	55	0	100.0
Abstinent	22	0	0.0
Overall Percentage			71.4

Table 49 Logistic regression of social support as a predictor of 3-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
Social Support	0.081	0.252	0.103	1	.749	1.084
Constant	-1.210	1.216	0.989	1	.320	0.298

*two-tailed p-values

Block 1: $\chi_{LR}2 = 3.767$, df = 1, p = .374; -2 Log likelihood = 93.253

Nagelkerke $R^2 = .002$; $\chi_{HL}2 = 4.689$, df = 2, and p = .096

Table 50 Classification social support as a predictor of 3-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	55	0	100.0
Abstinent	24	0	0.0
Overall Percentage			69.6

Table 51 Logistic regression of social support as a predictor of 6-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
Social Support	0.144	0.264	0.299	1	.584	1.155
Constant	-1.595	1.275	1.566	1	.211	0.203

*two-tailed p-values

Block 1: $\chi_{LR}2 = 2.779$, df = 1, p = .299; -2 Log likelihood = 89.355

Nagelkerke $R^2 = .006$;$\chi_{HL}2 = 4.052$, df = 2, and p = .132

Table 52 Classification of social support as a predictor of 6-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	55	0	100.0
Abstinent	22	0	0.0
Overall Percentage			71.4

Table 53 Depressive symptoms as a predictor of 3-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
Depressive symptoms & Dejection – POMS	-0.004	0.020	0.033	1	.856	0.996
Constant	-0.728	0.611	1.418	1	.234	0.483

*two-tailed p-values

Block 1: $\chi_{LR}2 = .033$, df = 1, p = .429; -2 Log likelihood = 96.987

Nagelkerke $R^2 = .001$; $\chi_{HL}2 = 8.791$, df = 8, and p = .360

Table 54 Classification of depressive symptoms as a predictor of 3-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	55	0	100.0
Abstinent	24	0	0.0
Overall Percentage			69.6

Table 55 Depressive symptoms as a predictor of 6-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
Depressive symptoms & Dejection – POMS	-0.002	0.022	0.010	1	.922	0.998
Constant	-0.857	0.652	1.729	1	.189	0.424

*two-tailed p-values

Block 1: $\chi_{LR}2 = .010$, df = 1, p = .462; -2 Log likelihood = 92.124

Nagelkerke $R^2 = .000$; $\chi_{HL}2 = 7.724$, df = 7, and p = .358

Table 56 Classification of depressive symptoms as a predictor of 6-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	55	0	100.0
Abstinent	22	0	0.0
Overall Percentage			71.4

The second secondary aim examined treatment-related variables, such as time to first smoking lapse, treatment adherence, self-efficacy (TASES), and smoking point prevalence. The first analysis presented is time to lapse. The goal of the treatment was to prevent relapse of subjects following discharge, particularly by preventing abstinence violation events. Next, treatment adherence was examined as a predictor of smoking behavior and for a potential relationship with self-efficacy (TASES). Finally, self-efficacy was examined over the measurement time points of this study by treatment assignment. Results are presented following the hypotheses statements. Overall level of significance for this secondary aim was .05, which was divided evenly across the five hypotheses to be tested to limit the inflation of type 1 error. Therefore, the testwise level of significance was set at .01.

Hypothesis 2f: The time to the first smoking lapse was hypothesized to be longer for subjects who were assigned the 12-week abstinence promotion and relapse management intervention as compared to subjects who were assigned to only enhanced usual care.

Based on the Kaplan-Meier method, the mean time to first lapse in smoking for the UC group was 54.40 days (SE = 8.81). Thirty percent of usual care subjects lapsed within the first week following hospitalization. Fifty percent of the usual care subjects experienced a lapse between 1 week following hospital discharge and the 12-week follow-up visit. Seven subjects in the UC group were right censored at 24 weeks. The SI group had a mean time to first lapse in smoking of 77.22 days (SE = 11.13), with 14 subjects being right censored at 24 weeks. Twenty-five percent of the special intervention group subjects lapsed within the first week following hospital discharge. Forty percent of the special intervention participants experienced a lapse between 1 week after hospital discharge and the 12-week follow-up visit. Figure 11 illustrates

the days to first lapse in smoking for both treatment groups. There was no difference between the treatment groups for the occurrence of the first lapse in smoking ($\chi_{Logrank}^2$ =1.79, df = 1, p = .181).

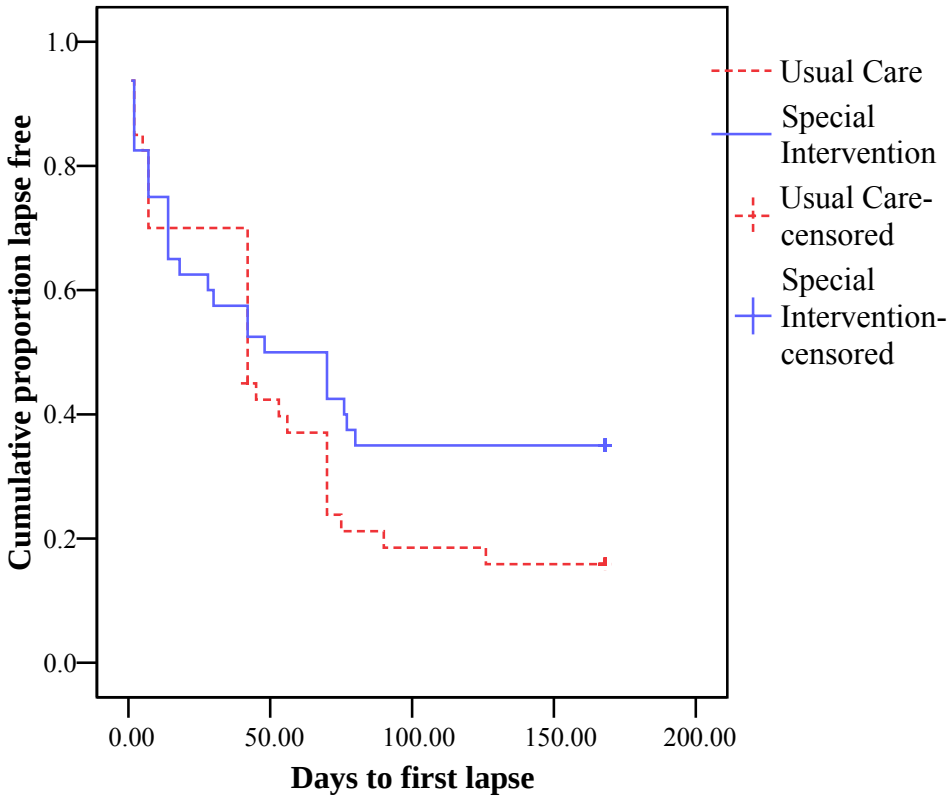


Figure 12 Event history for the to first lapse in smoking

Overall, SI participants in this study had poor adherence to the intervention treatment. Table 57 provides descriptive statistics for the overall rate of treatment adherence, adherence to the telephone calls, and adherence to homework assignments. Overall treatment adherence was on average 35.78% (SD=26.02). The mean adherence to the telephone calls was 41.25% (SD=26.58), while the mean homework adherence rate was 30.31% (SD=25.62). Adherence rates overall and for the telephone calls and homework assignments ranged from 0 – 100%. Only one subject achieved overall, telephone, and homework adherence rates at 100%. The mode (43.75) to overall treatment adherence was slightly higher than the mean and median. The frequency distribution was skewed to the left and did not have a "u" or "j" shape to the distribution more commonly observed with treatment adherence observations.

Hypothesis 2g(1): Smoking point prevalence at T_1 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with treatment adherence rates.

Tables 58 and 59 summarize the test statistics, estimated regression coefficients, and classification rates for overall treatment adherence entered into a logistic regression model to predict smoking point prevalence at T_1 for special intervention subjects (n = 40). The model was significant ($\chi_{LR}^2 = 8.26$, df = 1, p = .003) and the regression coefficient for treatment adherence suggested that overall treatment adherence was significantly related to abstinence from smoking at 12 weeks follow-up (one-sided p-value = .006). Sensitivity and specificity of this variable as a predictor of smoking status is presented in Table 59. Overall prediction was 70%. The model correctly classified positive smoking status at a rate of 87.5% and tobacco abstinence at a rate of 43.8%. These classification rates were similar to those observed for the covariate-adjusted predictive models for hypotheses associated with the primary study aim.

Hypothesis 2g(2): Smoking point prevalence at T_2 (smoking = 0; abstinence = 1) was hypothesized to be predicted by a positive relationship with treatment adherence rates.

Similar to the previous findings for smoking point prevalence at T_1, the logistic regression model for treatment adherence and smoking point prevalence at T_2 was significant ($\chi_{LR}^2 = 7.78$, df = 1, p = .003) (Refer to Table 60) and the regression coefficient for treatment adherence suggested that adherence to the components of the special intervention was related to smoking abstinence at 24 weeks follow-up (one-sided p=.007). As reported in Table 61, the overall correct classification rate was 68.4%, with smokers being correctly classified at a rate of 86.4% and abstinent participants classified at a rate of 43.8%. The fit of this model was similar to the model for T_1.

Hypothesis 2h: Treatment adherence was hypothesized to have a positive relationship with baseline (T_0) perceived self-efficacy as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES].

A one-tailed Spearman correlation, r_s, was used to assess the relationship between treatment adherence and baseline self-efficacy as measured by the TASES. There was no significant relationship between baseline self-efficacy and treatment adherence for the intervention subjects ($r_s = .029$, p = .861).

Hypothesis 2i: Compared to subjects in the enhanced usual care group, subjects in the treatment group were hypothesized to have a greater increase in self-efficacy, as measured by the Tobacco Abstinence Self-Efficacy Scale [TASES], from baseline (T_0) to follow-up measurements at T_1 and T_2.

A general linear model with repeated measures was used to examine TASES self-efficacy scores over time (T_0, T_1, and T_2) by treatment group (3 x 2 design). TASES self-efficacy scores was the within subject variable examined over time. The time by treatment interaction was not significant (F = .077, df = 1.049, p = .926) (Refer to Table 62). The main effect of time was also not significant regardless of treatment assignment (F = 3.321, df = 1.049, p = .07). There does appear to be a trend in the self-efficacy scores over time (Refer to Figure 12).

Table 57 Treatment adherence descriptive statistics for the intervention group

Variables	Special Intervention
Overall Treatment	M = 35.78 (SD = 26.02)
Adherence Rate (%)	Mdn = 31.25
n = 40	(Range = 0 – 100)
Rate of adherence to	M = 30.31 (SD 25.62)
homework (%)	Mdn = 25.00
n = 40	(Range = 0 – 100)
Adherence to phone	M = 41.25 (SD = 26.58)
appointment (%)	Mdn = 37.50
n = 40	(Range = 0 – 100)

Table 58 Treatment adherence as a predictor of treatment group 3-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
Adherence Rate	.041	.016	6.360	1	.012	1.042
Constant	-1.933	.718	7.253	1	.007	.145

*two-tailed p-values

Model: $\chi_{LR}2 = 8.26$, df = 1, p = .003*; -2 Log Likelihood = 45.584

Nagelkerke $R^2 = .252$; $\chi_{HL}2 = 13.233$, df = 5, and p = .021

Table 59 Classification of adherence as a predictor of treatment group 3-month smoking

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	21	3	87.5
Abstinent	9	7	43.8
Overall Percentage			70.0

Table 60 Treatment adherence as a predictor of treatment group 6-month smoking status

	b	SE(b)	Wald	df	p*	Odds Ratio
Adherence Rate	.040	.016	6.020	1	.014	1.041
Constant	-1.822	.721	6.378	1	.012	.162

*two-tailed p-values

Model: $\chi_{LR}2 = 7.78$ df = 1, p = .003*; -2 Log Likelihood = 43.946

Nagelkerke $R^2 = .249$; $\chi_{HL}2 = 12.160$, df = 5, and p = .033

Table 61 Classification of treatment adherence as a predictor of treatment group 6-month smoking status

	Predicted		
	Smoking status		Percentage Correct
Observed	Smoking	Abstinent	
Smoking	19	3	86.4
Abstinent	9	7	43.8
Overall Percentage			68.4

Table 62 Within-subject effects of self-efficacy (TASES) by treatment group

[a] Greenhouse-Geisser

Effect	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Self-efficacy	1992818.87	1.049	1899936.23	3.321	.070	.042
Self-efficacy x Treatment Group	45946.15	1.049	22973.07	.077	.926	.001
Error Self-efficacy	45003502.34	78.667	572079.32			

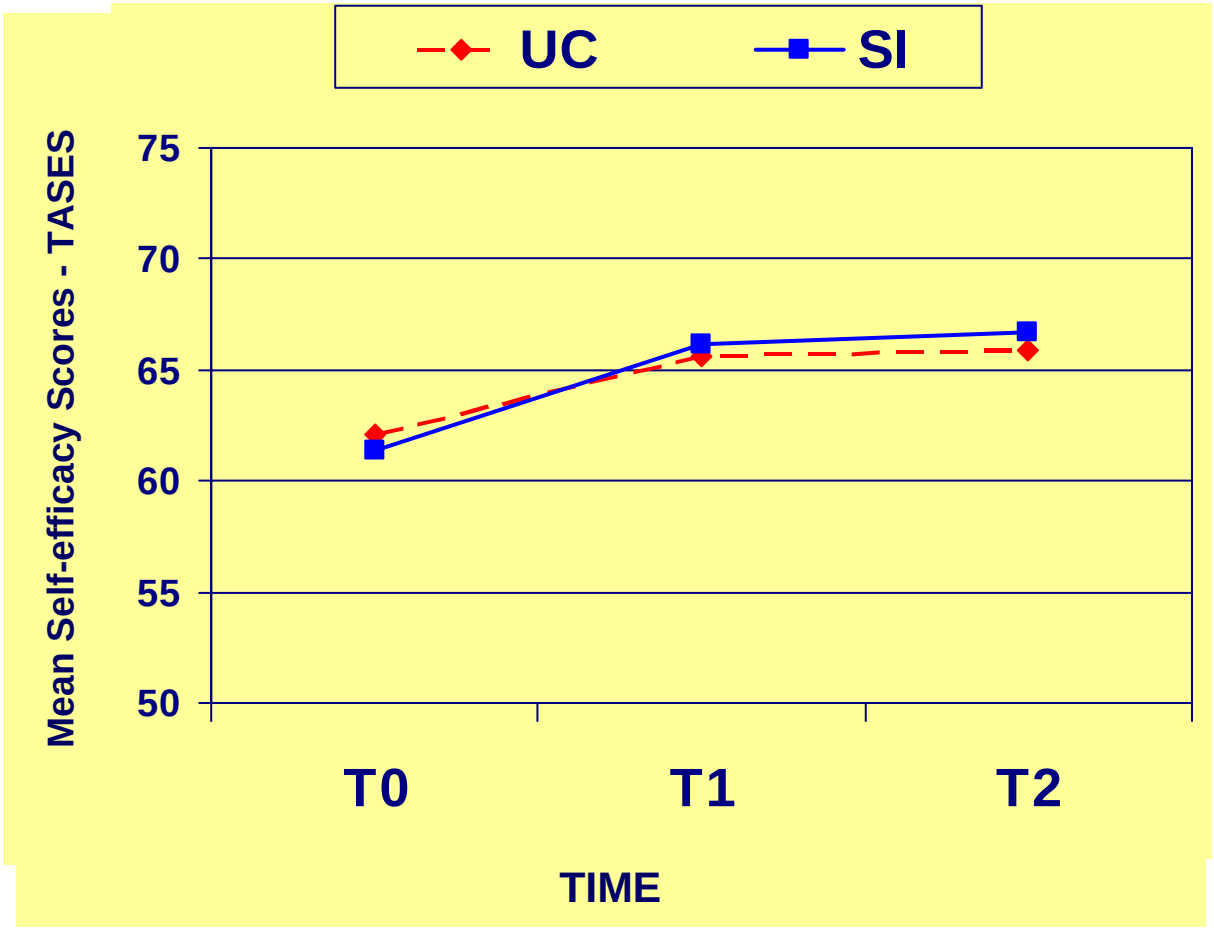


Figure 13 Self-efficacy across T0, T1, T2 by treatment group

5.1.5. Results of the exploratory analyses.

Recruitment cohort and site were not considered as possible stratification factors in the randomization process to assign study treatment to subjects. These exploratory analyses examined whether the recruitment site and cohort affected smoking point prevalence measured 12 and 24 weeks following hospital discharge and/or modified the effect of the treatment assignment. In addition, age was examined for a relationship with smoking behavior and as a potential influencing variable with the treatment assignment. Participants were divided into two cohorts. The first forty participants recruited to the study comprised the first recruitment cohort and the latter forty participants recruited for the project were assigned to the second cohort. Participants in the first cohort were recruited from one of two university medical hospitals used for recruitment. Participants from the second cohort were recruited across the Sites A, B, and C, which included the community hospital. At site A, thirty-nine participants were recruited; however, one participant was eliminated from the 24-week outcome data in Table 64 since their death occurred before the last smoking status measurement could be completed. There were thirty-eight participants recruited from site B. One person was eliminated for the 12-week outcome data due to death and additional subject was eliminated for the 24-week outcome data due to death prior to the last smoking status measurement. Site C had a total of three subjects recruited, which were all randomly assigned to special intervention. Tables 63 through 66 provide the smoking point prevalence for each cohort by treatment assignment. Both site and recruitment cohort were examined for differences with respect to the demographic baseline variables presented earlier. The only variable that significantly differed between the recruitment sites was employment status. A greater number of participants at site A were unemployed (χ^2 = 7.25, df = 2, p = .027). This variable was also significantly different between the recruitment

cohorts (χ^2 = 6.054, df = 1, p = .014). Two variables were found to be confounding variables to for the treatment assignment with respect to smoking behavior, which included current employment and hospital length of stay. Following the univariate regression models for site and recruitment cohort, these confounding variables were controlled in the analysis.

The examination of the site with smoking prevalence at 12-weeks will be presented first, which will be followed by the recruitment year cohort analyses. These results will be followed by the presentation of the site and recruitment year analyses in a similar fashion for their relationships with the 24-week smoking point prevalence.

Table 63 Smoking point prevalence by hospital site at 12 weeks

	Usual Care		Special Intervention		
	Smoking n (%)	Abstinent n (%)	Smoking n (%)	Abstinent n (%)	Odds Ratio*
Site A	21 (95 %)	1 (5 %)	11 (65 %)	6 (35 %)	11.455
Site B	10 (59 %)	7 (41 %)	12 (60 %)	8 (40 %)	0.952
Site C	0 (0 %)	0 (0 %)	1 (34 %)	2 (66 %)	0.000

*Probability of smoking/Probability of not smoking

Table 64 Smoking point prevalence by hospital site at 24 weeks

	Enhanced Usual Care		Special Intervention		
	Smoking n (%)	Abstinent n (%)	Smoking n (%)	Abstinent n (%)	Odds Ratio*
Site A	21 (95 %)	1 (5 %)	10 (63 %)	6 (37 %)	12.600
Site B	12 (71 %)	5 (29 %)	11 (58 %)	8 (42 %)	1.745
Site C	0 (0 %)	0 (0 %)	1 (34 %)	2 (66 %)	0.000

*Probability of smoking/Probability of not smoking

Table 65 Smoking point prevalence by cohort and treatment assignment at 12 weeks

	Enhanced Usual Care		Special Intervention		
	Smoking n (%)	Abstinent n (%)	Smoking n (%)	Abstinent n (%)	Odds Ratio*
Recruitment Cohort 1	14 (74 %)	5 (26 %)	10 (48 %)	11 (52 %)	3.080
Recruitment Cohort 2	17 (85 %)	3 (15 %)	14 (74 %)	5 (26 %)	2.024

*Probability of smoking/Probability of not smoking

Table 66 Smoking point prevalence by cohort and treatment assignment at 24 weeks

	Enhanced Usual Care		Special Intervention		
	Smoking n (%)	Abstinent n (%)	Smoking n (%)	Abstinent n (%)	Odds Ratio*
Recruitment Cohort 1	14 (82 %)	3 (18 %)	6 (35 %)	11 (65 %)	4.583
Recruitment Cohort 2	9 (75 %)	3 (25 %)	10 (83 %)	2 (17 %)	3.462

*Probability of smoking/Probability of not smoking

The results of a univariate logistic regression analysis suggested that recruitment site had a significant relationship with the smoking behavior measured at 12-weeks (χ_{LR}^2 =6.532, df = 2, p = .038) (Table 67) based on a two-tailed level of significance of .05. The overall site coefficient was significant. The first coefficient listed as Site 1 was a comparison of Site A and Site B. This comparison was significant for smoking behavior at Site B. The comparison between Site A and C was not significant, which suggested they were not different from one another. The difficulty with this analysis is the lack of usual care participants at Site C. This introduces cells with zeroes. When treatment assignment was added to this model, the block $\chi_{LR}2$ was 2.23 (df = 1, p = .135) and was not significant (Table 68). The Hosmer and Lemeshow test for goodness of fit was not significant with a $\chi_{HL}2$ of 3.92 (df = 2, p = .141) and did not suggest problems with the goodness of fit. The treatment group coefficient was not significant and the significance for other site coefficients increased. A third block added a treatment and site interaction variable. The block for this variable addition was significant (χ_{LR}^2 = 4.269, df = 1, p = .039). The Hosmer and Lemeshow goodness-of-fit test did not suggest problems with the fit of the model with the interaction term (χ_{HL}^2 =0.000 , df = 2, p = 1.00). The interaction coefficients for treatment and site were not significant, but the comparison between Site A and B was significant, as well as the treatment assignment (Refer to Table 69). The odds of smoking abstinence was 14.7 times more likely for participants located at Site B than other sites. The odds for smoking abstinence by special intervention participants was 11.5 times more likely than usual care participants. The interaction coefficient had a significance level of .061. These results suggested that treatment assignment and recruitment site were covariates with respect to smoking behavior measured 12 weeks following hospital discharge. Furthermore, there may be a

modifying affect between Site B and treatment assignment (Hosmer & Lemeshow, 2000). Sensitivity and specificity of these models was poor. They each predicted 98% of smoking behavior and only 8% of abstaining participants. Overall prediction was 71%.

Table 67 Univariate logistic regression of sites and 12-week smoking behavior

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Site[§]			5.991	2	.050			
Site 1	1.137	.535	4.515	1	.034	3.117	1.092	8.89
Site 2	2.213	1.294	2.925	1	.087	9.143	.724	115.46
Constant	-1.520	.417	13.267	1	.000	.219		

Model 1: $\chi_{LR}2 = 6.532$, df = 2, p = .038*; Block 1: $\chi_{LR}2 = 6.532$, df = 2, p = .038*
Block 1: -2 Log likelihood = 90.488; Nagelkerke $R^2 = .112$
§ Site A served as the reference

Table 68 Treatment assignment and sites at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Site[§]			4.830	2	.089			
Site 1	1.088	0.543	4.017	1	.045	2.968	1.024	8.600
Site 2	1.816	1.317	1.902	1	.168	6.146	0.465	81.161
Treatment Group	0.792	0.536	2.179	1	.140	2.208	0.771	6.318
Constant	-1.915	0.518	13.638	1	.000	0.147		

Model 2: $\chi_{LR}2 = 8.765$, df = 3, p = .033*; Block 2: $\chi_{LR}2 = 2.234$, df = 1, p = .135
Block 2: -2 Log likelihood = 88.254; Nagelkerke $R^2 = .149$; $\chi_{HL}2 = 3.920$, df = 2, p = .141
§ Site A served as the reference

Table 69 Interaction effect of Treatment assignment and sites at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Site[§]			6.559	2	.038			
Site 1	2.688	1.136	5.598	1	.018	14.700	1.586	136.23
Site 2	1.299	1.326	0.960	1	.327	3.667	0.273	49.29
Treatment Grp.	2.438	1.142	4.555	1	.033	11.455	1.220	107.50
Treatment Grp. x Site			3.522	1	.061			
Treatment Grp.(1) x Site(1)	-2.487	1.325	3.522	1	.061	0.083	0.006	1.117
Constant	-3.045	1.024	8.848	1	.003	0.048		

Model 3: $\chi_{LR}2 = 13.035$, df = 4, p = .011*; Block 3: $\chi_{LR}2 = 4.269$, df = 1, p = .039*
Block 3: -2 Log likelihood = 83.985; Nagelkerke $R^2 = .215$; $\chi_{HL}2 = 0.00$, df = 2, p = 1.000
[§] Site A served as the reference

Since there were two variables that had a confounding affect upon the treatment variable, the variables of employment status and hospital length of stay were controlled for with respect to the analysis previously presented. Furthermore, employment status had a potential confounding influence on the site and cohort variables. Employment and length of stay were entered in the first block of the logistic regression model with the dependent variable of smoking point prevalence at 12-weeks. The model and block ($\chi_{LR}2$ 16.766, df = 2, p = .000) were significant. The Hosmer and Lemeshow test for goodness of fit was not significant with a $\chi_{HL}2$ of 1.889 (df = 6, p = .930), which did not suggest problems with the fit of this step in the model. The beta coefficients for employment (OR = 0.167) and hospital stay (OR = 1.222). Sensitivity and specificity for this first block were improved over the previous discussed models, which included site, treatment, and an interaction term. The model predicted 95% of smokers, 42% of abstainers, and the overall model had sensitivity of 79%.

Site was entered into the model for block 2 (Refer to Table 70). This block was not significant ($\chi_{LR}2$ 3.412, df = 2, p = .182). Only the coefficients for employment and length of hospital stay were significant in this block of the model. Sensitivity of this block remained at 79%, but the specificity of the model predicted smokers at 93% and abstainers at 46%.

Treatment assignment was added in the third block, which was not significant (Block 3: $\chi_{LR}2$ 3.739, df = 1, p = .053). Again, the fit of the model was not problematic according to the Hosmer and Lemeshow test for goodness of fit ($\chi_{HL}2$ of 11.027, df = 8, p = .200). The treatment assignment coefficient was not significant (p = .061). Coefficients for employment (p = .006) and hospital stay (p = .007) were significant. Sensitivity and specificity decreased with this block of the model. The model predicted 87% of smokers, 38% of abstainers, and the overall model had sensitivity of 72%.

Since there was a significant finding with the addition of an interaction variable when site and treatment were previously examined, the interaction variable was added in a fourth block for this analysis, which was not significant ($\chi_{LR}2 = 1.791$, df = 1, p = .181). However, the overall model was significant ($\chi_{LR}2 = 25.708$, df = 6, p = .000). The Hosmer and Lemeshow goodness of fit statistic was not significant ($\chi_{HL}2$ of 1.423, df = 8, p = .994). The only significant coefficients were employment (OR = 0.134), hospital stay (OR = 1.196), and treatment assignment (OR = 12.053). Sensitivity and specificity improved with this block as compared to the third block. The model predicted 89% of smokers, 54% of abstainers, and the overall model had sensitivity of 79%. This model was more specific in predicting abstaining participants. The impact of the interaction on the model increased the level of significance on employment and hospital stay and decreased the treatment group significance (Table 72). Hosmer and Lemeshow (2000), suggest examining whether the significance of coefficients increase or decrease as variables are added to a model.

Table 70 Sites and confounding variables at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Site[§]			3.223	2	.200			
Site 1	1.019	0.615	2.747	1	.097	2.771	0.830	9.252
Site 2	1.536	1.338	1.318	1	.251	4.645	0.338	63.897
Employment	-1.548	0.668	5.377	1	.020	0.213	0.057	0.787
Hospital Stay	0.187	0.065	8.151	1	.004	1.206	1.060	1.371
Constant	-1.999	0.647	9.550	1	.002	0.135		

Model 2: $\chi_{LR}2 = 20.178$, df = 4, p = .000*; Block 2: $\chi_{LR}2 = 3.412$, df = , p = .182
Block 1: -2 Log likelihood = 76.842; Nagelkerke $R^2 = .319$; $\chi_{HL}2 = 10.157$, df = 8, p = .254
[§]Site A served as the reference

Table 71 Sites, treatment assignment, and confounding variables at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Site[§]			2.182	2	.336			
Site 1	0.924	0.631	2.144	1	.143	2.519	0.732	8.674
Site 2	0.861	1.397	0.380	1	.538	2.366	0.153	36.538
Employment	-2.077	0.762	7.441	1	.006	0.125	0.028	0.557
Hospital Stay	0.190	0.071	7.204	1	.007	1.209	1.053	1.389
Treatment Group(1)	1.244	0.663	3.523	1	.061	3.469	0.946	12.715
Constant	-2.402	0.720	11.115	1	.001	0.091		

Model 3: $\chi_{LR}2 = 23.917$, df = 5, p = .000*; Block 3: $\chi_{LR}2 = 3.739$, df = 1, p = .053
Block 3: -2 Log likelihood = 73.103; Nagelkerke $R^2 = .369$; $\chi_{HL}2 = 11.027$, df = 8, p = 0.200
[§] Site A served as the reference

Table 72 Interaction of treatment assignment and sites with confounding variables at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Employment	-2.009	0.771	6.785	1	.009	0.134	0.030	0.608
Hospital Stay	0.179	0.073	5.984	1	.014	1.196	1.036	1.38
Site[§]			3.072	2	.215			
Site 1	2.020	1.169	2.989	1	.084	7.540	0.763	74.48
Site 2	0.455	1.426	0.102	1	.750	1.576	0.096	25.76
Treatment Group(1)	2.489	1.256	3.927	1	.048	12.053	1.027	141.38
Treatment Grp x Site[§]			1.617	1	.204			
Treatment Group Grp(1) x Site 1	-1.807	1.421	1.617	1	.204	0.164	0.010	2.66
Constant	-3.166	1.099	8.306	1	.004	0.042		

Model 4: $\chi_{LR}2 = 25.708$, df = 6, p = .000*; Block 4: $\chi_{LR}2 = 1.791$, df = 1, p = .181
Block 4: -2 Log likelihood = 71.312; Nagelkerke $R^2 = .393$; $\chi_{HL}2 = 1.423$, df = 8, p = .994

The recruitment cohorts were examined to determine whether there was a relationship with smoking point prevalence at 12-weeks. The univariate logistic regression model was not significant ($\chi_{LR}2 = 3.599$, df = 1, p = .058). The fit of the model was not problematic according to the Hosmer and Lemeshow test for goodness of fit ($\chi_{HL}2$ of .000, df = 1, p = 1.00). The coefficient for cohort had a trend for significance (Table 73). The sensitivity and specificity of the model were poor. This step could not predict abstaining participants, although 100% of the smoking participants were predicted. This univariate logistic regression test suggested there was no direct relationship between recruitment cohort and smoking behavior measured 12 weeks following hospital discharge. Further analysis was conducted to evaluate the affect on treatment if cohort was controlled in the model. When the treatment assignment was entered within the next block, the model was significant, but the block was not (Model 2**:** $\chi_{LR}2 = 7.089$, df = 2, p = .029; Block 2: $\chi_{LR}2 = 3.490$, df = 1, p = .062) (Refer to Table 74). The coefficients were not significant in the model. The Hosmer and Lemeshow test for goodness of fit ($\chi_{HL}2$ of 7.259, df = 4, p = .123) was not significant. The sensitivity (71%) and specificity (smokers 82% and abstainers 46%) were improved over the last model. One must keep in mind this step was not significant, although the p-value suggested a trend.

A third step or block was entered containing an interaction term of recruitment cohort and treatment assignment in a similar manner conducted with the site analyses. Both the model and block were not significant (Model 3: $\chi_{HL}2$ of 7.245, df = 3, p = .064; Block 3 $\chi_{HL}2$ of 0.156, df = 1, p = .693) (Refer to Table 75). The coefficients were not significant. Sensitivity and specificity did not change from the previous block. The coefficients were not significant.

Again, the confounding variables were controlled in this next set of analyses with respect to recruitment cohort and smoking status at 12-weeks after discharge. The model and block for these variables were significant (Model and Block 1: $\chi_{LR}2$ = 16.766, df = 2, p = .000). Employment and hospital length of stay were significant coefficients. The Hosmer and Lemeshow goodness of fit statistic was not significant ($\chi_{HL}2$ of 1.889, df = 6, p = .930). Sensitivity of this particular model was 79%. Specificity to predict smoking participants was 96% and abstainers 42%. It was an improvement over the univariate test of recruitment year.

Cohort was added in the second block in a similar manner as the analysis described for Site. The block was not significant for the entry of the recruitment cohort (Block 2: $\chi_{LR}2$ = 0.480, df = 1, p = .488). The Hosmer and Lemeshow goodness of fit statistic was not significant ($\chi_{HL}2$ of 7.721, df = 8, p = .461) (Refer to Table 76). The coefficients for employment (p = .007), and hospital length of stay (p = .011) were significant. This model did not provide an improvement in sensitivity (overall prediction was 76%), specificity for smoking participants (91%) and abstainers (42%). Recruitment cohort was associated with smoking status. The sensitivity and specificity of this model was not an improvement.

In a third block, treatment assignment was added to the model. Both the block and model were significant (Model 3: $\chi_{LR}2$ = 22.039, df = 4, p = .000; Block 3: $\chi_{LR}2$ = 4.793, df = 1, p = .029). The treatment assignment coefficient was significant (p = .035) with an odds ratio of 3.726 (Refer to Table 77). Therefore, in this model, special intervention participants were 3.73 times more likely to be abstinent 12-weeks following hospital discharge than usual care participants. Current employment and hospital length of stay were also significant coefficients. There was no change in the overall sensitivity and specificity of this model. A fourth block examined an

interaction term between the treatment assignment and recruitment cohort. This block was not significant (Model 4: $\chi_{LR}2 = 22.329$, df = 5, p = .000*; Block 4: $\chi_{LR}2 = 0.290$, df = 1, p = .590) (Refer to Table 78).

Table 73 Univariate logistic regression of cohorts at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Cohort	-0.949	0.511	3.446	1	.063	0.387	0.142	1.054
Constant	-0.405	0.323	1.578	1	.209	0.667		

Model 1: $\chi_{LR}2 = 3.599$, df = 1, p = .058; Block 1: $\chi_{LR}2 = 3.599$, df = 1, p = .058
Block 1: -2 Log likelihood = 93.420; Nagelkerke $R^2 = .063$

Table 74 Cohorts and treatment assignment at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Cohort 1	-0.956	0.523	3.343	1	.067	0.384	0.138	1.071
Treatment Group(1)	0.956	0.523	3.343	1	.067	2.602	0.933	7.252
Constant	-0.931	0.448	4.320	1	.038	0.394		

Model 2: $\chi_{LR}2 = 7.089$, df = 2, p = .029*; Block 2: $\chi_{LR}2 = 3.490$, df = 1, p = .062
Block 2: -2 Log likelihood = 89.930; Nagelkerke $R^2 = .121$; $\chi_{HL}2 = 0.157$, df = 2, p = .924

Table 75 Interaction of treatment assignment and cohorts at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Cohort 1	-0.705	0.815	0.749	1	.387	0.494	0.100	2.439
Treatment Group(1)	1.125	0.680	2.737	1	.098	3.080	0.812	11.677
Treatment Grp x Cohort	-0.420	1.061	0.157	1	.692	0.657	0.082	5.258
Constant	-1.030	0.521	3.906	1	.048	0.357		

Model 3: $\chi_{LR}2 = 7.245$, df = 3, p = .064; Block 3: $\chi_{LR}2 = 0.156$, df = 1, p = .693
Block 3: -2 Log likelihood = 89.774; Nagelkerke $R^2 = .124$; $\chi_{HL}2 = 0.000$, df = 1, p = 1.000

Table 76 Cohorts and treatment confounding variables at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Employment	-1.674	0.655	6.525	1	.011	0.188	0.052	0.677
Hospital Stay	0.194	0.072	7.330	1	.007	1.214	1.055	1.397
Cohort	-0.398	0.575	0.479	1	.489	0.672	0.218	2.072
Constant	-1.161	0.536	4.684	1	.030	0.313		

Model 2: $\chi_{LR}2 = 17.246$, df = 3, p = .001*; Block 2: $\chi_{LR}2 = 0.480$, df = 1, p = .488
Block 2: -2 Log likelihood = 79.774; Nagelkerke $R^2 = .277$; $\chi_{HL}2 = 7.721$, df = 8, p = .461

Table 77 Cohorts, treatment assignment, & confounding variables at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Employment	-2.141	0.734	8.505	1	.004	0.118	0.028	0.496
Hospital Stay	0.192	0.076	6.438	1	.011	1.212	1.045	1.405
Cohort	-0.374	0.592	0.398	1	.528	0.688	0.216	2.197
Treatment Group(1)	1.315	0.624	4.437	1	.035	3.726	1.096	12.670
Constant	-1.681	0.623	4.278	1	.007	0.186		

Model 3: $\chi_{LR}2 = 22.039$, df = 4, p = .000*; Block 3: $\chi_{LR}2 = 4.793$, df = 1, p = .029*
Block 3: -2 Log likelihood = 74.980; Nagelkerke $R^2 = .344$; $\chi_{HL}2 = 7.064$, df = 8, p = .530

Table 78 Interaction of treatment assignment and cohorts with confounding variables at 12-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Employment	-2.146	0.731	8.612	1	.003	0.117	0.028	0.490
Hospital Stay	0.196	0.077	6.421	1	.011	1.216	1.045	1.415
Cohort	-0.017	0.877	0.000	1	.984	0.983	0.176	5.486
Treatment Group(1)	1.569	0.789	3.956	1	.047	4.800	1.023	22.524
Treatment Grp x Cohort	-0.633	1.174	0.291	1	.590	.531	0.053	5.299
Constant	-1.837	0.701	6.871	1	.009	0.159		

Model 4: $\chi_{LR}2 = 22.329$, df = 5, p = .000*; Block 4: $\chi_{LR}2 = 0.290$, df = 1, p = .590
Block 4: -2 Log likelihood = 74.690; Nagelkerke $R^2 = .348$; $\chi_{HL}2 = 2.700$, df = 8, p = .952

The variables were entered into similar logistic regression models with smoking behavior at 24-weeks as the dependent variable. These findings differed from the 12-week data previously presented. Tables 79 through 81 present the data for the examination of site with the dependent variable at 24-weeks. The sensitivity (overall prediction 73%) and specificity (prediction of smokers 98% and abstainers 9%) was poor. The univariate model with site was not significant ($p = .086$). When treatment assignment was added, the findings were similar to the unadjusted model presented in the primary aims. The model and block for the addition of the treatment variable was significant as noted on Table 80. The Hosmer and Lemeshow good of fit statistic was not significant ($\chi_{HL}2$ of 2.303, $df = 2$, $p = .316$). The sensitivity and specificity were no different then the first block with only the site variable tested. When an interaction variable was tested in a similar approach to the 12-week data, the block was not significant (Table 81) and the treatment assignment coefficient did not change significantly. Site was not associated with smoking status at 24-weeks as in the case of the 12-week results.

Tables 82 to 84 provide results of the examination of site and treatment assignment while controlling for the confounding variables of employment and hospital length of stay. Site was not associated with smoking behavior when confounding variables were controlled. The odds ratios presented for the treatment assignment through the analyses were similar to the odds ratio for the adjusted logistic model presented for the primary aim data at 24-weeks. Overall sensitivity throughout the analysis while controlling for confounding variables differed slightly, but was similar to the adjusted logistic regression presented for the primary aim results. The data presented in Tables 82 through 84 suggested that site does not impact smoking behavior or treatment assignment 24-weeks following hospital discharge.

Recruitment year was not significant in the univariate model tested with the 24-week dependent variable (Table 85), although the level of significance suggested a potential trend for a relationship. The model was not predictive of abstainers and only predicted smoking participants. As with the examination of recruitment site, findings for the cohort at 24-weeks following discharge were similar when the model was tested with the treatment group and confounding variables (Tables 86 and 90). Recruitment cohort was not related to the dependent variable and did not suggest a modifying affect on the treatment assignment. Sensitivity and specificity of the univariate model was poor. Sensitivity did not improve until the confounding variables were controlled and the treatment assignment coefficient was added (overall prediction 78%, specificity for smokers 93%, abstainers 41%).

In summary, Site B was related to smoking status at 12-weeks following hospitalization and there may be a modifying affect with respect to treatment assignment and recruitment Site B. By 24 weeks following hospitalization, this relationship of site to smoking is not present and there is no evidence suggesting that Site B has an affect upon treatment assignment 24-weeks following hospitalization. The recruitment cohort did not have a relationship to smoking status at 12 or 24 weeks following hospitalization. When confounding variables were controlled, the affect of these variables upon treatment assignment was similar to the adjusted models presented in the primary aim results.

The final analyses for this exploratory aim examined age since it was not controlled in this study with respect treatment assignment. As noted in Chapter 2, this older individuals may have more success with smoking cessation than younger adults. Therefore, age was examined for a relationship with smoking behavior at 12-weeks and 24-weeks. A univariate regression model was used to test for the association. The univariate model was not significant at 12-weeks

following hospital discharge (Model 1: $\chi_{LR}2$= 1.576, df = 1, p = .209). The coefficient for age was not significant (p = .217). Treatment assignment was added in the next block, which was not significant (Model 2: $\chi_{LR}2$= 4.548, df = 2, p = .103; Block 2: $\chi_{LR}2$ = 2.971, df = 1, p = .085). The treatment assignment was not significant (p = .090) and had an odds ratio of 2.405. This finding is close to the unadjusted model presented in the primary aim results. A univariate model was examined for the 24-week smoking status and age. The univariate model was not significant at 24-weeks following hospital discharge (Model 1: $\chi_{LR}2$= 2.103, df = 1, p = .147). The coefficient for age was not significant (p = .158). Treatment assignment was added in the next block, which was significant (Model 2: $\chi_{LR}2$= 5.841, df = 2, p = .016; Block 2: $\chi_{LR}2$ = 7.944, df = 1, p = .019). The treatment assignment was significant (p = .020) and had an odds ratio of 3.672. These findings were similar to those presented for the unadjusted model at 24-weeks in the primary aim results. The odds ratio is slightly smaller than the odds ratio reported in the primary aim results. Age was not related to smoking behavior in this study and did not impact the treatment assignment.

Table 79 Univariate logistic regression for sites and 24-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Site[§]			4.555	2	.103			
Site 1	0.918	0.544	2.849	1	.091	2.503	0.862	7.264
Site 2	2.181	1.294	2.840	1	.092	8.857	0.701	111.937
Constant	-1.488	.418	12.645	1	.000	0.226		

Model 1: $\chi_{LR}2 = 4.916$, df = 2, p = .086; Block 1: $\chi_{LR}2 = 4.916$, df = 2, p = .086
Block 1: -2 Log likelihood = 87.218; Nagelkerke $R^2 = .089$

§ Site A served as the reference

Table 80 Sites and treatment assignment at 24-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Site[§]			2.985	2	.225			
Site 1	0.848	0.563	2.270	1	.132	2.334	0.775	7.034
Site 2	1.578	1.317	1.436	1	.231	4.844	0.367	63.996
Treatment Grp(1)	1.251	0.571	4.805	1	.028	3.494	1.142	10.694
Constant	-2.136	0.555	14.788	1	.000	0.118		

Model 2: $\chi_{LR}2 = 10.056$, df = 3, p = .018*; Block 2: $\chi_{LR}2 = 5.140$, df = 1, p = .023*
Block 2: -2 Log likelihood = 82.077; Nagelkerke $R^2 = .175$; $\chi_{HL}2 = 2.303$, df = 2, p = .316
§ Site A served as the reference

Table 81 Sites, treatment assignment and interaction at 24-week outcome

	b	SE(b)	Wald	Df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Site[§]			4.355	2	.113			
Site 1	2.169	1.154	3.535	1	.060	8.750	0.912	83.949
Site 2	1.204	1.329	.821	1	.365	3.333	0.246	45.109
Treatment Group(1)	2.534	1.146	4.884	1	.027	12.600	1.332	119.181
Treatment Grp x Site			2.155	1	.142			
Treatment Grp(1) x Site 1	-1.977	1.347	2.155	1	.142	0.139	0.010	1.940
Constant	-3.045	1.024	8.848	1	.003	0.048		

Model 3: $\chi_{LR}2 = 12.547$, df = 4, p = .014*; Block 3: $\chi_{LR}2 = 2.491$, df = 1, p = .114
Block 2: -2 Log likelihood = 79.586; Nagelkerke $R^2 = .216$; $\chi_{HL}2 = 0.00$, df = 2, p = 1.000
§ Site A served as the reference

Table 82 Sites and confounding variables at 24-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI for Odds Ratio	
							Lower	Upper
Employment	-1.210	0.666	3.308	1	.069	0.298	0.081	1.099
Hospital Stay	0.180	0.065	7.682	1	.006	1.198	1.054	1.361
Site[§]			2.469	2	.291			
Site 1	0.841	0.624	1.817	1	.178	2.319	0.683	7.879
Site 2	1.572	1.340	1.377	1	.241	4.818	0.349	66.619
Constant	-2.091	0.655	10.184	1	.001	0.124		

Model 2: $\chi_{LR}2 = 16.732$, df = 4, p = .002*; Block 2: $\chi_{LR}2 = 2.604$, df = 2, p = .272
Block 2: -2 Log likelihood = 75.402; Nagelkerke $R^2 = .280$; $\chi_{HL}2 = 11.174$, df = 7, p = .131
§ Site A served as the reference

Table 83 Sites, treatment assignment & confounding variables at 24-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Employment	-1.854	.760	5.954	1	.015	.157	.035	.694
Hospital Stay	.186	.073	6.438	1	.011	1.204	1.043	1.391
Site[§]			1.220	2	.543			
Site 1	.711	.651	1.194	1	.275	2.036	.569	7.290
Site 2	.683	1.399	.238	1	.625	1.979	.128	30.705
Treatment Group (1)	1.642	.685	5.753	1	.016	5.166	1.350	19.767
Constant	-2.671	.761	12.331	1	.000	.069		

Model 3: $\chi_{LR}2 = 23.075$, df = 5, p = .000*; Block 3: $\chi_{LR}2 = 6.343$, df = 1, p = .012*
Block 3: -2 Log likelihood = 69.058; Nagelkerke $R^2 = .371$; $\chi_{HL}2 = 10.566$, df = 8, p = .228
§ Site A served as the reference

Table 84 Interaction of treatment assignment and sites with confounding variables at 24-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Employment	-1.808	0.768	5.535	1	.019	0.164	0.036	0.740
Hospital Stay	0.178	0.075	5.682	1	.017	1.195	1.032	1.384
Site[§]			1.651	2	.438			
Site 1	1.490	1.186	1.576	1	.209	4.436	0.434	45.388
Site 2	0.432	1.425	0.092	1	.762	1.541	0.094	25.178
Treatment Group (1)	2.479	1.252	3.920	1	.048	11.929	1.025	138.78
Treatment Grp x Site			0.730	1	.393			
Treatment Grp (1) x Site 1	-1.230	1.439	0.730	1	.393	0.292	0.017	4.909
Constant	-3.202	1.099	8.493	1	.004	0.041		

Model 4: $\chi_{LR}2 = 23.857$, df = 6, p = .001*; Block 4: $\chi_{LR}2 = 0.782$, df = 1, p = .377
Block 4: -2 Log likelihood = 68.277; Nagelkerke $R^2 = .382$; $\chi_{HL}2 = 5.142$, df = 7, p = .643
§ Site A served as the reference

Table 85 Univariate logistic regression of cohorts and 24-week outcome

	B	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Cohort	-1.018	0.532	3.657	1	.056	0.361	0.127	1.026
Constant	-0.470	0.329	2.039	1	.153	0.625		

Model 1: $\chi_{LR}2 = 3.857$, df = 1, p = .050; Block 1: 3.857, df = 1, p = .050
Block 1: -2 Log likelihood = 88.276; Nagelkerke $R^2 = .070$

Table 86 Cohorts and treatment assignment at 24-week outcome

	b	SE(b)	Wald	Df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Cohort	-1.059	0.558	3.599	1	.058	0.347	0.116	1.036
Treatment Group(1)	1.472	.577	6.520	1	.011	4.358	1.408	13.492
Constant	-1.142	.496	5.307	1	.021	.319		

Model 2: $\chi_{LR}2 = 10.720$, df = 2, p = .005*; Block 2: $\chi_{LR}2 = 6.862$, df = 1, p = .009*
Block 2: -2 Log likelihood = 81.414; Nagelkerke $R^2 = .186$; $\chi_{HL}2 = 0.058$, df = 2, p = .971

Table 87 Cohorts, treatment assignment, and interaction at 24-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Cohort	-0.875	0.934	0.879	1	.349	0.417	0.067	2.599
Treatment Group (1)	1.522	0.720	4.469	1	.035	4.583	1.117	18.803
Treatment Grp (1) x Cohort	-0.281	1.162	0.058	1	.809	0.755	0.077	7.371
Constant	-1.322	0.563	5.517	1	.019	0.267		

Model 3: $\chi_{LR}2 = 10.778$, df = 3, p = .013*; Block 3: $\chi_{LR}2 = 0.058$, df = 1, p = .810
Block 3: -2 Log likelihood = 81.356; Nagelkerke $R^2 = .187$; $\chi_{HL}2 = 0.000$, df = 2, p = 1.000

Table 88 Cohorts and confounding variables at 24-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Employment	-1.286	0.656	3.844	1	.050	0.276	0.076	1.000
Hospital Stay	0.188	0.072	6.870	1	.009	1.206	1.048	1.388
Cohort	-0.560	0.592	0.895	1	.344	0.571	0.179	1.822
Constant	-1.309	0.541	5.845	1	.016	0.270		

Model 2: $\chi_{LR}2 = 15.032$, df = 3, p = .002*; Block 2: $\chi_{LR}2 = 0.904$, df = 1, p = .342
Block 2: -2 Log likelihood = 77.101; Nagelkerke $R^2 = .254$; $\chi_{HL}2$ of 13.015, df = 7, p = .072

Table 89 Cohorts, treatment assignment, & confounding variables at 24-week outcome

	b	SE(b)	Wald	df	P	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Employment	-1.851	0.744	6.188	1	.013	0.157	0.037	0.675
Hospital Stay	0.187	0.077	5.843	1	.016	1.206	1.036	1.403
Cohort	-0.561	0.623	0.809	1	.368	0.571	0.168	1.936
Treatment Group(1)	1.710	0.658	6.748	1	.009	5.528	1.522	20.086
Constant	-2.028	0.662	9.383	1	.002	.132		

Model 3: $\chi_{LR}2 = 22.640$, df = 4, p = .000*; Block 3: $\chi_{LR}2 = 7.608$, df = 1, p = .006*
Block 3: -2 Log likelihood = 69.493; Nagelkerke $R^2 = .365$; $\chi_{HL}2$ of 4.972, df = 8, p = .761

Table 90 Cohorts, treatment assignment, confounding variables and interaction at 24-week outcome

	b	SE(b)	Wald	df	p	Odds Ratio	95% CI Odds Ratio	
							Lower	Upper
Employment	-1.854	0.742	6.246	1	.012	0.157	0.037	0.670
Hospital Stay	0.190	0.079	5.826	1	.016	1.210	1.036	1.412
Cohort	-0.253	0.982	0.066	1	.797	0.776	0.113	5.323
Treatment Group(1)	1.897	0.819	5.361	1	.021	6.668	1.338	33.231
Treatment Grp (1) x Cohort	-0.497	1.258	0.156	1	.693	0.608	0.052	7.153
Constant	-2.152	0.746	8.308	1	.004	0.116		

Model 4: $\chi_{LR}2 = 22.765$, df = 5, p = .000*; Block 4: $\chi_{LR}2 = 0.155$, df = 1, p = .694
Block 4: -2 Log likelihood = 69.338; Nagelkerke $R^2 = .367$; $\chi_{HL}2$ of 3.942, df = 7, p = .786

6. CHAPTER 6

6.1. SUMMARY AND DISCUSSION

The primary aim of this study was to examine the efficacy of a 12-week nurse delivered intervention program to hospitalized smokers to support tobacco abstinence and the prevention of relapse. Two additional secondary aims were proposed. The first secondary aim examined relationships between the conceptual variables of Self-efficacy Theory and the tobacco abstinence behavior. The second part of the secondary aim examined treatment related variables, such as smoking lapse, treatment adherence, self-efficacy, and smoking point prevalence as a measure of smoking behavior. An exploratory aim to examine potential cohort and recruitment site differences was added due to the 3-year length of recruitment and use of three hospitals for recruitment. Age was examined as a potential covariate or confounding variable.

A preliminary study provided important information pertinent to the selection of the conceptual framework, feasibility of follow-up beyond hospitalization, and guidance for recruitment efforts. The conceptual framework of Self-efficacy theory was selected to drive this current study due to the relationship of self-efficacy in the preliminary study with smoking cessation attempts and motivation to quit smoking. Furthermore, the preliminary study had difficulty recruiting female participants, which guided the current study to control for gender in the randomization process for treatment assignment. Follow-up methods during the preliminary study revealed a potential for loss of participants as the study moved beyond hospital discharge, which required a different mechanism in gathering addresses, secondary contacts, and telephone

numbers for follow-up contact. Recruitment efforts were difficult during the preliminary study, which identified that only 9% of all patients approached to participate in the preliminary signed consents for participation. This information was useful when forecasting recruitment efforts in the early part of the current study. Predictors of smoking cessation attempt from the preliminary study were included for measurement in this study, such as employment status. The preliminary study was also useful in organizing and using data collection tools, which were later modified for the current study.

6.1.1. Summary of the primary aim results

This study tested two directional hypotheses for the primary aim. The first hypothesis proposed treatment would be effective in promoting smoking abstinence behavior at the 12-week follow-up visit. The univariate logistic regression analysis of hypothesis 1a suggested the special intervention group did differ in abstinence response to the usual care group when measured 12 weeks following hospital discharge. The adjusted logistic regression controlled for baseline confounding variables was also significant and was a better predictor of smoking status than the univariate test of the treatment groups at 12-week follow-up. Current employment and longer hospital admission were independently related to smoking behavior. Controlling for these variables increased the significant effect of the treatment.

The second hypothesis proposed there would be a treatment effect of the intervention at the 24-week follow-up measurement of smoking behavior. Similar to the first hypothesis, the results of the univariate test were significant and indicated a treatment effect. The adjusted logistic regression test controlled for the confounding variables of current employment and length of hospital admission, which improved the prediction of the model and the significance of

the treatment. Both hypotheses were accepted. Given the small sample size, this intervention pilot study with hospitalized smokers is limited in its generalizability. Additional considerations are presented for future research with smokers in a hospital setting.

The results of the directional hypotheses suggested the study intervention was effective in promoting and maintaining tobacco abstinent behavior of hospitalized smokers 3 and 6 months following hospital discharge. Taylor et al. (1996) reported over 50% of the intervention participants relapsed within the first 90 days following hospital discharge. In an earlier study, Taylor et al. (1990) noted early relapse patterns within three weeks of discharge for some participants. In order to ascertain the effectiveness of the intervention, early assessment of smoking behavior was needed for this pilot study. Outcome measurements focused upon an immediate measurement following intervention delivery and a distanced time point following hospitalization. Therefore, the 12-week measurement in this study provided an indication of the effect of the intervention immediately following delivery of the last scheduled intervention before participants would be on their own in managing their smoking abstinence.

A small number of studies of hospitalized smokers examined abstinence rates at 3 months. The abstinence rates found by this study at the 12-week measurement were slightly higher than those found by Stevens, et al. (1993). In that study, measurements at 3 months indicated that the special intervention had a reported abstinent rate of 20.5% and the usual care 13.7%. The current study had 12-week abstinent rates of 40% for special intervention and 20% for usual care. The abstinent rates 24 weeks after discharge were 42% for special intervention and 15% for usual care. Taylor et al. (1996) used a hospitalized sample, which was not limited to cardiovascular diagnoses and reported self-reported abstinence rates of 48% for special intervention and 30% for usual care. Wewers et al. (1994) did not find significant results

between the experimental group and usual care group, but abstinent rates by diagnosis were reported. The experimental group among cardiovascular patients had an abstinent rate of 40% while usual care had an abstinent rate of 8.3% for those patients with oncological related admissions, experimental group abstinence was 64% and usual care was 50%. General surgery patients had the lowest abstinence rates with the experimental group reported at 7.7% and the usual care abstinence rate was 13.3%. When these groups were collapsed, experimental group abstinence was 35.8% and usual care was 25.6%. The abstinence rates from this current study fall between the rates listed by these studies for abstinence measures completed between 6 weeks and 3 months following hospital discharge for both the intervention and control conditions. All participants were provided enhanced usual care. Only one-fifth of the usual care group was abstinent 12 weeks after hospital discharge.

Unlike Rigotti et al. (1997), this study did not obtain self-reported abstinence rates 1 month following discharge. Rigotti et al. (1997) reported self-reported abstinence rates of 28.9% for the intervention group and 18.9% for the usual care group. Obtaining a measurement between 1 week and 1 month following discharge would have assisted in determining those participants who relapsed immediately following hospitalization. The mean days to lapse for the usual care group was 54.4 days. Special intervention subjects had a mean of 77.2 days. Thirty percent of the usual care subjects lapsed within 1 week or less after hospital discharge. Twenty-five percent of the special intervention subjects lapsed within this timeframe. Future studies should consider obtaining a measurement immediately following discharge if a relapse management program will be ongoing following this period. This measurement may help to identify recalcitrant smokers in the sample and further explain the process of relapse that occurs for this group of smokers.

Outcome measurements at 6 and 12 months following hospitalization have been more predominant among previous smoking cessation studies in this population of smokers (Dornelas et al., 2000; Rigotti et al., 1997; Simon et al., 1997; Taylor et al., 1996). In particular, selected studies have focused on reporting 12-month point prevalence rates or continuous abstinence rates (Bolman, de Vries, & van Breukelen, 2002; Froelicher, Sohn, Max, & Bacchetti, 2004b; Hajek, Taylor, & Mills, 2002; Hennrikus et al., 2005; Miller et al., 1997b; Reid, Pipe, Higginson, Johnson, D'Angelo, Cooke, & Dafoe, 2003; Simon et al., 1997; Sivarajan Froelicher et al., 2004). These studies tested the long-term effects of the treatment offered. The 24-week measurement captured smoking behavior while both the intervention and usual care groups were on their own in managing efforts to achieve or maintain abstinence. Future research is needed with behavioral intervention programs to ascertain potential long-term effectiveness and provide for better comparison with studies that used 12-month or longer outcome assessment intervals.

For this current study, a large portion (85%) of the usual care participants relapsed at 6 months. This rate of relapse is large in comparison to most studies of hospitalized smokers, however, Rigotti et al. (1997) had self-reported abstinence rates of 17.3% for special intervention and 14% for the usual care group. Validated abstinence rates were 8.1% and 8.7%, respectively for special intervention and usual care. It is clear that the significance of the 6-month (24-week) abstinence rates was driven by the difference between these two groups and particularly by the low rate of abstinence in the usual care group.

For this study, tobacco related admissions were comprised of pulmonary, cancer, cardiovascular, as well cardiac. Johnson et al. (1999) reported findings on a cardiac sample of hospitalized smokers. The 6-month data for the special intervention group (46% abstinence) was similar to the outcome measure for the special intervention participants (42%) in this current

study. Obtaining abstinence rate of less than 50% with intervention is not acceptable clinically. Clinicians may have difficulty promoting an intervention that has less than a 50% probability of assisting a patient to quit smoking. Future research is needed towards the development of behavioral interventions that can enhance the abstinence rates beyond those reported by this study, as well other abstinence rates in the literature. Future research is also needed to identify the difficulties or barriers usual care participants have with remaining abstinent. This topic will be revisited as the discussion continues.

Similar to other studies of hospitalized smokers, confounding variables were noted that affected smoking behavior following hospitalization. Previous studies have found length of stay to be a confounding variable with this population of smokers, which required statistical analysis methods to control for confounding variables to more appropriately assess the efficacy of the intervention (Rigotti et al., 1997; Stevens et al., 1993). The effect of a longer hospital stay was a predictor variable for post hospitalization smoking status in the preliminary study (Chapter 3). This current study also found longer hospital stays impacted smoking behavior following discharge. Persons with longer hospital stays were less likely to relapse. Future studies are needed to examine the mechanism by which longer hospital admissions influence future smoking behavior. Although experiencing nicotine withdrawal in a controlled setting has the potential for explaining this observation, other factors that may contribute over the course of a longer hospital stay need to be examined.

Employment was also a confounding variable in this study. Employed subjects were more likely to be successful with smoking abstinence following hospitalization than those on disability or unemployed. Future studies need to examine this influence within this population. The preliminary study also identified employment as a variable related to smoking cessation

attempts. The nature of this variable's impact upon the cessation and abstinence process is not clear. More research is needed to examine the affect this variable has on participant cessation efforts. Secondary analyses of previous studies would be helpful, as well as in examining this variable if it was measured. Future studies need to identify if employment status is related to social support at work, lack of time for the distraction of urges, controlled smoking environments at work, and financial resources used by participants to aid their efforts of abstinence and relapse prevention.

As previously noted, the findings from this study were consistent with other studies of hospitalized smokers, but there were similarities and differences in study methods between this study and previously reported studies (Dornelas et al., 2000; Froelicher et al., 2004a; Johnson et al., 1999; Jones, Griffiths, Skirrow, & Humphris, 2001; Miller et al., 1997b; Quist-Paulsen & Gallefoss, 2003; Ratner et al., 2004; Rigotti et al., 1999; Simon et al., 2003; Stevens et al., 2000; Taylor et al., 1996). This study used a conceptual framework of Self-efficacy Theory to drive the design and intervention. Taylor et al. (1990) reported using an intervention that assessed self-efficacy for relapse with significant abstinence rates one year following hospitalization. Self-efficacy scores for situations 70% or less were targeted in a review of strategies to prevent relapse. This current study used a similar method of assessing relapse risk by using the Relapse Situation Self-efficacy Questionnaire to determine a ranked profile of relapse risk accompanied with strategies pertinent to the relapse category. Use of this information was ongoing for the intervention activities following hospital discharge for the special intervention participants. Taylor et al. (1996) reported relapse rates of 50% in intervention while this study found 52% relapse at 24 weeks.

Other studies have used self-efficacy assessment and included counseling to reinforce confidence in quitting. Johnson et al. (1999) used the Transtheoretical Model with the incorporation of Self-efficacy Theory. The intervention reinforced intervention subjects to enhance their self-efficacy with tobacco abstinence, however, the study design was quasiexperimental and lacked biological validation. Unlike this study, results were not significant at 6 months. Dornelas et al. (2000) included an assessment of self-efficacy and found it to be a predictor with treatment of smoking behavior outcomes. As noted in Chapter 4 regarding study methods, this study used two scales to assess overall self-efficacy. Johnson et al. (1999) reduced the measure of self-efficacy to an ordinal classification for analysis. Simon et al. (1997) used a multi-component intervention, which aimed to increase self-efficacy and coping skills. Results indicated a difference at 12 months, but unlike this current study, results at 6 months were not significant. While these studies used similar outcome measures, the studies used different measures to examine self-efficacy, which limits the extent of comparisons of treatment between the studies. Future studies are needed with similar measures of self-efficacy to make treatment comparisons across studies, which focus upon enhancing self-efficacy to promote tobacco abstinence.

This study also had similarities and differences from other studies with respect to the use of phone calls as a means of following participants after hospital discharge with assessment and intervention activities. The primary difference in the use of phone calls between studies has been the number and schedule of the calls. This study used eight phone calls over 12 weeks to deliver intervention activities to special intervention subjects. These phone calls were structured with an algorithm related to whether a subject lapsed or remained abstinent from tobacco, however, the intervention that followed the dialogue was individualized to the participant's responses in terms

of strategies and direction. Each strategy offered was designed to meet one of the definitions of the four sources of self-efficacy (e.g. verbal persuasion, mastery, vicarious experience, or somatic experience). Dornealas, et al. (2000) used seven telephone calls to deliver brief counseling, but the structure of the counseling was not described. In addition, the calls occurred over 26 weeks as compared to the current study. Taylor, et al. (1990) and (1996) outlined the use of phone calls for three months, which was similar to the current study, but the schedule used in those studies differed from the current study. This study used weekly calls over the first five weeks following hospital discharge that tapered to every two weeks in the second month with a final call 11 weeks from the day of discharge. This schedule placed emphasis upon the initial period following discharge as a period of high risk for relapse. Calls occurred in those other studies on a monthly basis after the first call one week following hospital discharge. There schedule targeted the initial week following discharge, but following calls were spread out. In addition, participants having difficulty with abstinence were requested to see a nurse for a face-to-face counseling session. Quist-Paulsen and Gallenfoss (2003) also used phone calls and a return to face-to-face counseling six weeks after going home, which may have coincided with follow-up visits with the physician, but the methodology was not made clear by the authors. Rigotti, et al. (1997) used only four phone calls following hospital discharge to ascertain smoking status and offer encouragement. Other studies were similar to Rigotti, et al. (1997) in using phone calls with a target duration of 10 minutes (Miller et al., 1997b; Simon et al., 1997). As noted here, many studies have used phone calls, but the authors provided limited information as to the nature of the study phone calls. For example, if the phone calls were noted as a part of the intervention, information was not provided in most cases regarding whether all of the telephone contacts were completed and the number of attempts to contact a subject in order to

complete a call (Dornelas et al., 2000; Johnson et al., 1999; Quist-Paulsen & Gallefoss, 2003; Rigotti et al., 1997; Taylor et al., 1990; Taylor et al., 1996). In this study, participants who completed more of the intervention calls were also more likely to be abstinence 12 and 24 weeks following hospital discharge.

In meta-analysis reviews of intervention studies with hospitalized smokers, follow-up contact beyond hospitalization has been a hallmark of those studies with significant treatment effects (France et al., 2001; Rigotti et al., 2003). Future studies need to provide more clarity regarding the telephone intervention/follow-up contact. Given the limited information available, one must ask if telephone calls enhance treatment in these other studies due to the contact, content, or both. This study attempted to use a structured approach with the intervention telephone calls, but allowed for individualization in order to use strategies engaging sources of self-efficacy, such mastery with goal setting, verbal persuasion, and vicarious experience.

6.1.2. Summary of results for the secondary aims

6.1.2.1. Secondary aim I. Since this study was driven by Self-efficacy Theory, the first secondary aim examined the relationship of follow-up smoking behavior with the baseline data for the conceptual variables of self-efficacy, outcome expectancy, social support for smoking abstinence, and affect (depressive symptoms) (Refer to Figure 2). Self-efficacy and outcome expectancy are proposed by Self-efficacy theory to directly influence behavior, such as smoking cessation (Bandura, 1997). Conceptual variables for the most part were not significant in predicting smoking behavior as they were tested. Controlling the alpha level to the small value of .005 may have been problematic with the findings for hypotheses 2a through 2e of the secondary aim. For logistic regression analyses, one must review the confidence intervals for the coefficient's odds ratio and other diagnostics to assist in examining the ability to detect

differences at this preset alpha level (Hosmer & Lemeshow, 2000). For the logistic regression analyses involving the baseline values for the Tobacco Abstinence Self-efficacy Scale, Relapse Situation Efficacy Questionnaire, and Perceived Therapeutic Efficacy Scale, confidence intervals were small, which is desired in a confidence interval, but it should not contain 1.000. Since the hypotheses were directional, the upper confidence interval was of interest. For all of these tests, 1.000 was not within the upper end of the interval. Diagnostics suggested the fit of the models were questionable for the Tobacco Abstinence Self-efficacy Scale and Perceived Therapeutic Efficacy Scale. Therefore, the ability to detect significance at this alpha level of .005 was difficult. According to Hosmer and Lemeshow (2000), goodness of fit tests are not powerful enough for small to moderate sample sizes less than 400. Therefore, the Tobacco Abstinence Self-efficacy Scale and Perceived Therapeutic Efficacy Scale may have been hampered by Type II error. Results for baseline social support and depressive symptoms were clearly not within reach of being accepted even at an alpha of .05 and likely did not have implications of Type II error.

The Tobacco Abstinence Self-efficacy Scale was a measure designed for this study. Since this tool was new, results from this study provide information regarding the initial use of the TASES as a measurement for self-efficacy with hospitalized smokers. The measure may have contributed to borderline and nonsignificant results in predicting smoking behavior. Initial psychometric measures reported by this study for the Tobacco Abstinence Self-efficacy Scale indicate this tool was internally consistent (Cronbach $\alpha = .99$) and stable when tested three months after baseline measures within the control subjects ($r = .79$). The test-retest examination revealed minimal change in self-efficacy. The factor analysis for the TASES suggested there might have been multicollinearity within the items of this tool. Therefore, the tool may have

some redundancy in the information measured. The test for convergent validity for the TASES with the RSEQ suggested the two tools were measuring similar information. As expected, the TASES did not correlate with the Fagerstrom Test for Nicotine Dependence. Additional testing of the TASES, particularly after modifications to minimize redundant test items, is suggested. The TASES offered the ability to examine confidence in urge control in situations related to the hospitalized smoker, which has not been available by other tools, including the Relapse Situation Efficacy Questionnaire. Measures of self-efficacy and confidence continue to demonstrate significant relationships within conceptual models examining smoking abstinence in hospitalized and comorbid populations (Froelicher et al., 2004a; Johnson et al., 1999; Ratner et al., 2004). Self-efficacy did not definitively predict smoking behavior with every tool used in this study, but self-efficacy was associated with smoking behavior as measured by the Relapse Situation Efficacy Questionnaire. This finding adds further support of the importance of self-efficacy and its measurement in the process of smoking abstinence within the population of hospitalized smokers. Further work with the Tobacco Abstinence Self-efficacy tool will help to develop this tool for use in measuring self-efficacy more effectively in this population.

The Perceived Therapeutic Efficacy Scale for Tobacco Abstinence was a modified version of the CRCD measure for therapeutic expectancy. The Perceived Therapeutic Efficacy Scale was a reliable tool. A review of the validity and further testing may be necessary. Furthermore, this instrument as constructed, examined outcome expectancy of tobacco abstinence treatment in the control of heart problems. First, the context of cardiac disease may not have been relevant to all subjects. This study attempted to provide intervention across diagnoses related and not related to tobacco use. On the other hand, the high mean scores suggested the smokers in this sample were sensitive to and knowledgeable about tobacco effects

on health. Future use of this version of the instrument is recommended after further examination of the validity is conducted. In addition, future studies of hospitalized smokers with the tool may need outcome expectancy measures relevant to the health status of the participant.

Baseline Relapse Situation Efficacy Questionnaire scores were predictive of smoking behavior at 12-week follow-up. These findings were supportive of the conceptual underpinnings of this study and design of this measurement tool (Bandura, 1997; Gwaltney et al., 2001). According to Bandura (1997), self-efficacy is better at predicting behavior proximal to the assessment of self-efficacy. Since smokers are more apt to relapse within the first 30 to 90 days of smoking cessation than 6 months later, the trends in the predictive models for the 12-week follow-up of smoking behavior were supportive of the theory. One must also keep in mind that self-efficacy can be influenced by a variety of other variables. Self-efficacy is temporal in nature in that it can change over time due to other influencing factors, such as sources of self-efficacy and ongoing events related to the behavior. This tool was not used to determine if the relapse situations ranked for profiling were related to the situations that led to relapse. Future studies of the self-efficacy intervention need to examine the specificity of this self-efficacy tool as a predictor of relapse events, as observed by the developers of this tool (Gwaltney et al., 2001).

Social support was related to outcome smoking behavior in the preliminary study. Previous studies have identified social support as a key variable necessary to the tobacco abstinence process (Murray et al., 1995; Roski et al., 1996), although social support was not found to be a predictor of smoking abstinence in patients who received coronary artery bypass grafts (Rigotti et al., 1994). Johnson, et al. (1999) measured social support with two questions, but did not report social support as a significant predictor of smoking behavior. Ratner et al. (2004) used similar measures of social support and had a lack of significance with respect to this

variable of interest. The method of assessing perceived social support in this study was also not a standardized instrument, which may have contributed to the lack of significant findings. Future studies with similar interventions require a more sensitive measure of perceived social support for tobacco abstinence promotion. Due to the lack of a standardized measure of social support for tobacco abstinence, research is needed in the development of such a measure. Qualitative studies may be necessary to explore the nature of this concept with smoking abstinence in the hospitalized smoker and/or smoker with chronic disorders. Several intervention subjects during the course of the intervention delivery discussed concerns for disclosure of study participation since their smoking status was not known either at home or at work. They shared concerns regarding their lack of social support in these situations. Additionally, this variable tested the ability of social support to impact directly upon behavior. If social support is a source of self-efficacy through verbal persuasion and modeling, then appropriate analyses are needed to examine this path of influence through self-efficacy in influencing smoking behavior outcomes (Bandura, 1997).

Social support has the potential of requiring greater depth in measurement than was conducted in this study and previous studies of hospitalized smokers. For example, the network of support at work for abstinence was not accounted for in the current study or previous studies. Findings from this research suggested employment was related to abstinence. It is not clear if employment provided distraction or an interruption from craving cues and/or if social support networks were also available and helpful to subjects in their abstinence efforts. At least one subject within the intervention group commented upon the social support from a supervisor at work. Another subject identified concerns for disclosure of their former smoking status since co-

workers were not aware the participant had been smoking prior to study entry. These points were brought forth during the intervention process and were not measured as part of this study. Future research is needed to identify and measure social support for this population of smokers.

The use of the Profile of Mood States to test the relationship of affect, specifically depressive symptoms, may not have been adequate for this population. During the course of administration of the instruments, a few participants asked for definitions of words they did not recognize like "muddled." The POMS has been used previously with smokers receiving cancer therapy (Gritz et al., 1999a). Future secondary analyses may be needed to assess whether this instrument was detecting difficulties patients were having with their illness, such as fatigue and depressive symptoms. Studies of hospitalized smokers have not examined relationships between affect, nicotine withdrawal symptoms, smoking behavior, and manifestations of the diseases experienced by hospitalized smokers. Participants in the study with severe illnesses requiring higher acuity of care or diagnosed with terminal conditions were outlier cases with respect to fatigue, anger, depression, and confusion on the POMS. This finding suggested the need to examine whether the results for the POMS were also representative of the affect of subjects' disease state, as well as smoking behavior. This examination failed to demonstrate a relationship between depressive symptoms and smoking behavior. Further analysis is needed to examine whether depressive symptoms mediate behavior through self-efficacy as expected by the theory (Bandura, 1997).

6.1.2.2. Secondary aim II. The second secondary aim tested treatment related variables. Survival analysis results were not suggestive that the treatment was effective in promoting relapse prevention by preventing smoking lapses, but one must not ignore the finding that more UC subjects lapsed than SI subjects. Of those SI subjects who were abstinent at 12 and 24-weeks

following hospital discharge, only two SI participants experienced lapses. Those subjects did not proceed to a relapsed smoking state, but were successful in attempting to regain abstinence. Further analysis is needed with respect to the concept of lapse and relapse in this sample, particularly since the treatment assignment and treatment adherence were predictive of smoking behavior.

Treatment adherence in this sample population was not different with respect to average rates of adherence in other populations (Dunbar-Jacob, Schlenk, & Caruthers, 2002a). Slightly less than 50% of the subjects adhered to the intervention treatment. The frequency distribution as depicted by histogram had a "j" shape often noted for rates of treatment adherence (Dunbar-Jacob et al., 1998b). This finding suggests a similarity to studies that have examined treatment adherence with other types of treatments, such as medication taking. Adherence data is rarely reported for smoking cessation treatment research as it has been with medication, diet, and exercise adherence. As noted in Chapter 2, Kamarck and Lichtenstein (1988) reported on adherence rates of behavioral intervention for smoking. Treatment adherence has been examined with regard to medication adherence within a smoking cessation study (Killen et al., 1997). Killen et al. (1997) found at the 6-month follow-up visit that adherence to the use of nicotine patch in the treatment group was similar to that of the group assigned to placebo gum; however, a study limitation was the use of self-report measures for treatment adherence. Furthermore, Killen et al. (1997) noted the significance of effort, commitment, and expectancy in changing health behaviors. This study is the first to describe and examine treatment adherence with a population of hospitalized smokers receiving a behavioral intervention. The findings from this research raise various questions that need to be addressed in future research. Is the adherence rate observed in this study consistent with future studies? Can adherence intervention strategies

improve adherence to a tobacco abstinence intervention? What is the effect of improving treatment adherence upon abstinence outcomes? Can treatment adherence strategies target smoking behavior and other treatment management required by this hospital population?

The small sample size of this study limits the extent to which predictors of treatment adherence from the current study can be examined as part of a secondary analysis. Findings from this study suggest that the behavioral intervention may have a dose response. Participants who had higher rates of adherence were more likely to be abstinent at the follow-up visits 12 and 24 weeks following hospital discharge. Furthermore, these findings raise the question of whether adherence intervention would alter these adherence responses in future research. If smoking cessation at the time of hospitalization is a "window of opportunity," it is necessary to test methods to maximize the effectiveness of treatment aimed to manage tobacco abstinence and prevent relapse by addressing treatment adherence. By not addressing adherence, an opportunity for effective intervention approaches to be successful may be missed in this population of smokers. There is a paucity of treatment adherence related literature within the research realm of smoking behavior and nicotine addiction.

6.1.3. Summary of results for the exploratory analyses

The relationship of age was examined to smoking behavior and treatment assignment. No relationships were found, however, this current study had a homogeneous sample, reducing the ability to detect any relationships. The age of subjects did not differ between treatment groups, recruitment sites, and recruitment cohorts. On average, subjects were middle aged. However, a few previous studies have identified that older smokers had greater success with attempts to quit

smoking than younger adult smokers (Fortmann & Killen, 1995; McWhorter et al., 1990; Murray et al., 2000; Ockene et al., 1994). Future studies, therefore, need to continue to examine results for the potential influence of age on smoking behavior.

The site from which a subject was recruited appeared to have a relationship with smoking behavior at the first follow-up visit 12 weeks following hospital discharge. Furthermore, there may have been a modifying affect by site upon the treatment assignment. Data are not available to determine how these sites differed in ways that they modified the treatment effect. Site effects would be useful to explore in post hospitalization studies.

Employment status was the only variable to statistically differ across the recruitment sites and recruitment cohorts. Statistically, there were more unemployed subjects recruited from site A than B and C. Unemployment was associated with smoking relapse following hospitalization. In addition, more subjects at site A relapsed than at site B and C. It is unclear what process occurred between employment status and the recruitment sites with respect to smoking status measured 12-weeks following hospital discharge.

An indepth examination of usual care practices was not conducted as part of this study. At all three sites, cardiopulmonary rehabilitation healthcare providers were expected by their institutions to be actively engaged with smoking patients with cessation advice and assistance. Site B differed from hospitals A and C with respect to the visibility of cardiopulmonary staff on the cardiopulmonary patient care units. Hospital B had two satellite stations on two different cardiopulmonary patient care units, which contained needed patient education materials, which provided opportunity for the staff to remain on the unit for longer periods than if they were required to gather supplies at the cardiopulmonary offices located some distance from the patient care units. The other hospitals did not have this unique physical configuration, which may have

increased the visibility and efforts of their staff with smoking cessation assistance to patients. In addition, physician messages may have differed as part of usual care. The investigator was witness to many physicians providing smoking cessation information. The impact of these messages was not measured and the impact remains unknown. Delivery of physician messages was observed at all three hospital sites. This study did not have a measure for differences in the intensity of messages from the physicians for all of the participants in this study. Future research may need to measure and examine usual care practices as an intervention is being delivered to this smoking population to better interpret outcome findings.

A study limitation was the lack of controlling for site in the randomization of treatment assignment. Other studies have met with difficulty in controlling site treatment assignment due to the requests by hospitals for inclusion within the treatment group assignment or concern for participants housed within a double occupancy room within the hospital (Bolman et al., 2002). This study did not have problems with the study subjects assigned to the same room during the same time for hospitalization. Most patients were admitted to private rooms within all three institutions or were not assigned to a room with another hospitalized smoker. Future research needs to incorporate control of the effect of recruitment sites on treatment outcome if more than one hospital site is used.

Recruitment cohorts were not related to smoking behavior and did not have a significant direct impact on the effect of treatment assignment, although the first year of the study had the largest treatment effect when examined independently by year (odds ratio = 5.107). The analysis suggested that over the course of three years required to obtain a sample for this study, year of recruitment did not influence the overall results of this study. Future studies with similar

recruitment requirements may need to build into their methods section a method to periodically examine recruitment efforts for balance between years of recruitment and to include a cohort analysis of the findings.

Site and recruitment cohorts as variables entered together were not associated with 24-week smoking behavior. The site affect noted at 12-weeks was not apparent with smoking status at 24-weeks and treatment assignment. The treatment effect observed in the site and cohort analyses for this timeframe of measurement for smoking status did not differ from the adjusted model presented in the results of the primary aim.

6.1.4. Concluding remarks

What have we learned from the results of this study? This pilot study demonstrated that the treatment assignment was significantly related to 24-week smoking status. Findings were impacted by the confounding variables of employment and hospital length of stay. The mechanism by which abstinence was increased remains unclear. Logistic regression models of recruitment sites and cohorts together did not independently affect the treatment effect observed at 24-weeks, but recruitment site alone had an independent relationship with smoking behavior at 12 weeks following hospital discharge. One must keep in mind that the inclusion of site as a variable encompasses an array of variables that were and were not measured by this study. For example, current employment was a confounding variable for both site and treatment assignment with respect to smoking behavior measured 12 weeks following discharge. Since usual care practices were not measured for the effect of their intensity on study outcomes, it is not clear whether the examination by recruitment sites might have captured this type and other potential differences between the recruitment sites. The delivery of smoking intervention at the bedside was feasible, but the combination of collecting baseline data and providing intervention prior to

discharge for short hospital stays was time consuming to the participant. Participants were required to partition their attention to the completion of study procedures with attention needed by their nurses and doctors for discharge instructions. The amount of information collected at baseline may need to be streamlined for future studies to limit subject burden in this process. Twenty-eight percent (n = 22) of the hospitalized smokers across treatment groups were able to maintain abstinence over the course of this research study. The large amount of relapse experienced within the usual care group contributed to the treatment effect observed at 12 and 24 weeks following hospital discharge, although, the percentage in the usual care group who relapsed is similar to other studies of hospitalized smokers. The percentage of abstinent participants from the treatment group was comparative to findings from other studies. Baseline self-efficacy as measured by the RSEQ was a predictor of smoking status at 12-weeks following hospital discharge, but the TASES measure was a not significant predictor of smoking status. This finding for the relationship of the proximal measure of smoking status with the baseline RSEQ will add further support for the predictive nature of baseline self-efficacy and smoking behavior in the literature. This study was one of few to report self-efficacy over time following hospital discharge and found it did not differ by treatment groups. This study is one of the first to examine adherence to behavioral intervention for smoking abstinence in a population of hospitalized smokers. Adherence to the special intervention was related to smoking behavior measured at both time points. The findings for this study regarding adherence to behavioral intervention for smoking abstinence raised various questions for future research, including what strategies are needed to increase adherence to behavioral interventions for smoking abstinence in this population when intervention delivery occurs by telephone.

What do future research efforts need to address? Conceptual variables, such as social support for smoking abstinence need more exploration for definition and measurement in this population of smokers. As noted earlier, the impact of individual physician messages were not measured as part of this study. Future research efforts need to find accurate methods to measure usual care practices by healthcare providers, such as physicians, nurses, and cardiopulmonary rehabilitation healthcare providers. Recruitment from various hospital sites may have different usual care practices that interact with the treatment assignment for the research project. Barriers and influencing factors experienced by usual care and intervention participants needs further study in this population. These factors are similar to side effects of other medical treatment modalities; they interfere with the adherence to prescribed treatment. This population of smokers is complex in that research has yet to address patient's needs more individually. This study attempted to address the relapse risks on a more individual basis, as well as with enhancing sources of self-efficacy. Future research needs to consider additional factors that were not addressed in this study and previous literature that may also influence the smoking behavior outcomes of hospitalized smokers offered assistance to remain tobacco abstinent following their discharge. Adherence needs to be further addressed in future studies and strategies to increase treatment adherence in this population is necessary. Finally, this study had abstinence success for 42% of the intervention group and 15% of the usual care group at 24 weeks following hospitalization. The results of this study suggest relapse occurred for more than 50% of both treatment assignments. Those abstinence rates need to increase for clinical application. Providers and hospitalized smokers need future research to extend what has been learned thus far to develop an intervention that has greater than a 50% chance of being effective to quit smoking and remain abstinent.

APPENDIX A

INTERVENTION BOOK

STAY QUIT STUDY

INTERVENTION BOOK

Smoking, Quitting Smoking, and Your Heart

Smoking is the leading cause of:

1. **Heart disease → Heart attacks and High Blood Pressure (hypertension)**
2. **Vascular disease (blood vessels) →Strokes, Peripheral artery disease, carotid disease**
3. **Cancer → Lung, Head/Neck, Bladder, Cervix, etc.**
4. **Chronic Obstructive Pulmonary Disease → Emphysema, Chronic Bronchitis**

Effects of smoking on the heart and blood vessels:

1. **Constriction (tightening) of the blood vessels**
2. **Decreased variability of the heart rhythm**
3. **Increase in bad cholesterol levels**
4. **Decrease in good cholesterol levels**
5. **Increased stickiness of platelets resulting in greater risk for clotting in the blood vessels.**

These effects on the heart and blood vessels lead to increased blockage in the blood vessels, hypertension, and changes in the heart's ability to perform.

Due to the cardiovascular effects of nicotine and increase in carbon monoxide, wound healing is impaired for individuals who smoke, such as healing from surgical wounds as well as those that develop from peripheral vascular disease.

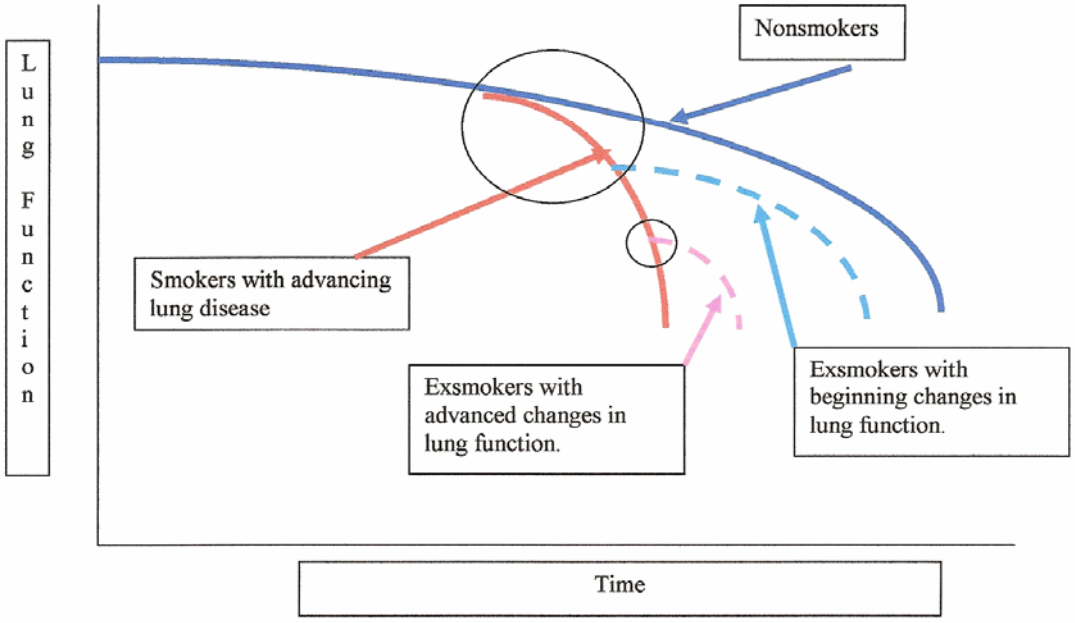
Smoking, Quitting, and your Lungs

Your lungs use two methods to get rid of debris or clearing mechanisms:

1. **Cough**
2. **Mucociliary escalator**

In your airways, before you started smoking, small hair-like projections lined your airways. These hair-like projections (known as cilia), move a mucus layer on upward. This mucus layer is like a conveyor belt that carries debris to the top of the lungs so that an individual can cough out the debris. Unfortunately, this mucociliary escalator is destroyed when someone smokes tobacco. This leaves the airways unprotected and in contact with cancer causing debris/toxins. In addition, the debris only can be taken out of the lungs if it gets near the top of the airway near the windpipe to be coughed out. When someone quits smoking, this mucociliary escalator returns. It may not occur immediately, but does begin to return. Most exsmokers in this phase of recovery notice they have a more intense cough as the mucociliary escalator returns. Using cough/throat lozengers can be helpful during this time to control temporary increased coughing.

Impaired clearing mechanisms can place a patient at risk for the development of chronic obstructive pulmonary disease, such as chronic bronchitis.


Lung Function
Nonsmokers
Smokers with advancing lung disease
Exsmokers with advanced changes in lung function.
Exsmokers with beginning changes in lung function.
Time

What is your motivation to quit smoking?

_____

Insert applied to page 1 of the "You Can Quit" booklet.

Although a lapse (just one puff of a cigarette) can trigger a full relapse to smoking, it is important that you do not think of a lapse as a failure. If a lapse occurs, look at what you can learn from it to stay free of tobacco.

Insert applied to page 2 of the "You Can Quit" booklet.

Social Support is not nagging. Social Support should provide encouragement for your abstinence and assistance to help you when you have urges to smoke.

Insert applied to page 3 of the "You Can Quit" booklet.

Inserts applied to page 4 of the "You Can Quit" booklet.

<u>First Goal</u>: Stay quit from tobacco for 24 hours following your hospital discharge.

Plan: Avoid Hunger,
Get Adequate Rest,
Drink Plenty of Fluids, &

Distraction, Avoidance, Change of Routines, Social Support, Stress Management

Urges to smoke are intense and short in duration. These urges may come close together at first, but less frequently over time as you stay abstinent.

Inserted with page 4 of the "You Can Quit" booklet.

STRATEGIES TO COMBAT SMOKING URGES

Distraction:

***Tasks that distract you with your hands and/or your mind.**
These tasks can be tasks around your house, at work, computer activity, jigsaw puzzles, crossword puzzles, crafts, reading, watching TV, playing games.
***Oral substitutes, such as gum, coffee stirrers, straws, cinnamon sticks. Drinking water.**
***Aroma therapy.**
***Exercise, such as walking, biking, running.**

Change of Routines:

Examples include:
***changing where you sit to eat your meals;**
***changing your morning routine to include getting washing your face, brushing teeth, shower, dressing before breakfast;**
***change what you do after meals like getting up from the table and taking a walk, read the paper, play a game, watch TV instead of sitting at the table to smoke.**

Stress Management:

***Visual Imagery.**
***Progressive Muscle Relaxation.**
***Deep Breathing.**

Reward Yourself:

***You use to reward yourself with a cigarette.**
***You need a new reward, which can be a new hobby, saving the money you would have spent on smoking and rewarding yourself with a treat of some kind (movie, book, and outing).**

Avoidance:

You can use avoidance for prevention of lapsing/relapsing or as a last resort when other strategies are not working in the face of an urge. For example, if you are out with others who are smoking and you are getting the urge to smoke, you can excuse yourself and take a walk outside or depart for the evening.

Insert applied to page 5 of the "You Can Quit" booklet.

Work with your doctor if you want to use a medication to quit smoking.

Insert applied with page 6 of the "You Can Quit" booklet.

#__Negative Moods→ Strategies:
Stress management
Distraction
Avoidance

#__Positive Moods → Strategies:
Distraction
Avoidance

#__Restrictive Situations → Strategies:
Change of Routines
Avoidance
Distraction

#__Idle/Boredom Times → Strategies:
Distraction
Change of Routines

#__Social/Food Situations → Strategies:
Change of Routines
Avoidance
Distraction

#__Low Arousal/Autopilot → Strategies:
Distraction
Change of Routines

#__Craving → Strategies:
Distraction
Change of Routines

Websites added to page 10 of the “You Can Quit” booklet

www.americanheart.org - American Heart Association

www.cancer.org - American Cancer Society

www.lungusa.org - American Lung Association

www.cancer.gov - National Cancer Institute

www.cdc.gov - Center for Disease Control & Prevention

Additional information was provided by web pages from Ashline. Use link to view available web pages. www.ashline.org

APPENDIX B

CORRESPONDENCE AND FORMS

University of Pittsburgh

Institutional Review Board

3500 Fifth Avenue
Ground Level
Pittsburgh, PA 15213
(412) 383-1480
(412) 383-1508 (fax)

MEMORANDUM:

TO: Donna Caruthers, MSN, RN, PhD (c)

FROM: Christopher Ryan, Ph.D., Vice Chair

DATE: May 5, 2004

SUBJECT: IRB #020454: Enhancing Tobacco Abstinence Following Hospitalization

Your renewal with modifications of the above-referenced proposal has received expedited review and approval by the Institutional Review Board under 45 CFR 46.110 (6) (7).

Please include the following information in the upper right-hand corner of all pages of the consent forms.

Approval Date: May 5, 2004
Renewal Date: May 4, 2005
University of Pittsburgh
Institutional Review Board
IRB #020454

Adverse events which occur during the course of the research study must be reported to the IRB Office. Please call the IRB Adverse Event Coordinator at 412-383-1145 for the current policy and forms.

The protocol and consent forms, along with a brief progress report must be resubmitted at least **one month prior** to the expiration date noted above for annual renewal as required by FWA00006790 (University of Pittsburgh), FWA00006735 (University of Pittsburgh Medical Center), FWA00000600 (Children's Hospital of Pittsburgh).

Please be advised that your research study may be audited periodically by the University of Pittsburgh Research Conduct and Compliance Office.

CR:cc

University of Pittsburgh

Institutional Review Board

3500 Fifth Avenue
Ground Level
Pittsburgh, PA 15213
412-578-3424
Fax: 412-578-8553

MEMORANDUM:

TO: Donna Caruthers, MSN

FROM: Christopher Ryan, Ph.D., Vice Chair

DATE: May 9, 2002

SUBJECT: IRB #020454
Enhancing Tobacco Abstinence Following Hospitalization

The above-referenced proposal has received expedited review and approval from the Institutional Review Board under 45 CFR 46.110 (6, 7).

Approval Date: **May 9, 2002**

Renewal Date: **May 8, 2003**

Please be advised that only the IRB approved and stamped consent form can be used to make copies to enroll subjects. If you did not include an original, un-highlighted consent form with this submission, please forward one to the IRB with a copy of this letter to have it stamped.

Adverse events which occur during the course of the research study must be reported to the IRB Office. Please call the IRB Adverse Event Coordinator at 578-8569 for the current policy and forms.

The protocol and consent forms, along with a brief progress report must be resubmitted at least **one month prior** to the expiration date noted above for annual renewal as required by Assurance No. IORG0000196, given to DHHS by the University of Pittsburgh.

Please be advised that your research study may be audited periodically by the University of Pittsburgh Research Conduct and Compliance Office.

University of Pittsburgh

Institutional Review Board

3500 Fifth Avenue
Ground Level
Pittsburgh, PA 15213
(412) 578-3424
(412) 578-8553 (fax)

MEMORANDUM

TO: Donna Caruthers, MSN, RN, Ph.D.(c)

FROM: Philip Troen, M.D., Chair PT

DATE: December 11, 2002

SUBJECT: IRB #020454: Enhancing Tobacco Abstinence Following Hospitalization

The Institutional Review Board reviewed the recent modifications to your protocol and consent form(s) and find them acceptable for expedited review. These changes, noted in your submission of 12/05/02, are approved.

Please include the following information in the upper right-hand corner of all pages of the consent forms:

Approval Date: December 11, 2002
Renewal Date: May 8, 2003
University of Pittsburgh
Institutional Review Board
IRB #020454

Adverse events which occur during the course of the research study must be reported to the IRB Office. Please call the IRB Adverse Event Coordinator at 412-578-8569 for the current policy and forms.

The protocol and consent form(s) together with a brief progress report must be resubmitted **prior to the date of expiration listed above** by the General Assurance No. IORG0000196 given to DHHS by the University of Pittsburgh.

If your research proposal involves an investigational drug, please forward a copy of this approval letter along with a copy of the Cover Sheet, protocol, consent form(s) and drug brochure to Investigational Drug Service, PUH Pharmacy.

Please be advised that your research study may be audited periodically by the University of Pittsburgh Research Conduct and Compliance Office.

PT/ay

University of Pittsburgh

Institutional Review Board

3500 Fifth Avenue
Ground Level
Pittsburgh, PA 15213
(412) 578-3424
(412) 578-8553 (fax)

MEMORANDUM:

TO: Donna D. Caruthers, MSN, RN

FROM: Christopher Ryan, Ph.D, Vice Chair

DATE: May 1, 2003

SUBJECT: IRB #980660: Characteristics of Hospitalized Smokers

Your renewal of the above-referenced proposal has received expedited review and approval by the Institutional Review Board. **This approval is for analysis of data only.**

Approval Date: **May 1, 2003**

Expiration Date: **April 30, 2004**

The protocol and consent forms, along with a brief progress report must be resubmitted at least **one month prior** to the expiration date noted above for annual renewal as required by Assurance No. M-1259, given to DHHS by the University of Pittsburgh.

Please be advised that your research study may be audited periodically by the University of Pittsburgh Research Conduct and Compliance Office.

CR:cc

University of Pittsburgh

Institutional Review Board

3500 Fifth Avenue
Ground Level
Pittsburgh, PA 15213
412-578-0464
Fax: 412-578-8553

MEMORANDUM:

TO: Donna Caruthers, MSN, RN

FROM: Christopher Ryan, Ph.D., Vice Chair

DATE: May 2, 2002

SUBJECT: IRB #980660: Characteristics of Hospitalized Smokers

Your renewal of the above referenced proposal has received expedited review and approval by the Institutional Review Board. **This approval is for analysis of data only.**

Approval Date: **May 2, 2002**

Renewal Date: **May 1, 2003**

The protocol and consent forms, along with a brief progress report must be resubmitted at least **one month prior** to the expiration date noted above for annual renewal as required by Assurance No. IORG0000196, given to DHHS by the University of Pittsburgh.

Please be advised that your research study may be audited periodically by the University of Pittsburgh Research Conduct and Compliance Office.

CR:jmb

INTER

Jefferson Regional Medical Center

OFFICE

MEMO

To: Donna Carothers, MSN, RN, Phd (c)
Stay Quit Study, University of Pittsburgh, School of Nursing
3500 Victoria Street, Room 360R, Pittsburgh, PA 15261

From: William R. Sims, President, Medical Staff

Subject: **IRB #020454: Enhancing Tobacco Abstinence Following Hospitalization**

Date: July 8, 2004

At the July 6, 2004 Medical Executive Staff Committee meeting, the IRB #020454 Research Project entitled Enhancing Tobacco Abstinence Following Hospitalization was approved as presented.

If you have any questions, please contact the Medical Staff Services Office at 412-469-5100.

cc: Marc Irwin

H:\Diana\MEC\2004\Memo\7-[illegible]-04IRB#020454.doc

University of Pittsburgh

School of Nursing

460 Victoria Building
3500 Victoria Street
Pittsburgh, Pennsylvania 15261
412-624-9935
Fax: 412-624-1508

Approval Date: May 5, 2004
Renewal Date: May 4, 2005
University of Pittsburgh
Institutional Review Board
IRB #020454

CONSENT TO ACT AS A SUBJECT IN A RESEARCH STUDY

TITLE: Enhancing Tobacco Abstinence Following Hospitalization

PRINCIPAL INVESTIGATOR: Donna D. Caruthers, MSN, RN
Doctoral Candidate
University of Pittsburgh School of Nursing
Rm. 360R, Victoria Building
Telephone: 412-624-9935 and 412-558-8463

CO-INVESTIGATORS:

Jacqueline Dunbar-Jacob, PhD, RN
Professor
University of Pittsburgh School of Nursing
Rm. 350, Victoria Building
Telephone: 412-624-7838

Susan Sereika, PhD
Associate Professor
University of Pittsburgh School of Nursing
Rm. 360, Victoria Building
Telephone: 412-624-0799

Kenneth Perkins, PhD
Professor
University of Pittsburgh
Rm. 1225 Thomas Detre Hall of the
Western Psychiatric Institute and Clinic
Telephone: 412-624-1716

Susan Albrecht, PhD, RN
Associate Professor
University of Pittsburgh School of Nursing
Rm. 415, Victoria Building
Telephone: 412-624-2403

Jill Landsbaugh
Recruitment Coordinator
University of Pittsburgh School of Nursing
Rm. 360R, Victoria Building
Telephone: 412-624-9935

SOURCE OF SUPPORT: National Institutes of Health (NIH),
National Institute of Nursing Research (NINR)
1 F31 NR07343 (Predoctoral Fellowship Award)
Eta Chapter, Sigma Theta Tau
Pauline Thompson Clinical Research Award,
Nursing Foundation of Pennsylvania

Why is this research being done?

Since you smoke cigarettes, we are asking you to join a research study. This study will test two treatment programs offering help to quit smoking. If you join the study you will be assigned by chance to one of these two programs to help you quit smoking. Smokers selected for the first program, will be given a message from a nurse to quit smoking and a self-help book about quitting smoking. Smokers selected for the second program will be given a message from a nurse to quit smoking and a self-help book like the first group. They will also take part in a 12-week telephone program with a nurse after they go home from the hospital. We will compare these two programs to see what effect they have in helping patients stay free of smoking 24 weeks after they go home from the hospital.

Who is being asked to take part in this research study?

You are invited to take part in this research study because you are a patient admitted to the hospital and smoke cigarettes. To take part, you may be either male or female and must be at least 18 years old. The study will screen up to 396 current smokers persons admitted to either the Oakland or Shadyside UPMC medical centers. The first 150 persons who qualify for the study will continue with the study following the screening procedures.

What procedures will be performed for research purposes?

If you decide to take part in this research study, you will undergo the following procedures that are not part of your standard medical care:

Screening Procedures:

Procedures to determine if you are eligible to take part in a research study are called "screening procedures". The following screening procedures will occur for this study.

1. We will interview you and review your medical chart for medical or personal conditions that would make it difficult for you to participate with this study. Examples include a stroke, life-threatening condition or illness, evaluation for organ transplantation or awaiting transplantation, senile dementia, Alzheimer disease, non-English speaking patients, lack of a home telephone, lack of a mailing address, lack of any ability to participate with self-care activities, and planned transfer to a rehabilitation hospital or nursing home following your hospital admission at UPMC.

Experimental Procedures:

If you qualify to take part in this research study, you will undergo the following study procedures.

1. We will interview you and review your medical history and treatments ordered by your doctors.

2. You will complete a brief interview, paper and pencil tests, and a breath test for exhaled carbon monoxide during your hospital admission. Exhaled carbon monoxide will be used in this study to measure your exposure or use of inhaled tobacco. The interview, paper and pencil tests, and the carbon monoxide breath test will require about 45 minutes of your time. The following paper pencil tests will be used as part of this study:

   a. Tobacco Abstinence Self-efficacy Scale tests how you view your ability to stay free of tobacco.
   b. Perceived Therapeutic Efficacy Scale for Tobacco Abstinence tests how you view the therapy you receive to stay free of tobacco.
   c. Relapse Situation Efficacy Questionnaire measures your view of your ability to stay smoke free in different situations when you may consider smoking.
   d. Sociodemographic Questionnaire collects information such as your age, job, gender, and marital status.
   e. Tobacco Consumption Questionnaire collects information about your use of tobacco, such as how much you smoke, for how long, if you want to quit smoking, quit smoking treatment you have used in the past and if you smoked even a puff of cigarette during the study.
   f. Fagerstrom Test for Nicotine Dependence measures your level of nicotine dependence.
   g. Tobacco Withdrawal Form measures if you are experiencing nicotine withdrawal symptoms.
   h. Profile of Mood States measures different moods you may be experiencing during the study as you try to stay free of tobacco.
   i. CRCD Comorbidity Questionnaire collects information about any medical disorders you have experienced.

3. You will receive a message from a nurse urging you to quit smoking with your hospital admission and a self-help book about quitting smoking. This will require 10 minutes of your time.

4. You will receive assignment to one of two study treatment programs. This will take 5 minutes of your time.

5. Everyone who qualifies for the study will receive a message about quitting smoking and a self-help guide to quitting smoking. A review of the materials you will be provided and a message regarding quitting smoking will take 30 minutes with the study nurse.

   If you are selected to receive the first study treatment program, you will be asked to use the self-help book to assist your efforts in quitting smoking and remaining free from tobacco after you leave the hospital. There will be no additional contact with the study nurse until you are called to schedule your follow-up visits.

If you receive the second study treatment program, you will receive the quit smoking message, self-help guide, and will be asked to take part in 9 counseling sessions with a study nurse. The first session will occur in the hospital before you leave. The study nurse will speak to you directly about planning to stay free from tobacco when you leave the hospital. The next 8 sessions will occur by telephone over 11 weeks after you leave the hospital. The study nurse will arrange the schedule for these telephone sessions. The telephone counseling sessions will review what you go through to stay free of tobacco, how your plans to stay free of tobacco worked, and future goals and plans you set to stay free of tobacco. Each of these telephone sessions will require 15 to 20 minutes of your time. The study nurse will be tape recorded to check that you receive the quality of information planned by study researchers. Your comments from the conversation will not be recorded. Also, the study nurse will only use your first name so that you cannot be identified from the recording of the study nurse.

Monitoring/Follow-up Procedures:

Procedures performed to evaluate the safety and effectiveness of the experimental procedures are called "monitoring " or "follow-up" procedures. The monitoring/follow-up procedures include the following for this study.

1. We will interview you and review your medical history and treatments ordered by your doctors.

2. You will complete a brief interview, paper and pencil tests, and a breath test for exhaled carbon monoxide during your hospital admission. Exhaled carbon monoxide will be used in this study to measure your exposure or use of inhaled tobacco. The interview, paper and pencil tests, and the carbon monoxide breath test will require about 45 minutes of your time. The following paper pencil tests will be used as part of this study:

   a. Tobacco Abstinence Self-efficacy Scale tests how you view your ability to stay free of tobacco.
   b. Perceived Therapeutic Efficacy Scale for Tobacco Abstinence tests how you view the therapy you receive to stay free of tobacco.
   c. Relapse Situation Efficacy Questionnaire measures your view of your ability to stay smoke free in different situations when you may consider smoking.
   d. Sociodemographic Questionnaire collects information such as your age, job, gender, and marital status.
   e. Tobacco Consumption Questionnaire collects information about your use of tobacco, such as how much you smoke, for how long, if you want to quit smoking, quit smoking treatment you have used in the past and if you smoked even a puff of cigarette during the study.
   f. Fagerstrom Test for Nicotine Dependence measures your level of nicotine dependence.
   g. Tobacco Withdrawal Form measures if you are experiencing nicotine withdrawal symptoms.

h. Profile of Mood States measures different moods you may be experiencing during the study as you try to stay free of tobacco.
i. CRCD Comorbidity Questionnaire collects information about any medical disorders you have experienced.

2. You will complete two follow-up visits with the study staff. The first visit will occur 12 weeks after you leave the hospital. The second visit will occur 24 weeks after you leave the hospital. These visits will take place at the University of Pittsburgh School of Nursing. You will complete a brief interview, paper and pencil tests (described above), and a breath test for exhaled carbon monoxide (described above). This will require about 45 minutes of your time.

What are the possible risks, side effects, and discomforts of this research study?

There are possible risks or discomforts related to taking part in this study. You may feel fatigued during the process of completing forms for the study. Efforts will be made to allow plenty of rest periods during the completion of these forms.

You may feel nicotine withdrawal symptoms as you quit smoking tobacco. These symptoms are more likely to occur within the first three days of not smoking. You may feel any of the following as nicotine withdrawal symptoms: cigarette craving, depressed mood, irritability, frustration, anger, anxiety, restlessness, increased hunger, decreased heart rate, difficulty concentrating, difficulty sleeping, dizziness, constipation, and headaches. If you are experiencing severe nicotine withdrawal symptoms you will be withdrawn from study participation and encouraged to work with your physician in managing severe withdrawal symptoms.

What are possible benefits from taking part in this study?

You will likely receive no direct benefit from taking part in this research study. Should either study treatment help you to quit smoking, it is possible that you may receive some health benefit by quitting smoking. However, such a benefit cannot be guaranteed.

What treatments or procedures are available if I decide not to take part in this research study?

If you decide not to take part in this research study, you may ask your doctor and nurses for help to quit smoking or seek out help from other quit smoking programs. This study will have no effect on the medical care you receive from the doctors and nurses at the hospital.

If I agree to take part in this research study, will I be told of any new risks that may be found during the course of the study?

You will be promptly notified if any new information develops during the conduct of this research study, which may cause you to change your mind about continuing to participate. You will also be notified of any new benefits found during the course of the study.

Will my insurance provider or I be charged for the costs of any procedures performed as part of this research study?

Neither you, nor your insurance provider, will be charged for the costs of any of the procedures performed for the purpose of this research study (i.e., the Screening Procedures, Experimental Procedures, or Monitoring/Follow-up Procedures described above). You will be charged, in the standard manner, for any procedures ordered by your doctors and performed for your routine medical care.

Will I be paid if I take part in this research study?

You will be paid $10 for each follow-up visit you attend. A total of $20 dollars will be paid to you if you attend both follow-up visits. The first visit will occur 12 weeks after you go home from the hospital. The second visit will take place 24 weeks after you go home from the hospital. This money is paid to defray your parking and travel expenses for these study visits. If, for whatever reason, you do not attend a follow-up visit, the $10 payment for parking and travel will not made.

Who will pay if I am injured as a result of taking part in this study?

University of Pittsburgh researchers and their associates who provide services at the UPMC Health System (UPMC HS) recognize the importance of your voluntary participation in their research studies. These individuals and their staffs will make reasonable efforts to minimize, control, and treat any injuries that may arise as a result of this research. If you believe that you are injured as a result of the research procedures being performed, please contact immediately the Principal Investigator or a co-investigator listed on the first page of this form.

Emergency medical treatment for injuries solely and directly related to your participation in this research study will be provided to you by the hospitals of the UPMC HS. It is possible that the UPMC HS may bill your insurance provider for the costs of this emergency treatment, but none of these costs will be charged directly to you. If your research-related injury requires medical care beyond this emergency treatment, you will be responsible for the costs of this follow-up care unless otherwise specifically stated below. You will not receive any monetary payment for, or associated with, any injury that you suffer in relation to this research.

Who will know about my participation in this research study?

Any information about you obtained from this research will be kept as confidential (private) as possible. You will not be identified by name in any publication of research results unless you sign a separate form giving your permission (release). All records related to your involvement in this research study will be stored in a locked file cabinet. Your identity on these records will be indicated by a case number rather than by your name, and the information linking these case numbers with your identity will be kept separate from the research records. Only the

researchers listed on the first page of this form and their staff will have access to your research records. Your research records will be maintained at least 5 years following completion of this study.

Will this research study involve the use or disclosure of my identifiable medical information?

This research study will involve the recording of current and/or future identifiable medical information from your hospital and/or other (e.g., physician office) records. The information that will be recorded will be limited to information concerning the reason for your hospital admission and medical history. This information will be used to evaluate the study among comparable patients for the purpose of evaluating the safety and effectiveness of the relapse prevention intervention.

You may choose whether you want the study team to notify your medical team of your participation in this smoking relapse prevention research. If your choice is to permit our research team to notify your medical team, identifiable information will be placed into your medical records held this UPMC healthcare facility. The nature of the identifiable information resulting from your participation in this research study that will be recorded in your medical record includes your completion of the assessment and hospital-based smoking intervention received by all patients who participate in this research. No other information obtained by the study will be released to the medical team.

Do you want the research team to notify your medical team when you have completed baseline assessments and the hospital-based smoking intervention?

_____Yes, inform my medical team.
_____No, do not inform my medical team.

Who will have access to identifiable information related to my participation in this research study?

In addition to the investigators listed on the first page of this authorization (consent) form and their research staff, the following individuals will or may have access to identifiable information (which may include your identifiable medical information) related to your participation in this research study:

Authorized representatives of the University of Pittsburgh Research Conduct and Compliance Office may review your identifiable research information (which may include your identifiable medical information) for the purpose of monitoring the appropriate conduct of this research study.

It is also possible that authorized representatives for the study sponsor (National Institute for Nursing Research), may inspect your research records. If the researchers learn that you or someone with whom you are involved is in serious danger or harm, they will need to inform the appropriate agencies as required by Pennsylvania law.

In unusual cases, the investigators may be required to release identifiable information (which may include your identifiable medical information) related to your participation in this research study in response to an order from a court of law. If the investigators learn that you or someone with whom you are involved is in serious danger or potential harm, they will need to inform, as required by Pennsylvania law, the appropriate agencies.

Authorized representatives of the UPMC Health System hospitals or other affiliated health care providers may have access to identifiable information (which may include your identifiable medical information) related to your participation in this research study for the purpose of (1) fulfilling orders, made by the investigators, for hospital and health care services (e.g., laboratory tests, diagnostic procedures) associated with research study participation; (2) addressing correct payment for tests and procedures ordered by the investigators; and/or (3) for internal hospital operations (i.e. quality assurance).

For how long will the investigators be permitted to use and disclose identifiable information related to my participation in this research study?

The investigators may continue to use and disclose, for the purposes described above, identifiable information (which may include your identifiable medical information) related to your participation in this research study for a minimum of 5 years and for as long (indefinite) as it may take to complete this research study.

May I have access to my medical information that results from my participation in this research study?

In accordance with the UPMC Health System Notices of Privacy Practices document that you have been provided, you are permitted access to information (including information resulting from your participation in this research study) contained within your medical records filed with your health care provider.

Is my participation in this research study voluntary?

Your participation in this research study is completely voluntary. You do not have to take part in this research study and, should you change your mind, you can withdraw from the study at any time. Your current and future care at a UPMC HS facility and any other benefits for which you qualify will be the same whether you participate in this study or not.

Before entering this study or at any time during the research, you may discuss your care with your physician who is in no way associated with this research project. You are not under any obligation to participate in any research study.

May I withdraw, at a future date, my consent for participation in this research study?

You may withdraw, at any time, your consent for participation in this research study, to include the use and disclosure of your identifiable information for the purposes described above. (Note, however, that if you withdraw your consent for the use and disclosure of your identifiable medical record information for the purposes described above, you will also be withdrawn, in general, from further participation in this research study.) Any identifiable research or medical information recorded for, or resulting from, your participation in this research study prior to the date that you formally withdrew your consent may continue to be used and disclosed by the investigators for the purposes described above.

To formally withdraw your consent for participation in this research study you should provide a written and dated notice of this decision to the principal investigator of this research study at the address listed on the first page of this form.

Your decision to withdraw your consent for participation in this research study will have no affect on your current or future relationship with the University of Pittsburgh. Your decision to withdraw your consent for participation in this research study will have no affect on your current or future medical care at a UPMC Health System hospital or affiliated health care provider or your current or future relationship with a health care insurance provider.

If you decide to withdraw from study participation after you have received the study drug, you should participate in described monitoring follow-up procedures directed at evaluating the safety of the study drug.

If I agree to take part in this research study, can I be removed from the study without my consent?

It is possible that you may be removed from the research study by the researchers if, for example, the investigators finds you are not eligible to participate during the screening process, you experience severe nicotine withdrawal symptoms, and/or you are unable to be reached to schedule follow-up visits. Even if you are withdrawn from the study by the researchers, data collected up to that point in time would be used for the study.

Approval Date: May 5, 2004
Renewal Date: May 4, 2005
University of Pittsburgh
Institutional Review Board
IRB #020454

****

VOLUNTARY CONSENT

All of the above has been explained to me and all of my current questions have been answered. I understand that I am encouraged to ask questions about any aspect of this research study during the course of this study, and that such future questions will be answered by the researchers listed on the first page of this form.

Any questions which I have about my rights as a research participant will be answered by the Human Subject Protection Advocate of the IRB Office, University of Pittsburgh (412-578-8570).

By signing this form, I agree to participate in this research study. A copy of this consent form will be given to me.

_____ _____

Participant's Signature Date

CERTIFICATION of INFORMED CONSENT

I certify that I have explained the nature and purpose of this research study to the above-named individual(s), and I have discussed the potential benefits and possible risks of study participation. Any questions the individual(s) have about this study have been answered, and we will always be available to address future questions as they arise."

_____ _____

Printed Name of Person Obtaining Consent Role in Research Study

_____ _____

Signature of Person Obtaining Consent Date

****

We are planning more studies about smokers like this one. Would you be interested in receiving information regarding these studies?

[ ] Yes [ ] No

What does this study involve?

Everyone participating in this study will:

- *be asked to complete an interview* ***before*** *leaving the hospital.*
- *be asked to quit smoking* ***while*** *in the hospital.*
- *receive a "quit smoking" message from a study nurse.*
- *receive a "quit smoking" manual.*
- *be asked to meet with a study nurse for two individually arranged follow-up contacts at 12 and 24 weeks after leaving the hospital.*

75 patients will be asked:

- *to participate with a home-based "stay quit" program after leaving the hospital.*
- *This additional program provides 8 follow-up calls over the first three months after discharge from the hospital.*

Smoking Relapse Prevention Project

Please place sealed brochures in the Stay Quit Study Inbox on this patient care unit.

Any questions, please call:
412-624-9935
or
412-558-8463.

Stay Quit Study

Donna D. Caruthers, MSN, RN
Principal Investigator

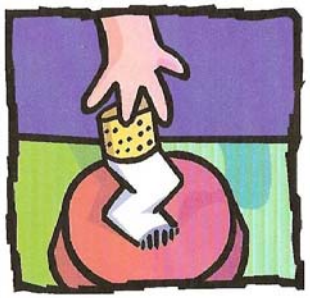
Stay Quit Study
University of Pittsburgh
School of Nursing
3500 Victoria Street, Room 360R
Pittsburgh, PA, 15261

Phone: 412-558-8463
Fax: 412-624-1508

Smoking Relapse Prevention Project

University of Pittsburgh

School of Nursing



Stay Quit Study

Donna D. Caruthers, MSN, RN
Principal Investigator

Telephone:
412-558-8463

- *Are you at least 18 years old?*
- *Did you smoke at least one cigarette within the last month?*

Are you admitted to this hospital for inpatient care?

If you answered yes to these questions, we would like to talk with you about a smoking relapse prevention research project.

How can I find out more about this research study?

You can:

- **call us directly, or**
- **complete the brochure panel.**

Call : 412-558-8463.

- **Please provide your name, hospital, and room number.**
- **If you would like to participate with this project, we will need at least an hour of your time before you are released from the hospital.**

Yes, I would like more information. Please stop to see me in my hospital room.

First Name: _____

Last Name: _____

Room Number: _____

Date: _____

Seal the brochure and give it to your nurse to place in our mailbox located on this unit.

UPMC HEALTH SYSTEM

UPMC Presbyterian/UPMC Shadyside

AUTHORIZATION FOR THE SHARING OF HEALTH INFORMATION RELATED TO POSSIBLE PARTICIPATION IN A RESEARCH STUDY

Title of Research Study: Enhancing Tobacco Abstinence Following Hospitalization

UPMC Presbyterian
200 Lothrop Street
Pittsburgh, PA 15213-2582

UPMC Shadyside
5230 Centre Avenue
Pittsburgh, PA 15232

RESEARCH STUDY INVESTIGATOR(S):
PRINCIPAL INVESTIGATOR:
Donna D. Caruthers, MSN, RN
Doctoral Candidate
University of Pittsburgh School of Nursing
Rm. 360R, Victoria Building
Telephone: 412-624-9935

CO-INVESTIGATORS:

Jacqueline Dunbar-Jacob, PhD, RN, Professor
University of Pittsburgh School of Nursing
Rm. 350, Victoria Building
Telephone: 412-624-7838

Kenneth Perkins, PhD, Professor
University of Pittsburgh
Rm. 1225 Thomas Detre Hall of the
Western Psychiatric Institute and Clinic
Telephone: 412-624-1716

Jill Landsbaugh, Recruitment Coordinator
University of Pittsburgh School of Nursing
Rm. 360R, Victoria Building
Telephone: 412-624-9935

Susan Sereika, PhD, Associate Professor
University of Pittsburgh School of Nursing
Rm. 360, Victoria Building
Telephone: 412-624-0799

Susan Albrecht, PhD, RN, Associate Professor
University of Pittsburgh School of Nursing
Rm. 415, Victoria Building
Telephone: 412-624-2403

What is the purpose of this authorization?

Your doctor or a member of your doctor's health care staff has discussed with you that you may be eligible to take part in the above-named research study. You have indicated an interest in learning more about this research study from the researchers who are involved in conducting the study. Thus, your authorization (permission) is being requested to:

- share the fact that you are interested in hearing more about this study from the involved researchers;
- allow the involved researchers to contact you so as to permit additional discussions of this study with you and/or to provide you with information on how you may take part in this study.

Patient's Initials:_____

Item# 02157

What information about me will be shared with the researchers?

If you give your permission, the following information about you will be shared (for example, by telephone or FAX) with the researchers involved in the conduct of the above-named research study:

- your name and hospital room number

To whom will the above information be given?

We will share this information with one of the researchers listed above or a member of their research staff. This information will be used by the researchers to evaluate if you are eligible to participate in this research study and/or to contact you to further discuss this research study with you.

These researchers recognize the importance of maintaining the confidentiality (privacy) of your health information, however it is not possible for us to guarantee its confidentiality after we have provided it to them.

For how long is authorization valid?

Once this information has been shared with the researchers, this authorization form will expire. We will not continue to share your future health information with these researchers, nor will we share your health information with any other researchers unless you sign a separate authorization form that permits us to do so.

Is my permission to provide this information to the researchers voluntary?

Your permission to provide this information to the researchers is completely voluntary. Whether or not you provide your permission will have no affect on your current or future medical care or your relationship with your doctor or health care provider. Whether or not you provide your permission will have no affect on your current or future relationship with the University of Pittsburgh or University of Pittsburgh Medical Center.

May I withdraw, at a future date, my permission to provide this information to the researchers?

You may withdraw, at any time, your permission to provide this information to the researchers. However, once this information has been shared with the researchers, the information will be in their possession. Hence, should you decide to withdraw your permission after your information has been given to the researchers you should send a written and dated notice of this decision to the principal investigator of this research study at the address listed above. Upon receipt of this request, the researchers will destroy your information that was provided to them. If you wish to withdraw your permission to provide this information to the researchers before it is given to them, you should contact, by telephone, your doctor or a member of your doctor's health care staff. With receipt of this request, your information will not be shared with the researchers.

Your decision to withdraw your permission to provide this information to the researchers will have no affect on your current or future medical care or your relationship with your doctor or health care provider. Your decision to withdraw your permission will have no affect on your current or future relationship with the University of Pittsburgh or University of Pittsburgh Medical Center.

****

VOLUNTARY AUTHORIZATION

All of the above has been explained to me. By signing below I give my permission to share the information, specified above, with the researchers, identified above, for the purposes described.

_____

Printed Name of Patient

_____ _____

Signature of Patient Date

Univeristy of Pittsburgh School of Nursing
Donna Caruthers, MSN, RN - Study Investigator

If you smoked at least one cigarette in the last 30 days and you are a patient admitted to UPMC Presbyterian/Montefiore or UPMC Shadyside, you may be eligible for a study assisting patients to quit smoking.

WE NEED YOUR HELP WITH THIS STUDY

- Everyone participating in the study will be asked to complete an interview before leaving the hospital to go home.
- Everyone participating in the study will be asked to quit smoking while admitted to the hospital.
- Everyone will receive a "quit smoking" message from a study nurse and "quit smoking" manual.
  - 75 patients will be asked to participate in a home-based "stay quit" program after leaving the hospital.
  - After hospital discharge, patients in the home-based "stay quit" program will receive 9 telephone counseling calls from the study nurse over 11 weeks.
- Everyone participating in the study will be asked to meet with a study nurse for two follow-up study visits at 12 and 24 weeks after leaving the hospital.

If you ARE interested in hearing more about this study, please check the box below, sign your name, and give this flyer to your nurse. You may ask your nurse to contact us at 412-624-9935. A member of the research team will stop by your room to tell you about this study and answer any questions you may have about this study. You may contact us directly at 412-624-9935 for information or request for us to contact you.

☐ I am not interested. Date: ☐☐ / ☐☐ / ☐☐

☐ I am interested in more information.

Patient Name

Unit Contact Nurse

Office use only

○1 ○2

Sigma Theta Tau International, Inc.
HONOR SOCIETY OF NURSING

Eta Chapter
School of Nursing, University of Pittsburgh
Victoria Building

Pittsburgh, Pennsylvania 15261

Donna Caruthers, MSN, RN

June 19, 2001

Dear Ms. Caruthers,

It is with great enthusiasm that Eta Chapter is bestowing upon you the 2002 Research Award for your proposed study entitled "Psychometric Examination of the Tobacco Abstinence Self-Efficacy Scale." Enclosed please find a check in the amount of $1,500 to help defray the costs of conducting your investigation. As you well know, a research career is built upon a foundation of scientifically sound and meritorious scholarly work. Your efforts clearly reflect commitment and focus toward that end. May I wish you every success as you advance nursing practice. You are a definite asset to our profession and most deserving of this honor.

Sincerely,

Judith Tabolt Matthews

Judith Tabolt Matthews, PhD, MPH, RN
Assistant Professor
Department of Health and Community Systems

Nursing Foundation of Pennsylvania

June 11, 2002

Donna D. Caruthers, PhD(c), MSN, RN

Dear Ms. Caruthers:

Congratulations! This letter is to confirm the Nursing Foundation of Pennsylvania (NFP) Awards Committee has selected you to receive the Pauline Thompson Clinical Research Award in the amount of $1,000. The Awards Committee was impressed by your application and the Board of Trustees and I are looking forward to meeting you.

The scholarship will be presented during the Pennsylvania State Nurses Association Awards Banquet to be held Friday, **October 4**, 2002, at Marriott Hotel located at 4650 Lindle Road, Harrisburg, PA 17111, telephone 717-558-4615. A President's Reception will be held beginning at 6:30 p.m. with the Awards Banquet following at 7:30 p.m. It would be appreciated if you could send a photograph (good quality) of yourself. We would like to have your picture printed in the Awards Banquet Booklet.

Please complete the enclosed response form and return in the enclosed postage-paid envelope no later than **August 16, 2002.**

Sincerely,

Michele P Campbell

Michele P. Campbell, MSN, RN, C
Chief Executive Officer

MPC:fm

Enclosures

cc: Dr. Dunbar-Jacob
University of Pittsburgh

2578 Interstate Drive, Suite 101 • P.O. Box 68525 • Harrisburg, PA 17106-8525 • Phone: 717-657-1222 • Fax: 717-657-3796

Subject: RE: pdf downloads
From: Susan Larsen <smlarsen@u.arizona.edu>
Date: Thu, 14 Feb 2002 11:59:15 -0700
To: Donna Caruthers <caru@pitt.edu>

Ms. Carruthers:

The download files can be used and reproduced as long as they are not altered in any fashion. There is no limit to the number of copies you make.

Thank you for your interest and good luck on your project.

Sue Larsen, Webmaster
Network for Information and Counseling
University of Arizona College of Public Health
520 318-7212 ext 204 smlarsen@u.arizona.edu
http://www.tepp.org
http://www.ashline.org
Cessation Counseling at
ashline1@u.arizona.edu

-----Original Message-----
From: Donna Caruthers [mailto:caru+@pitt.edu]
Sent: Wednesday, February 13, 2002 9:55 AM
To: nicnet@u.arizona.edu
Subject: pdf downloads

To whom it may concern,
I was interested in the one page help sheets listed on the following web page: http://www.ashline.org/ASH/free/downloads.html . These pages are really well done. I have attached one of the pdf files to this email so that you can see an example of what I was referring to in this message. I would like to make copies of these "free download" hint sheets to give to hospitalized smokers in my dissertation project, which is attempting to intervene with hospitalized smokers. I would like to use these sheets along with the program book from the CDC regarding smoking cessation. Your coping hint sheets are very well done and my interest is not to reinvent the wheel. Furthermore, my program is designed to build upon materials such as these hint sheets and the CDC book. I my plan is to use these across all study subjects as a standard of care for smoking cessation and my special intervention group is going to receive a relapse prevention program in addition to these materials. I know that the web site says these materials may be reproduced, but I was not sure if that was by individual or a blanket of repetitive copies. Therefore, in order to satisfy our school's policy and protocols, I need to have this information clarified and/or to obtain permission to reproduce these web materials for 150 subjects and the project team.

Thank you for your assistance in this matter. I look forward to hearing from you.
Sincerely,
Donna Caruthers
412-624-9935

Instrument Number:

T A S

(For internal use only)

Study ID: 0 5 6

TOBACCO ABSTINENCE SELF- EFFICACY

Center for Research in Chronic Disorders

ID Number: | | | | | | | | | | | Administration Date: | | / | | / | | | | |

(month) (day) (year)

0 ○ 1 ○ 2 ○

(FOR STAFF USE ONLY)

Please use the following example to answer all questions:

Shade circles like this: ●

Not like this: ⊗ ✓

Please turn to the next page to answer the following questions.

ID Number: _ _ _ _ _ _ _ _ _ _ _ (for internal use only) Date: _ _ / _ _ / _ _ (for internal use only) Study ID: 056

In the following questions, we'd like to know how your tobacco dependence affects you. For each of the following questions, please fill in the circle that corresponds best to your confidence that you can now perform the following tasks.

	Not Very Confident				Moderately Confident					Very Confident
	10	20	30	40	50	60	70	80	90	100
1. How confident are you that you can decrease your urges to smoke quite a bit?	○	○	○	○	○	○	○	○	○	○
2. How confident are you that you can continue most of your daily activities while quitting smoking?	○	○	○	○	○	○	○	○	○	○
3. How confident are you that you can keep urges to smoke from interfering with your daily activities?	○	○	○	○	○	○	○	○	○	○
4. How confident are you that you can make small-to-moderate decreases in your urges to smoke by using methods other than taking medication to aid quitting smoking (such as nicotine gum, patch, inhaler, or Zyban)?	○	○	○	○	○	○	○	○	○	○
5. How confident are you that you can make large decreases in your urges t smoke by using methods other than taking medication to aid quitting smoking (such as nicotine gum, patch, inhaler, or Zyban?	○	○	○	○	○	○	○	○	○	○
6. How confident are you that you can control your urges to smoke?	○	○	○	○	○	○	○	○	○	○
7. How confident are you that you can control your daily activities so as to lessen the impact of urges to smoke?	○	○	○	○	○	○	○	○	○	○
8. How confident are you that you can do something to help yourself from giving in to urges to smoke when you feel blue or down?	○	○	○	○	○	○	○	○	○	○
9. As compared to others quitting tobacco use, howconfident are you that you can manage your urges to smoking during your daily activities?	○	○	○	○	○	○	○	○	○	○
10. How confident are you that you can manage urges to smoke so that you can enjoy doing things you like to do without smoking?	○	○	○	○	○	○	○	○	○	○

ID Number: _ _ _ _ _ _ _ _ _ _ _ (for internal use only) Date: _ _ / _ _ / _ _ (for internal use only) Study ID: 056

In the following questions, we would like to know how confident you are in your ability to resist using tobacco after you are discharged from a hospital admission.

For each of the following questions, please fill in the circle that corresponds to your confidence that you can perform the tasks as of now. Please consider what you routinely can do, not what would require a single extraordinary effort.

As of now, how confident are you that you can resist using tobacco (smoking) ....

	Not Very Confident				Moderately Confident					Very Confident
	10	20	30	40	50	60	70	80	90	100
11. after you are discharged from the hospital?	○	○	○	○	○	○	○	○	○	○
12. for one day after you are discharged from the hospital?	○	○	○	○	○	○	○	○	○	○
13. for one week after you are discharged from the hospital?	○	○	○	○	○	○	○	○	○	○
14. for the next three months?	○	○	○	○	○	○	○	○	○	○
15. for the next six months?	○	○	○	○	○	○	○	○	○	○
16. for the next 12 months?	○	○	○	○	○	○	○	○	○	○

	Not Very Confident				Moderately Confident					Very Confident
	10	20	30	40	50	60	70	80	90	100
17. How confident are you that you can resist taking one puff of a cigarette after you are after you are discharged from the hospital?	○	○	○	○	○	○	○	○	○	○
18. If you do take one puff of a cigarette, how confident are you that you can resist taking one puff of a cigarette the next day?	○	○	○	○	○	○	○	○	○	○
19. If you do take a puff of a cigarette, how confident are you that you can resist taking one of a cigarette the next week?	○	○	○	○	○	○	○	○	○	○
20. If you do take a puff of a cigarette, how confident are you that you can resist taking one of a cigarette the next month?	○	○	○	○	○	○	○	○	○	○

ID Number: _ _ _ _ _ _ _ _ _ _ _ (for internal use only) Date: _ _ / _ _ / _ _ (for internal use only) Study ID: 0 5 6

In the following questions, we would like to know your confidence in your ability to control your desire to use tobacco in specific situations. For each of the following questions, please fill in the circle that corresponds best to your certainty that you can <u>now</u> perform the following activities or tasks.

How confident are you that you will not take one puff of a cigarette when.....

	Not Very Confident				Moderately Confident					Very Confident
	10	20	30	40	50	60	70	80	90	100
21. you are in your home?	○	○	○	○	○	○	○	○	○	○
22. you are with others who are smoking ?	○	○	○	○	○	○	○	○	○	○
23. you are with others who are not smoking, but have cigarettes with them?	○	○	○	○	○	○	○	○	○	○
24. you are in pain?	○	○	○	○	○	○	○	○	○	○
25. you have just finished a meal ?	○	○	○	○	○	○	○	○	○	○
26. you are having a drink, such as coffee or tea?	○	○	○	○	○	○	○	○	○	○
27. you can smell tobacco in the air or on others who have just smoked?	○	○	○	○	○	○	○	○	○	○
28. you are riding in a car or truck?	○	○	○	○	○	○	○	○	○	○
29. you are craving tobacco?	○	○	○	○	○	○	○	○	○	○
30. you are angry or irritable?	○	○	○	○	○	○	○	○	○	○
31. you are anxious?	○	○	○	○	○	○	○	○	○	○
32. you are having difficulty concentrating?	○	○	○	○	○	○	○	○	○	○
33. you are restless?	○	○	○	○	○	○	○	○	○	○
34. you are impatient?	○	○	○	○	○	○	○	○	○	○
35. you are hungry?	○	○	○	○	○	○	○	○	○	○

ID Number: _ _ _ _ _ _ _ _ _ _ (for internal use only) Date: _ _ / _ _ / _ _ (for internal use only) Study ID: 056

How confident are you that you will not take one puff of a cigarette when.....

	Not Very Confident				Moderately Confident					Very Confident
	10	20	30	40	50	60	70	80	90	100
36. you cannot sleep?	○	○	○	○	○	○	○	○	○	○
37. you are drowsy and should not sleep?	○	○	○	○	○	○	○	○	○	○
38. you have a headache?	○	○	○	○	○	○	○	○	○	○
39. you are constipated?	○	○	○	○	○	○	○	○	○	○
40. you are sad?	○	○	○	○	○	○	○	○	○	○
41. you are depressed?	○	○	○	○	○	○	○	○	○	○

In the following questions, we would like to know your confidence in your ability to resist using tobacco during a hospital admission.

	Not Very Confident				Moderately Confident					Very Confident
	10	20	30	40	50	60	70	80	90	100
42. How confident are you that you can stop using tobacco (smoking) in the hospital?	○	○	○	○	○	○	○	○	○	○
43. How confident are you that you can stop using tobacco (smoking) for one day in the hospital?	○	○	○	○	○	○	○	○	○	○
44. How confident are you that you can resist taking one puff of a cigarette while in the hospital?	○	○	○	○	○	○	○	○	○	○
45. How confident are you that you can resist taking one puff of a cigarette while other people/visitors in your hospital room have cigarettes with them?	○	○	○	○	○	○	○	○	○	○

Instrument Number:

P T E S

(For internal use only)

Study ID: 0 5 6

PERCEIVED THERAPEUTIC EFFICACY SCALE- SMOKING ABTINENCE TREATMENT

Center for Research in Chronic Disorders

ID Number: | | | | | | | | | | | Administration Date: | | / | | | / | | | | |

(month) (day) (year)

0 ○ 1 ○ 2 ○

(FOR STAFF USE ONLY)

Please use the following example to answer all questions:

Shade circles like this: ●
Not like this: ⊗ ✓

For each statement listed below, please fill in the circle for the number that best describes your level of confidence in your "quit tobacco" treatment and how it may control your heart health, where 0 = No Confidence and 10 = Highest Confidence.

	No Confidence 0	1	2	3	4	5	6	7	8	9	Highest Confidence 10
1. My level of confidence in the ability of my "quit tobacco" treatment to control my heart health is:	○	○	○	○	○	○	○	○	○	○	○
2. My level of confidence in the ability of my "quit tobacco" treatment to prevent episodes of heart disease symptoms (such as chest pain and shortness of breath) is:	○	○	○	○	○	○	○	○	○	○	○
3. My level of confidence in the ability of my "quit tobacco" treatment to limit the number of heart disease complications (such as heart attacks and strokes) :	○	○	○	○	○	○	○	○	○	○	○
4. My level of confidence in the ability of my "quit tobacco" treatment to **prevent**from getting (more) heart disease complications is:	○	○	○	○	○	○	○	○	○	○	○
5. My level of confidence in my ability to control my heart health by abstaining from tobacco is:	○	○	○	○	○	○	○	○	○	○	○

ID Number: _ _ _ _ _ _ _ _ _ _ (for internal use only)

Date: _ _ / _ _ / _ _ (for internal use only)

Study ID: 0 5 6

	No Confidence 0	1	2	3	4	5	6	7	8	9	Highest Confidence 10
6. My level of confidence in the need to totally quit using tobacco each day to control my heart health is:	○	○	○	○	○	○	○	○	○	○	○
7. My overall level of confidence in the value of "quit tobacco" treatment is:	○	○	○	○	○	○	○	○	○	○	○
8. My level of confidence in the ability of my "quit tobacco" treatment general to control my health is:	○	○	○	○	○	○	○	○	○	○	○
9. My level of confidence in my doctor's/nurse's advice about my "quit tobacco" treatment is:	○	○	○	○	○	○	○	○	○	○	○
10. My overall level of confidence in my ability to manage my heart health is:	○	○	○	○	○	○	○	○	○	○	○

BIBLIOGRAPHY

AARDEX, L. (1998). Electronic Drug Exposure Monitor (pp. 1-6). Bundesplatz: AARDEX.

Abrams, D., Borrelli, B., Shadel, W., King, T., Bock, B., & Niaura, R. (1998). Adherence to treatment for nicotine dependence. In S. Shumaker, E. Shron, J. Ockene & W. McBee (Eds.), *The handbook of health behavior change* (2nd ed., pp. 137-165). New York, NY: Springer Publishing Company.

Abrams, D. B., & Biener, L. (1992). Motivational characteristics of smokers at the workplace: a public health challenge. *Preventive Medicine, 21*(6), 679-687.

Abrams, D. B., Herzog, T. A., Emmons, K. M., & Linnan, L. (2000). Stages of change versus addiction: a replication and extension. *Nicotine & Tobacco Research, 2*(3), 223-229.

Ad Hoc Working Group on Treatment of Tobacco, D. (2001). Realignment of the nation's tobacco agenda: the need to treat tobacco dependence. *Preventive Medicine, 32*(2), 95-100.

Adams, J. A. (1995). Smoking cessation counseling in adults and children: the clinician's role. *Maryland Medical Journal, 44*(10), 779-787.

Afifi, A. A., & Clark, V. (1996). *Computer-aided mulitivariate analysis*. Boca Raton: Chapman & Hall/CRC.

Ahijevych, K., & Gillespie, J. (1997). Nicotine dependence and smoking topography among black and white women. *Research in Nursing & Health, 20*(6), 505-514.

Ahijevych, K., & Parsley, L. A. (1999). Smoke constituent exposure and stage of change in black and white women cigarette smokers. *Addictive Behaviors, 24*(1), 115-120.

Albandar, J. M., Streckfus, C. F., Adesanya, M. R., & Winn, D. M. (2000). Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. *Journal of Periodontology, 71*(12), 1874-1881.

Albrecht, S., Payne, L., Stone, C. A., & Reynolds, M. D. (1998). A preliminary study of the use of peer support in smoking cessation programs for pregnant adolescents. *Journal of the American Academy of Nurse Practitioners, 10*(3), 119-125.

Allen, S. S., Hatsukami, D., Christianson, D., & Brown, S. (2000). Effects of transdermal nicotine on craving, withdrawal and premenstrual symptomatology in short-term smoking abstinence during different phases of the menstrual cycle. *Nicotine & Tobacco Research, 2*(3), 231-241.

Alterman, A. I., Gariti, P., Cook, T. G., & Cnaan, A. (1999). Nicodermal patch adherence and its correlates. *Drug & Alcohol Dependence, 53*(2), 159-165.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: APA Press.

American Psychiatric Association. (1996). Practice guidelines for the treatment of patients with nicotine dependence. *American Journal of Psychiatry, 153*(suppl), 1-31.

American Psychiatric Association. (2001). *Disease definition, epidemiology, and natural history*. Retrieved 08/08/2001, from http://www.psych.org/clin_res/pg_nicotine_2.cfm

American Thoracic Society. (1996). Cigarette smoking and health. *American Journal of Respiratory and Critical Care Medicine, 153*, 861-865.

Anonymous. (1996). *Pennsylvania County Health Profiles*. Harrisburg: The Hospital Association of Pennsylvania and Health Care Finance and Research Department.

Anonymous. (2001). *Mosby's GENRx - The complete reference for generic and brand drugs.* Retrieved May 1, 2001, 2001, from http://136.142.56.239/STATRef/tserver.dll?command=doGetDoc&sUI=&database=MEDICINE&useScheme=STATREFIntranet.Scheme&getDoc=default.html

Anthonisen, N. R., Skeans, M. A., Wise, R. A., Manfreda, J., Kanner, R. E., & Connett, J. E. (2005). The effects of a smoking cessation intervention on 14.5-year mortality: a randomized clinical trial. *Annals of Internal Medicine, 142*(4), 233-239.

Antonuccio, D. O., Boutilier, L. R., Ward, C. H., Morrill, G. B., & Graybar, S. R. (1992). The behavioral treatment of cigarette smoking. *Progress in Behavior Modification, 28*, 119-181.

Atkins, P. M., Mion, L. C., Mendelson, W., Palmer, R. M., Slomka, J., & Franko, T. (1997). Characteristics and outcomes of patients who self-extubate from ventilatory support: a case-control study. *Chest, 112*(5), 1317-1323.

Bandura, A. (1977). *Social learning theory*. Upper Saddle River, NJ: Prentice Hall.

Bandura, A. (1982). Self-efficacy mechanism of human agency. *American Psychologist, 37*(2), 122-147.

Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory.* Englewood Cliffs, NJ: Prentice-Hall.

Bandura, A. (1989). Human agency in social cognitive theory. *American Psychologist, 44*(9), 1175-1184.

Bandura, A. (1992). Self-efficacy mechanism in psychobiologic functioning. In R. Schwarzer (Ed.), *Self-efficacy: Thought control in action.* (pp. 355-394). Washington: Hemisphere Publishing Corporation.

Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: W. H. Freeman and Company.

Bandura, A., Cioffi, D., Taylor, C. B., & Brouillard, M. E. (1988). Perceived self-efficacy in coping with cognitive stressors and opioid activation. *Journal of Personality & Social Psychology, 55*(3), 479-488.

Bandura, A., Taylor, C. B., Williams, S. L., Mefford, I. N., & Barchas, J. D. (1985). Catecholamine secretion as a function of perceived coping self-efficacy. *Journal of Consulting & Clinical Psychology, 53*(3), 406-414.

Barnett, S. A. (1955). Displacement behavior. *Lancet, 2*, 1203-1208.

Barnum, H. (1994). *The economic burden of the global trade in tobacco.*, from http://www.worldbank.org/html/extdr/hnp/hddflash/other/oth0004.html

Bartecchi, C. E., MacKenzie, T. D., & Schrier, R. W. (1994). The human cost of tobacco use (first of two parts). *New England Journal of Medicine, 330*, 970-912.

Battaglia, G., Di Mario, F., Piccoli, A., Vianello, F., Farinati, F., & Naccarato, R. (1987). Clinical markers of slow healing and relapsing gastric ulcer. *Gut, 28*(2), 210-215.

Baudry, J. (1988). *Jean Nicot*. Lyon: La Manufacturer.

Beck, A., Wright, F., Newman, C., & Liese, B. (1993). *Cognitive therapy of substance abuse.* New York: Guilford Press.

Beck, G., Doyle, C., & Schachter, E. (1981). Smoking and lung function. *American Review of Respiratory Disease, 123*, 149-155.

Becona, E., & Garcia, M. P. (1995). Relation between the Tolerance Questionnaire (nicotine dependence) and assessment of carbon monoxide in smokers who participated in treatment for smoking. *Psychological Reports, 77*(3 Pt 2), 1299-1304.

Beemer, B. R. (1993). Hospital psychiatric units. Nonsmoking policies. *Journal of Psychosocial Nursing & Mental Health Services, 31*(4), 12-14.

Benowitz, N. (1992). Cigarette smoking and nicotine addiction. *Medical Clinics of North America, 76*, 415-437.

Benowitz, N. (1994). Acute biological effects of nicotine and its metabolites. In P. Clarke, M. Quik, F. Adlkofer & K. Thurau (Eds.), *Effects of nicotine on biological systems, II.* (pp. 9-16). Basel, Switzerland: Birkhauser Verlag.

Benowitz, N. (1998a). Cardiovascular toxicity of nicotine: Pharmacokinetic and pharmacodynamic considerations. In N. Benowitz (Ed.), *Nicotine safety and toxicity* (pp. 19-27). New York: Oxford University Press.

Benowitz, N. (1998b). Nicotine pharmacology and addiction. In N. Benowitz (Ed.), *Nicotine safety and toxicity* (pp. 3-16). New York: Oxford University Press.

Benowitz, N. L. (1988a). Pharmacologic aspects of cigarette smoking and nicotine addition. *New England Journal of Medicine, 319*(20), 1318-1330.

Benowitz, N. L. (1988b). Toxicity of nicotine: implications with regard to nicotine replacement therapy. *Progress in Clinical & Biological Research, 261*, 187-217.

Benowitz, N. L., Porchet, H., Sheiner, L., & Jacob, P., 3rd. (1988). Nicotine absorption and cardiovascular effects with smokeless tobacco use: Comparison with cigarettes and nicotine gum. *Clinical Pharmacology & Therapeutics, 44*(1), 23-28.

Berg, J., Dunbar-Jacob, J., & Sereika, S. M. (1997). An evaluation of a self-management program for adults with asthma. *Clinical Nursing Research, 6*(3), 225-238.

Bergler, E. (1946). Psychopathology of compulsive smoking. *Psychiatric Quarterly, 20*, 297-321.

Bergstrom, J., Eliasson, S., & Dock, J. (2000). A 10-year prospective study of tobacco smoking and periodontal health. *Journal of Periodontology, 71*(8), 1338-1347.

Biener, L., & Abrams, D. (1991). The contemplation ladder. Validation of a measure of readiness to consider smoking cessation. *Health Psychology, 10*, 360-365.

Bjornson, W., Rand, C., Connett, J. E., Lindgren, P., Nides, M., Pope, F., Buist, A. S., Hoppe-Ryan, C., & P, O. H. (1995). Gender differences in smoking cessation after 3 years in the Lung Health Study. *American Journal of Public Health, 85*(2), 223-230.

Blondal, T., Gudmundsson, L. J., Tomasson, K., Jonsdottir, D., Hilmarsdottir, H., Kristjansson, F., Nilsson, F., & Bjornsdottir, U. S. (1999). The effects of fluoxetine combined with nicotine inhalers in smoking cessation--a randomized trial. *Addiction, 94*(7), 1007-1015.

Bobo, J. K., Lando, H. A., Walker, R. D., & McIlvain, H. E. (1996). Predictors of tobacco quit attempts among recovering alcoholics. *Journal of Substance Abuse, 8*(4), 431-443.

Bohadana, A., Nilsson, F., Rasmussen, T., & Martinet, Y. (2000). Nicotine inhaler and nicotine patch as a combination therapy for smoking cessation: a randomized, double-blind, placebo-controlled trial. *Archives of Internal Medicine, 160*(20), 3128-3134.

Bolliger, C. T., Zellweger, J. P., Danielsson, T., van Biljon, X., Robidou, A., Westin, A., Perruchoud, A. P., & Sawe, U. (2000). Smoking reduction with oral nicotine inhalers: double blind, randomised clinical trial of efficacy and safety. *British Medical Journal, 321*(7257), 329-333.

Bolman, C., & de Vries, H. (1998). Psycho-social determinants and motivational phases in smoking behavior of cardiac inpatients. *Preventive Medicine, 27*(5 Pt 1), 738-747.

Bolman, C., de Vries, H., & van Breukelen, G. (2002). A minimal-contact intervention for cardiac inpatients: long-term effects on smoking cessation. *Preventive Medicine, 35*(2), 181-192.

Borland, R. (1992). Relationships between mood around slip-up and recovery of abstinence in smoking cessation attempts. *International Journal of the Addictions, 27*(9), 1079-1086.

Brill, A. A. (1922). Tobacco and the individual. *International Journal of Psychoanalysis, 3*, 430-444.

Brown, D. C. (1996). Smoking cessation in pregnancy. *Canadian Family Physician, 42*, 102-105.

Burchfiel, C. M., Marcus, E. B., Curb, J. D., Maclean, C. J., Vollmer, W. M., Johnson, L. R., Fong, K. O., Rodriguez, B. L., Masaki, K. H., & Buist, A. S. (1995). Effects of smoking and smoking cessation on longitudinal decline in pulmonary function. *American Journal of Respiratory & Critical Care Medicine, 151*(6), 1778-1785.

Burke, L. E., & Dunbar-Jacob, J. (1995). Adherence to medication, diet, and activity recommendations: from assessment to maintenance. *Journal of Cardiovascular Nursing, 9*(2), 62-79.

Burke, L. E., Dunbar-Jacob, J., Sereika, S., & Ewart, C. K. (2003). Development and testing of the Cholesterol-Lowering Diet Self-Efficacy Scale. *European Journal of Cardiovascular Nursing, 2*(4), 265-273.

Campbell, I. A., Prescott, R. J., & Tjeder-Burton, S. M. (1996). Transdermal nicotine plus support in patients attending hospital with smoking-related diseases: a placebo-controlled study. *Respiratory Medicine, 90*(1), 47-51.

Catley, D., O'Connell, K. A., & Shiffman, S. (2000). Absentminded lapses during smoking cessation. *Psychology of Addictive Behaviors, 14*(1), 73-76.

Center for Disease Control and Prevention. (1992). Public health focus: Effectiveness of smoking-control strategies--United States. *Morbidity & Mortality Weekly Report, 41*(35), 645-647, 653.

Center for Disease Control and Prevention. (1993a). Cigarette smoking-attributable mortality and years of potential life lost--United States, 1990. *Morbidity & Mortality Weekly Report, 42*(33), 645-649.

Center for Disease Control and Prevention. (1993b). Smoking cessation during previous year among adults--United States, 1990 and 1991. *Morbidity & Mortality Weekly Report, 42*(26), 504-507.

Center for Disease Control and Prevention. (1993c). Use of smokeless tobacco among adults - United States, 1991. *Morbidity & Mortality Weekly Report, 42*(14), 263-266.

Center for Disease Control and Prevention. (1994). Prevalence of disability and associated health conditions - United States, 1991-1992. *Morbidity & Mortality Weekly Report, 43*(40), 730-731-, 737-739.

Center for Disease Control and Prevention. (1995a). Smoking-attributable mortality--Mexico, 1992. *Morbidity & Mortality Weekly Report, 44*(19), 372-373, 379-381.

Center for Disease Control and Prevention. (1995b). Symptoms of substance dependence associated with use of cigarettes, alcohol, and illicit drugs - United States, 1991-1992. *Morbidity & Mortality Weekly Report, 44*(44), 830-831, 837-839.

Center for Disease Control and Prevention. (1997a). Efforts to quit smoking among persons with a history of alcohol problems--Iowa, Kansas, and Nebraska, 1995-1996. *Morbidity & Mortality Weekly Report, 46*(48), 1144-1148.

Center for Disease Control and Prevention. (1997b). Impact of promotion of the Great American Smokeout and availability of over-the-counter nicotine medications, 1996. *Morbidity & Mortality Weekly Report, 46*(37), 867-871.

Center for Disease Control and Prevention. (1997c). Perspectives in disease prevention and health promotion smoking attributable mortality and years of potential life lost - United States, 1984. *Morbidity & Mortality Weekly Report, 46*(20), 444-451.

Center for Disease Control and Prevention. (1998). Selected cigarette smoking initiation and quitting behaviors among high school students--United States, 1997. *Morbidity & Mortality Weekly Report, 47*(19), 386-389.

Center for Disease Control and Prevention. (1999a). Chronic diseases and their risk factors: the nation's leading causes of death. Atlanta, GA: U.S. Department of Health and Human Services Centers for Disease Control and Prevention.

Center for Disease Control and Prevention. (1999b). Cigarette smoking among adults-United States, 1997. *Morbidity & Mortality Weekly Report, 48*(43), 986-993.

Center for Disease Control and Prevention. (1999c). Cigarette smoking among adults--United States, 1997. *Morbidity & Mortality Weekly Report, 48*(43), 993-996.

Center for Disease Control and Prevention. (1999d). *State tobacco control highlights - 1999*. Atlanta, GA: Center for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, CDC Publication No. 099-5621.

Center for Disease Control and Prevention. (1999e). State-specific prevalence of current cigarette and cigar smoking among adults--United States, 1998. *Morbidity & Mortality Weekly Report, 48*(45), 1034-1039.

Center for Disease Control and Prevention. (1999f). Tobacco use--United States, 1900-1999. *Morbidity & Mortality Weekly Report, 48*(43), 986-993.

Center for Disease Control and Prevention. (2000a). State-specific prevalence of current cigarette smoking among adults and the proportion of adults who work in a smoke-free environment--United States, 1999. *Morbidity Mortality Weekly Report, 49*(43), 978-982.

Center for Disease Control and Prevention. (2000b). You can quit smoking - a consumer guide. Silver Spring, MD: Publications Clearinghouse, U.S. Dept. of Human and Health Services, Public Health Service.

Center for Disease Control and Prevention. (2002). Annual smoking-attributable mortality, years of potential life lost, and economic costs--United States, 1995-1999. *Morbidity Mortalality Weekly Report, 51*(14), 300-303.

Center for Disease Control and Prevention. (2004a). Cigarette Smoking-Attributable Morbidity — United States, 2000. *Morbidity Mortality Weekly Report, 52*(35), 842-844.

Center for Disease Control and Prevention. (2004b). Prevalence of Cigarette Use Among 14 Racial/Ethnic Populations - United States, 1999–2001. *Morbidity & Mortality Weekly Report, 53*(3), 49-52.

Center for Disease Control and Prevention. (2004c). State medicaid coverage for tobacco-dependence treatments--United States, 1994-2002. *Morbidity Mortalality Weekly Report, 53*(3), 54-57.

Center for Disease Control and Prevention. (2004d). State-Specific Prevalence of Current Cigarette Smoking Among Adults — United States, 2003. *Morbidity Mortality Weekly Report, 53*(44), 1035-1037.

Chandola, T., Head, J., & Bartley, M. (2004). Socio-demographic predictors of quitting smoking: how important are household factors? *Addiction, 99*(6), 770-777.

Chiang, V. L., Castleden, W. M., & Leahy, M. F. (1992). Detection of reversible platelet aggregates in the blood of smokers and ex-smokers with peripheral vascular disease. *Medical Journal of Australia, 156*(9), 601-603.

Chollat-Traquet, C. (1992). Tobacco or health: a WHO programme. *European Journal of Cancer, 28*(2-3), 311-315.

Christen, W. G., Manson, J. E., Seddon, J. M., Glynn, R. J., Buring, J. E., Rosner, B., & Hennekens, C. H. (1992). A prospective study of cigarette smoking and risk of cataract in men [see comments]. *Journal of the American Medical Association, 268*(8), 989-993.

Christensen, A. J., Moran, P. J., Ehlers, S. L., Raichle, K., Karnell, L., & Funk, G. (1999). Smoking and drinking behavior in patients with head and neck cancer: effects of behavioral self-blame and perceived control. *Journal of Behavioral Medicine, 22*(5), 407-418.

Cinciripini, P. M., Lapitsky, L., Seay, S., Wallfisch, A., Kitchens, K., & Van Vunakis, H. (1995). The effects of smoking schedules on cessation outcome: can we improve on common methods of gradual and abrupt nicotine withdrawal? *Journal of Consulting & Clinical Psychology, 63*(3), 388-399.

Cinciripini, P. M., Lapitsky, L., Seay, S., Wallfisch, A., Meyer, W. J., 3rd, & van Vunakis, H. (1995). A placebo-controlled evaluation of the effects of buspirone on smoking cessation: differences between high- and low-anxiety smokers. *Journal of Clinical Psychopharmacology, 15*(3), 182-191.

Cinciripini, P. M., Lapitsky, L. G., Wallfisch, A., Mace, R., Nezami, E., & Van Vunakis, H. (1994). An evaluation of a multicomponent treatment program involving scheduled smoking and relapse prevention procedures: initial findings. *Addictive Behaviors, 19*(1), 13-22.

Cinciripini, P. M., Wetter, D. W., & McClure, J. B. (1997). Scheduled reduced smoking: effects on smoking abstinence and potential mechanisms of action. *Addictive Behaviors, 22*(6), 759-767.

Clarke, P. (1993). Nicotine dependence-mechanisms and therapeutic strategies. In S. Wonnacott & G. Lunt (Eds.), *Neurochemistry of drug dependence.* (pp. 83-96). London, England: Portland Press.

Coburn, R. F., Forster, R. E., & Kane, P. B. (1965). Considerations of the physiological variables that determine the blood carboxyhemoglobin concentration in man. *Journal of Clinical Investigations, 44*(11), 1899-1910.

Cohen, D., & Barton, G. (1998). The cost to society of smoking cessation. *Thorax, 53*(Suppl 2), S38-42.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health Society and Behavior, 24*(4), 385-396.

Colletti, G., Payne, T. J., & Rizzo, A. A. (1987). Treatment of cigarette smoking. In *Nirenberg, Ted D. (Ed); Maisto, Stephen A. (Ed). (1987). Developments in the assessment and treatment of addictive behaviors. Developments in clinical psychology.* (pp. 243-276). Stamford, CT, US: Ablex Publishing Corp.

Collins, R. L., Emont, S. L., & Zywiak, W. H. (1990). Social influence processes in smoking cessation: postquitting predictors of long-term outcome. *Journal of Substance Abuse, 2*(4), 389-403.

Condiotte, M. M., & Lichtenstein, E. (1981). Self-efficacy and relapse in smoking cessation programs. *Journal of Consulting & Clinical Psychology, 49*(5), 648-658.

Conover, W. J. (1980). *Practical nonparametric statistics* (2nd ed.). New York: John Wiley & Sons.

Conrad, K. M., Campbell, R. T., Edington, D. W., Faust, H. S., & Vilnius, D. (1996). The worksite environment as a cue to smoking reduction. *Research in Nursing & Health, 19*(1), 21-31.

Cooper, T. V., Klesges, R. C., Debon, M. W., Zbikowski, S. M., Johnson, K. C., & Clemens, L. H. (2005). A placebo controlled randomized trial of the effects of phenylpropanolamine and nicotine gum on cessation rates and postcessation weight gain in women. *Addiction Behavior, 30*(1), 61-75.

Corrao, M. A., Guindon, G. E., Cokkinides, V., & Sharma, N. (2000). Building the evidence base for global tobacco control. *Bulletin of the World Health Organization, 78*(7), 884-890.

Cottone, M., Rosselli, M., Orlando, A., Oliva, L., Puleo, A., Cappello, M., Traina, M., Tonelli, F., & Pagliaro, L. (1994). Smoking habits and recurrence in Crohn's disease. *Gastroenterology, 106*(3), 643-648.

Covey, L. S., Glassman, A. H., & Stetner, F. (1997). Major depression following smoking cessation. *American Journal of Psychiatry, 154*(2), 263-265.

Covey, L. S., Glassman, A. H., & Stetner, F. (1998). Cigarette smoking and major depression. *Journal of Addictive Diseases, 17*(1), 35-46.

Covey, L. S., Sullivan, M. A., Johnston, J. A., Glassman, A. H., Robinson, M. D., & Adams, D. P. (2000). Advances in non-nicotine pharmacotherapy for smoking cessation. *Drugs, 59*(1), 17-31.

Craig, D., Parrott, A., & Coomber, J. A. (1992). Smoking cessation in women: effects of the menstrual cycle. *International Journal of the Addictions, 27*(6), 697-706.

Craig, S., & Rees, T. D. (1985). The effects of smoking on experimental skin flaps in hamsters. *Plastic Reconstructive Surgery, 75*(6), 842-846.

Crooks, P., & Dwoskin, L. (1997). Contribution of CNS nicotine metabolites to the neuropharmacological effects of nicotine and tobacco smoking. *Biochemical Pharmacology, 54*, 743-753.

Cuff, M. J., McQuade, M. J., Scheidt, M. J., Sutherland, D. E., & Van Dyke, T. E. (1989). The presence of nicotine on root surfaces of periodontally diseased teeth in smokers. *Journal of Periodontology, 60*(10), 564-569.

Cummings, S. R., & Richard, R. J. (1988). Optimum cutoff points for biochemical validation of smoking status. *American Journal of Public Health, 78*(5), 574-575.

Curry, S. (1993). Self-help interventions for smoking cessation. *Journal of Consulting & Clinical Psychology, 61*, 790-803.

Dalack, G. W., Glassman, A. H., Rivelli, S., Covey, L., & Stetner, F. (1995). Mood, major depression, and fluoxetine response in cigarette smokers. *American Journal of Psychiatry, 152*(3), 398-403.

Dale, L. C., Olsen, D. A., Patten, C. A., Schroeder, D. R., Croghan, I. T., Hurt, R. D., Offord, K. P., & Wolter, T. D. (1997). Predictors of smoking cessation among elderly smokers treated for nicotine dependence. *Tobacco Control, 6*(3), 181-187.

Daughton, D. M., Fortmann, S. P., Glover, E. D., Hatsukami, D. K., Heatley, S. A., Lichtenstein, E., Repsher, L., Millatmal, T., Killen, J. D., Nowak, R. T., Ullrich, F., Patil, K. D., & Rennard, S. I. (1999). The smoking cessation efficacy of varying doses of nicotine patch delivery systems 4 to 5 years post-quit day. *Preventive Medicine, 28*(2), 113-118.

David, S. P., Niaura, R., Papandonatos, G. D., Shadel, W. G., Burkholder, G. J., Britt, D. M., Day, A., Stumpff, J., Hutchison, K., Murphy, M., Johnstone, E., Griffiths, S. E., & Walton, R. T. (2003). Does the DRD2-Taq1 A polymorphism influence treatment response to bupropion hydrochloride for reduction of the nicotine withdrawal syndrome? *Nicotine Tobacco Research, 5*(6), 935-942.

de Vries, H., & Backbier, E. (1994). Self-efficacy as an important determinant of quitting among pregnant women who smoke: the phi-pattern. *Preventive Medicine, 23*(2), 167-174.

de Vries, H., Mudde, A., Dijkstra, A., & Willemsen, M. (1998). Differential beliefs, perceived social influences, and self-efficacy expectations among smokers in various motivational phases. *Preventive Medicine, 27*(5 Pt 1), 681-689.

DeBon, M., Klesges, R. C., & Klesges, L. M. (1995). Symptomatology across the menstrual cycle in smoking and nonsmoking women. *Addictive Behaviors, 20*(3), 335-343.

DeBusk, R. F., Miller, N. H., Superko, H. R., Dennis, C. A., Thomas, R. J., Lew, H. T., Berger, W. E., 3rd, Heller, R. S., Rompf, J., & Gee, D. (1994). A case-management system for coronary risk factor modification after acute myocardial infarction. *Annals of Internal Medicine, 120*(9), 721-729.

Devins, G. M., & Edwards, P. J. (1988). Self-efficacy and smoking reduction in chronic obstructive pulmonary disease. *Behavioral Research & Therapy, 26*(2), 127-135.

Dew, M. A., Roth, L. H., Thompson, M. E., Kormos, R. L., & Griffith, B. P. (1996). Medical compliance and its predictors in the first year after heart transplantation. *Journal of Heart & Lung Transplantation, 15*(6), 631-645.

Doll, R., & Hill, A. B. (1954). Smoking and lung cancer. *British Medical Journal, 2*, 808-809.

Dornelas, E. A., Sampson, R. A., Gray, J. F., Waters, D., & Thompson, P. D. (2000). A randomized controlled trial of smoking cessation counseling after myocardial infarction. *Preventive Medicine, 30*(4), 261-268.

Dunbar-Jacob, J., Schlenk, E., Burke, L., & Matthews, J. (1998a). Predictors of patient adherence: Patient characteristics. In S. Shumaker, E. Schron & e. al. (Eds.), *The handbook of health behavior change.* (2nd ed., pp. 491-511). New York, NY, US: Springer Publishing Co, Inc.

Dunbar-Jacob, J., Schlenk, E., & Caruthers, D. (2002a). Adherence in the management of chronic disorders. In A. Christensen (Ed.), *Chronic medical disorders: Behavioral medicine's perspective* (pp. 69-82). Oxford: Blackwell Publishers, Limited.

Dunbar-Jacob, J., Schlenk, E. A., & Caruthers, D. (2002b). Adherence in the management of chronic disorders. In A. J. Christiansen & M. H. Antoni (Eds.), *Chronic medical disorders: Behavioral medicine's perspective* (pp. 69-82). Oxford: Blackwell Publishers, Ltd.

Dunbar-Jacob, J., Sereika, S., Burke, L., Kwoh, C. K., Rosella, J., McCall, M., Locke, C., Holmes, J., Bondi, K., Canty, T., & Starz, T. (1993). Perceived treatment efficacy: Assessment in rheumatoid arthritis. *Annals of Behavioral Medicine, 15 (Supplement)*, S147.

Dunbar-Jacob, J., Sereika, S., Rohay, J., & Burke, L. (1998b). Electronic methods in assessing adherence to medical regimens. In D. S. Krantz & A. Baum (Eds.), *Technology and methods in behavioral medicine.* (pp. 95-113). Mahwah, NJ, US: Lawrence Erlbaum Associates, Inc., Publishers.

Elder, J., Ayala, G., & Harris, S. (1999). Theories and intervention approaches to health-behavior change in primary care. *American Journal of Preventive Medicine, 17*(4), 275-284.

Emmons, K. M., & Goldstein, M. G. (1992). Smokers who are hospitalized: A window of opportunity for cessation interventions. *Preventive Medicine, 21*(2), 262-269.

Emmons, K. M., Goldstein, M. G., Roberts, M., Cargill, B., Sherman, C. B., Millman, R., Brown, R., & Abrams, D. B. (2000). The use of nicotine replacement therapy during hospitalization. *Annals of Behavioral Medicine, 22*(4), 325-329.

Emmons, K. M., Kawachi, I., & Barclay, G. (1997). Tobacco control: A brief review of its history and prospects for the future. *Hematology - Oncology Clinics of North America, 11*(2), 177-195.

Fagerstrom, K. O., & Schneider, N. G. (1989). Measuring nicotine dependence: a review of the Fagerstrom Tolerance Questionnaire. *Journal of Behavioral Medicine, 12*(2), 159-182.

Ferry, L. H. (1999). Non-nicotine pharmacotherapy for smoking cessation. *Primary Care; Clinics in Office Practice, 26*(3), 653-669.

Finnegan, J., Larson, P., & Haag, H. (1945). The role of nicotine in the cigarette habit. *Science, 102*, 94-96.

Fiore, M. C., Bailey, W. C., Cohen, S. J., & et al. (1996). *Smoking cessation. Clinical practice guideline.* Rockville, MD: U.S. Department of Health and Human Services. Public Health Service.

Fiore, M. C., Bailey, W. C., Cohen, S. J., & et al. (2000). *Treating tobacco use and dependence. Clinical practice guideline.* Rockville, MD: U.S. Department of Health and Human Services. Public Health Service.

Fiore, M. C., Croyle, R. T., Curry, S. J., Cutler, C. M., Davis, R. M., Gordon, C., Healton, C., Koh, H. K., Orleans, C. T., Richling, D., Satcher, D., Seffrin, J., Williams, C., Williams, L. N., Keller, P. A., & Baker, T. B. (2004). Preventing 3 million premature deaths and helping 5 million smokers quit: a national action plan for tobacco cessation. *American Journal of Public Health, 94*(2), 205-210.

Fiore, M. C., & Jorenby, D. E. (1992). Smoke-free hospitals. A time for universal action. *Chest, 102*(5), 1317-1318.

Fiore, M. C., Jorenby, D. E., Schensky, A. E., Smith, S. S., Bauer, R. R., & Baker, T. B. (1995). Smoking status as the new vital sign: effect on assessment and intervention in patients who smoke. *Mayo Clinic Proceedings, 70*(3), 209-213.

Fiore, M. C., Novotny, T. E., Pierce, J. P., Giovino, G. A., Hatziandreu, E. J., Newcomb, P. A., Surawicz, T. S., & Davis, R. M. (1990). Methods used to quit smoking in the United States. Do cessation programs help? *Journal of the American Medical Association, 263*(20), 2760-2765.

Fortmann, S. P., & Killen, J. D. (1995). Nicotine gum and self-help behavioral treatment for smoking relapse prevention: results from a trial using population-based recruitment. *Journal of Consulting & Clinical Psychology, 63*(3), 460-468.

Fowler, J. S., Volkow, N. D., Logan, J., Pappas, N., King, P., MacGregor, R., Shea, C., Garza, V., & Gatley, S. J. (1998). An acute dose of nicotine does not inhibit MAO B in baboon brain in vivo. *Life Sciences, 63*(2), PL19-23.

Fowler, J. S., Volkow, N. D., Wang, G. J., Pappas, N., Logan, J., MacGregor, R., Alexoff, D., Shea, C., Schlyer, D., Wolf, A. P., Warner, D., Zezulkova, I., & Cilento, R. (1996). Inhibition of monoamine oxidase B in the brains of smokers. *Nature, 379*(6567), 733-736.

Fowler, J. S., Volkow, N. D., Wang, G. J., Pappas, N., Logan, J., MacGregor, R., Alexoff, D., Wolf, A. P., Warner, D., Cilento, R., & Zezulkova, I. (1998). Neuropharmacological actions of cigarette smoke: brain monoamine oxidase B (MAO B) inhibition. *Journal of Addictive Diseases, 17*(1), 23-34.

Fowler, J. S., Wang, G. J., Volkow, N. D., Franceschi, D., Logan, J., Pappas, N., Shea, C., MacGregor, R. R., & Garza, V. (1999). Smoking a single cigarette does not produce a measurable reduction in brain MAO B in non-smokers. *Nicotine & Tobacco Research, 1*(4), 325-329.

France, E. K., Glasgow, R. E., & Marcus, A. C. (2001). Smoking cessation interventions among hospitalized patients: what have we learned? *Preventive Medicine, 32*(4), 376-388.

Freedman, B. (1948). Conditioned reflex and psychodynamic equivalent in alcohol addiction. An illustration of psychoanalytic neurology, with rudimentary equations. *Quarterly Journal of Alcohol, 9*, 53-71.

Freund, K. M., D'Agostino, R. B., Belanger, A. J., Kannel, W. B., & Stokes, J., 3rd. (1992). Predictors of smoking cessation: the Framingham Study. *American Journal of Epidemiology, 135*(9), 957-964.

Friedman, L., Furberg, C., & De Mets, D. (1996). *Fundamentals of clinical trials.* (3rd ed.). New York: Springer.

Froelicher, E. S., Li, W. W., Mahrer-Imhof, R., Christopherson, D., & Stewart, A. L. (2004a). Women's initiative for non-smoking (WINS) VI: reliability and validity of health and psychosocial measures in women smokers with cardiovascular disease. *Heart Lung, 33*(3), 162-175.

Froelicher, E. S., Sohn, M., Max, W., & Bacchetti, P. (2004b). Women's Initiative for Nonsmoking-VII: evaluation of health service utilization and costs among women smokers with cardiovascular disease. *Journal of Cardiopulmonary Rehabilitation, 24*(4), 218-228.

Gamberino, W. C., & Gold, M. S. (1999). Neurobiology of tobacco smoking and other addictive disorders. *Psychiatric Clinics of North America, 22*(2), 301-312.

Garvey, A. J., Bliss, R. E., Hitchcock, J. L., Heinold, J. W., & Rosner, B. (1992). Predictors of smoking relapse among self-quitters: a report from the Normative Aging Study. *Addictive Behaviors, 17*(4), 367-377.

Gentry, M. V., Hammersley, J. J., Hale, C. R., Nuwer, P. K., & Meliska, C. J. (2000). Nicotine patches improve mood and response speed in a lexical decision task. *Addictive Behaviors, 25*(4), 549-557.

Gilbert, D. G., Crauthers, D. M., Mooney, D. K., McClernon, F. J., & Jensen, R. A. (1999). Effects of monetary contingencies on smoking relapse: influences of trait depression, personality, and habitual nicotine intake. *Experimental & Clinical Psychopharmacology, 7*(2), 174-181.

Glasgow, R. E., Stevens, V. J., Vogt, T. M., Mullooly, J. P., & Lichtenstein, E. (1991). Changes in smoking associated with hospitalization: quit rates, predictive variables, and intervention implications. *American Journal of Health Promotion, 6*(1), 24-29.

Glassman, A. H. (1993). Cigarette smoking: implications for psychiatric illness. *American Journal of Psychiatry, 150*(4), 546-553.

Glassman, A. H., Covey, L. S., Dalack, G. W., Stetner, F., Rivelli, S. K., Fleiss, J., & Cooper, T. B. (1993). Smoking cessation, clonidine, and vulnerability to nicotine among dependent smokers. *Clinical Pharmacology & Therapeutics, 54*(6), 670-679.

Glassman, S. D., Anagnost, S. C., Parker, A., Burke, D., Johnson, J. R., & Dimar, J. R. (2000). The effect of cigarette smoking and smoking cessation on spinal fusion. *Spine, 25*(20), 2608-2615.

Glynn, R. J., Buring, J. E., Manson, J. E., LaMotte, F., & Hennekens, C. H. (1994). Adherence to aspirin in the prevention of myocardial infarction. The Physicians' Health Study. *Archives of Internal Medicine, 154*(23), 2649-2657.

Goldstein, A. (1994). *Addiction: From biology to drug policy.* New York, NY: W.H. Freeman and Co.

Goldstein, A., Hellier, A., Fitzgerald, S., Stegall, T., & FIshcher, P. (1987). Hospital nurse counseling of patients who smoke. *American Journal of Public Health, 77*(10), 1333-1334.

Goldstein, A. O., Westbrook, W. R., Howell, R. E., & Fischer, P. M. (1992). Hospital efforts in smoking control: Remaining barriers and challenges. *Journal of Family Practice, 34*(6), 729-734.

Goldstein, M., Niaura, R., Willey-Lessne, C., DePue, J., Eaton, C., Rakowski, W., & Dube, C. (1997). Physicians counseling smokers. A population-based survey of patients' perceptions of health care provider-delivered smoking cessation interventions. *Archives of Internal Medicine, 157*(12), 1313-1319.

Goodman, J. (1993). *Tobacco in history: The culture of dependence.* London: Routledge, Inc.

Gori, G., Benowitz, N., & Lynch, C. (1986). Mouth versus deep airways absorption of nicotine in cigarette smokers. *Pharmacology Biochemistry, and Behavior, 25*, 1181-1184.

Griebel, B., Wewers, M. E., & Baker, C. A. (1998). The effectiveness of a nurse-managed minimal smoking-cessation intervention among hospitalized patients with cancer. *Oncology Nursing Forum, 25*(5), 897-902.

Gritz, E. R., Carmack, C. L., de Moor, C., Coscarelli, A., Schacherer, C. W., Meyers, E. G., & Abemayor, E. (1999a). First year after head and neck cancer: quality of life. *Journal of Clinical Oncology, 17*(1), 352-360.

Gritz, E. R., Schacherer, C., Koehly, L., Nielsen, I. R., & Abemayor, E. (1999b). Smoking withdrawal and relapse in head and neck cancer patients. *Head Neck, 21*(5), 420-427.

Grossi, S. G., Zambon, J., Machtei, E. E., Schifferle, R., Andreana, S., Genco, R. J., Cummins, D., & Harrap, G. (1997). Effects of smoking and smoking cessation on healing after mechanical periodontal therapy. *Journal of the American Dental Association, 128*(5), 599-607.

Groudine, S. B., & Morley, J. N. (1996). Recent problems with paracervical vasopressin: a possible synergistic reaction with nicotine. *Medical Hypotheses, 47*(1), 19-21.

Gulliver, S. B., Hughes, J. R., Solomon, L. J., & Dey, A. N. (1995). An investigation of self-efficacy, partner support and daily stresses as predictors of relapse to smoking in self-quitters. *Addiction, 90*(6), 767-772.

Gwaltney, C., Shiffman, S., Norman, G., Paty, J., Kassel, J., Gnys, M., Hickcox, M., Waters, A., & Balabanis, M. (2001). Does smoking abstinence self-efficacy vary across situations? Identifying context-specificity within the relapse situation efficacy questionnaire. *Journal of Consulting & Clinical Psychology, 69*(3), 516-527.

Haaga, D. A. (1990). Issues in relating self-efficacy to smoking relapse: importance of an "Achilles' heel" situation and of prior quitting experience. *Journal of Substance Abuse, 2*(2), 191-200.

Haaga, D. A., & Stewart, B. L. (1992). Self-efficacy for recovery from a lapse after smoking cessation. *Journal of Consulting & Clinical Psychology, 60*(1), 24-28.

Hajek, P., & Stead, L. F. (2000). Aversive smoking for smoking cessation. *Cochrane Database of Systematic Reviews [computer file]*(2), CD000546.

Hajek, P., Taylor, T. Z., & Mills, P. (2002). Brief intervention during hospital admission to help patients to give up smoking after myocardial infarction and bypass surgery: randomised controlled trial. *British Medical Journal, 324*(7329), 87-89.

Hall, S. M., Munoz, R. F., Reus, V. I., & Sees, K. L. (1993). Nicotine, negative affect, and depression. *Journal of Consulting & Clinical Psychology, 61*(5), 761-767.

Hall, S. M., Munoz, R. F., Reus, V. I., Sees, K. L., Duncan, C., Humfleet, G. L., & Hartz, D. T. (1996). Mood management and nicotine gum in smoking treatment: a therapeutic contact and placebo-controlled study. *Journal of Consulting & Clinical Psychology, 64*(5), 1003-1009.

Hall, S. M., Reus, V. I., Munoz, R. F., Sees, K. L., Humfleet, G., Hartz, D. T., Frederick, S., & Triffleman, E. (1998). Nortriptyline and cognitive-behavioral therapy in the treatment of cigarette smoking. *Archives of General Psychiatry, 55*(8), 683-690.

Halpern, M. T., Schmier, J. K., Ward, K. D., & Klesges, R. C. (2000). Smoking cessation in hospitalized patients. *Respiratory Care, 45*(3), 330-336.

Hammond, E. C., & Horn, D. (1954). The relationship between human smoking habits and death rates. A follow-up study of 187,766 men. *Journal of the American Medical Association, 155*, 1316-1328.

Hance, A. J., Basset, F., Saumon, G., Danel, C., Valeyre, D., Battesti, J. P., Chretien, J., & Georges, R. (1986). Smoking and interstitial lung disease. The effect of cigarette smoking on the incidence of pulmonary histiocytosis X and sarcoidosis. *Annals of the New York Academy of Sciences, 465*, 643-656.

Hasdai, D., Garratt, K. N., Grill, D. E., Lerman, A., & Holmes, D. R., Jr. (1997). Effect of smoking status on the long-term outcome after successful percutaneous coronary revascularization. *New England Journal of Medicine, 336*(11), 755-761.

Hasdai, D., Garratt, K. N., Grill, D. E., Mathew, V., Lerman, A., Gau, G. T., & Holmes, D. R., Jr. (1998). Predictors of smoking cessation after percutaneous coronary revascularization. *Mayo Clinic Proceedings, 73*(3), 205-209.

Haukkala, A., Uutela, A., Vartiainen, E., McAlister, A., & Knekt, P. (2000). Depression and smoking cessation: the role of motivation and self-efficacy. *Addictive Behaviors, 25*(2), 311-316.

Haverstock, B. D., & Mandracchia, V. J. (1998). Cigarette smoking and bone healing: implications in foot and ankle surgery. *Journal of Foot and Ankle Surgery, 37*(1), 69-74; discussion 78.

Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerstrom, K. O. (1991). The Fagerstrom Test for Nicotine Dependence: a revision of the Fagerstrom Tolerance Questionnaire. *British Journal of Addiction, 86*(9), 1119-1127.

Helgason, A. R., Tomson, T., Lund, K. E., Galanti, R., Ahnve, S., & Gilljam, H. (2004). Factors related to abstinence in a telephone helpline for smoking cessation. *European Journal of Public Health, 14*(3), 306-310.

Hennekens, C., & Buring, J. E. (1987). *Epidemiology in medicine.* (First ed.). Boston: Little, Brown and Company.

Henningfield, J., Cohen, C., & Pickworth, W. (1993). Psychopharmacology of nicotine. In C. Orleans & J. Slade (Eds.), *Nicotine addiction: principles of management.* (pp. 24-45). New York, NY: Oxford University Press.

Henningfield, J. E. (1995). Nicotine medications for smoking cessation. *New England Journal of Medicine, 333*(18), 1196-1203.

Henningfield, J. E., Clayton, R., & Pollin, W. (1990). Involvement of tobacco in alcoholism and illicit drug use. *British Journal of Addiction, 85*(2), 279-291.

Henningfield, J. E., & Keenan, R. M. (1993). Nicotine delivery kinetics and abuse liability. *Journal of Consulting & Clinical Psychology, 61*(5), 743-750.

Henningfield, J. E., & Woodson, P. P. (1989). Dose-related actions of nicotine on behavior and physiology: review and implications for replacement therapy for nicotine dependence. *Journal of Substance Abuse, 1*(3), 301-317.

Hennrikus, D. J., Lando, H. A., McCarty, M. C., Klevan, D., Holtan, N., Huebsch, J. A., Jestus, S., Pentel, P. R., Pine, D., Sullivan, S., Swenson, K., & Vessey, J. (2005). The TEAM project: the effectiveness of smoking cessation intervention with hospital patients. *Preventive Medicine, 40*(3), 249-258.

Hill, A., & Doll, R. (1956). Lung cancer and tobacco. *British Medical Journal, 1*, 1160-1163.

Hill, G. E., Ruggeroli, A., Pohorecki, R., Alonso, A., & Robbins, R. A. (1995). Cigarette smoking reduces endogenous airway nitric oxide production during cardiopulmonary bypass in humans. *Anesthesia & Analgesia, 81*(1), 170-172.

Hill, H. A., Schoenbach, V. J., Kleinbaum, D. G., Strecher, V. J., Orleans, C. T., Gebski, V. J., & Kaplan, B. H. (1994). A longitudinal analysis of predictors of quitting smoking among participants in a self-help intervention trial. *Addictive Behaviors, 19*(2), 159-173.

Hjalmarson, A., Franzon, M., Westin, A., & Wiklund, O. (1994). Effect of nicotine nasal spray on smoking cessation. A randomized, placebo-controlled, double-blind study. *Archives of Internal Medicine, 154*(22), 2567-2572.

Hjalmarson, A., Nilsson, F., Sjostrom, L., & Wiklund, O. (1997). The nicotine inhaler in smoking cessation. *Archives of Internal Medicine, 157*(15), 1721-1728.

Ho, R. (1998). The intention to give up smoking: disease versus social dimensions. *Journal of Social Psychology, 138*(3), 368-380.

Hodgson, T. (1992). Cigarette smoking and lifetime medical expenditures. *Milbank Quarterly, 70*(1), 81-125.

Hollinger, J. O., Schmitt, J. M., Hwang, K., Soleymani, P., & Buck, D. (1999). Impact of nicotine on bone healing. *Journal of Biomedical Material Research, 45*(4), 294-301.

Holm, K. J., & Spencer, C. M. (2000). Bupropion: a review of its use in the management of smoking cessation. *Drugs, 59*(4), 1007-1024.

Holmes, E., Mateczun, J. M., & Pentzien, R. J. (1991). Guidelines for conducting a hospital-based smoker's consult service. *Military Medicine, 156*(11), 585-589.

Hosmer, D. W., & Lemeshow, S. (2000). *Applied logistic regression* (2nd ed.). New York: John Wiley & Sons, Inc.

Hovell, M. F., Jones, J. A., & Adams, M. A. (2001). The feasibility and efficacy of tobacco use prevention in orthodontics. *Journal of Dental Education, 65*(4), 348-353.

Hueston, W. J., & Hubbard, E. T. (2000). Preventive services for rural and urban African American adults. *Archives of Family Medicine, 9*(3), 263-266.

Hughes, J., & Hatsukami, D. (1986). Signs and symptoms of tobacco withdrawal. *Archives of General Psychiatry, 43*, 289-294.

Hughes, J. R. (1993). Risk-benefit assessment of nicotine preparations in smoking cessation. *Drug Safety, 8*(1), 49-56.

Hughes, J. R., Gust, S. W., Skoog, K., Keenan, R. M., & Fenwick, J. W. (1991). Symptoms of tobacco withdrawal. A replication and extension. *Archives of General Psychiatry, 48*(1), 52-59.

Hughes, J. R., Stead, L. F., & Lancaster, T. (2000). Anxiolytics and antidepressants for smoking cessation. *Cochrane Database of Systematic Reviews [computer file]*(2), CD000031.

Hutchinson, D., Shepstone, L., Moots, R., Lear, J. T., & Lynch, M. P. (2001). Heavy cigarette smoking is strongly associated with rheumatoid arthritis (RA), particularly in patients without a family history of RA. *Annals of Rheumatic Disease, 60*(3), 223-227.

Hyman, D. J., Simons-Morton, D. G., Dunn, J. K., & Ho, K. (1996). Smoking, smoking cessation, and understanding of the role of multiple cardiac risk factors among the urban poor. *Preventive Medicine, 25*(6), 653-659.

Jacobson, E. (1943). Muscular tension and the smoking cigarettes. *American Journal of Psychology, 56*, 559-574.

James I King of England (1566-1625). (1900). *A royal rhetorician: A treatise on Scottis poesie, a counterblaste to tobacco, ect.* Westminster: A. Constable.

Jarvis, M. J., Tunstall-Pedoe, H., Feyerabend, C., Vesey, C., & Saloojee, Y. (1987). Comparison of tests used to distinguish smokers from nonsmokers. *American Journal of Public Health, 77*(11), 1435-1438.

Johnson, J. L., Budz, B., Mackay, M., & Miller, C. (1999). Evaluation of a nurse-delivered smoking cessation intervention for hospitalized patients with cardiac disease. *Heart Lung, 28*(1), 55-64.

Johnston, L. (1942). Tobacco smoking and nicotine. *Lancet, 2*, 742.

Jones, C., Griffiths, R. D., Skirrow, P., & Humphris, G. (2001). Smoking cessation through comprehensive critical care. *Intensive Care Med, 27*(9), 1547-1549.

Jonsson, T., Thorsteinsson, J., & Valdimarsson, H. (1998). Does smoking stimulate rheumatoid factor production in non-rheumatic individuals? Acta Pathologica, Microbiologica et Immunologica Scandinavica, *106*(10), 970-974.

Jorenby, D. E., Hatsukami, D. K., Smith, S. S., Fiore, M. C., Allen, S., Jensen, J., & Baker, T. B. (1996). Characterization of tobacco withdrawal symptoms: transdermal nicotine reduces hunger and weight gain. *Psychopharmacology, 128*(2), 130-138.

Jorenby, D. E., Leischow, S. J., Nides, M. A., Rennard, S. I., Johnston, J. A., Hughes, A. R., Smith, S. S., Muramoto, M. L., Daughton, D. M., Doan, K., Fiore, M. C., & Baker, T. B. (1999). A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. *New England Journal of Medicine, 340*(9), 685-691.

Joseph, A. M., Nichol, K. L., & Anderson, H. (1993). Effect of treatment for nicotine dependence on alcohol and drug treatment outcomes. *Addictive Behaviors, 18*(6), 635-644.

Joseph, A. M., Norman, S. M., Ferry, L. H., Prochazka, A. V., Westman, E. C., Steele, B. G., Sherman, S. E., Cleveland, M., Antonnucio, D. O., Hartman, N., & McGovern, P. G. (1996). The safety of transdermal nicotine as an aid to smoking cessation in patients with cardiac disease. *New England Journal of Medicine, 335*(24), 1792-1798.

Kabat, G., & Wynder, E. (1987). Determinants of quitting smoking. *American Journal of Public Health, 77*, 1301-1305.

Kamarck, T. W., & Lichtenstein, E. (1988). Program adherence and coping strategies as predictors of success in a smoking treatment program. *Health Psychology, 7*(6), 557-574.

Kanner, R. (1996). Early intervention in chronic obstructive pulmonary disease. A review of the Lung Health Study results. *Medical Clinics of North America, 80*, 523-544.

Karanci, N. A. (1992). Self-efficacy-based smoking situation factors: the effects of contemplating quitting versus relapsing in a Turkish sample. *International Journal of the Addictions, 27*(7), 879-886.

Kawachi, I., Colditz, G. A., Stampfer, M. J., Willett, W. C., Manson, J. E., Rosner, B., Speizer, F. E., & Hennekens, C. H. (1993). Smoking cessation and decreased risk of stroke in women. *Journal of the American Medical Association, 269*(2), 232-236.

Keeley, E. C., Pirwitz, M. J., Landau, C., Lange, R. A., Hillis, L. D., Foerster, E. H., Conrad, K., & Willard, J. E. (1996). Intranasal nicotine spray does not augment the adverse effects of cigarette smoking on myocardial oxygen demand or coronary arterial dimensions. *American Journal of Medicine, 101*(4), 357-363.

Kenford, S. L., Fiore, M. C., Jorenby, D. E., Smith, S. S., Wetter, D., & Baker, T. B. (1994). Predicting smoking cessation. Who will quit with and without the nicotine patch. *Journal of the American Medical Association, 271*(8), 589-594.

Kharitonov, S., & Barnes, P. (2001). Exhaled markers of pulmonary disease. *American Journal of Respiratory and Critical Care Medicine., 163*(7), 1693-1722.

Kiernan, V. G. (1991). *Tobacco: A history.* London: Hutchinson Radius.

Killen, J. D., Fortmann, S. P., Davis, L., & Varady, A. (1997). Nicotine patch and self-help video for cigarette smoking cessation. *Journal of Consulting & Clinical Psychology, 65*(4), 663-672.

Killen, J. D., Fortmann, S. P., Kraemer, H. C., Varady, A., & Newman, B. (1992). Who will relapse? Symptoms of nicotine dependence predict long-term relapse after smoking cessation. *Journal of Consulting & Clinical Psychology, 60*(5), 797-801.

Killen, J. D., Fortmann, S. P., Newman, B., & Varady, A. (1990). Evaluation of a treatment approach combining nicotine gum with self-guided behavioral treatments for smoking relapse prevention. *Journal of Consulting & Clinical Psychology, 58*(1), 85-92.

Kinnunen, T., Doherty, K., Militello, F. S., & Garvey, A. J. (1996). Depression and smoking cessation: characteristics of depressed smokers and effects of nicotine replacement. *Journal of Consulting & Clinical Psychology, 64*(4), 791-798.

Klerman, L. (2004). Protecting children: reducing their environmental tobacco smoke exposure. *Nicotine Tobacco Research, 6 Suppl 2*, S239-253.

Kwiatkowski, T. C., Hanley, E. N., Jr., & Ramp, W. K. (1996). Cigarette smoking and its orthopedic consequences. *American Journal of Orthopedics, 25*(9), 590-597.

Lachin, J. M. (2000). Statistical considerations in the intent-to-treat principle. *Controlled Clinical Trials, 21*(3), 167-189.

Lambe, R., Osier, C., & Franks, P. (1986). A randomized controlled trial of hypnotherapy for smoking cessation. *Journal of Family Practice, 22*(1), 61-65.

Lams, B. E., Sousa, A. R., Rees, P. J., & Lee, T. H. (1998). Immunopathology of the small-airway submucosa in smokers with and without chronic obstructive pulmonary disease. *American Journal of Respiratory & Critical Care Medicine, 158*(5 Pt 1), 1518-1523.

Langley, J. N., & Dickinson, W.L. (1889). On the local paralysis of the peripheral ganglia and on the connexion of different classes of nerve fibers with them. *Proceedings of the Royal Society of London, 46*, 423-431.

Langlois, M. A., Petosa, R., & Hallam, J. S. (1999). Why do effective smoking prevention programs work? Student changes in social cognitive theory constructs. *Journal of School Health, 69*(8), 326-331.

Lavernia, C. J., Sierra, R. J., & Gomez-Marin, O. (1999). Smoking and joint replacement: resource consumption and short-term outcome. *Clinical Orthopedics, 367*, 172-180.

Le, C. T. (1997). *Applied survival analysis*. New York: John Wiley & Sons, Inc.

Le Souef, P. N. (2000). Pediatric origins of adult lung diseases. 4. Tobacco related lung diseases begin in childhood. *Thorax, 55*(12), 1063-1067.

Lena, C., Changeux, J., & Mulle, C. (1993). Evidence of "preterminal" nicotinic receptors in GABAergic axons in the rat interpeduncular nucleus. *Journal of Neuroscience, 13*, 2680-2688.

Lerman, C., Caporaso, N., Main, D., Audrain, J., Boyd, N. R., Bowman, E. D., & Shields, P. G. (1998). Depression and self-medication with nicotine: The modifying influence of the dopamine D4 receptor gene. *Health Psychology, 17*(1), 56-62.

Lerman, C., Caporaso, N. E., Audrain, J., Main, D., Bowman, E. D., Lockshin, B., Boyd, N. R., & Shields, P. G. (1999). Evidence suggesting the role of specific genetic factors in cigarette smoking. *Health Psychology, 18*(1), 14-20.

Lerman, C., Caporaso, N. E., Audrain, J., Main, D., Boyd, N. R., & Shields, P. G. (2000). Interacting effects of the serotonin transporter gene and neuroticism in smoking practices and nicotine dependence. *Molecular Psychiatry, 5*(2), 189-192.

Levin, F., Levin, H., & Nagoshi, C. (1992). Autonomic functioning and cigarette smoking heart rate spectral analysis. *Biological Psychiatry, 31*, 639-643.

Lewis, S. F., Piasecki, T. M., Fiore, M. C., Anderson, J. E., & Baker, T. B. (1998). Transdermal nicotine replacement for hospitalized patients: a randomized clinical trial. *Preventive Medicine, 27*(2), 296-303.

Liao, T. F. (1994). *Interpreting probability models: logit, probit, and other generalized linear models.* Thousand Oaks: Sage Publications.

Lieberman, E., Gremy, I., Lang, J. M., & Cohen, A. P. (1994). Low birthweight at term and the timing of fetal exposure to maternal smoking. *American Journal of Public Health, 84*(7), 1127-1131.

Lightwood, J. M., & Glantz, S. A. (1997). Short-term economic and health benefits of smoking cessation: myocardial infarction and stroke. *Circulation, 96*(4), 1089-1096.

Lind, J., Kramhoft, M., & Bodtker, S. (1991). The influence of smoking on complications after primary amputations of the lower extremity. *Clinical Orthopedics*(267), 211-217.

Lindqvist, R., & Aberg, H. (2001). Who stops smoking during pregnancy? *Acta Obstetricia et Gynecologica Scandinavica, 80*(2), 137-141.

Lovich, S. F., & Arnold, P. G. (1994). The effect of smoking on muscle transposition. *Plastic Reconstructive Surgery, 93*(4), 825-828.

Lyon, E. R. (1999). A review of the effects of nicotine on schizophrenia and antipsychotic medications. *Psychiatric Services, 50*(10), 1346-1350.

MacArthur, C., Waldron, E., & Dickinson, J. (1958). The psychology of smoking. *Journal of Abnormal Psychology, 56*, 267-275.

MacKenzie, T. D., Bartecchi, C. E., & Schrier, R. W. (1994). The human cost of tobacco use (second of two parts). *New England Journal of Medicine, 330*, 975-980.

Mahamarian, J., Moye, L., Nasser, G., Nagueh, S., Bloom, M., Benowitz, N., Verani, M., Byrd, W., & Pratt, C. (1997). Nicotine patch therapy in smoking cessation reduces the extent of exercise-induced myocardial ischemia. *Journal of the American College of Cardiology, 30*(1), 125-130.

Maio, F., Dallman, M., Benowitz, N., Bashbaum, A., & Levine, J. (1993). Adrenal medullary modulation of the inhibition of bradykinin-induced plasma extravasion by intrathecal nicotine. *Journal of Pharmacology & Experimental Therapeutics, 53*, 6-14.

Manfredi, C., Lacey, L. P., Warnecke, R., & Petraitis, J. (1998). Sociopsychological correlates of motivation to quit smoking among low-SES African American women. *Health Education and Behavior, 25*(3), 304-318.

Manley, M. W., Epps, R. P., & Glynn, T. J. (1992). The clinician's role in promoting smoking cessation among clinic patients. *Medical Clinics of North America, 76*(2), 477-494.

Marlatt, G. A. (1979). A cognitive-behavioral model of the relapse process. *NIDA Research Monograph*(25), 191-200.

Martin, K., Froelicher, E. S., & Miller, N. H. (2000). Women's initiative for nonsmoking (WINS) II: the intervention. *Heart Lung, 29*(6), 438-445.

Matheny, K. B., & Weatherman, K. E. (1998). Predictors of smoking cessation and maintenance. *Journal of Clinical Psychology, 54*(2), 223-235.

Maxwell, C., & Hirdes, J. (1993). The prevalence of smoking and implications for quality of life among the community-based elderly. *American Journal of Preventive Medicine, 9*(6), 338-345.

McBride, C. M., Curry, S. J., Grothaus, L. C., Nelson, J. C., Lando, H., & Pirie, P. L. (1998). Partner smoking status and pregnant smoker's perceptions of support for and likelihood of smoking cessation. *Health Psychology, 17*(1), 63-69.

McCann, J. (2000). Anti-smoking effort seeks global implementation. *Journal of the National Cancer Institute, 92*(7), 523-524.

McGinnis, J., & Foege, W. (1993). Actual causes of death in the United States. *Journal of the American Medical Association, 270*, 2207-2212.

McIntyre, K. O., Lichtenstein, E., & Mermelstein, R. J. (1983). Self-efficacy and relapse in smoking cessation: a replication and extension. *Journal of Consulting & Clinical Psychology, 51*(4), 632-633.

McWhorter, W. P., Boyd, G. M., & Mattson, M. E. (1990). Predictors of quitting smoking: the NHANES I followup experience. *Journal of Clinical Epidemiology, 43*(12), 1399-1405.

Mermelstein, R., Cohen, S., Lichtenstein, E., Baer, J. S., & Kamarck, T. (1986). Social support and smoking cessation and maintenance. *Journal of Consulting & Clinical Psychology, 54*(4), 447-453.

Micro Medical Limited. (1998). MicroCO. Kent, UK: Micro Medical Limited.

Miller, C., Johnson, J. L., Mackay, M., & Budz, B. (1997a). The challenges of clinical nursing research: strategies for successful conduct. *Clinical Nurse Specialist, 11*(5), 213-216.

Miller, L., Zhang, X., Novotny, T., Rice, D., & Max, W. (1998). State estimates of Medicaid expenditures attributable to cigarette smoking, fiscal year 1993. *Public Health Reports, 113*(2), 140-151.

Miller, L., Zhang, X., Rice, D., & Max, W. (1998). State estimates of total medical expenditures attributable to cigarette smoking, 1993. *Public Health Reports, 113*(5), 448-458.

Miller, N. H., Smith, P. M., DeBusk, R. F., Sobel, D. S., & Taylor, C. B. (1997b). Smoking cessation in hospitalized patients. Results of a randomized trial. *Archives of Internal Medicine, 157*(4), 409-415.

Moadel, A. B., Lederberg, M. S., & Ostroff, J. S. (1999). Nicotine dependence and withdrawal in an oncology setting: a risk factor for psychiatric comorbidity and treatment non-adherence. *Psycho-Oncology, 8*(3), 264-267.

Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual causes of death in the United States, 2000. Journal of the American Medical Association*, 291*(10), 1238-1245.

Molyneux, A., Lewis, S., Leivers, U., Anderton, A., Antoniak, M., Brackenridge, A., Nilsson, F., McNeill, A., West, R., Moxham, J., & Britton, J. (2003). Clinical trial comparing nicotine replacement therapy (NRT) plus brief counseling, brief counseling alone, and minimal intervention on smoking cessation in hospital inpatients. *Thorax, 58*(6), 484-488.

Monardes, N. (1967). *Joyful newes out of newe founde worlde. Translated by John Frampton.* (J. Frampton, Trans.). NY: AMS Press.

Mosely, L. H., Finseth, F., & Goody, M. (1978). Nicotine and its effect on wound healing. *Plastic Reconstructive Surgery, 61*(4), 570-575.

Murray, R. P., Gerald, L. B., Lindgren, P. G., Connett, J. E., Rand, C. S., & Anthonisen, N. R. (2000). Characteristics of participants who stop smoking and sustain abstinence for 1 and 5 years in the Lung Health Study. *Preventive Medicine, 30*(5), 392-400.

Murray, R. P., Johnston, J. J., Dolce, J. J., Lee, W. W., & O'Hara, P. (1995). Social support for smoking cessation and abstinence: the Lung Health Study. Lung Health Study Research Group. *Addictive Behaviors, 20*(2), 159-170.

Narsavage, G., & Idemoto, B. K. (2003). Smoking cessation interventions for hospitalized patients with cardio-pulmonary disorders. *Online Journal of Issues in Nursing, 8*(2), 8.

National Institutes of Health. (1997). *Monograph 8 - Changes in cigarette - related disease risks and their implication for prevention and control.* (No. NIH Publication No. 97-1213). Rockville: National Institute of Health, National Cancer Institute.

Neighbor, W. E., Jr., Stoop, D. H., & Ellsworth, A. (1994). Smoking cessation counseling among hospitalized smokers. *American Journal of Preventive Medicine, 10*(3), 140-144.

Nelson, D. E., Emont, S. L., Brackbill, R. M., Cameron, L. L., Peddicord, J., & Fiore, M. C. (1994). Cigarette smoking prevalence by occupation in the United States. A comparison between 1978 to 1980 and 1987 to 1990. *Journal of Occupational Medicine, 36*(5), 516-525.

Neter, J., Kutner, M., Nachtsheim, C., & Wasserman, W. (1996). *Applied linear statistical models* (4th ed.). Chicago: Irwin.

Nides, M., Rand, C., Dolce, J., Murray, R., O'Hara, P., Voelker, H., & Connett, J. (1994). Weight gain as a function of smoking cessation and 2-mg nicotine gum use among middle-aged smokers with mild lung impairment in the first 2 years of the Lung Health Study. *Health Psychology, 13*(4), 354-361.

Nides, M. A., Rakos, R. F., Gonzales, D., Murray, R. P., Tashkin, D. P., Bjornson-Benson, W. M., Lindgren, P., & Connett, J. E. (1995). Predictors of initial smoking cessation and relapse through the first 2 years of the Lung Health Study. *Journal of Consulting & Clinical Psychology, 63*(1), 60-69.

Nielsen, K., & Fiore, M. C. (2000). Cost-benefit analysis of sustained-release bupropion, nicotine patch, or both for smoking cessation. *Preventive Medicine, 30*(3), 209-216.

Norman, P., Conner, M., & Bell, R. (1999). The theory of planned behavior and smoking cessation. *Health Psychology, 18*(1), 89-94.

Norregaard, J., Tonnesen, P., & Petersen, L. (1993). Predictors and reasons for relapse in smoking cessation with nicotine and placebo patches. *Preventive Medicine, 22*(2), 261-271.

Ockene, J. K., Kristeller, J., Pbert, L., Hebert, J. R., Luippold, R., Goldberg, R. J., Landon, J., & Kalan, K. (1994). The physician-delivered smoking intervention project: can short-term interventions produce long-term effects for a general outpatient population? *Health Psychology, 13*(3), 278-281.

O'Hara, P., Portser, S. A., & Anderson, B. P. (1989). The influence of menstrual cycle changes on the tobacco withdrawal syndrome in women. *Addictive Behaviors, 14*(6), 595-600.

Orleans, C. T., Kristeller, J. L., & Gritz, E. R. (1993). Helping hospitalized smokers quit: new directions for treatment and research. *Journal of Consulting & Clinical Psychology, 61*(5), 778-789.

Osler, M., & Jespersen, N. B. (1993). The effect of a community-based cardiovascular disease prevention project in a Danish municipality. *Danish Medical Bulletin, 40*(4), 485-489.

Overall, J. E. (1990). Tests of one-sided versus two-sided hypotheses in placebo-controlled clinical trials. *Neuropsychopharmacology, 3*(4), 233-235.

Padula, C. A. (1992). Nurses and smoking: review and implications. *Journal of Professional Nursing, 8*(2), 120-132.

Pallonen, U. E., Leskinen, L., Prochaska, J. O., Willey, C. J., Kaariainen, R., & Salonen, J. T. (1994). A 2-year self-help smoking cessation manual intervention among middle-aged Finnish men: an application of the transtheoretical model. *Preventive Medicine, 23*(4), 507-514.

Parrott, A. C. (2004). Heightened stress and depression follow cigarette smoking. *Psychological Reports, 94*(1), 33-34.

Patten, C., Schneekloth, T., Morse, R., Herrick, L., Offord, K., Wolter, T., Williams, B., & Hurt, R. (2001). Effect of current tobacco use and history of an alcohol problem on health status in hospitalized patients. *Addictive Behaviors, 26*, 129-136.

Patten, C. A., Martin, J. E., Calfas, K. J., Brown, S. A., & Schroeder, D. R. (2000). Effect of three smoking cessation treatments on nicotine withdrawal in 141 abstinent alcoholic smokers. *Addictive Behaviors, 25*(2), 301-306.

Pauwels, R. A., & Rabe, K. F. (2004). Burden and clinical features of chronic obstructive pulmonary disease (COPD). *Lancet, 364*(9434), 613-620.

Pearl, R. (1938). Tobacco smoking and longevity. *Science, 87*, 216-217.

Pederson, L. L., Wanklin, J. M., & Lefcoe, N. M. (1991). The effects of counseling on smoking cessation among patients hospitalized with chronic obstructive pulmonary disease: a randomized clinical trial. *International Journal of the Addictions, 26*(1), 107-119.

Perkins, K. A. (1995). Individual variability in responses to nicotine. *Behavior Genetics, 25*(2), 119-132.

Perkins, K. A. (2001). Smoking cessation in women. Special considerations. *CNS Drugs, 15*(5), 391-411.

Perkins, K. A., Epstein, L. H., & Jennings, J. R. (1991). Smoking as a cue for subjective and behavioral responses to a stressor. *Journal of Substance Abuse, 3*(1), 29-38.

Perkins, K. A., Levine, M., Marcus, M., Shiffman, S., D'Amico, D., Miller, A., Keins, A., Ashcom, J., & Broge, M. (2000). Tobacco withdrawal in women and menstrual cycle phase. *Journal of Consulting & Clinical Psychology, 68*(1), 176-180.

Perkins, K. A., Levine, M. D., Marcus, M. D., & Shiffman, S. (1997). Addressing women's concerns about weight gain due to smoking cessation. *Journal of Substance Abuse Treatment, 14*(2), 173-182.

Perz, C. A., DiClemente, C. C., & Carbonari, J. P. (1996). Doing the right thing at the right time? The interaction of stages and processes of change in successful smoking cessation. *Health Psychology, 15*(6), 462-468.

Philaretes. (1936). *Work for chimny-sweepers, or A warning for tabacconists, with an introduction by S.H. Atkins, 1602.* London: Oxford University Press.

Piasecki, T. M., Fiore, M. C., & Baker, T. B. (1998). Profiles in discouragement: two studies of variability in the time course of smoking withdrawal symptoms. *Journal of Abnormal Psychology, 107*(2), 238-251.

Piasecki, T. M., Niaura, R., Shadel, W. G., Abrams, D., Goldstein, M., Fiore, M. C., & Baker, T. B. (2000). Smoking withdrawal dynamics in unaided quitters. *Journal of Abnormal Psychology, 109*(1), 74-86.

Picciotto, M., Zoli, M., Lena, C., Marubio, E., Merlo-Pich, E., Fuxe, K., & Chageux, J. (1998). Acetylcholine receptors containing the beta2 subunit are involved in reinforcing properties of nicotine. *Nature, 391*, 173-177.

Pinel, J. (1997). *Biopsychology* (3rd ed.). Boston: Allyn and Bacon.

Pirie, P. L., McBride, C. M., Hellerstedt, W., Jeffery, R. W., Hatsukami, D., Allen, S., & Lando, H. (1992). Smoking cessation in women concerned about weight. *American Journal of Public Health, 82*(9), 1238-1243.

Pirie, P. L., Murray, D. M., & Luepker, R. V. (1991). Gender differences in cigarette smoking and quitting in a cohort of young adults. *American Journal of Public Health, 81*(3), 324-327.

Polednak, A. P. (2000). Hospital-based smoking cessation programs: a statewide survey. *Connecticut Medicine, 64*(11), 683-685.

Pollack, H. A. (2001). Sudden infant death syndrome, maternal smoking during pregnancy, and the cost-effectiveness of smoking cessation intervention. *American Journal of Public Health, 91*(3), 432-436.

Pomerleau, C. S., Carton, S. M., Lutzke, M. L., Flessland, K. A., & Pomerleau, O. F. (1994). Reliability of the Fagerstrom Tolerance Questionnaire and the Fagerstrom Test for Nicotine Dependence. *Addictive Behaviors, 19*(1), 33-39.

Pomerleau, C. S., Majchrzak, M. J., & Pomerleau, O. F. (1989). Nicotine dependence and the Fagerstrom Tolerance Questionnaire: a brief review. *Journal of Substance Abuse, 1*(4), 471-477.

Pomerleau, O. F. (1992). Nicotine and the central nervous system: Biobehavioral effects of cigarette smoking. *American Journal of Medicine, 93*(1A), 2S-7S.

Pomerleau, O. F., & Kardia, S. L. (1999). Introduction to the featured section: genetic research on smoking. *Health Psychology, 18*(1), 3-6.

Pomerleau, O. F., Pomerleau, C. S., & Marks, J. L. (2000). Abstinence effects and reactivity to nicotine during 11 days of smoking deprivation. *Nicotine & Tobacco Research, 2*(2), 149-157.

Prochaska, J. (1995). An eclectic and integrative approach: Transtheoretical therapy. In A. S. Gurman, & Messer, S.B. (Ed.), *Essential Psychotherapies.* (pp. 403-440). New York, NY: The Gulford Press.

Prochaska, J. O. (2000). Stages of change model for smoking prevention and cessation in schools. Authors applied adult dose for smoking to adolescents when smoking behaviour is different in the two. *British Medical Journal, 320*(7232), 447.

Prochaska, J. O., DiClemente, C. C., Velicer, W. F., Ginpil, S., & Norcross, J. C. (1985). Predicting change in smoking status for self-changers. *Addictive Behaviors, 10*(4), 395-406.

Prochaska, J. O., & Velicer, W. F. (1997). The transtheoretical model of health behavior change. *American Journal of Health Promotion, 12*(1), 38-48.

Prokhorov, A. V., De Moor, C., Pallonen, U. E., Hudmon, K. S., Koehly, L., & Hu, S. (2000). Validation of the modified Fagerstrom tolerance questionnaire with salivary cotinine among adolescents. *Addictive Behaviors, 25*(3), 429-433.

Quattrocki, E., Baird, A., & Yurgelun-Todd, D. (2000). Biological aspects of the link between smoking and depression. *Harvard Review of Psychiatry, 8*(3), 99-110.

Quist-Paulsen, P., & Gallefoss, F. (2003). Randomised controlled trial of smoking cessation intervention after admission for coronary heart disease. *British Medical Journal, 327*(7426), 1254-1257.

Radzius, A., Moolchan, E. T., Henningfield, J. E., Heishman, S. J., & Gallo, J. J. (2001). A factor analysis of the Fagerstrom Tolerance Questionnaire. *Addictive Behaviors, 26*, 303-310.

Rand, C. S., Nides, M., Cowles, M. K., Wise, R. A., & Connett, J. (1995). Long-term metered-dose inhaler adherence in a clinical trial. The Lung Health Study Research Group. *American Journal of Respiratory & Critical Care Medicine, 152*(2), 580-588.

Ratner, P. A., Johnson, J. L., Richardson, C. G., Bottorff, J. L., Moffat, B., Mackay, M., Fofonoff, D., Kingsbury, K., Miller, C., & Budz, B. (2004). Efficacy of a smoking-cessation intervention for elective-surgical patients. *Research in Nursing Health, 27*(3), 148-161.

Reid, R., Pipe, A., Higginson, L., Johnson, K., D'Angelo, M. S., Cooke, D., & Dafoe, W. (2003). Stepped care approach to smoking cessation in patients hospitalized for coronary artery disease. *Journal of Cardiopulmonary Rehabilitation, 23*(3), 176-182.

Reid, R. D., Pipe, A., & Dafoe, W. A. (1999). Is telephone counseling a useful addition to physician advice and nicotine replacement therapy in helping patients to stop smoking? A randomized controlled trial. *Canadian Medical Association Journal, 160*(11), 1577-1581.

Rice, V. H., Templin, T., Fox, D. H., Jarosz, P., Mullin, M., Seiggreen, M., & Lepczyk, M. (1996). Social context variables as predictors of smoking cessation. *Tobacco Control, 5*(4), 280-285.

Richmond, R. L., Kehoe, L. A., & Webster, I. W. (1993). Multivariate models for predicting abstention following intervention to stop smoking by general practitioners. *Addiction, 88*(8), 1127-1135.

Rigotti, N. A., Arnsten, J. H., McKool, K. M., Wood-Reid, K. M., Pasternak, R. C., & Singer, D. E. (1997). Efficacy of a smoking cessation program for hospitalized patients. *Archives of Internal Medicine, 157*, 2653-2660.

Rigotti, N. A., Arnsten, J. H., McKool, K. M., Wood-Reid, K. M., Pasternak, R. C., & Singer, D. E. (2000). Smoking by patients in a smoke-free hospital: prevalence, predictors, and implications. *Preventive Medicine, 31*(2 Pt 1), 159-166.

Rigotti, N. A., Arnsten, J. H., McKool, K. M., Wood-Reid, K. M., Singer, D. E., & Pasternak, R. C. (1999). The use of nicotine-replacement therapy by hospitalized smokers. *American Journal of Preventive Medicine, 17*(4), 255-259.

Rigotti, N. A., McKool, K. M., & Shiffman, S. (1994). Predictors of smoking cessation after coronary artery bypass graft surgery. Results of a randomized trial with 5-year follow-up. *Annals of Internal Medicine, 120*(4), 287-293.

Rigotti, N. A., Munafo, M. R., Murphy, M. F., & Stead, L. F. (2003). Interventions for smoking cessation in hospitalized patients. *Cochrane Database System Review*(1), CD001837.

Rigotti, N. A., Singer, D. E., Mulley, A. G., Jr., & Thibault, G. E. (1991). Smoking cessation following admission to a coronary care unit. *Journal of General Internal Medicine, 6*(4), 305-311.

Robbins, R. A., Millatmal, T., Lassi, K., Rennard, S., & Daughton, D. (1997). Smoking cessation is associated with an increase in exhaled nitric oxide. *Chest, 112*(2), 313-318.

Robinson, M. D., Laurent, S. L., & Little, J. M., Jr. (1995). Including smoking status as a new vital sign: it works! *Journal of Family Practice, 40*(6), 556-561.

Rohren, C. L., Croghan, I. T., Hurt, R. D., Offord, K. P., Marusic, Z., & McClain, F. L. (1994). Predicting smoking cessation outcome in a medical center from stage of readiness: contemplation versus action. *Preventive Medicine, 23*(3), 335-344.

Rose, J. E., Behm, F. M., Westman, E. C., Levin, E. D., Stein, R. M., & Ripka, G. V. (1994). Mecamylamine combined with nicotine skin patch facilitates smoking cessation beyond nicotine patch treatment alone. *Clinical Pharmacology & Therapeutics, 56*(1), 86-99.

Rosecrans, J. A., & Karan, L. D. (1993). Neurobehavioral mechanisms of nicotine action: role in the initiation and maintenance of tobacco dependence. *Journal of Substance Abuse Treatment, 10*(2), 161-170.

Roski, J., Schmid, L. A., & Lando, H. A. (1996). Long-term associations of helpful and harmful spousal behaviors with smoking cessation. *Addictive Behaviors, 21*(2), 173-185.

Royce, J. M., Corbett, K., Sorensen, G., & Ockene, J. (1997). Gender, social pressure, and smoking cessations: the Community Intervention Trial for Smoking Cessation (COMMIT) at baseline. *Social Science & Medicine, 44*(3), 359-370.

Ruggiero, L., Tsoh, J. Y., Everett, K., Fava, J. L., & Guise, B. J. (2000). The transtheoretical model of smoking: comparison of pregnant and nonpregnant smokers. *Addictive Behaviors, 25*(2), 239-251.

Saag, K. G., Cerhan, J. R., Kolluri, S., Ohashi, K., Hunninghake, G. W., & Schwartz, D. A. (1997). Cigarette smoking and rheumatoid arthritis severity. *Annals of Rheumatic Disease, 56*(8), 463-469.

Sabol, S. Z., Nelson, M. L., Fisher, C., Gunzerath, L., Brody, C. L., Hu, S., Sirota, L. A., Marcus, S. E., Greenberg, B. D., Lucas, F. R. t., Benjamin, J., Murphy, D. L., & Hamer, D. H. (1999). A genetic association for cigarette smoking behavior. *Health Psychology, 18*(1), 7-13.

Samet, J. M. (1992). The health benefits of smoking cessation. *Medical Clinics of North America, 76*(2), 399-414.

Sarna, L., Brown, J. K., Lillington, L., Wewers, M. E., & Brecht, M. L. (2000). Tobacco-control attitudes, advocacy, and smoking behaviors of oncology nurses. *Oncology Nursing Forum, 27*(10), 1519-1528.

Schlenk, E. (2001). Psychometric properties of the perceived therapeutic efficacy scale.(Personal Communication). Pittsburgh, PA.

Schmitz, J. M., Spiga, R., Rhoades, H. M., Fuentes, F., & Grabowski, J. (1999). Smoking cessation in women with cardiac risk: a comparative study of two theoretically based therapies. *Nicotine & Tobacco Research, 1*(1), 87-94.

Schneider, N. G., Olmstead, R., Nilsson, F., Mody, F. V., Franzon, M., & Doan, K. (1996). Efficacy of a nicotine inhaler in smoking cessation: a double-blind, placebo-controlled trial. *Addiction, 91*(9), 1293-1306.

Scholte, R. H., & Breteler, M. H. (1997). Withdrawal symptoms and previous attempts to quit smoking: associations with self-efficacy. *Substance Use & Misuse, 32*(2), 133-148.

Schwartz, J. (1992). Methods of smoking cessation. *Medical Clinics of North America, 76*, 451-476.

Sereika, S., & Davis, C. (2001). The analysis of clinical trials and treatment nonadherence. In L. Burke & I. Ockene (Eds.), *Compliance in healthcare and research*: Blackwell Futura.

Sethi, J. M., & Rochester, C. L. (2000). Smoking and chronic obstructive pulmonary disease. *Clinics in Chest Medicine, 21*(1), 67-86, viii.

Shiffman, S. (1993). Assessing smoking patterns and motives. *Journal of Consulting & Clinical Psychology, 61*(5), 732-742.

Shiffman, S., Balabanis, M. H., Paty, J. A., Engberg, J., Gwaltney, C. J., Liu, K. S., Gnys, M., Hickcox, M., & Paton, S. M. (2000). Dynamic effects of self-efficacy on smoking lapse and relapse. *Health Psychology, 19*(4), 315-323.

Shiffman, S., Gnys, M., Richards, T. J., Paty, J. A., Hickcox, M., & Kassel, J. D. (1996a). Temptations to smoke after quitting: a comparison of lapsers and maintainers. *Health Psychology, 15*(6), 455-461.

Shiffman, S., Hickcox, M., Paty, J. A., Gnys, M., Kassel, J. D., & Richards, T. J. (1996b). Progression from a smoking lapse to relapse: prediction from abstinence violation effects, nicotine dependence, and lapse characteristics. *Journal of Consulting & Clinical Psychology, 64*(5), 993-1002.

Shiffman, S., Hickcox, M., Paty, J. A., Gnys, M., Kassel, J. D., & Richards, T. J. (1997). The abstinence violation effect following smoking lapses and temptations. *Cognitive Therapy and Research, 21*(5), 497-523.

Shiffman, S., Paty, J. A., Gnys, M., Kassel, J. A., & Hickcox, M. (1996c). First lapses to smoking: within-subjects analysis of real-time reports. *Journal of Consulting & Clinical Psychology, 64*(2), 366-379.

Shipley, R. H., Rosen, T. J., & Williams, C. (1982). Measurement of smoking: surveys and some recommendations. *Addictive Behaviors, 7*(3), 299-302.

Silagy, C., Mant, D., Fowler, G., & Lancaster, T. (2000). Nicotine replacement therapy for smoking cessation. *Cochrane Database System Review*(3), CD000146.

Silcox, D. H., 3rd, Daftari, T., Boden, S. D., Schimandle, J. H., Hutton, W. C., & Whitesides, T. E., Jr. (1995). The effect of nicotine on spinal fusion. *Spine, 20*(14), 1549-1553.

Simmons, M. S., Nides, M. A., Rand, C. S., Wise, R. A., & Tashkin, D. P. (1996). Trends in compliance with bronchodilator inhaler use between follow-up visits in a clinical trial. *Chest, 109*(4), 963-968.

Simon, J. A., Carmody, T. P., Hudes, E. S., Snyder, E., & Murray, J. (2003). Intensive smoking cessation counseling versus minimal counseling among hospitalized smokers treated with transdermal nicotine replacement: a randomized trial. *American Journal of Medicine, 114*(7), 555-562.

Simon, J. A., Solkowitz, S. N., Carmody, T. P., & Browner, W. S. (1997). Smoking cessation after surgery. A randomized trial. *Archives of Internal Medicine, 157*(12), 1371-1376.

Sivarajan Froelicher, E. S., Miller, N. H., Christopherson, D. J., Martin, K., Parker, K. M., Amonetti, M., Lin, Z., Sohn, M., Benowitz, N., Taylor, C. B., & Bacchetti, P. (2004). High rates of sustained smoking cessation in women hospitalized with cardiovascular disease: the Women's Initiative for Nonsmoking (WINS). *Circulation, 109*(5), 587-593.

Smith, P. M., Reilly, K. R., Houston Miller, N., DeBusk, R. F., & Taylor, C. B. (2002). Application of a nurse-managed inpatient smoking cessation program. *Nicotine Tobacco Research, 4*(2), 211-222.

Snively, T. A., Ahijevych, K. L., Bernhard, L. A., & Wewers, M. E. (2000). Smoking behavior, dysphoric states and the menstrual cycle: results from single smoking sessions and the natural environment. *Psychoneuroendocrinology, 25*(7), 677-691.

Snyder, F., Davis, F., & Henningfield, J. (1989). The tobacco withdrawal syndrome: performance decrements assessed on a computerized test battery. *Drug & Alcohol Dependence, 23*(3), 259-266.

Spanier, C. A., Shiffman, S., Maurer, A., Reynolds, W., & Quick, D. (1996). Rebound following failure to quit smoking: The effects of attributions and self-efficacy. *Experimental & Clinical Psychopharmacology, 4*(2), 191-197.

Stapleton, J. A., Russell, M. A., Feyerabend, C., Wiseman, S. M., Gustavsson, G., Sawe, U., & Wiseman, D. (1995). Dose effects and predictors of outcome in a randomized trial of transdermal nicotine patches in general practice. *Addiction, 90*(1), 31-42.

Stead, L. F., & Hughes, J. R. (2000). Lobeline for smoking cessation. *Cochrane Database of Systematic Reviews [computer file]*(2), CD000124.

Stead, L. F., & Lancaster, T. (2001). Telephone counseling for smoking cessation (Cochrane Review). *Cochrane Database System Review, 2*, CD002850.

Stefanadis, D., Tsiamis, E., Vlachopoulos, C., Stratos, C., Toutouzas, K., Pitsavos, C., Marakas, S., Boudoulas, H., & Toutouzas, P. (1997). Unfavorable effect of smoking on the elastic properties of the human aorta. *Circulation, 95*(1), 31-38.

Stevens, V. J., Glasgow, R. E., Hollis, J. F., Lichtenstein, E., & Vogt, T. M. (1993). A smoking-cessation intervention for hospital patients. *Medical Care, 31*(1), 65-72.

Stevens, V. J., Glasgow, R. E., Hollis, J. F., & Mount, K. (2000). Implementation and effectiveness of a brief smoking-cessation intervention for hospital patients. *Medical Care, 38*(5), 451-459.

Stillman, F. A., Hartman, A. M., Graubard, B. I., Gilpin, E. A., Murray, D. M., & Gibson, J. T. (2003). Evaluation of the American Stop Smoking Intervention Study (ASSIST): a report of outcomes. *Journal of the National Cancer Institute, 95*(22), 1681-1691.

Stollerman, I. (1993). Components of drug dependence: reinforcement, discrimination, and adaptation. In S. Wonnacott & G. Lunt (Eds.), *Neurochemistry of drug dependence.* (pp. 1-2). London, England: Portland Press.

Swan, G. E. (1999). Implications of genetic epidemiology for the prevention of tobacco use. *Nicotine & Tobacco Research*(1SUPPL1), S49-56.

Swan, G. E., Roby, T. J., Hodgkin, J. E., Mittman, C., Peters, J. A., & Jacobo, N. (1994). Relationship of cytomorphology to spirometric findings in cigarette smokers. *Acta Cytologica, 38*(4), 547-553.

Swan, G. E., Valdes, A. M., Ring, H. Z., Khroyan, T. V., Jack, L. M., Ton, C. C., Curry, S. J., & McAfee, T. (2004). Dopamine receptor DRD2 genotype and smoking cessation outcome following treatment with bupropion SR. *Pharmacogenomics Journal,* 5(1), 21-29.

Swan, G. E., Ward, M. M., & Jack, L. M. (1996). Abstinence effects as predictors of 28-day relapse in smokers. *Addictive Behaviors, 21*(4), 481-490.

Tashkin, D., Kanner, R., Bailey, W., Buist, S., Anderson, P., Nides, M., Gonzales, D., Dozier, G., Patel, M. K., & Jamerson, B. (2001). Smoking cessation in patients with chronic obstructive pulmonary disease: a double-blind, placebo-controlled, randomised trial. *Lancet, 357*(9268), 1571-1575.

Tashkin, D. P., Rand, C., Nides, M., Simmons, M., Wise, R., Coulson, A. H., Li, V., & Gong, H., Jr. (1991). A nebulizer chronolog to monitor compliance with inhaler use. *American Journal of Medicine, 91*(4A), 33S-36S.

Taylor, C. B., Houston-Miller, N., Killen, J. D., & DeBusk, R. F. (1990). Smoking cessation after acute myocardial infarction: Effects of a nurse-managed intervention. *Annals of Internal Medicine, 113*(2), 118-123.

Taylor, C. B., Miller, N. H., Herman, S., Smith, P. M., Sobel, D., Fisher, L., & DeBusk, R. F. (1996). A nurse-managed smoking cessation program for hospitalized smokers. *American Journal of Public Health, 86*(11), 1557-1560.

Tilley, N. M. (1948). *The bright-tobacco industry, 1860-1929*. Chapel Hill: University of North Carolina Press.

Toljamo, M., & Hentinen, M. (2001). Adherence to self-care and glycaemic control among people with insulin-dependent diabetes mellitus. *Journal of Advanced Nursing, 34*(6), 780-786.

Tsoh, J. Y., Humfleet, G. L., Munoz, R. F., Reus, V. I., Hartz, D. T., & Hall, S. M. (2000). Development of major depression after treatment for smoking cessation. *American Journal of Psychiatry, 157*(3), 368-374.

Tyndale, R. F., & Sellers, E. M. (2001). Variable CYP2A6-mediated nicotine metabolism alters smoking behavior and risk. *Drug Metabolism and Disposition, 29*(4 Pt 2), 548-552.

U.S. Census Bureau. (2002). *State and County Quick Facts for Allegheny County of Pennsylvania*, from http://quickfacts.census.gov/qfd/states/42/42003.html

U.S. Department of Health and Human Services. (1981). *The health consequences of smoking - the changing cigarette - a report of the Surgeon General.* (No. DHHS (PHS) Publication No. 81-50156). Washington, D.C.: U.S. Government Printing Office.

U.S. Department of Health and Human Services. (1982). *The health consequences of smoking - cancer - a report of the Surgeon General.* (No. DHHS (PHS) Publication No. 82-50178). Rockville: U.S. Department of Health and Human Services, Public Health Service, Office on Smoking and Health.

U.S. Department of Health and Human Services. (1983). *The health consequences of smoking - cardiovascular disease - a report of the Surgeon General.* (No. DHHS (PHS) Publication No. 84-50204). Washington, D.C.: U.S. Government Printing Office.

U.S. Department of Health and Human Services. (1984). *The health consequences of smoking - chronic obstructive lung disease - a report of the Surgeon General.* (No. DHHS (PHS) Publication No. 84-50205). Rockville: U.S. Department of Health and Human Services, Public Health Service, Office on Smoking and Health.

U.S. Department of Health and Human Services. (1985). *The health consequences of smoking - cancer and chronic lung disease in the workplace - a report of the Surgeon General. (DHHS (PHS) 85-50207)*. Rockville, MD: U.S. Department of Health and Human Services.

U.S. Department of Health and Human Services. (1986). *Clinical opportunities for smoking intervention. A guide for the busy physician.* (No. NIH publication No. 86-2178). Washington, DC: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health.

U.S. Department of Health and Human Services. (1988). *The health consequences of smoking - nicotine addiction - a report of the Surgeon General.* (No. DHHS (CDC) Publication No. 88-8406). Rockville: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Centers for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

U.S. Department of Health and Human Services. (1989). *Reducing the health consequences of smoking: 25 years of Progress: A report of the Surgeon General.* (No. DHHS (CDC) Publication No. 89-8411). Rockville: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Centers for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

U.S. Department of Health and Human Services. (1990). *The health benefits of smoking cessation - a report of the Surgeon General.* (No. DHHS (CDC) Publication No. 90-8416). Rockville: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Chronic Disease Prevention and Promotion, Office on Smoking and Health.

U.S. Department of Health and Human Services. (1991). *Healthy people 2000: National health promotion and disease prevention objectives.* (No. DHHS Publication No. (PHS) 91-50212). Washington: U.S. Department of Health and Human Services, Public Health Service.

U.S. Department of Health and Human Services. (2000). *Reducing tobacco use: A report of the surgeon general—executive summary.* Atlanta, Georgia: U.S.: Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health.

U.S. Department of Health, E., and Welfare. (1964). *Smoking and health; report of the Advisory Committee to the Surgeon General of the Public Health Service.* (No. PHS Publication No. 1103). Washington, D.C.: U.S. Dept. of Health, Education, and Welfare, Public Health Service.

U.S. Preventive Services Task Force. (1996). Counseling to prevent tobacco use. In *Guide to clinical preventive services* (2nd ed., pp. 597-609). Baltimore: Williams & Wilkins.

Vander Ark, W., DiNardo, L. J., & Oliver, D. S. (1997). Factors affecting smoking cessation in patients with head and neck cancer. *Laryngoscope, 107*(7), 888-892.

Vaur, L., Vaisse, B., Genes, N., Elkik, F., Legrand, C., & Poggi, L. (1999). Use of electronic pill boxes to assess risk of poor treatment compliance: results of a large-scale trial. *American Journal of Hypertension, 12*(4 Pt 1), 374-380.

Vernon, J. D., Crane, L. A., Prochazka, A. V., Fairclough, D., & MacKenzie, T. D. (1999). Smokers hospitalized in an urban, public hospital: addiction, stages of change, and self-efficacy. *Preventive Medicine, 28*(5), 488-495.

Villarreal, F. J., Hong, D., & Omens, J. (1999). Nicotine-modified postinfarction left ventricular remodeling. *American Journal of Physiology, 276*(3 Pt 2), H1103-1106.

Wada, E., Wada, K., Boulter, J., Deneris, E., Heinemann, S., Patrick, J., & Swanson, L. (1989). Distribution of alpha2, alpha3, alpha4, and beta2 neuronal nicotinic receptor subunit mRNAs in the central nervous system: a hybridization histochemical study in the rat. *Journal of Comparative Neurology, 284*, 314-355.

Wahlgren, D. R., Hovell, M. F., Meltzer, E. O., & Meltzer, S. B. (2000). Involuntary smoking and asthma. *Current Opinion in Pulmonary Medicine, 6*(1), 31-36.

Waite, N. R., & Clough, J. B. (1998). A single-blind, placebo-controlled trial of a simple acupuncture treatment in the cessation of smoking. *British Journal of General Practice, 48*(433), 1487-1490.

Warner, D., Warner, M., Offord, K., Schroeder, D., Maxson, R., & Scanlon, P. (1999). Airway obstruction and perioperative complications in smokers undergoing abdominal surgery. *Anesthesiology, 90*(2), 372-379.

Warner, M., Divertie, M., & Tinker, J. (1984). Preoperative cessation of smoking and pulmonary complications in coronary artery bypass patients. *Anesthesiology, 60*, 380-383.

Watkins, S., Koob, G., & Markou, A. (2000). Neural mechanisms underlying nicotine addiction: acute positive reinforcement and withdrawal. *Nicotine & Tobacco Research, 2*(1), 19-37.

Watson, D., & Tharp, R. (1997). *Self-directed behavior: self-modification for personal adjustment* (7th ed.). Pacific Grove, CA: Brooks/Cole Publishing Company.

Weir, M. R., Maibach, E. W., Bakris, G. L., Black, H. R., Chawla, P., Messerli, F. H., Neutel, J. M., & Weber, M. A. (2000). Implications of a health lifestyle and medication analysis for improving hypertension control. *Archives of Internal Medicine, 160*(4), 481-490.

Westman, E. C., Behm, F. M., Simel, D. L., & Rose, J. E. (1997). Smoking behavior on the first day of a quit attempt predicts long-term abstinence. *Archives of Internal Medicine, 157*(3), 335-340.

Wewers, M., & Gonyon, D. (1989). Cigarette craving during the immediate postcessation period. *Research Briefs, 2*(1), 46-51.

Wewers, M. E., & Ahijevych, K. L. (1996). Smoking cessation interventions in chronic illness. *Annual Review of Nursing Research, 14*, 75-93.

Wewers, M. E., Ahijevych, K. L., & Sarna, L. (1998). Smoking cessation interventions in nursing practice. *Nursing Clinics of North America, 33*(1), 61-74.

Wewers, M. E., Bowen, J. M., Stanislaw, A. E., & Desimone, V. B. (1994). A nurse-delivered smoking cessation intervention among hospitalized postoperative patients--influence of a smoking-related diagnosis: a pilot study. *Heart & Lung, 23*(2), 151-156.

Wewers, M. E., Jenkins, L., & Mignery, T. (1997). A nurse-managed smoking cessation intervention during diagnostic testing for lung cancer. *Oncology Nursing Forum, 24*(8), 1419-1422.

White, A. R., Resch, K. L., & Ernst, E. (1998). Randomized trial of acupuncture for nicotine withdrawal symptoms. *Archives of Internal Medicine, 158*(20), 2251-2255.

White, A. R., Resch, K. L., & Ernst, E. (1999). A meta-analysis of acupuncture techniques for smoking cessation. *Tobacco Control, 8*(4), 393-397.

Woehlck, H. J., Connolly, L. A., Cinquegrani, M. P., Dunning, M. B., 3rd, & Hoffmann, R. G. (1999). Acute smoking increases ST depression in humans during general anesthesia. *Anesthesia & Analgesia, 89*(4), 856-860.

Working group for the study of transdermal nicotine in patients with coronary artery disease. (1994). Nicotine replacement therapy for patients with coronary artery disease. *Archives of Internal Medicine, 154*, 989-995.

World Bank. (1999). Curbing the epidemic: Governments and the economics of tobacco control. *Tobacco Control, 8*(2), 196-201.

World Health Organization. (1999). *Making a difference, world report 1999.* Geneva: World Health Organization.

Yates, A. J., & Thain, J. (1985). Self-efficacy as a predictor of relapse following voluntary cessation of smoking. *Addictive Behaviors, 10*(3), 291-298.

Yiming, C., Changxin, Z., Ung, W. S., Lei, Z., & Kean, L. S. (2000). Laser acupuncture for adolescent smokers--a randomized double-blind controlled trial. *American Journal of Chinese Medicine, 28*(3-4), 443-449.

Yotsukura, M., Koide, Y., Fujii, K., Tomono, Y., Katayama, A., Ando, H., Suzuki, J., & Ishikawa, K. (1998). Heart rate variability during the first month of smoking cessation. *American Heart Journal, 135*(6 Pt 1), 1004-1009.

Zabaneh, R. I., Ejaz, A. A., & Christiansen, B. (1994). Nicotine scores again: in-hospital withdrawal. *Journal of the American Medical Association, 272*(20), 1576-1577.

Zabetian, C. P., Gelernter, J., & Cubells, J. F. (2000). Functional variants at CYP2A6: new genotyping methods, population genetics, and relevance to studies of tobacco dependence. *American Journal of Medicine & Genetics, 96*(5), 638-645.

Zhu, S. H., Tedeschi, G., Anderson, C. M., Rosbrook, B., Byrd, M., Johnson, C. E., & Gutierrez-Terrell, E. (2000). Telephone counseling as adjuvant treatment for nicotine replacement therapy in a "real-world" setting. *Preventive Medicine, 31*(4), 357-363.

Zoli, M., Lena, C., Picciotto, M., & Changeux, J. (1998). Identification of four classes of brain nicotinic receptors using Beta 2 mutant mice. *Journal of Neuroscience, 18*, 4461-4472.