SMOKING CESSATION USING AN INTERPERSONAL COPING SKILLS PROGRAM

by

Mary-Beth Minthorn-Biggs

A thesis submitted in conformity with the requirements for the degree of Doctor of Education
Department of Adult Education, Community Development and Counselling Psychology
Ontario Institute for Studies in Education of the University of Toronto

© Copyright by Mary-Beth Minthorn-Biggs 1999
The author has granted a non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.
SMOKING CESSION USING AN INTERPERSONAL COPING SKILLS PROGRAM
Mary-Beth Minthorn-Biggs, Ed.D., 1999
Department of Adult Education, Community Development and Counselling Psychology
University of Toronto

ABSTRACT

The present study investigated the effectiveness of Christensen and Pass' (1983) interpersonal coping skills model as a tool for facilitating smoking cessation and relapse prevention. It was hypothesized that the implementation of the seven skills from the interpersonal coping skills program (Social Interaction Program) would assist in the acquisition of the skills conducive to quit smoking and to deal more effectively with the high risk situations which lead individuals to relapse, post cessation.

Seventy-five subjects (51 female and 24 male) who responded to a local advertisement for a smoking cessation study were randomly assigned to a Social Interaction Program group, The Lung Association Countdown Programme group, or to the No Treatment Control group. At the designated quit day, approximately 62% of the individuals in the Social Interaction Program group had quit smoking, while only 22% of those in the Lung Association Countdown Programme group and 10% of those in the No Treatment group had quit smoking. At the six month post cessation assessment period, the Social interaction Program group had significantly more quitters than the No Treatment group, whereas the Lung Association Programme group did not. At the one year post cessation assessment period, there was no significant difference in the percent of
individuals who smoked between the Social Interaction Program group and the Lung Association Countdown Programme group. However, those individuals in the Social Interaction Program group were smoking significantly fewer cigarettes than those individuals in the Lung Association Countdown Program group.

There were no significant differences on any of the pretreatment variables between those individuals who quit smoking and those who did not quit smoking. With respect to relapse, those individuals who relapsed, scored significantly higher on the depression subscale of the Profile of Mood States Questionnaire (POMS), than did those who did not relapse. The reason for relapse provided by subjects in the study closely paralleled the findings of Marlatt and Gordon (1980, 1985). That is, a majority relapsed due to peer pressure and depression.

The Social Interaction Program was effective in promoting smoking cessation. It is probable that the effectiveness of the program could be maintained past six months with the incorporation of pharmacological intervention to address depression, which has been previously demonstrated to significantly impede successful smoking cessation (Abrams et al., 1987; Carmody, 1992; Covey et al., 1990; Lerman et al., 1996).

The Social Interaction Program could also be modified and utilized as a skill teaching program for adolescent prevention programs. Research suggests that between 33% and 50% of individuals who start smoking, become regular smokers (McNeil, 1991). Since adolescent smoking is often viewed as a collective, peer behavior (King et al., 1996), it would be prudent to provide prevention which incorporates effective coping skills to deal with the pressure to commence smoking.
ACKNOWLEDGEMENTS

I can not begin to express my gratitude to my supervisor, Dr. Niva Piran, for her unconditional support as I attempted to complete this project. She encouraged me to finish my thesis, although I had let it sit for a considerable period of time. Niva, thank you very much, your patience and confidence allowed me to finish something I did not think I could complete. I would also like to thank Dr. Bill Melnyk, my friend and mentor. He provided friendship and comfort during this unending project. Thank you Bill for your gentle prodding (sometimes on a daily basis!) to keeping me on task. You have inspired me to continue learning.

I am grateful to my husband Barry, and my daughters Mallory and Hayley for their support and love. You kindly allowed me the time to finish this project, even when it necessitated that I miss some family events. I would also like to thank my good friend Antoinette Savas, who stood by me during these last few years, quietly encouraging me to finish. Finally, I would like to acknowledge the spirit of Keith Gamble, who's zest for life will not be forgotten.
# Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABSTRACT</td>
<td>ii</td>
</tr>
<tr>
<td>ACKNOWLEDGMENTS</td>
<td>iv</td>
</tr>
<tr>
<td>List of Tables</td>
<td>vii</td>
</tr>
<tr>
<td>List of Figures</td>
<td>viii</td>
</tr>
<tr>
<td>List of Appendices</td>
<td>ix</td>
</tr>
<tr>
<td>Chapter 1. INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>Chapter 2. LITERATURE REVIEW</td>
<td>9</td>
</tr>
<tr>
<td>2.1 Theoretical rationale for why individuals start and continue to smoke</td>
<td>9</td>
</tr>
<tr>
<td>2.1.1 Physiological Theories for Smoking Initiation and Maintenance</td>
<td>9</td>
</tr>
<tr>
<td>2.1.2 Psychological Theories for Smoking Initiation and Maintenance</td>
<td>18</td>
</tr>
<tr>
<td>2.1.3 Psychobiological Theories for Smoking Initiation and Maintenance</td>
<td>25</td>
</tr>
<tr>
<td>2.1.4 Smoking as an Addiction</td>
<td>30</td>
</tr>
<tr>
<td>2.2 Methods of Smoking Intervention</td>
<td>33</td>
</tr>
<tr>
<td>2.2.1 Cognitive-Behavioral Approaches to Smoking Intervention</td>
<td>33</td>
</tr>
<tr>
<td>2.2.2 Hypnosis as an Approaches to Smoking Intervention</td>
<td>36</td>
</tr>
<tr>
<td>2.2.3 Nicotine Replacement Therapy for Smoking Intervention</td>
<td>37</td>
</tr>
<tr>
<td>2.2.4 Other Pharmacological Therapies for Smoking Intervention</td>
<td>40</td>
</tr>
<tr>
<td>2.2.5 Multi-component Techniques for Smoking Intervention</td>
<td>41</td>
</tr>
<tr>
<td>2.3 Stages of Change Model</td>
<td>44</td>
</tr>
<tr>
<td>2.4 Theories of Relapse</td>
<td>45</td>
</tr>
<tr>
<td>2.4.1 The Marlatt and Gordon Relapse Prevention Model</td>
<td>45</td>
</tr>
<tr>
<td>2.5 Implications for the Prevention and Treatment of Smoking</td>
<td>49</td>
</tr>
</tbody>
</table>
Table of Contents (Continued)

2.6 The Social Interactional Approach to Counselling/Psychotherapy 51
2.7 Focus of the Present Study
2.7.1 Research Questions and Hypothesis 55

Chapter 3. METHODOLOGY
3.1 Procedure 59
3.2 Research Participants 60
3.3 Measures
3.3.1 Outcome Measures 63
3.3.2 Dependent Measures 66
3.4 Procedure
3.4.1 Initial Contact 74
3.4.2 Initial Assessment 75
3.4.3 Treatment
3.4.3.1 The Social Interaction Coping Skills Program 76
3.4.3.2 The Lung Association Countdown Programme 77
3.4.3.3 The No Treatment Control Group 77
3.4.4 Post Treatment Assessments 77
3.4.5 Follow-up Assessment Session 78
3.5 Analysis 78

Chapter 4. RESULTS
4.1 Smoking Cessation
4.1.1 Hypothesis 1: Increase the success of smoking cessation 80
4.1.2 Hypothesis 2: Time of relapse 80
4.1.3 Hypothesis 3: Number of cigarettes smoked post quit 83
4.1.4 Hypothesis 4: Predictors of smoking cessation 83
4.2 Daily Measures Post Quit
4.2.1 Personality Variables 94
4.3 Relapse Data 97

Chapter 5. DISCUSSION
5.1 Smoking Cessation 98
5.2 Demographic and Personality Variables 100
5.3 Relapse 103
5.4 Limitations of the Present Study and Suggestion for Future Research 107
5.5 Implications of the Present Study
5.5.1 Research 111
5.5.2 Clinical Practice and Prevention 111
5.6 Conclusion 115

REFERENCES vi

vi
## List of Tables

<table>
<thead>
<tr>
<th>Table</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Table 1-1.</td>
<td>Summary of Direct and Indirect Costs for Tobacco Use in Ontario 1992 (in millions of dollars)</td>
<td>3</td>
</tr>
<tr>
<td>Table 3-1.</td>
<td>Demographic and Smoking Comparisons of Initial Sample</td>
<td>61</td>
</tr>
<tr>
<td>Table 3-2.</td>
<td>Demographic and Smoking Comparisons of Drop Outs</td>
<td>62</td>
</tr>
<tr>
<td>Table 3-3.</td>
<td>Demographic and Smoking Comparisons of Main Sample</td>
<td>64</td>
</tr>
<tr>
<td>Table 3-4.</td>
<td>Comparisons Among the Groups on the Pretreatment Variables</td>
<td>65</td>
</tr>
<tr>
<td>Table 3-5.</td>
<td>Administration of Assessment Tools</td>
<td>73</td>
</tr>
<tr>
<td>Table 4-1.</td>
<td>Percentage of Individuals Who Quit Per Group Post Cessation</td>
<td>81</td>
</tr>
<tr>
<td>Table 4-2.</td>
<td>Group Comparisons for Cigarettes Smoked at Post Quit Assessment Periods</td>
<td>84</td>
</tr>
<tr>
<td>Table 4-3.</td>
<td>Symptom Segment Scores By Group</td>
<td>89</td>
</tr>
<tr>
<td>Table 4-4.</td>
<td>POMS Segment Scores By Group</td>
<td>90</td>
</tr>
<tr>
<td>Table 4-5.</td>
<td>Post Cessation Smoking Confidence Questionnaire Scores</td>
<td>95</td>
</tr>
<tr>
<td>Table 4-6.</td>
<td>Urge Ratings 30 Day Post Cessation for SI Group</td>
<td>96</td>
</tr>
</tbody>
</table>
# List of Figures

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 1-1.</td>
<td>Diagram of the exposure model of tolerance</td>
<td>14</td>
</tr>
<tr>
<td>Figure 1-2.</td>
<td>Diagram of the sensitivity model</td>
<td>16</td>
</tr>
<tr>
<td>Figure 2-1.</td>
<td>A Cognitive-Behavioral Model of the Relapse Process</td>
<td>48</td>
</tr>
<tr>
<td>Figure 2-2.</td>
<td>Relapse Prevention: Specific Intervention Strategies</td>
<td>50</td>
</tr>
<tr>
<td>Figure 4-1.</td>
<td>The Percentage of Subjects Quitting in Each Group</td>
<td>82</td>
</tr>
<tr>
<td>Figure 4-2.</td>
<td>Mean Number of Cigarettes Smoked Per Group Post Cessation</td>
<td>85</td>
</tr>
<tr>
<td>Figure 4-3.</td>
<td>Group Comparisons on Symptoms Ratings</td>
<td>87</td>
</tr>
<tr>
<td>Figure 4-4.</td>
<td>Group Comparisons on POMS Scores</td>
<td>88</td>
</tr>
<tr>
<td>Figure 4-5.</td>
<td>Symptom Ratings for Quitters and Non-Quitters</td>
<td>92</td>
</tr>
<tr>
<td>Figure 4-6.</td>
<td>POMS Ratings for Quitters and Non-Quitters</td>
<td>93</td>
</tr>
</tbody>
</table>
List of Appendices

<table>
<thead>
<tr>
<th>Appendix</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Categories for Classification of Relapse Episodes</td>
<td>135</td>
</tr>
<tr>
<td>B</td>
<td>Social Interactional Coping Skills Program for Smoking Cessation and Relapse Prevention</td>
<td>137</td>
</tr>
<tr>
<td>C</td>
<td>Lung Association Countdown Calendar</td>
<td>141</td>
</tr>
<tr>
<td>D</td>
<td>Personal Consent Form</td>
<td>142</td>
</tr>
<tr>
<td>F</td>
<td>Smoking Confidence Questionnaire</td>
<td>143</td>
</tr>
<tr>
<td>G</td>
<td>Profile of Mood States Questionnaire</td>
<td>144</td>
</tr>
<tr>
<td>H</td>
<td>Anger Expression Scale</td>
<td>145</td>
</tr>
<tr>
<td>I</td>
<td>Self-report Symptom Checklist</td>
<td>146</td>
</tr>
<tr>
<td>J</td>
<td>Personal Information</td>
<td>147</td>
</tr>
<tr>
<td>K</td>
<td>Situations for smoking temptation</td>
<td>148</td>
</tr>
<tr>
<td>L</td>
<td>Reason for Relapse</td>
<td>149</td>
</tr>
<tr>
<td>M</td>
<td>Table Comparing Quitter and Non-quitters on Demographic, Smoking History and Personality Variables</td>
<td>150</td>
</tr>
</tbody>
</table>

ix
Chapter 1

INTRODUCTION

The World Health Organization (WHO) estimates that there are approximately 1.1 billion smokers in the world today, with almost three times the number of smokers in developing countries as in developed countries (World Health Organization, 1998). WHO (1998) also estimates that six people worldwide die every minute from a smoking related illness and that one out of two smokers will eventually die from a smoking related illness. It is expected that by the year 2020, smoking will kill 10 million people worldwide annually.

Currently, tobacco kills three times more Canadians than the combination of car accidents, AIDS, suicide, murder, illicit drugs and alcohol (Peto, Lopez, Boreham and Thun, 1994). In Canada, it is estimated that more than 40,000 people over the age of 35 years, die annually as a direct result of smoking. The Canadian statistics on young smokers (Survey on Smoking in Canada (SOSIC), 1994) indicate a dramatic increase in youth smoking. The Health Promotion Survey results found that in 1990, 21% of both males and females in the 15 to 19 year age range were current smokers. However, by 1994 the rate for males had increased to 26% and to 29% for females (SOSIC, 1994). The Survey on Smoking in Canada (1994) also found that 41% of males and 38% of females in
the age ranges of 20 to 24 years of age were current smokers. Ninety-eight percent of the current youth smokers indicated that they had had their first cigarette by the age of 17.

The increase in youth smoking suggests that the current load on the health care system will increase should these cohort groups continue to smoke. The economic costs of smoking on the health care system are significant and will continue to escalate if there is not a mechanism in place to promote early smoking intervention and prevention. The total cost for substance abuse related health care in Ontario in 1992 was $7,030,532 million (Xie, Rehm, Single and Robson, 1996). Of this, $3,683,737 million was related to tobacco. Xie et al. (1996) estimate that the per capita cost of substance use disorders in Ontario is approximately $662, of which $347.00 is related to smoking (see Table 1 for a summary of the Direct and Indirect Costs for tobacco in Ontario in 1992). Similarly, Xie et al. (1996) estimate that the monetary burden on the health care system in Ontario has increased from approximately 1.35 billion dollars in 1979 to 3.62 billion dollars in 1988.

Tobacco use increases the risk of cancers, cardiovascular diseases, respiratory diseases, gastrointestinal problems and high risk pregnancies (WHO, 1995). WHO (1995) estimates that there are at least 16,000 cardiovascular deaths each year in Canada which are related to smoking. It is estimated that cardiovascular disease is two to four times greater in smokers than non-smokers. Similarly, smokers have a two to four times greater risk of dying from stroke than
### Table 1-1

**Summary of Direct and Indirect Costs for Tobacco Use in Ontario 1992**

*(in millions of dollars)*

<table>
<thead>
<tr>
<th>Direct Costs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Health Care</strong></td>
<td></td>
</tr>
<tr>
<td>Costs of Hospital Care</td>
<td>687.409</td>
</tr>
<tr>
<td>Cost of Ambulance Services</td>
<td>23.010</td>
</tr>
<tr>
<td>Cost of Physician Services</td>
<td>157.513</td>
</tr>
<tr>
<td>Prescription Drugs Out of Hospital</td>
<td>170.622</td>
</tr>
<tr>
<td>Home Care</td>
<td>34.621</td>
</tr>
<tr>
<td><strong>Health Care Total:</strong></td>
<td>1073.175</td>
</tr>
<tr>
<td><strong>Research, Prevention and Training</strong></td>
<td></td>
</tr>
<tr>
<td>Research</td>
<td>13.200</td>
</tr>
<tr>
<td>Prevention</td>
<td>9.800</td>
</tr>
<tr>
<td>Training Costs</td>
<td>0.0</td>
</tr>
<tr>
<td><strong>Research, Prevention and Training Total:</strong></td>
<td>23.000</td>
</tr>
<tr>
<td><strong>Fire and Accident Damage</strong></td>
<td></td>
</tr>
<tr>
<td>Forest Fire Damage</td>
<td>2.346</td>
</tr>
<tr>
<td>Property Fire Losses</td>
<td>4.059</td>
</tr>
<tr>
<td><strong>Fire and Accident Damage Total:</strong></td>
<td>6.405</td>
</tr>
<tr>
<td><strong>Direct Costs - Total:</strong></td>
<td>1102.580</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indirect Costs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Productivity Losses due to Morbidity</td>
<td>30.260</td>
</tr>
<tr>
<td>Productivity Losses due to Mortality</td>
<td>2550.896</td>
</tr>
<tr>
<td><strong>Indirect Costs - Total:</strong></td>
<td>2581.156</td>
</tr>
</tbody>
</table>

**Total Costs:** 3683.736

---

The Economic Costs of Alcohol, Tobacco and Illicit Drug Abuse in Ontario: 1992
do non-smokers. More than 2,000 smokers die each year in Canada from strokes. Smoking is also responsible for more than 16,000 cancer deaths in Canada (WHO, 1995). Almost 30% of all cancers are related to smoking. It is estimated that 80%-85% of lung cancers are related to smoking. It is the most common cancer among men and in 1993, it surpassed breast cancer as the leading cancer in women. However, in the mid 1980's, lung cancer rates began to level off in males (Xie et al., 1996). It is estimated that the lung cancer rates will not level off for women until after the year 2000. Recognizing the increase in smoking rates in the youth in Canada, it is likely that lung cancer will continue as the leading cause of cancer death for at least the next twenty-five years. Smoking is also the major cause of mouth cancers and cancers of the larynx and esophagus. Similarly, smoking is related to cancers of the pancreas, stomach, kidneys and bladder. With respect to respiratory diseases, smoking is responsible for more than 8,000 deaths annually in Canada (WHO, 1995). Smoking accounts for 80 - 90% of all chronic obstructive pulmonary disease (COPD) deaths. Smokers also appear to be at greater risk for respiratory infections and the common cold. Smoking has been related to significantly lower birth rates in pregnant women who smoke than those women who do not smoke (WHO, 1995). As well, pregnant women who smoke are at a higher risk for miscarriage, premature delivery, stillbirth, and abnormal placentas.

Environmental tobacco smoke (ETS) or second hand smoke has also been demonstrated to cause significant health problems (Ashley and Ferrence, 1995).
Ashley and Ferrence (1995) estimate that in the United States alone, between 150,000 and 300,000 cases of bronchitis and pneumonia in infants and toddlers under the age of 18 months are attributable to environmental tobacco smoke. Each year, up to 1,000,000 asthmatic children in the United States are adversely affected by exposure to second hand smoke. The Environmental Protection Agency (EPA) has reported a relationship between environmental tobacco smoke and sudden infant death syndrome (SIDS) as have several paediatric researchers (Mitchell, Ford, Stewart, Taylor, Becroft, Thompson, Scragg, Hassall, Barry, Allen and Roberts, 1993; Scragg, Mitchell, Taylor, Stewart, Ford, Thompson, Allen and Becroft, 1993; Scragg, Stewart, Mitchell, Ford and Thompson, 1995; Cohen-Klonoff, Edelstein, Lefkowitz, Srinivasan, Kagei, Chang and Wiley, 1995). The EPA (1993) declared environmental tobacco smoke to be a human carcinogen, stating that it was responsible for approximately 3,000 lung cancer deaths a year in the United States for non-smokers. Health Canada estimated that there are more than 300 deaths from lung cancer in non-smokers annually, caused from environmental tobacco smoke (Ashley, 1995).

Environmental tobacco smoke is composed of the exhaled mainstream smoke from the smoker; the sidestream smoke from the smoker; and contaminants which are diffused through the cigarette paper (EPA, 1992). Sidestream smoke also contains all of the toxins and the carcinogens which are found in mainstream smoke, which is inhaled by the smoker. Environmental tobacco smoke is found in restaurants, homes, workplaces, cars, bars and
nightclubs. The American Environmental Protection Agency (1992) declared that environmental tobacco smoke belongs in the category of Group A carcinogens, stating that it is a human carcinogen and a cause of lung cancer in non-smokers.

Although many individuals understand the health risks associated with smoking, 31% of Canadians smoke more than 15 cigarettes per day (SOSIC, 1994). In Canada, smoking rates dropped from 61% to 31% from 1965 to 1990 (HPS, 1990). Similarly, the rate in females dropped from 38% to 28%. Health Canada (1994) estimates that 5.9 million Canadians are former smokers. The province of Quebec has the highest rate of smokers at 38%, whereas British Columbia has the lowest rate at 25% (SOSIC, 1994). Prince Edward Island has the highest rate of male smokers at 48%, while Quebec has the highest rate of female smokers at 38% (SOSIC, 1994). According to Millar (1992), Aboriginal persons have the highest smoking rate of any ethnic group in Canada, at 59%. The lowest rate is found in Vietnamese and South East Asian groups at approximately 9%.

The research document, Facts on Tobacco (1996) indicates that the average age of smoking initiation in Ontario is 15 years of age. Women in the 18-34 age group have the highest rates of current smoking at 35%, while women who are over 55 years of age have the lowest rates of smoking at 21%. Men in the middle age group of 34-55 years of age have the highest rate of smoking in Ontario at 36%, while those over the age of 55 years have the lowest rate at 17% (Facts on Tobacco, 1996). In 1995, 61% of Ontarians aged 18 years and older
who had ever smoked had quit, an increase from 54% in 1991 (Millar, 1992).
The World Health Organization (1995) estimates that in developing countries, 50% of males smoke and 8% of females smoke. The proportion of females smoking in developing countries is increasing, whereas the rate of smoking in adults in developed countries is declining (Peto et al., 1994). Peto et al. (1994) estimate that there are approximately 3 million tobacco related deaths worldwide, with almost one third of them in developing countries. They estimate that worldwide within the next 30 to 40 years, there will be 10 million tobacco related deaths per year, with 70% of them occurring in developing countries.

The tobacco industry has changed its market focus to the markets in developing countries (WHO, 1995), where the trends for smoking are on the increase. Due to the increase of smoking in developing countries, the world wide production of tobacco products has remained unchanged over the last 30 years, despite the fact that smoking rates are decreasing in developed countries. In Canada, four main tobacco product manufactures: Imperial Tobacco Limited, Rothmans, Benson & Hedges Inc., and RJR-Macdonald Inc., control 99% of all tobacco products sold in Canada (Statistics Canada, 1996). In 1991 (Statistics Canada, 1996) the tobacco industry directly employed 4,404 Canadians. It had reduced the production facilities from nine factories to five between 1981 and 1991, with no change in the magnitude of tobacco product production (Statistics Canada, 1996).

The tobacco industry spends considerable money in advertising. Cigarette
advertising has become the primary source of advertising revenue for Russian television (WHO, 1995). However, the governments of the developed countries are attempting to change the laws pertaining to the advertising of cigarettes and tobacco products. In Strasbourg, the European parliament approved significant bans on tobacco advertising in the 15 nations which it represents (Time Magazine, May 25, 1998). In Canada, the federal government has passed the controversial Tobacco Act (1991), which is regulating the use of the tobacco industry sponsorships of sporting and cultural events, as well as advertising. However, due to significant pressure, the federal government will give the tobacco industry, who is sponsoring and organizing sporting and cultural events, up to five years to withdraw their sponsorships. There will be no restrictions for the next two years (1998 and 1999), where upon, curbs leading to total ban will start to take over in the subsequent three years (Globe and Mail, June 3, 1998).
Chapter 2  
LITERATURE REVIEW  
2.1 Theoretical rationale for why individuals start to and continue to smoke

Recognizing the health risks, it is somewhat difficult to understand why individuals choose to start smoking and why they continue to smoke, despite information on tobacco-related illnesses. Van Roosmalen and McDaniel (1989) indicate that peer groups are crucially important to the initiation of smoking behavior in adolescents. Why do adolescents and young adults continue to smoke, despite the initial adverse affects of nausea and respiratory irritation? There are several different approaches to explaining the maintenance of smoking behavior. For ease of discussion, the prominent theories have been divided into three categories: physiological, psychological, and psychobiological.

2.1.1 Physiological Theories for Smoking Initiation and Maintenance

The physiological effects of nicotine were first studied by Langley and Dickinson in 1889. However, it was not until the early 1900's that the deleterious effects of nicotine upon the human body were investigated. Further, few studies prior to the 1950's looked at the possible rewarding properties of nicotine with respect to the smoking habit.

The essential reason why individuals experience significant difficulty quitting smoking is due to the addictive properties of nicotine, which maintains smoking behavior. Nicotine is the primary alkaloid of tobacco and it represents almost 95\%
of the total alkaloid content of tobacco (Le Houezec and Benowitz, 1991). Nicotine is a tertiary amine, which is composed of a pyridine ring and a pyrrolidine ring. It is absorbed very quickly from cigarette smoke, taking between 10 and 20 seconds to pass through to the brain (Benowitz, 1996).

Recent research has found that nicotine activates the mesocorticolumbic dopamine (DA) system in a manner similar to that of cocaine (Pich, Pagluisi, Tessari, Talabot-Ayer, van Huijsduijnen and Chiamulera, 1997). Studies in the area of addiction indicate a strong link between the mesocorticolumbic DA system stimulation and drug seeking behavior (Di Chiara, 1995). The neuronal nicotinic acetylcholine receptors have a primary role in nicotine addiction. Neuronal nicotinic acetylcholine receptors are found to have the greatest number of binding sites in the amygdala, septum, and brain stem motor nuclei and locus ceruleus (Role and Berg, 1996). The number of different receptors sites may contribute to the many different effects of nicotine in individuals.

Nicotine is readily absorbed by the body and crosses the blood-brain barrier with ease, and as a result is readily distributed across the brain very quickly. Absorption of nicotine can be both via passive diffusion and active transport via the choroid plexus (Spector and Goldberg, 1982). Nicotine binds stereospecifically to the acetylcholine receptors of the autonomic ganglia, the adrenal medulla, neuromuscular junctions, and the brain (Benowitz, 1988). Two types of nicotinic receptors have been differentiated - C10 and C8. These two receptors act primarily on the central nervous system rather than the peripheral nervous system. Clarke
states that recent studies have found that central nicotinic receptors are a distinct subtype. Nicotine modulates several central nervous system pathways by stimulating the presynaptic receptors (Le Houezec and Benowitz, 1991). This leads to the release of acetylcholine, norepinephrine, dopamine, serotonin, vasopressin, growth hormone, and ACTH. It is believed that the effects on the central nervous system are primarily related to the direct action nicotine has on the brain.

The sympathetic and parasympathetic nervous system are both stimulated via the nicotine found in cigarettes. Le Houezec and Benowitz (1991) indicate that the effects of nicotine are dose dependent and biphasic. That is, small doses of nicotine excite the nervous system, while large doses of nicotine produce inhibition after the initial excitation. This biphasic response appears to have a direct relationship with the acute and chronic tolerance of nicotine in humans and laboratory animals. The level of nicotine and its relational effects has been a topic of recent research (Le Houezec and Benowitz, 1991; Benowitz, 1996). The relationship between the dose of nicotine and the corresponding physiological response and the development of dose tolerance is relatively complicated.

Nicotine has an elimination half life of two hours. However, it accumulates over six to eight hours of regular smoking. Cotinine, a primary metabolite of nicotine, has a longer half life, approximately 16 hours. As a result, it is often used as a biochemical marker for nicotine intake in smoking cessation studies. However, with the recent increase in the utilization of nicotine replacement therapy
in smoking cessation treatment, the measuring of cotinine as an indicator of cessation will be confounded since it is also a metabolite of the replacement treatment. An active smoker will be exposed to significant levels of nicotine over the average day. Tolerance to many of the side effects of nicotine can therefore develop relatively quickly (Le Houezec and Benowitz, 1991). However, due to the short half life, the average individual who abstains from smoking overnight, eliminates the accumulated nicotine, which is mainly metabolized by the liver. Between 70% and 80% of nicotine is metabolized into cotinine and about 4% into nicotine N'-oxide (Benowitz, 1996). Cotinine is excreted in urine. However, there appears to be significant individuation in the ability for individuals to metabolize nicotine.

Cotinine apparently has no effect on nicotine receptors. However, it does cause decreased blood pressure in animals (Benowitz, 1996). This may explain why smokers often have lower blood pressure than non-smokers. Cotinine is currently being studied for possible use in nicotine replacement therapy.

Addiction to nicotine can occur relatively quickly. For the regular smokers, the first cigarette smoked during the day produces significant arousal, however, at the same time tolerance begins to develop. Tolerance is defined as when, after repeated doses, a given dose of a drug produces less effect, or when increasing doses are required to achieve the effect observed with the first dose (Benowitz, Porchet and Jacob III, 1990). Tolerance is lost to a certain degree after overnight abstinence.
The 1988 U.S. Surgeon General's Report, *The Health Consequence of Smoking: Nicotine Addiction* (USDHHAS, 1988) differentiated tolerance into four subtypes. Pharmacokinetic tolerance relates to accelerated drug elimination as a mechanism for explaining diminished effects after repeated dosing. Behavioral tolerance is related to the decreased effect of a given dose of a drug as a result of compensatory behaviors which reduce adverse effects on performance or activity. Conditional tolerance refers to responses elicited by association with drug administration; while functional tolerance (pharmacodynamic) refers to the loss of responsivity to dosing observed when dispositional and behavioral tolerance can be ruled out. That is, a condition in which drug concentration at the receptor site produces a smaller effect as a result of exposure. If after one or two exposures tolerance develops, acute tolerance is said to have been acquired. Tolerance which occurs after prolonged exposure is referred to as chronic tolerance.

McNeil (1991), suggests that between 33% and 50% of those individuals who experiment with smoking, become regular smokers. Early self-administration is presumed to be maintained by social reinforcement - peer pressure. Nausea, dizziness, and vomiting are common side effects initially experienced by the novel smoker. However, the smoker quickly becomes tolerant to these aversive side effects. Pomerleau, Collins, Shiffman and Pomerleau (1993) proposed an exposure tolerance model (Figure 1) which suggests that those individuals who are more sensitive to the initial aversive side effects of smoking are less likely to become smokers. McNeil (1991) posits that the initial positive social
reinforcement experienced by the smoker is replaced by avoidance of nicotine withdrawal.

Although the exposure model of tolerance lends itself to ease of understanding, there exists minimal empirical data to support this hypothesis. As a result, Pomerleau et al. (1993) presented a second model, the sensitivity model of tolerance (Figure 2). This model suggests that individuals with high innate sensitivity experience both aversive effects, but also reinforcing consequences (such as social reinforcement, temporary improvement in performance and attention, termination of withdrawal symptoms). In this model, Pomerleau et al., hypothesized that heavy smokers are nicotine sensitive individuals who have been able to develop tolerance to the aversive effects to a degree which permits chronic use. The discontinuation of use leads to loss of tolerance and physical withdrawal (Pomerleau et al., 1993).

Individuals who do experience minimal side effects upon initial exposure, those who are less sensitive, may remain nonsmokers or minimal smokers, depending on the existing social conditions surrounding use. Contrary to the exposure model, the sensitivity model suggests that constitutional factors determine the degree of dependence. These are sets of biobehavioral adaptions made up of both learned associative factors and nonassociative factors.

Although individuals differ, does genetics have a role in nicotine dependence? An initial twin study 40 years ago, demonstrated that concordance for smoking behavior was higher in monozygotic twins than in dizygotic twins
<table>
<thead>
<tr>
<th>Initial Sensitivity</th>
<th>Self-Administration Pattern</th>
<th>Degree of Tolerance</th>
</tr>
</thead>
<tbody>
<tr>
<td>High sensitivity</td>
<td>no further exposure after</td>
<td>no tolerance</td>
</tr>
<tr>
<td>(aversive effects)</td>
<td>initial experimentation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(nonsmoker)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>exposure via lowgradual</td>
<td></td>
</tr>
<tr>
<td></td>
<td>nicotine self-administration development of some (chipper or light smoker)</td>
<td>some tolerance</td>
</tr>
<tr>
<td>Low sensitivity</td>
<td>exposure via high</td>
<td>grad ual development</td>
</tr>
<tr>
<td>(minimal aversive effects)</td>
<td>nicotine self-administration</td>
<td>extensive tolerance</td>
</tr>
<tr>
<td></td>
<td>(dependent smoker)</td>
<td></td>
</tr>
</tbody>
</table>

*Figure 1* Diagram of the exposure model of tolerance (Pomerleau et al., 1993)
<table>
<thead>
<tr>
<th>Initial Sensitivity</th>
<th>Degree of Tolerance</th>
</tr>
</thead>
<tbody>
<tr>
<td>high sensitivity</td>
<td>exposure rapidly induces self-administration</td>
</tr>
<tr>
<td>(mix of aversive and rewarding effects)</td>
<td>tolerance to aversive effects</td>
</tr>
</tbody>
</table>

Intermittent administration smoker

<table>
<thead>
<tr>
<th>low sensitivity</th>
<th>exposure leads to minimal change in initial sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>(minimal effects)</td>
<td>no further exposure after initial experiment</td>
</tr>
</tbody>
</table>

Figure 2. Diagram of the sensitivity model (from Pomerleau et al., 1993).
These findings have been replicated several times, even with twins who have been reared apart. Eaves and Eysenck (1975) suggest that genetics also plays a role in determining the age of smoking onset and the number of cigarettes smoked daily. Eysenck (1980) proposed that 60% of the variance in age of onset of smoking loaded on environmental factors, while 68% of the variance in persistence in smoking loaded on genetic factors. One may conclude then, that environmental factors may contribute to the initiation of smoking, but that biological factors contribute to the maintenance of the behavior.

The issue of physical dependence resulting from continue smoking is still not agreed upon. According to Schachter (1980), a smoker smokes to regulate the level of nicotine. He suggests that there exists a central mechanism which can sense the level of nicotine and that this mechanism provides the impetus for the individual’s behavior. Physical dependency is measured by a withdrawal or abstinence syndrome (Jarvik, 1970). The controversy pertaining to the dependency issue revolves around the issue of nicotine withdrawal. There are several symptoms which have been attributed to smoking cessation such as: craving for tobacco, irritability, anxiety, increased appetite, insomnia, constipation, lightheadedness, and restlessness (Cummings, Giovino, Jaen, and Emrich, 1985; Hughes, Hatsukami, Picens, Krahn, Malin and Luknic, 1984; Hughes and Hatsukami, 1986). Although these findings have been replicated, Jarvik (1977) postulates that the changes which occur during smoking cessation could be explained as the adjustment of the body to changes in the level of arousal, rather
than as a withdrawal syndrome.

There are problems with the nicotine regulation model offered by the proponents of the biological theory of smoking maintenance. The nicotine regulation model does not adequately explain why there is no significant difference between heavy smokers and light smokers (with the exception of the self-reported measures of irritability) on self-reported and observed measures of withdrawal during smoking cessation programs (Cummings et al., 1985; Hatsukami et al., 1984). Although Lucchesi (1976), found that intravenous infusion of nicotine diminished the frequency and amount of cigarettes consumed by the subjects in his study, the relationship between blood plasma levels and cravings for cigarettes has yet to be clearly established. Similarly, why people relapse after considerable time in which nicotine has been absent from their system, can not be explained by the nicotine regulation model.

2.1.2. Psychological Theories of Smoking Initiation and Maintenance

Why do individuals begin to smoke? According to Russell (1977), smoking behavior is usually acquired through a routine of social reinforcement (e.g. peer pressure). Although initially the inhalation of smoke can be aversive, (causing nausea, dizziness, and/or lightheadedness), after a period of time, habituation to these effects occurs (Pomerleau, 1981).

Why is it that some people smoke, while others do not? Many researchers have attempted to differentiate smokers from non-smokers. Larson, Hagg and Silvette (1973) suggested that smokers tend to be impatient, somewhat more
angry and much more easily bored than non-smokers. Eysenck (1973) suggested that those individuals who smoke are extroverted and they are somehow not functioning at their optimal level of arousal. Although studies have consistently reported that smokers' personalities can be characterized by extroversion and sensation-seeking (ie. Lester, 1974), few have investigated whether those differences are related to different motivational factors for smoking.

King, Connop, and Mercer (1996) investigated the factors which may contribute to adolescent female smoking. Health Canada (1995) reported that there are more females in the age group of 15 years to 19 years smoking, than males in that same age group. This is despite the fact that legislation has increased the legal age for tobacco purchase. The research conducted by King et al. (1996) suggests that smoking is a collective behaviour which is reflective of the group norm and values. Once a young person becomes affiliated with a smoking peer group, the pressure to also smoke is almost insurmountable. The pressure is often indirect and covert. However, King et al. (1996) propose that young females have a higher need for social acceptance and therefore quickly adopt the smoking behaviors of the peer group. The initial findings of the King study supports their hypothesis that adolescent females who have a lower level of academic achievement and other probable related difficulties at school and at home are more likely to seek acceptance via a peer group affiliation. King et al. (1996) also found that the adolescent female smoker spends more time than non-smokers with her peer group and that her friends are often experiencing similar
academic and home difficulties. Pederson, Koval and O'connor (1996) investigated the psychosocial factors related to smoking in grade six students. The sample consisted of 1552 grade six students from 107 schools in Ontario. The smokers perceived themselves to be below average or average in school and had a parent or sibling who smoked. Those youth who smoked also missed more school in the previous two months than the non-smokers and had fewer concerns about second hand smoke than did the non-smokers. The smokers had more close friends who smoked and they spent more time with their peer group, than did the non-smokers in the study. The grade sixers who tried smoking or who were current smokers in the Pederson et al. study (1996), scored higher on the scales related to depression and rebelliousness. They also reported more life events in the last year with lower scores for social support than the non-smokers.

The findings from the Pederson et al. study (1996) are consistent with the findings of Wang, Fitzhug, Westerfield and Eddy (1993) and Bates and Pandina (1991) who found that smokers report lower self-esteem and lower levels of peer and family support. Pederson, Koval and O'Connor suggest that perhaps once a youth utilizes smoking as a coping mechanism, they are less likely to develop healthier coping strategies.

Lichtenstein and Brown (1980) have described four stages in the development and maintenance of smoking behavior. They suggest that the reasons individuals commence smoking are psychosocial. That is, an individual may start out of curiosity, social confidence, social pressure, or a variety of other
socially reinforcing reasons. However, social pressure is likely the prime instigator for experimenting with cigarettes (Hill, 1971; Gorsuch and Buthler, 1976). Leventhal and Cleary (1980) view this initial experimentation with smoking as the critical step to becoming a smoker. They report that approximately 85% - 90% of those individuals who smoke more than four cigarettes will eventually become smokers. The smoking behavior is maintained or continues due to psychosocial reinforcements. Lichtenstein and Brown (1980) categorize this maintenance phase as the second stage in their model. The third stage in the development and maintenance of the smoking habit is the stopping phase. At this stage, an individual may attempt to quit for psychosocial reasons, such as: health, social support, or expense. However, they resume (stage four) for psychosocial or physiological factors.

Social learning theory puts much emphasis on the influence of modelling, symbolism, and self-regulatory processes (Bandura, 1977). Smoking maintenance, within a social learning framework, is thought to be acquired by social reinforcement, usually peer pressure. During the initiation phase, the cigarette is likely to be found aversive, but after many trials, the individual habituates or learns to tolerate the aversive effects. After the habituation process, the new smoking behavior starts to provide the individual with adequate positive reinforcement. As a result, the social reinforcement which originally lead to the initial smoking behavior is not needed in order to maintain the smoking. As the smoking continues, the individual's smoking behavior gradually becomes under the
control of other external and internal cues, which act as discriminators for situations that are punishing, neutral, or reinforcing.

Within the social learning model, internal cues, such as an emotion, can influence smoking behavior. Initially, an individual may smoke a cigarette if he or she is angry or anxious. Smoking the cigarette could have been part of a coping response to the stimulus. However, it then leads to an avoidance/escape response to the internal cue. After much reinforcement, the escape response, that is, the smoking behavior, generalizes to other internal cues. In general, smoking behavior, within this framework is maintained as it gives both positive and negative reinforcement to many internal and external cues.

Hunt and Matarazzo (1970) do not view smoking maintenance behavior in terms of dependence or addiction. They explore smoking behavior in terms of habit mechanisms. Hunt and Matarazzo suggest that more emphasis should be placed on the importance of secondary reinforcement in maintaining the smoking behavior. They define habit as “a fixed behaviour pattern over learned to the point of becoming automatic and marked by decreasing awareness and increasing dependence on secondary, rather than primary reinforcement” (p.67). A habit then, according to the authors, can be differentiated from other behaviors by over learning. Many habitual behaviors are often more over learned than one would expect. Take for example, habitual smoking behavior. If the inhalation or puffing of the cigarette is the unit which is reinforced or reinforcing the act of smoking, and the individual smokes 25 cigarettes daily (taking ten puffs per cigarette), within one
year, that individual has received 91,100 reinforcements. It would appear then, that the amount of reinforcement the smoker receives (ie. puffs of cigarette) is higher than that which users of other drugs would receive.

Hunt and Matarazzo (1970) theorize, that although reinforcement is the primary process in the acquisition of a new behaviour, associative learning is the important contributing factor in the maintenance of the behavior. That is, the behavior pattern of smoking becomes over learned to the point of becoming automatic. This automatic habitual behavior will occur spontaneously, independent of reinforcement. These automatic behavior processes are short-circuited as the behavior becomes a function of associative learning. The associative learning is the important factor in the maintenance of the smoking behavior.

Tompkins (1966) on the other hand, in his affect reduction model, postulates that smoking behavior is used to manage affect. According to Tompkins, there are eight primary affects - excitement, enjoyment, distress, anger, fear, shame, and contempt. He further suggests that these affects are innate, in that they are not learned. However, the stimuli which activate the affects, may be either innate or learned (Tompkins, 1966). Basic to the affect reduction model is the premise that there is an innate motivation to maximize positive affect and minimize negative affect.

Tompkins (1966) proposes four types of smoking behavior: 1) habitual smoking, 2) positive affect smoking, 3) negative affect smoking, and 4) addictive
smoking. Tompkins suggests that the habitual smoker may have initially smoked in order to produce a positive affect or to reduce a negative affect. However, the smoker no longer smokes because of affect, and may not be aware that he or she is smoking a cigarette and has likely incorporated smoking into a daily routine. Habitual smoking occurs with a minimal degree of awareness and little reward (Ikard, Green and Horn, 1969).

The positive affect smoker smokes to obtain pleasure. Tompkins divides positive affect smoking into two categories; the cigarette as a stimulant and the cigarette as a relaxant. The stimulant smoker smokes for the positive affect of the excitement associated with it, whereas the relaxant smoker smokes within a relaxing atmosphere (such as after eating, or during a party). Horn (1969) suggests that there is an additional type of positive affect smoking, that which is associated with the sensorimotor aspects of smoking. Here, the individual receives gratification from the process of having a cigarette, that is, holding it, lighting it and watching it burn.

The negative affect smoker smokes when he or she is experiencing one of five negative affects (distress, fear, anger, shame or contempt). This individual smokes primarily to reduce these negative affects. Tompkins labels the negative affect smoking behavior as sedative smoking. He further distinguishes between two types of sedative smokers - the partial smoker and the complete sedative smoker. The partial sedative smoker has a cigarette to help reduce a negative affect so that he or she may better face a problem and attempt to solve it. The
complete sedative smoker does not face his or her problem or attempt to solve it, thereby relying on the cigarette exclusively to reduce the negative affect.

The addictive type of smoker, smokes to alleviate negative affect and to increase positive affect. The individual categorized in this general classification type is aware of not smoking whenever he or she is without a cigarette. The awareness of not smoking is accompanied by negative affect. The experience of negative affect increases until the individual thinks he or she can no longer tolerate it if a cigarette is not available. When the individual finally smokes a cigarette, the negative affect should decrease immediately, and the positive affect would be evoked. The subsequent positive affect confirms the smoker's belief that only a cigarette could reduce negative affect.

2.1.3. Psychobiological Theories for Smoking Initiation and Maintenance

The opponent-process model proposed by Solomon (1977, 1980) attempts to incorporate concepts from both social learning and biological models. Although the opponent-process theory of acquired motivation has been used to explain cigarette smoking behavior, it has yet to be empirically tested as a model for smoking in humans (Pomerleau, 1980). The model postulates that central nervous system mechanisms operate homoeostatically, so as to reduce significant departures from affective equilibrium. The affective departures may be either positive or negative. The return to the homoeostatic balance occurs through the activation of an opposing process. Therefore, if the initial change in the
equilibrium is caused by a positive increase in affect, then a negative process would be initiated. The disequilibrium is usually brought about by unconditional stimuli (UCS) or primary reinforcers. The UCS is an affect-arousing stimulus which can initiate the elicitation of primary affective processes (UCR), that are referred to as the a-processes. Depending upon the UCS which elicits it, the a-process may be a pleasant or an unpleasant affect. When the a-process is elicited, a second response called the b-process is triggered. The b-process is an opposite affective response to the a-process. The a-process is closely associated with the UCS and does not show much habituation. The b-process, on the other hand, is much slower to respond than the a-process, and is slower to peak or to return to baseline following the termination of the UCS (Pomerleau, 1980). According to Solomon (1977), the affective condition of the organism is the algebraic sum of the intensities of the a-process and b-process (where the b-process has a negative sign since it is in opposition to the a-process). As a result, whenever $a$ is greater than $b$, the organism is in State A. That is, the affective state is dictated by the a-process. Whenever $b$ is greater than $a$, the organism is in State B, and the affective state reflects the b-process. If being in State A is positively reinforcing, then being in State B will be negatively reinforcing (Solomon, 1977).

Through his use of imprinting experiments, Solomon was able to establish that b-processes are strengthened by increases of intensity, frequency and duration of the UCS. They are weakened however, through reduction or disuse of the UCS. The a and b processes are non associative and are not based on
conditioning or learning.

In order for an addictive cycle to be maintained, there must be a critical decay duration. The critical decay duration refers to the interval between the UCS presentations. It must be short enough that the complete decay of the b-process is anticipated.

Another model which attempts to integrate psychological and biological concepts is the multiple-regulation model proposed by Leventhal and Cleary (1980). Their theory relies on a developmental framework in which they describe stages of smoking. They suggest that the developmental history of a smoker starts with the preparation stage, and continues through the stages of initiation, becoming a smoker, and maintenance of smoking. This is followed by stages of dissatisfaction, decision to stop and the adoption and maintenance of the self-image of an ex or non-smoker. The first four stages shall be discussed briefly with respect to smoking behaviour maintenance.

The individual usually enters the initiation stage due to social pressure (Hill, 1971; Leventhal and Cleary, 1980). Salber, Freeman, and Abelin (1968) state that 86% to 90% of people who smoke more than four cigarettes become regular smokers. However, data also shows that many people never get to their fourth cigarette. The number of children, adolescents, and teens who smoke while still attending an educational setting usually oscillates around 50% (Leventhal and Cleary, 1980). Yet, Palmer (1970) reports that over 80% of students state trying at least one cigarette. It would appear then, that not all individuals who attempt
smoking continue to smoke.

Those individuals who do not quit at the initiation stage develop a tolerance to the side effects of cigarettes and gradually begin to increase the number of cigarettes consumed. It may take two years or longer for smoking to become consistent and frequent (Leventhal and Cleary, 1980). During this ‘becoming a smoker’ stage, the aversive effects of smoking cigarettes such as dizziness, sore throat, and nausea have not faded. The authors postulate that biological factors are very important in this stage, however they also suggest that social, cognitive and behavioral factors contribute to the process of becoming a smoker. Integral to the fourth stage, the maintenance stage, is that smoking has become a necessary part of self-regulation in many different situations.

Leventhal and Cleary’s multiple-regulation model is an integration of Tompkins affect regulation model and Solomon’s opponent-process model. The primary and most important assumption of the model is the belief that emotional regulation, not nicotine regulation, is critical in smoking behavior. The individual regulates nicotine intake not because of a drop or decrease in his or her nicotine level, but because of the negative emotional state associated with the experience of craving. The second assumption is that several emotional processes may be operating simultaneously. The third assumption, that these affective states can be added algebraically at any given point, is also a main component of Solomon’s model.

The fourth assumption is that changes in plasma nicotine level generates
a variety of bodily sensations that can be conditioned to the emotional states experienced. The fifth assumption is that smoking can be used as a coping skill, as it enhances relaxation and helps to control negative affect. The sixth and final assumption postulates that there is an emotional memory. Thus, the smoking experience will be strongly associated with memories of smoking to cope with aversive events.

Although physicians, psychologists, physiologists, sociologists and many other investigators in the health field theorize about the acquisition and maintenance of smoking behavior, it remains unclear how pharmacological, social, psychological, and biological factors interact and contribute to its maintenance (Borgatta and Evans, 1968; Hunt, 1970; Dunn, 1973). Hunt and Matarazzo (1982) suggest that a major weakness in the smoking research to date is that many of the intervention studies have not based their method of intervention on any comprehensive model of smoking behavior. They further conclude that intervention procedures should include components that would combine behavioral, cognitive, physiological, and affective factors, rather than continuing with studies that are built on hunches.

Although many of the theories pertaining to why individuals start and continue to smoke have remained constant in the last few years, the general theories of drug addiction have developed significantly over the last fifty years. The evolution of theories related to drug addiction have progressed from a focus on the personality factors in the 1940's; the search for psychopathology in the
1950's and 1960's; to the reinforcement models of the 1970's and 1980's and the neuroadaptive models of the 1990's.

2.1.4 Smoking as an Addiction

Jaffe (1975) used the term addiction as a "behavioural pattern of compulsive drug use, characterized by overwhelming involvement with the use of a drug, the securing of its supply, and a high tendency to relapse after withdrawal". Jaffe differentiated between drug addiction and drug dependence. Bozarth (1990) defined drug addiction as an extreme example of compulsive drug use associated with strong motivational effects of the drug. The U.S. Surgeon General (1988) used the following criteria to evaluate tobacco as an addictive substance: 1) that a highly controlled or compulsive patterns of drug taking occur; 2) that a psychoactive or mood-altering drug is ingested by user of the substance and is involved in the resulting pattern of behavior and 3) that the drug is capable of functioning as a reinforcer that can directly strengthen behavior leading to further drug ingestion. Addicting drugs can be characterized by other properties that include the following: they can produce pleasurable effects in users, they can cause tolerance and physical dependence, and they can have adverse or toxic effects (p.149).

Gilman, Goodman, Rall and Murad (1985) suggest that two characteristics are required to demonstrate the physical component of addiction. The first is dependence, which is defined by the experience of withdrawal symptoms and
craving when drug use is terminated. The second, tolerance, is defined as the need for more amounts of the drug in order to obtain the same effect. According to Gilman et al. (1985), nicotine meets these two criteria for an addiction. Jarvik and Henningfield (1988) demonstrated that nicotine tolerance is lost and gained on a daily basis. That is, when the individual has not smoked while sleeping, their tolerance decreases and as a result, the first cigarette of the day has the strongest effect. However, it would appear that the physical dependence on nicotine is not the only variable involved in maintaining smoking.

Wise and Bozarth’s (1987) psychomotor stimulant theory of addiction attempts to isolate commonalities across a broad range of drug classes in order to present a unified theory of addiction. Their theory combines three major assertions: 1) that all addictive drugs have psychomotor stimulant actions, 2) that the stimulant actions of these different drugs have a shared biological mechanism, and 3) that the biological mechanism of these stimulant actions is homologous with the biological mechanism of positive reinforcement (1987). Although the psychomotor stimulant theory of addiction is based on the premises of operant reinforcement, it differs from the basic Skinnerian paradigm in that the reinforcing effects of a drug is directly related to its ability to produce psychomotor stimulation. The drugs which Wise and Bozarth define as psychomotor stimulants are amphetamines, cocaine, nicotine, caffeine, opiates, barbiturates, alcohol, benzodiazepines, cannabis, and phencyclidine. The primary mechanism of the psychomotor stimulant involves the catecholaminergic synapse of the central and
peripheral nervous system. Norepinephrine, dopamine and epinephrine are the primary catecholamine transmitters. Psychomotor stimulants increase the amount of dopamine concentrations at both the autoreceptors and at the postsynaptic receptors. It is the increase of dopamine at the postsynaptic receptors which accounts for the rewarding properties of psychomotor stimulants. Yoon, Gessa, Boi, Nates, Mereu, and Westfall (1986) suggest that nicotine acts on the same mechanism which is associated with the psychomotor stimulation effects of amphetamine. However, Wise and Bozarth (1987) caution that nicotine is likely a weak psychomotor stimulant. Nonetheless, they have found that there is strong evidence that for all psychomotor stimulants, nicotine included, that there is a common brain mechanism which mediates their reinforcing properties and their psychomotor stimulant properties. In essence, Wise and Bozarth (1987) propose that all addictive drugs have psychomotor stimulant properties and that the mechanism which mediates this process has the same properties as the mechanism which mediates the biological reinforcing properties of the drugs. That is, there exists a common mechanism which mediates both the psychomotor stimulant properties and the reinforcing properties.

Does understanding the multitude of theories of addiction and more specifically, smoking, lead to the successful creation of a smoking cessation program? As stated earlier, Hunt and Matarazzo (1982) suggest that a major difficulty with current smoking cessation research is that many methods of intervention are not based on comprehensive addiction models. As a result, the
modes of treatment or intervention often only look at one component (either behavioral, cognitive, physiological, and affect) which contribute to the maintenance of smoking behavior.

2.2 Methods of Smoking Intervention

2.2.1 Cognitive-Behavioral Approaches to Smoking Intervention

Historically, one of the more widely researched approaches to smoking cessation is that of behavioral intervention. The behavior therapies do not treat the smoker per se, they treat the smoking response. The initial behavioral techniques usually included methods which reinforced nonsmoking or included a stimulus control, aversion technique, or affect/tension reduction. Current behavioral techniques used are aversive smoking, self-management procedures and relapse prevention strategies (Hall, Hall, and Ginsberg, 1990).

The stimulus control method, which is based on operant conditioning principles, teaches the client to gradually eliminate locations where he or she smokes. The key is that smoking occurs in many settings, and as a result, there are many controlling environmental cues. When smoking becomes restricted to fewer and fewer environments, the cues which elicitate the smoking behavior in other locations, should diminish in power. Over a period of time, the smoking environment becomes smaller and smaller, until the individual abstains from smoking in all environments. A difficulty with the stimulus control program is that as the smoker reduces the number of cigarettes daily, the enjoyment for the remaining cigarettes increases (Shapiro, Tursky, Schwartz and Schnidman, 1971).
As a result, the method does not provide either significant initial or long-term effects in the reduction of smoking, compared to other methods (Shapiro et al., 1971). Glasgow and Bernstein (1981) report very high drop-out rates in the stimulus control intervention programs.

Leventhal and Cleary (1980) suggest that the sensitization and/or desensitization procedures are the most commonly used of the behavioral therapies. These methods attempt to eliminate the urge to smoke and to control the smoking response. In the sensitization treatments, noxious stimuli, such as electric shock, aversive taste stimuli and smoke are used to pair with actual or imagined smoking (Meichenbaum and Best, 1970). Pechacek (1979) concluded in his literature review that laboratory administration of electric shock was ineffective because humans were well able to differentiate between situations in which they would receive an electric shock and those in which they would not. As a result, the electric shock smoking cessation experiments, have failed to show long-term results.

Cognitive sensitization methods attempt to pair an unpleasant sensation (such as images of nausea) with smoking. This method, although promising in its early trials, again failed to produce long-term abstinence (Carlin and Armstrong, 1968). The results of aversive taste stimulus methods have not been able to produce any significant results in smoking cessation intervention studies.

The use of smoke itself has been utilized as a behavioral therapy method for smoking cessation. A rapid smoking technique which requires the individual
to take more puffs of the cigarette at a faster than normal pace (inhale every six seconds to the point of physical discomfort), has shown mixed results (Lando, 1975; Lando, 1976). There are some questions about the relative safety of this method of cessation, as it can produce potentially dangerous physiological side effects. Rapid smoking can lead to significant increases in heart rate, blood pressure, carboxy hemoglobin levels, and some cardiac abnormalities (Horan, Hackett, Nicholas, Linberg, Stone, and Lukoski, 1977). However, of the aversion procedures, rapid smoking appears to have generated the most research. The early studies (Harris and Lichtenstein, 1971; Schmal, Lichtenstein and Harris, 1978) showed almost 100% abstinence rates at the termination of treatment and almost 60% abstinence rates at the six month follow-up (Lichtenstein et. al, 1973, Schmal et. al., 1978). Danaher (1977) reviewed 22 rapid smoking studies and found that the abstinence rates varied from 0% - 81% at three month follow-up and 6.7% - 55% at six month follow-up. However, no further studies were able to replicate that high abstinence rates of Lichtenstein (Hall et al., 1990). Nonetheless, rapid smoking continues to be a popular behavioral technique for smoking cessation. Schwartz (1987) reviewed the post 1977 literature pertaining to rapid smoking and found that when used with other procedures, the results produced abstinence rates as high as 50% at the one year follow-up.

Self-management strategies have also been utilized as program strategies for smoking cessation. Schwartz (1987) suggested the following purposes for self-management: 1) the individual is to record his/her own smoking behavior, 2)
attempt to change the antecedent consequences of one’s smoking response, and
3) to develop awareness of and changing cue-elicited smoking patterns. The
primary objective is to educate the smoker to the stimuli which controls his or her
smoking behavior. As a result, the smoker starts to monitor all of their smoking
behaviors, such as the number of cigarettes smoked per day; the time of day they
smoke; the activity occurring before the smoking; their mood before smoking and
after smoking; where they are smoking etcetera (Moss, Prue, Lomax, and Martin,
1982). The goal of the self-management strategy of smoking cessation is the
gradual reduction of the number of cigarettes smoked daily, combined with
increased control of the stimuli associated with smoking.

Desensitization therapies are often combined with the self-monitoring
strategies (Paxton, 1980). They teach the individual relaxation so they may reduce
the stress and anxiety produced by internal or external cues which stimulate
smoking behavior. This method although theoretically attractive, has not produced
long-term abstinence rates (Pechacek, 1979; Pechacek and McAlister, 1980).
Other methods of behavioral therapy such as contracting and self-monitoring
procedures have also been used in smoking intervention studies. Used on their
own, they have not been successful in producing long-term results.

2.2.2 Hypnosis as Intervention for Smoking

Individual therapies such as hypnosis and drug treatments have also
emerged in the research of smoking cessation interventions. Hypnosis has been
used for many years in the treatment of smoking cessation. Although many
clinicians have claimed from a 0% to 94% success rate (Dedenroth, 1968), most of these claims have not been substantiated in controlled research (Pechacek, 1979). Barkley, Hastings and Jackson (1977) found that hypnosis did not differ significantly from attention-placebo control groups in mean smoking rates at any point during treatment or follow-up. Perry and Mullen (1975) suggest that a 15% to 20% success rate for hypnosis may be attributed to the expected percentage or proportion of individuals who are highly susceptible to hypnosis.

2.2.3 Nicotine Replacement Therapy for Smoking Intervention

Nicotine replacement therapy is one of the newer treatment methods for smoking cessation. It is based on the premise that nicotine is the primary factor associated with continued smoking (de Wit and Zacny, 1995). Nicotine replacement therapies include nicotine gum, nicotine nasal spray, and sustained-released transdermal nicotine-delivery system. In 1993, thirty-two million American smokers (70% of all adult smokers) reported that they wanted to quit smoking completely (Center for Disease Control, Tobacco Information and Prevention Source, 1993). It can therefore, be safely concluded that nicotine replacement therapy has great economic potential for drug companies. The most effective replacement therapy is the patch (sustained released transdermal nicotine-delivery system).

It has been estimated that in the United States alone, that sales for the patch reached $300 million (Fiore, Smith, Jerenby and Baker, 1994). This economic impact is significant considering that the patch has only been available
in the U.S. since December 1991. Two nicotine patches were among the top 100 prescriptions in the United States in 1992 (Haxby, Sinclair, Eiff, McQueen, and Toffler, 1994).

Fiore et al. (1994) reviewed the effectiveness of the nicotine patch for smoking cessation by completing a meta-analysis on all nicotine efficacy studies published in September 1993, identified through MEDLINE, Psychological Abstracts, and Food and Drug Administrations new drug applications. The studies were required to meet the criteria of being double-blind, placebo-controlled nicotine patch studies of four weeks or longer, with random assignment of subjects. Seventeen studies (n=5098) met the inclusion criteria. The 16 hour and the 24 hour patch studies had very similar outcomes; both performed significantly better than the placebo in terms of smoking cessation at the end of treatment and at six months post treatment. Patch users were abstinent 2.5 times that of placebo subjects at the end of the study and three times more abstinent at six months than that of the placebo subjects. The study also found that intensive counselling enhanced clinical success with the patch. The analysis also found no support for the continuation of the nicotine patch past eight weeks. This finding is of economic interest considering that the majority of marketers of the patch recommend that the patch be used for between 10 and 18 weeks. Considering the cost of each week of patch administration, prolonged use of the patch will have considerable financial impact on the pharmaceutical companies.

One reported difficulty with the patch is the rapidity with which nicotine is
absorbed into the system. The transdermal nicotine patch provides serum levels of nicotine which may take from five to ten hours to reach the maximum serum concentration level of 13 to 15 ng per mL. (McKenna and Cox, 1992). This translates to between one third and one half of the level obtained from smoking. Although the nicotine patch provides a steady rate of nicotine, the user can not titrate its dosage. The nicotine spray on the other hand, is absorbed rapidly (Sutherland, et. al., 1992) and its dosage can be controlled. This may be an advantage for those individuals who find it difficult to control their withdrawal symptoms and/or cravings with the patch or nicotine gum.

Sutherland et al. (1992) found that the one year abstinence rate with the nasal spray to be similar to that of the nicotine gum, 26%. The nasal spray appeared to be the most effective with the heaviest smokers. A difficulty with the nasal spray is that it may be perceived as less socially acceptable than the nicotine gum or the patch. Its use has decreased significantly since the introduction of the patch.

Nicotine gum was approved by the Food and Drug Administration as an aid for smoking cessation in 1984 (McGovern and Lando, 1992). Similar to the serum blood levels achieved by the patch, nicotine gum raises serum concentration levels to approximately half of what would be obtained via smoking. Numerous studies have found that nicotine gum used in conjunction with behavioral therapy is more successful for abstinence rates than using behavioral therapy alone (Hall, Tunstall, Ginsburg, Benowitz, and Jones, 1987; Tonnesen, Fryd, Hanses, Helsted,
Gunnersen, Forchhammer, and Stockner, 1988; Jarvis, 1988). Cepeda-Benito (1993) performed a meta-analysis review of the efficacy of the nicotine chewing gum and smoking cessation programs. The review entailed calculating effect sizes from studies conducted between 1973 and 1991; 33 studies were included in the meta-analysis. The analysis found that the groups who received nicotine gum performed better than those in the control groups. The nicotine gum treatment groups produced a higher quit rate than those in a placebo-controlled treatment, with both brief and more intensive treatment strategies.

It would appear that nicotine replacement therapy has great potential as a tool for smoking cessation. However, studies have found that nicotine replacement therapies need to be utilized in the proper manner in order to be optimally effective. With all three replacement therapies, the outcome rate is significantly improved when adjunct therapy is provided.

**2.2.4 Other pharmacological Therapies for Smoking Intervention**

There have been other pharmacological /over-the-counter treatment products offered for smoking cessation. Hall et. al. (1990) and Schwartz (1987) reviewed the literature pertaining to the three primary products in use (lobeline sulfate, silver acetate, and Water Pik filters). The lobeline sulfate (e.g. Nikoban, Bantron) was found to be no more effective than a placebo for smoking cessation. Similarly, there was little empirical support for silver acetate (which leaves a metallic taste in the mouth after one smokes). When investigating the effectiveness of the Water Pik filters, Hymowitz, Lasser and Safirstein (1982)
found that the rates for the individuals assigned to the quit smoking on own group were significantly higher than the rates for those individuals who were assigned to the Water Pik filter group and the placebo filter group.

2.2.5 Multi-component Techniques for Smoking Intervention

Multi-component interventions which incorporate techniques and procedures from more than one theoretical model are showing the most promise for smoking cessation. These procedures are usually quite comprehensive programs, which utilize the adjunct behavior techniques such as contracting and self-monitoring. Although many of the multi-component interventions have shown an improvement in long-term abstinence rates, treatment programs which utilize behavioral techniques alone (e.g. self-control methods), have not been as effective. Many of these intervention programs are combining social learning theories and behavior modification approaches jointly in the creation of smoking intervention models. However, the psychological-behavioral intervention strategies have had limited success in the facilitation of smoking cessation (Hunt and Bespelac, 1974; Pechacek, 1979). Epstein and Collins (1977) suggest that the difficulty in these programs may be in the lack of understanding of the stimulus variables which elicit the smoking behaviors in the natural environment.

Pomerleau, Adkins, and Peryschuk (1978) attempted to determine predictors of success in a multi-component behavioral treatment program for smoking cessation. Initially, their study showed that 61% of those participating in
the program were not smoking after the eight weekly treatment sessions. However, by the one year anniversary follow-up, only 32% had remained abstinent. Pomerleau et. al. suggest that the only significant predictors of recidivism is “negative affect”. That is, those smokers classified as negative affect smokers, showed a higher recidivism rate in the first 4-6 weeks after the eight weekly treatment sessions and continued to relapse at a higher rate compared to non-negative affect smokers. The Pomerleau study also suggested that years smoked, rate and percent overweight did not predict recidivism during the follow-up periods, although smoking rate and years smoked has predicted outcome in non-behavioral programs. Research is finding that smokers who report high levels of affective distress fail to quit smoking and also fail to maintain abstinence (Hall et. al, 1990; U.S. PHS, 1980, 1988). Hall, Bachman, Henderson, Barstow and Jones (1983) found that even in smokers who are critically ill, elevated mood disturbance predicted relapse at follow-up. Tompkins(1966) proposed a topology of smoking, which included affect. However the scales derived from that topology have not been successful in predicting treatment outcome (Flaxman, 1978, Hall et. al., 1983).

Self-help smoking cessation manuals, such as those produced by Dananher and Lichetenstien (1978) and Pomerleau and Pomerleau (1977) are based on multi-component programs. Glasgow, Schafer and O’Neill (1982) evaluated the two self-help therapy books for smoking cessation. They compared the books to a widely available minimal treatment program which was an “I quit"
kit published by the American Cancer Society. The subjects were randomly assigned to either a self-administered condition or a therapist-administered condition. All of the three treatment programs produced modestly significant changes in smoking behavior. All three groups were subject to approximately equal relapse rates.

Commercial stop smoking programs are no longer limited to the printed media. Televised intervention programs are also available to assist with smoking cessation. Warnecke, Langerberg, Gruer, Faly and Jason (1989) investigated 1,354 individuals who registered to receive smoking cessation assistance through a televised intervention program. Although televised self-help programs may be effective in reaching large segments of the smoking population, results of the Warnecke et al. (1989) investigation indicated that only 10% of participants from the study remained abstinent at 12 months.

Smoking cessation clinics are also increasing in popularity. Early studies reported short-term cessation rates as high as 76% and 31% for long-term outcomes (Leventhal and Cleary, 1980). However, many of the clinic run programs failed to provide data on the number of successes relative to the number of patients treated, nor did they incorporate the use of control groups in their research. West, Graham, Swanson, and Wilkinson (1977) in a five year follow-up of a smoking withdrawal clinic population, found that initial cessation rates were high, but so were the rates of relapse.
2.3 Stages of Change Model

Research has demonstrated that successful smoking cessation is often preceded by several failed attempts to quit (Pederson et al., 1996). Prochaska and DiClemente (1992) have proposed the Stages of Change Model, which they use to explain the process of quitting smoking. Prochaska and DiClemente suggest that there are five basic stages of change. Individuals in the first stage, precontemplation, are not currently thinking about quitting smoking. Once they enter the second stage, contemplation, they are actively thinking about quitting and may be acquiring information about cessation. In the third stage, preparation, the individual appears ready to quit and will likely proceed with an attempt in the near to immediate future. Action, the fourth stage, involves an actual cessation attempt. In the final stage, maintenance, the ex-smoker works toward abstinence.

It would appear, that in spite of the many varied types of smoking intervention methods available, the final stage of maintenance is difficult to sustain. The majority of smokers relapse (Hunt and Matarazzo, 1982). Siegel (1979) proposed that conditioned withdrawal reactions lead to relapse, while Cummings, Gordon and Marlatt (1980) suggested that relapses occur in the absence of conditioned withdrawal symptoms. Relapse prevention has now become an important component in smoking cessation treatment models. Initially, relapse prevention was dealt with via group meetings or support meetings after the treatment program was over (Tiffany, Martin, and Baker, 1986). However, their effectiveness as a prevention strategy is not significant (Brandon, Zelman, and
Baker, 1987). More complex models needed to be proposed to effectively incorporate relapse prevention in smoking cessation treatment.

2.4 Theories of relapse

2.4.1 The Marlatt and Gordon Model

Marlatt and Gordon (1980, 1985) likely offer the most influential theory of relapse (Brandon, Tiffany, Obremski and Baker, 1990). Their theory departs from traditional approaches of the analysis of relapse, in that it does not focus on pre-existing factors that could predict relapse. Instead, they are interested in determining the conditions which lead to relapse. The focus of the model is the determination of recidivism (Marlatt and Gordon, 1980).

Hunt, Barnett and Brand (1971) investigated relapse rates in addiction programs. In comparing the data curves of follow-up relapse information from heroin, alcohol, and smoking cessation studies, they suggest that the curves are very similar. All three curves show a marked decline during the first three months, followed by a gradual leveling off. Approximately two-thirds of all of the relapses, in all three of the different addiction type programs, occurred within the first 90 days. Marlatt and Gordon (1980) propose that since the relapse rate is so similar between the addictions, that there is likely a common behavioral and cognitive component associated with any relapse.

Relapse, according to the Marlatt and Gordon model, is a process rather than a discrete, irreversible event (Brandon, Tiffany, Obremski, and Baker, 1990).
Relapse is defined as “any discrete violation of an imposed rule or set of rules governing the rate or pattern of consumption behaviours” (Marlatt and Gordon 1980, p.413). The primary goal of the model is to attempt to establish the relationship between the initial relapse episode and subsequent use of the substance. The model does not focus on internal or physiological factors which may be associated with the addictive properties of the substance in question.

Marlatt and Gordon’s (1980) attempt to determine the relapse circumstances of chronic alcoholics, who had successfully completed an aversion condition treatment, revealed that over 50% of all relapse situations fell into one of two categories. The analysis of the data suggested that the individual relapsed when: a) situations in which the patient was frustrated or angered, usually in an interpersonal or social situation; and b) situations in which the patient was confronted by social pressures to resume drinking, usually from a drinking partner or family member.

Further research revealed that relapse episodes are first categorized in one of two major classes: 1) intrapersonal/environmental determinants; and 2) interpersonal determinants. In their study, Marlatt and Gordon found that 76% of all relapses fell into one of three categories: coping with negative emotional states (37% of relapses), social pressure (24%), and coping with interpersonal conflict (15%). The data specific to smokers showed that 43% relapsed due to negative emotional states, followed by 25% due to social pressure. No smokers indicated that they relapsed as a result of negative physical states. This would then cast
some doubt on Wikler's (1973) proposed theory of physiological basis of relapse.

The Marlatt and Gordon (1980) relapse model applies to individuals who choose to abstain from a substance, following a voluntary termination of the use of the substance. A major assumption of the model is that the individual experiences a sense of personal control over the target behavior while remaining abstinent. This perception will continue, until the individual encounters a high-risk situation. A high-risk situation is any situation which is a potential threat to the individual's perceived sense of control. A threat to the individual's feeling of control may subsequently lead him or her to relapse. Marlatt and Gordon constructed a list of high-risk situations for the occurrence of relapse (Appendix A).

A major determinant to the relapse process, is an individual's ability to engage in an adequate coping response while confronted with a high-risk situation. An effective coping response significantly reduces the potency of the high-risk situation, thereby reducing the probability of relapse. If the individual is successful in coping with the high-risk situation, the authors propose that an expectation that they will be able to deal successfully with the next situation is formulated. The formulation of expectancy is based on Bandura's (1977) theory of self-efficacy.

If a successful coping mechanism is not utilized, or attempted by the individual when presented with a high-risk situation, Marlatt and Gordon suggest that there is a decrease in the individual's perceived self-efficacy. This decrease in self-efficacy may increase the likelihood of relapse as the individual perceives himself or herself as having no control over the situation. Similarly, if the
Figure 2-1  A Cognitive-Behavioral Model of the Relapse Process

individual's usual method of coping is to use the substance he or she is attempting to quit/avoid, then he or she is at higher risk to relapse (Figure 2-1). Marlatt and Gordon also suggest that if the individual believes that the effects of a substance will enhance his or her feelings of power or control, the individual will be more likely to relapse if the high-risk situation has decreased his or her self-efficacy. If no coping mechanism is utilized at this moment, the individual will relapse.

The individual's cognitive/affective reaction to a relapse determines subsequent substance use. The "Abstinence Violation Effect" (AVE), is a label created by Marlatt (1978) to describe a possible cognitive/affective reaction to a lapse. The AVE has two principal components: 1) a cognitive dissonance component, in which the relapse behavior is in direct conflict with the individual's new self-image; and 2) a personal attribution component, in which the individual attributes the cause of the lapse to internal failure or personal failure. Marlatt and Gordon propose that the cognitive dissonance and attribution components somewhat overlap, and that the additive effects of both reactions will greatly increase the probability of the individual repeating the behavior, whether it be smoking, drinking and/or gambling (1985).

2.5 Implications for the Prevention and Treatment Smoking

Shiffman (1982) found that post cessation coping responses correlated with successful abstinence. However, Wewers (1988) reported that problem-focused coping responses were used significantly more often than emotion-focused coping responses in abstinent subjects. Marlatt and Gordon suggest that there are ideal
Figure 2-2  Relapse Prevention: Specific Intervention Strategies

Self-monitoring + Behaviour Assessment (e.g. situational competency test)

Relaxation Training, Stress Management, + Efficacy Enhancing Imagery

Contract to Limit Extent of Use + Reminder Card (What to do when you have a slip)

High-risk Situation

No Coping Response(329,174),(505,507)

Decreased Self-efficacy

No Positive Outcome Expectations

Initial Use of substance

Abstinence Violation Effect

Relapse Fantasies + Descriptions of Past Relapses

Skill Training + Relapse Rehearsal

Education About Immediate vs. Delayed Effects Of Substance; Use of Decision Matrix

Programmed Relapse

Cognitive Restructuring (Slip = Mistake. Attribution to Situation vs. Self

Marlatt and Gordon (1980) p. 437
points of coping skills intervention for prevention of relapse in their model (Figure 2-2).

The implementation of effective coping skills is crucial in maintaining abstinent behaviors (Marlatt and Gordon, 1980). The social interactional (SI) approach to counselling (Christensen and Pass, 1983) offers a problem-solving or coping-skills training program as an alternative to traditional psychotherapy. The SI approach focuses on the client's interactions and transactions with his or her environment. The SI treatment, in effect, teaches the client to change his or her action or reaction to the specific aspects of the social environment which is troublesome.

The SI framework can be utilized effectively as the main component of a smoking cessation program, as it teaches the individual how to identify inappropriate and ineffectual response behaviors to various stimuli in the environment. Once identified, the individual is then taught how to modify his or her behavior. The primary method of modifying behavior is through reappraisal of the social stimuli which are related to the inappropriate behaviors. The SI identification and reappraisal skills will increase the individual's probability of success when placed in a high-risk situation.

2.6 The Social Interactional Approach to Counselling/Psychotherapy

Christensen and Pass' (1983) Social Interactional (SI) model takes into account both situational and person or cognitive variables. Inherent to the approach is the basic assumption that "psychopathology is conceived of as
ineffective or inappropriate ways of interacting with the social environment". "Ineffective' or 'inappropriate' is essentially a judgment, either by the individual that the results are not satisfying, or by others that the coping behaviour and results are not acceptable" (Christensen and Pass, 1983, p. 52). The teaching of coping/social skills will allow the individual to interact more effectively in the social environment.

Bandura's proposal that some learning can be either obtained observationally or vicariously, contributes significantly to the SI model which suggests that a response pattern can be acquired through the observation of another individual's performance. Similarly, an emotional response, such as fear, anger, or joy, can be conditioned observationally. However, just as an individual learns to model appropriate responses through observation, he or she may acquire inappropriate responses to the environment. The appropriateness of an individual's response pattern directly affects how well he or she will interact socially. Christensen and Pass (1983) propose that four processes - observing, inferring, appraising, and speaking, determine how a person copes within the environment. Key to the individual's effective coping is how he or she appraises the stimulus or situation.

Christensen and Pass incorporate the theories of Arnold (1970) and Lazarus (1968) to explain the role of appraisal within the SI model. Arnold and Lazarus postulate that the experience of an emotion is a byproduct of an appraisal
process. That is, emotions result from the mediation of the individual's cognitive system. Lazarus (1968) suggests that there are two kinds of appraisal processes, primary and secondary. The primary appraisal could be referred to as an orienting response. When responding to a stimulus, the individual must determine if the stimulus is relevant or irrelevant. If the stimulus is deemed irrelevant, the individual either does not attend to it any longer, or habituation takes place. If the stimulus is believed to be relevant, a secondary appraisal occurs. The resultant of the secondary appraisal is an action or an action tendency. If the individual cannot respond overtly, an internal response, or an action tendency results. This action tendency, according to Arnold (1960), is experienced as an emotion. Therefore, it is not the behavior which causes the emotion, but the individual's appraisal of the stimulus or event (which results in an action or an action tendency), which is experienced as an emotion. Christensen (1974) proposed that by changing an individual's appraisal of a situation or stimulus, you may effectively change the corresponding emotion, as the reappraisal will produce a different action tendency. The implementation of effective reappraisal skills would lead to more appropriate social interaction when presented with previously troublesome stimuli.

Christensen (1974) theorizes that in order to function effectively, an individual must be able to recognize social stimuli and learn about their effect on his or her behavior. Social stimuli refer "to the appearance, speech, and actions of others; that is, a social stimulus is what a person looks like, says, and does"
One individual is capable of supplying a plethora of social stimuli. Two children may respond to a parent in identical manner and still present different stimuli. Social stimuli are specific and concrete.

Christensen’s SI theory proposes that there are seven social stimuli, generated by four significant others, which are likely to be of concern for most individuals. The social stimuli are expressions of anger, criticism, depression, unresponsiveness, impulsivity, affection, and commands. The four significant others usually include a teacher or supervisor, spouse or close friend, mother and father. Not all individuals have difficulty with these specific social stimuli, and others have difficulty with social stimuli which have not been mentioned. However, clinical experience suggests that these specified social stimuli are troublesome to many individuals.

Troublesome social stimuli are defined as those stimuli which lead individuals to: a) avoid the stimulus, b) attempt to change the stimulus, c) freeze in the presence of the stimulus. Any of the three responses can restrict a person’s behaviour significantly. Not all troublesome social stimuli are external, that is, troublesome stimuli may also be internal. Christensen (1974) indicates that there are two types of internal stimuli. The first category of internal stimuli is usually referred to as bodily sensations. That is, those physical sensations derived from varied muscle activity. When describing this type of internal stimulus, an individual may report that they have butterflies in their stomach, or that their stomach is in a knot, or that their heart rate is racing, and so on. Urges or action tendencies
make up the second group of internal stimuli. They are specific muscle activities which are experienced as feelings by the individual to perform a specific action in response to some external stimuli. Arnold (1960) suggested that these urges are the basis for emotional experiences. Just as with external stimuli, internal stimuli have the ability to cause the individual to attempt to avoid or change them, or the individual may be controlled by them.

Christensen and Pass (1983) postulate that there are seven interpersonal coping skills which are essential in effective social interaction. The authors propose further that psychopathology is the manifestation of a lack or inability to implement these seven skills: 1) the ability to discriminate and attend to social stimuli; 2) the ability to distinguish among observation, inference, and appraisal; 3) the ability to accurately observe self-other interactions; 4) the ability to infer accurately; 5) the ability to appraise appropriately; 6) the ability to vary behaviour and observe the consequence; and 7) the ability to use language in a concrete and specific way.

It is the implementation of the seven coping skills which is integral to the success of the SI approach to smoking cessation and relapse prevention.

2.7 Focus of the Present Study

The purpose of the present study was to investigate the effectiveness of social interactional coping skills in a smoking cessation and relapse prevention program and compare it to the success rate of a national smoking cessation program (The Lung Association Countdown Program, Appendix C) and a control
The Lung Association Countdown Program was used as a comparative smoking cessation program as it was recognized as one most effective publically available treatment program in Canada (Health Canada, 1995). The program is widely available throughout Canada and it is available in both French and English. It is targeted to the general population with an emphasis on behaviour modification.

The use of the SI model as a treatment mechanism for smoking cessation is partially based on the premise that Leventhal and Cleary's (1980) multiple-regulation model for the maintenance of smoking behaviour is a good, however incomplete explanation for an addiction model. However, the additional incorporation of Wise and Bozarth's (1987) psychomotor stimulant theory is essential in terms of explaining the neuro-biological components of nicotine addiction, thereby providing a more comprehensive addiction model. Similarly, to provide holistic treatment, it is important that the SI treatment program also utilizes the teaching of the social interaction skills as components of relapse prevention, as proposed by Marlatt and Gordon (1980, 1985).

Christensen's interpersonal coping skills program has been found effective in a variety of therapeutic settings. Sanchez-Craig (1976), while working with elementary school children demonstrated a decrease in the level of discomfort experienced by the children while in the presence of previously aversive stimuli. Briedis (1976) used the interpersonal coping skills approach to counsel high
school

students. Pre and post self-report measures of social confidence, indicated that the students perceived themselves as more confident after participating in the coping skills program. These self-report measures were validated by independent teacher ratings of effectiveness. Cole (1979) compared the interpersonal coping skills program to a stress-management treatment and a non-directive patient-centered treatment of adult psychiatric patients. Using a variety of therapy effectiveness measures, Cole's study indicated that the interpersonal coping skills program was therapeutically more effective than either the stress-management treatment or the non-directive patient-centered programs.

2.7.1 Research Questions and Hypothesis

The main hypothesis of the present study was that successful implementation of the seven social interaction skills from the Christensen and Pass SI model, along with various cognitive-behavioural tactics, would significantly increase the success of smoking cessation and significantly reduce the relapse rate of smokers (see Appendix B for program outline). The clients were taught appropriate and effective coping skills which would be utilized when they were presented with a high risk situation. That is, clients were taught effective mechanisms of reappraisal to deal with troublesome internal and external stimuli. Similarly, the clients were taught effective and appropriate social interaction skills to assist them when dealing with significant individuals in their social environment.

The second hypothesis proposed that those individuals assigned to the SI
treatment group who relapsed, would relapse later post cessation when compared to those individuals who relapsed in the Lung Association treatment group. Marlatt and Gordon's (1980) and Hunt, Barnett and Brand's (1971) cross addiction research indicated that most relapses occur within the first 90 days post cessation. This study predicted that fewer individuals in the SI group compared to the Lung Association group would relapse within the first three follow up assessments. This would be due to the successful incorporation of the positive coping skills and relapse prevention skills taught as part of their treatment in the SI group.

The third hypothesis predicted that those individuals who did relapse in the SI treatment group, would smoke fewer cigarettes post relapse than those individuals who relapsed in the Lung Association group. That is, they would be less likely to smoke cigarettes as a means to cope with troublesome internal and external stimuli.

The fourth hypothesis predicted that those individuals who demonstrated high self-efficacy, as measured by a lower score on the Smoking Confidence Questionnaire, would be more likely to quit smoking. Similarly, the fourth hypothesis predicted that those individuals who quit would be significantly different from those individuals who did not quit smoking on The Profile of Mood States scale and The Anger Expression Scale.
Chapter 3

METHODOLOGY

3.1 Procedure

A total of 75 subjects, 51 females and 24 males, who fulfilled the inclusion criteria, responded to an advertisement for a research-treatment smoking cessation program in two community newspapers. A smoker was defined as anyone who currently smoked more than five cigarettes per day. In addition to the currently smoking criterion, the following conditions had to be met in order for respondents to be selected:

1) no concurrent participation in other smoking cessation programs.
2) willingness to be randomly assigned to one of three conditions.
3) willing to attend weekly treatment sessions, and
4) willingness to make a nonrefundable payment for treatment if assigned to one of the treatment conditions.

The last criterion was implemented to guard against potential attrition and to partially fund one of the treatment conditions. Those subjects assigned to the treatment groups were asked to make a payment of $45.00 at the first treatment session. All subjects signed a consent form (Appendix D).

The 75 subjects were evenly and randomly assigned to one of three conditions; a Social Interaction Coping Skills Program (SI), the Lung Association Countdown Program (LA), or a No-Treatment Control Condition. Each group
contained 25 subjects after the random assignment. All members of the No Treatment Control Group were offered post-study treatment in the Lung Association Countdown Program.

3.2 Research Participants

The subjects ranged in age from 21 to 71 years, with a mean age of 40.45 years and a standard deviation of 10.57 years. The mean number of prior attempts at quitting smoking was 3.62 times, with a standard deviation of 3.06 times; while the mean number of cigarettes smoked per day was 25.46, with a standard deviation of 12.08. On average, the participants started smoking at age 16.36 years (standard deviation of 3.10 years) and had been smoking for 23.73 years (standard deviation of 10.84 years).

Pretreatment comparisons were performed for age, years smoked, and number of cigarettes smoked per day, to examine whether the groups differed prior to the administration of treatment. The means and standard deviations for each group are summarized in Table 3-1. One way analyses of variance, performed on each variable failed to reveal any significant differences between groups on any of the pretreatment variables (see Table 3-1).

Table 3-2 contains the demographic information for those individuals who dropped out of the study. A dropout is defined as a subject who ended his or her
Table 3-1

Demographic and Smoking Comparisons of Initial Sample (n=59)

<table>
<thead>
<tr>
<th>Variable</th>
<th>SI Group (n=21)</th>
<th>LA Group (n=18)</th>
<th>Control Group (n=20)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>M=41.36, SD=11.19</td>
<td>M=40.80, SD=11.91</td>
<td>M=39.20, SD=8.66</td>
<td>F(2,72)=.276 n.s.</td>
</tr>
<tr>
<td>Age Started</td>
<td>M=16.48, SD=2.54</td>
<td>M=16.04, SD=3.41</td>
<td>M=16.56, SD=3.40</td>
<td>F(2,72)=.198 n.s.</td>
</tr>
<tr>
<td>Years Smoked</td>
<td>M=24.60, SD=11.67</td>
<td>M=24.28, SD=12.17</td>
<td>M=22.32, SD=8.69</td>
<td>F(2,72)=.318 n.s.</td>
</tr>
<tr>
<td>Cigs per day</td>
<td>M=26.84, SD=10.05</td>
<td>M=27.93, SD=16.05</td>
<td>M=21.64, SD=8.25</td>
<td>F(2,72)=1.98 n.s.</td>
</tr>
<tr>
<td>Times Quit</td>
<td>M=3.36, SD=1.93</td>
<td>M=3.64, SD=4.20</td>
<td>M=3.88, SD=2.71</td>
<td>F(2,72)=.177 n.s.</td>
</tr>
<tr>
<td>Max. length of quit in days</td>
<td>M=167.16, SD=249.39</td>
<td>M=145.64, SD=237.38</td>
<td>M=200.28, SD=295.02</td>
<td>F(2,72)=.759 n.s.</td>
</tr>
<tr>
<td>%Female</td>
<td>64</td>
<td>64</td>
<td>72</td>
<td>χ(2)=1.10 n.s.</td>
</tr>
<tr>
<td>% Married</td>
<td>64</td>
<td>44</td>
<td>60</td>
<td>χ(10)=9.04 n.s.</td>
</tr>
<tr>
<td>% College or University</td>
<td>68</td>
<td>64</td>
<td>68</td>
<td>χ(8)=7.43 n.s.</td>
</tr>
</tbody>
</table>
Table 3-2

Demographic and Smoking Comparisons of Drop Outs (n=16)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SI Group (n=4)</td>
<td>LA Group (n=7)</td>
</tr>
<tr>
<td>Age Started</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>14.25</td>
<td>15.28</td>
</tr>
<tr>
<td>SD</td>
<td>(3.59)</td>
<td>(3.09)</td>
</tr>
<tr>
<td>Years Smoked</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>19.75</td>
<td>25.00</td>
</tr>
<tr>
<td>SD</td>
<td>(5.73)</td>
<td>(9.46)</td>
</tr>
<tr>
<td>Cigs per day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>33.75</td>
<td>31.57</td>
</tr>
<tr>
<td>SD</td>
<td>(13.76)</td>
<td>(12.06)</td>
</tr>
<tr>
<td>Times Quit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>3.75</td>
<td>4.85</td>
</tr>
<tr>
<td>SD</td>
<td>(2.21)</td>
<td>(6.74)</td>
</tr>
<tr>
<td>Max length of Quit in Days Prior to study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>30.25</td>
<td>185.28</td>
</tr>
<tr>
<td>SD</td>
<td>(43.55)</td>
<td>(361.51)</td>
</tr>
<tr>
<td>% Female</td>
<td>75</td>
<td>86</td>
</tr>
<tr>
<td>% College or University</td>
<td>75</td>
<td>43</td>
</tr>
<tr>
<td>Sample size</td>
<td>4</td>
<td>7</td>
</tr>
</tbody>
</table>
participation in the study prior to sixty days from the commencement of the study. A total of 16 individuals dropped out of the study prior to the 60 day inclusion criteria, leaving 59 subjects in the study. The rate of drop out was evenly distributed across the groups ($\chi^2(2, n = 16) = 1.11$, n.s.). The groups did not differ with respect to the demographic variables and smoking history (see Table 3-2). The remaining 59 subjects will now be referred to as the main sample.

Several demographic comparisons were made to evaluate the equivalence of the three groups which comprise the main sample. Table 3-3 contains the means and the standard deviations of the three groups on demographic and smoking history variables. Those in the main sample remained equivalent on these variables.

The means and the standard deviations of the other pretreatment variables are summarized in Table 3-4. The SI group and the no treatment control group differed significantly with respect to the Smoking Confidence Pre Score ($f(2,56) = 4.15$, $p < .05$), with the no treatment control group, indicating that they had less confidence that they could resist the urge to smoke a cigarette. However, the two treatment groups did not differ statistically on any of the demographic, smoking history, or personality measures.

3.3 Measures

3.3.1 Outcome Measures

Smoking cessation, measured by percent quit in each group, represented the primary goal of treatment. However, overall number of cigarettes smoked per
Table 3-3

Demographic and Smoking Comparisons of Main Sample (n=59)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Significance</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SI (n=21)</td>
<td>LA (n=18)</td>
<td>Control (n=20)</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>42.76</td>
<td>40.66</td>
<td>40.35</td>
</tr>
<tr>
<td>SD</td>
<td>(11.61)</td>
<td>(12.76)</td>
<td>(8.83)</td>
</tr>
<tr>
<td>Age Started</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>16.90</td>
<td>16.33</td>
<td>16.55</td>
</tr>
<tr>
<td>SD</td>
<td>(2.14)</td>
<td>(3.56)</td>
<td>(3.21)</td>
</tr>
<tr>
<td>Years Smoked</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>25.52</td>
<td>24.00</td>
<td>23.55</td>
</tr>
<tr>
<td>SD</td>
<td>(12.35)</td>
<td>(13.31)</td>
<td>(9.10)</td>
</tr>
<tr>
<td>Cigs per day</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>25.52</td>
<td>26.50</td>
<td>22.60</td>
</tr>
<tr>
<td>SD</td>
<td>(9.02)</td>
<td>(17.45)</td>
<td>(8.76)</td>
</tr>
<tr>
<td>Times Quit</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>3.28</td>
<td>3.16</td>
<td>3.95</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Female</td>
<td>61.9</td>
<td>55.5</td>
<td>75.0</td>
</tr>
<tr>
<td>% Married</td>
<td>66.6</td>
<td>38.8</td>
<td>50.0</td>
</tr>
<tr>
<td>% College or University</td>
<td>66.6</td>
<td>72.2</td>
<td>65.0</td>
</tr>
</tbody>
</table>
Table 3-4
Comparisons Among The Groups On the Pretreatment Variables (n=59)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Groups</th>
<th>One-Way ANOVA F(2,56)</th>
<th>Newman Keuls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SI (n=21)</td>
<td>LA (n=18)</td>
<td>Control (n=20)</td>
</tr>
</tbody>
</table>

Smoking Confidence
Pre Score

<table>
<thead>
<tr>
<th>M</th>
<th>SD</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>184.09</td>
<td>(60.33)</td>
<td>212.05</td>
<td>(69.71)</td>
<td>235.65</td>
</tr>
</tbody>
</table>

POMS Pre Score

<table>
<thead>
<tr>
<th>M</th>
<th>SD</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>30.71</td>
<td>(26.54)</td>
<td>30.72</td>
<td>(32.27)</td>
<td>32.25</td>
</tr>
</tbody>
</table>

Anger Expression Scores

<table>
<thead>
<tr>
<th>M</th>
<th>SD</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>14.76</td>
<td>(3.74)</td>
<td>14.88</td>
<td>(3.80)</td>
<td>15.15</td>
</tr>
<tr>
<td>16.52</td>
<td>(4.49)</td>
<td>19.11</td>
<td>(6.60)</td>
<td>16.25</td>
</tr>
<tr>
<td>22.71</td>
<td>(5.06)</td>
<td>24.33</td>
<td>(13.27)</td>
<td>23.55</td>
</tr>
<tr>
<td>24.47</td>
<td>(8.30)</td>
<td>25.66</td>
<td>(10.96)</td>
<td>22.40</td>
</tr>
</tbody>
</table>

* p<.05
** p<.01
a=SI Group significantly different from Control Group;
b=SI Group significantly different from LA Group;
c=LA Group significantly different from Control Group
day was also of interest, as the third hypothesis predicted that those individuals who relapsed in the SI group would smoke fewer cigarettes than those individuals who relapsed in the LA group. Similarly, each participant was required to record the date and reason for relapse.

The number of cigarettes each participant smoked prior to the commencement of the study was recorded. During the first 30 days post cessation, each participant was required to record daily how many cigarettes they smoked. Similarly, they were required to record the number of cigarettes smoked daily at two, three, four, five, six and 12 months post cessation. If a participant in the study relapsed during the one year assessment period, reason for relapse and date of relapse was to be recorded. Several other variables both directly related to smoking behaviour and personality were measured.

3.3.2 Dependent Measures

The Smoking Confidence Questionnaire (Appendix F) was created by Conditte and Lichtenstein in 1981. It was designed to assess the magnitude, strength, and generality of expectations in smoking situations. The questionnaire offers 43 situations in which the individual is to assess the probability that he or she would be able to resist the urge to smoke in that particular situation. The individual rates the probability of smoking using a 10 point Likert scale. A score of 1 indicates that they would be very unlikely to smoke, whereas a score of 10 indicates that they would very likely smoke. The lower the individual's score, the higher the self-efficacy. In their 1981 study, Conditte and Lichtenstein found a
.59 correlation. The validity of the scale was also demonstrated by Brown, Lichtenstein, McIntyre, and Harrington-Koster (1984).

Self-efficacy ratings have successfully predicted smoking status for up to six months post cessation (Baer, Holt and Lichtenstein, 1986; Carey, Snel, Carey and Richards, 1989; Colletti, Supnick, and Payne, 1985; Condiotte and Lichtenstein, 1981; DiClemente, 1981; Garcia, Schmitz and Doerfler, 1990; McIntyre, Lichtenstein and Mermelstein, 1983; Mudde, Kok, and Strecher, 1995). These studies demonstrated that the self-efficacy of successful quitters was significantly higher than the self-efficacy of those who were not successful quitters. Similarly, self-efficacy expectations at post-treatment were effective predictors for short-term maintenance of smoking cessation for up to six month after treatment (Pederson, Strickland and DesLauriers, 1991; Haaga, 1989; Haaga and Stewart, 1992.

Becona, Frojan and Lista (1988) compared the self-efficacy scales of DiClemente and Condiotte and Lichtenstein. They found that the Condiotte and Lichtenstein Confidence Questionnaire clearly predicted the smoking status at one, two, three, four, five, six and 12 month follow-up assessments, whereas the DiClemente Self-efficacy Questionnaire did not. Similarly, Borrelli and
Mermelstein (1994) demonstrated that the level of self-efficacy during treatment was the only significant variable to predict follow-up abstinence in smoking cessation programs.

Self-efficacy scores as measured by the Condotte and Lichtenstein Self-efficacy Questionnaire were obtained for all participants in the study at the onset of the study, once group assignment was completed. Self-efficacy was also obtained from all participants once a week for five weeks during the initial 30 days post cessation.

The Profile of Mood States (POMS, Appendix G) measures an individual's level of emotional functioning. The POMS contains 65 adjectives and measures the moods of tension, anxiety, depression-dejection, anger-hostility, vigor-activity, fatigue-inertia, and confusion-bewilderment. Participants rate each adjective on a five point scale from not at all (score of 0) to extremely (score of 4). A total score is obtained from the POMS by summing the scores of each of the six mood categories with the category of vigor-activity weighted negatively. The range of scores on the POMS are from 32 (no emotional distress) to 200 (indicating high emotional distress). The POMS has shown moderate to high correlations with other mood measures (McNair and Lorr, 1964; McNair, Lorr and Droppelman, 1981). As well, McNair and Lorr (1964) demonstrated that the POMS had an internal reliability of .90 and all six of the mood scales. A validity study by Lorr, Daston, and Smith (1967) found strong evidence for factorial validity. Similarly, Gibson (1997) also found strong support for concurrent validity, internal
consistency and test retest reliability. The POMS has become one of the most frequently used tools to assess mood in smoking cessation studies (Gilbert, 1997; Gilbert, McClenon, Rabinovich, Plath, Jensen, and Meliska, 1998; Levin, Conners, Sparrow, Hinton, Erhardt, Meck, Rose and March, 1995; Sharpe and Gilbert, 1998).

Gilbert (1997) used the POMS to measure mood repeatedly every 48 hours during a 30 day smoking abstinence phase. Gilbert found repeated-measures effects to be no greater than those found in the Beck Depression Inventory, The Zung depression scales and the Multiple Affect Adjective Check List anxiety scores.

The Anger Expression Scale (AX, Appendix H), created by Charles Spielberger (1986) has 24 questions. The scale was constructed to assess how people generally react or behave when they feel angry or furious. The AX Scale is comprised of 24 items and yields four different scores. The Anger Expression (AX/EX) score, which is based on all 24 items, provides a general index of how often anger is aroused and expressed or suppressed. The three AX subscales assess individual differences in the tendency to: 1) express anger toward other people or objects in the environment; 2) experience but hold in (suppress) angry feelings (AX/in); and 3) control the experience and expression of anger (AX/Con). The individual reads the statement and circles the number which corresponds to how often he or she generally reacts or behaves in the manner described. The scale used is a four point Likert scale, with 1 representing almost never, 2 -
sometimes, 3 - often, and 4 - almost always. The AX/In, AX/Out, and the AX/Con subscale scores are computed by summing the items which correspond to each scale. The range of possible scores for the three subscales varies from a minimum of eight to a maximum of 32. The scale has been demonstrated to be a valid and reliable tool for a measure of anger expression (Johnson, 1984; Jamieson and Roger, 1986; Spielberger, 1996; Spielberger, Johnson, Russell, Crane, Jacobs, and Warden; 1985; Spielberger, Krasner, and Solomon, 1988).

Anger has consistently been reported in the literature as predictor of relapse in smoking cessation studies (Hatsukami, Hughes, Pickens, and Svikis, 1984; Hughes, Hatsukami, Pickens and Svikis, 1984; Hughes, Gust, Skoog, Keenan and Fenwick, 1991). The Anger-Expression Scale was utilized to measure a participants experience and expression of anger in order to attempt to establish the existence of a relationship between anger and relapse.

An item symptom checklist (Appendix I) containing 10 of the most frequently reported withdrawal symptoms (Hatsukami et al., 1984; Brandon et al., 1990; Cummings et al., 1980) was created. All participants rated each symptom (weight gain, irritability, anxiety, sleeplessness, poor concentration, restlessness, anger, nausea, depression, urge to have a cigarette) on a six point Likert scale (ranging from not present - 0, to very strong 5) for the 30 consecutive days post designated quit day. These post smoking cessation symptoms have been demonstrated to be reliable and consistent across most smoking literature (Hughes, 1992; Hughes, Higgins and Hatsukami, 1990; Hughes, Hatsukami, Pickens and Svikis, 1984;

Hughes et al. (1991) assessed 315 smokers who wished to quit for symptoms of nicotine withdrawal prior to cessation and at follow-up at one week, two weeks, one month and six months. They found that anger, impatience and restlessness were the most prominent symptoms of tobacco withdrawal. Those individuals who reported more intense withdrawal symptoms were more likely to relapse. Whereas Hughes (1992) assessed 830 smokers and found that post cessation self-reported depression predicted relapse. However, the withdrawal symptoms of anxiety, difficulty concentrating, weight gain, irritability, restlessness and hunger were also consistently reported during the post cessation follow-up.

The primary focus of the self-reported symptom checklist was to determine whether there was a relationship between severity of symptoms and relapse. The ten most frequently cited symptoms from the literature on nicotine withdrawal were presented to the participants to rate in a self-reporting format.

Those individuals who were assigned to the SI group were asked to record on a daily basis the strength of their urge to have a cigarette for the 30 consecutive days post designated quit day. Since a component of the SI program was to teach effective coping skills to deal with internal stimuli, it was proposed that there would be no relationship between strength of urge and relapse.

Table 3-5 details when each assessment tool was administered to the participants in the study. The demographic information was obtained prior to the onset of treatment in order to ensure equivalency across groups. Similarly, the
Anger Expression Scale and Smoking Confidence Questionnaire were administered prior to the onset of treatment for the investigation of predictive validity for these tools. All participants were required to complete the POMS and Post Cessation Symptom Checklist daily for 30 consecutive days post designated quit day. The purpose of completing these measures of mood and withdrawal symptoms was to investigate whether a relationship between scores on these measures and relapse existed. All participants in the SI group were also required to complete a daily urge to smoke a cigarette rating for 30 consecutive days post designated quit day. It was assumed that those individuals in the SI group would not relapse despite high urge ratings, due to the specific training received in their treatment program.

The number of cigarettes smoked and reason for relapse were recorded for all participants at the two month, three month, four month, five month and six month post quit assessments. The frequent recording of this information allowed for direct comparison to other smoking cessation research and to investigate whether there was a difference between the two treatment groups with regard to length of abstinence. Brigham, Henningfield and Stitzer (1991) in their review of smoking relapse studies found that more than one-third of all quit attempts fail within a month of cessation. Hunt and Bespalec (1974) found that 80% of smokers who succeed in quitting will relapse within a year of quitting. Therefore, the monthly reports of relapse, number of cigarettes smoked and reason for relapse were important for comparison to relapse literature. Similarly, those
Table 3-5
Administration of Assessment Tools

<table>
<thead>
<tr>
<th>Time Administered</th>
<th>Demographic information</th>
<th>POMS</th>
<th>Anger Expression Scale</th>
<th>Self-report Symptom Checklist</th>
<th>Smoking Confidence Questionnaire</th>
<th>Urge to smoke</th>
<th>Number of cigarettes smoked and date</th>
<th>reason for relapse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>daily for the first 30 days</td>
<td></td>
<td>✓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>post cessation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>all participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>daily for the first 30 days</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>post cessation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SI group only</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>when required- all participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>end of weeks two months post</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>cessation all participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>three months post cessation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>all participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>four months post cessation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>all participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>five months post</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>cessation all participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>six months post</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>cessation all participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>one year post</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>cessation SI and LA groups only</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
participants in the SI group and the LA group were required to record the number of cigarettes smoked, date of relapse and reason for relapse at the one year follow up assessment.

3.4 Procedure

The procedure for the present investigation consisted of the following steps:
1) Initial Contact
2) Initial Assessment
3) Treatment
4) Post-Treatment Assessments
5) Follow-up Assessment Session

3.4.1 Initial Contact

Initial telephone contact was initiated by potential participants who responded to advertisements placed to two local newspapers for a smoking cessation program. During the initial telephone contact with each potential participant, the investigator introduced herself as a doctoral student in Applied Psychology at the Ontario Institute for Studies in Education, University of Toronto conducting research as part of her doctoral dissertation.

All respondents were informed of the participation criterion at the time of the initial contact. They were also informed that should they choose to participate in the research study that they would be required to complete research questionnaires, attend weekly treatment sessions ranging from seven to ten weeks in duration. Information would also be collected on a daily basis for 30 consecutive days at a date designed once the study commenced. As well, they would be required to record information at two, three, four, five, six, and twelve months following the designated quit day in the treatment programs. The potential
Participants were also informed that there would be a $45.00 participation fee. Individuals who indicated that they were able to commit themselves to partake in the study were invited to the initial assessment meeting.

3.4.2 Initial Assessment

During the initial assessment, the investigator again introduced herself and explained that the research was part of the requirements for a doctoral degree from the Ontario Institute for Studies in Education, University of Toronto. The criteria for inclusion in the investigation were presented to the potential participants. Those individuals who were able and willing to commit themselves to the investigation were randomly assigned to one of the treatment conditions. The participants were informed that one third of them would not be offered treatment immediately, as they would act as the control group. They were informed that treatment from the Lung Association Countdown Programme would be offered post-study to those offered no treatment at the initial phase.

Individuals assigned to Group One were informed that they were designated to the Social Interaction Coping Skills group. Individuals assigned to Group Two were informed that they were designated to the Lung Association Countdown Programme, while those individuals assigned to Group Three were informed that they were designated to the no treatment control group.

After assignment to groups, each participant was asked to fill out a personal history questionnaire (see Appendix J). As well, the Smoking Confidence Questionnaire (see Appendix F), the Anger Expression Questionnaire (see
Appendix H), and the Profile of Mood States (see Appendix G) were administered. Table 3-5 charts when the tests were administered to the participants. The subjects were informed that all information gathered through the questionnaires and treatment sessions was to be treated in confidence and that the results of the study would be shared with those interested participants.

3.4.3 Treatment

3.4.3.1 The Social Interaction Coping Skills Program

The Social Interaction Coping Skills Program (SI) was designed to teach individuals how to improve their interpersonal functioning through the acquisition of new skills which would allow them to identify stimuli and situations which were related to their smoking behaviour. They were also taught how their cognitions, behaviours, and feelings interact to maintain ineffective coping skills, as well as the techniques of reappraisal. There was a substantial focus on urge/craving control.

The program was administered to the participants by the author of the study.

The program was assembled into 16 sessions. The group met twice weekly for the first six weeks, and once a week for the subsequent four weeks. The program is outlined in Appendix B. The start date of the SI program was arranged so that the proposed quit date matched that of the quit date for the comparative Lung Association Countdown Programme.

The participants in the SI program were required to complete various questionnaires for the 30 day period post cessation. They completed the Self-
reported Symptoms Checklist and the POMS daily to monitor their withdrawal symptoms and their moods during the first 30 days post smoking cessation. As well, each subject in the SI group was required on a daily basis for this same time period, to rate their urge to have a cigarette, using a 10 point Likert scale (1 - no urge; 10- great urge).

3.4.3.2 Lung Association Countdown Programme

The Lung Association Countdown Programme is a seven week program which focuses on the gradual cutting down of cigarette consumption over a period of time. Individuals in this group were given a predesignated quit day, which treatment was geared toward. As part of the study, but not part of the Lung Association Countdown Programme, each participant was required to complete the Self-reported Symptoms Checklist and the POMS, on a daily basis, for the 30 day post-cessation assessment period. This program was administered by regular program facilitator with the Lung Association.

3.4.3.3. No Treatment Control Group

Throughout the treatment phase, contact was made with those individuals assigned to the control group, once the participants in the two treatment groups had reached the quit day in their respective programs. Subjects in the control group were also required to complete the symptoms checklist and the POMS on a daily basis for the same 30 day period of time.

3.4.4 Post Treatment Assessments

At two, three, four, five and six months post-treatment, all subjects were
mailed questionnaires (Appendix K and Appendix L) regarding smoking cessation, relapse and reason for relapse (if applicable). If responses were not received within two weeks of mailing, a pick up of the questionnaire was arranged.

After the six month post-treatment assessment, arrangements were made for treatment via the Lung Association Countdown Programme for those individuals in the no treatment control group.

At one year post-treatment, only those subjects in the two treatment groups were contacted via the mail and asked to fill out the questionnaires regarding smoking cessation, relapse and reason for relapse. The no treatment control group was also contacted, however their questionnaires were not utilized in the 12 month analysis.

3.4.5 Follow-up Assessment Session

Thirteen months post-treatment, all participants in the research project were contacted and invited to an information session regarding the outcome of the investigation.

3.5 Analysis

The main purpose of this study was to investigate the effectiveness of the Social Interaction Model as a means to achieve and maintain smoking cessation. Pairwise chi-square analyses were performed to compare the percentage of quitters in each of the groups. An analysis of variance technique was used (ANOVA) to test for differences between the three groups on the demographic, dependent measures and number of cigarettes smoked post cessation.
CHAPTER 4

Results

As previously described, the data was analyzed using the 7.5 version of the SPSS statistical software programme (SPSS Incorporated). Descriptive statistics focusing on the means and standard deviations were calculated for the demographic variables and the assessment tools.

Analysis of variance was performed on the means of total scores and subscale scores of the POMS, Smoking Confidence Questionnaire, Anger Expression Scale, Self-reported Symptom Checklist, and number of cigarettes smoked. T-tests were used to compare the means of total scores and subscale scores between those participants who successfully quit smoking and those who did not. Group comparisons of success rates (percent abstinent) were made using chi-square analysis.

4.1 Smoking Cessation

Quit day is defined in each of the treatment programs as the predesignated day in which the participants were to cease smoking. Appendix M contains the comparative data between those individuals who quit smoking (the quitters) and those who did not quit smoking (the non-quitters) on the demographic, personality, and smoking history variables. There were no significant differences between these two groups relative to the demographic or smoking history variables.
Similarly, there were no statistically significant differences between the quitters and non-quitters on any of the personality variables.

4.1.1 Hypothesis 1: Increase the success of smoking cessation

By the designated quit day, 19 participants quit smoking, while 40 did not quit. 61.9% of the participants in the SI group quit smoking, 22.2% of participants in the LA group quit smoking and 10% of participants in the no treatment control group quit smoking. Table 4-1 displays the percentage of quitters per group over time.

There was a significant difference between the three groups from the quit day (Figure 4-1) to the six month follow up session. At the one year follow up period, only the SI group and the LA group were compared, as the no treatment control group had the option of receiving treatment after the six month follow up assessment. There was no significant difference between the SI group and the LA group at the one year follow up session $\chi^2 = 1.20$, n.s.

4.1.3 Hypothesis 2: Time of relapse

In order to investigate the difference between the percentage of quitters between the groups, pair wise chi-square analysis was performed. The SI group had significantly more quitters than did the LA group, $\chi^2 = 6.20$, p<.5, and the no treatment control group, $\chi^2 = 11.89$, p<.01, at the designated quit day. The SI group maintained a significant difference over the no treatment control group at the six month follow up analysis. There was no difference between the SI group and the LA group with respect to time of relapse for those individuals who had
### Table 4-1

**Percentage of Individuals Who Quit Per Group Post Cessation**

<table>
<thead>
<tr>
<th>Time</th>
<th>SI Group (N)</th>
<th>LA Group (N)</th>
<th>Control Group (N)</th>
<th>X</th>
<th>Pairwise  X</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quit Day (N)</td>
<td>61.9 (21)</td>
<td>22.2 (18)</td>
<td>10.0 (20)</td>
<td>13.8**</td>
<td>a,b</td>
</tr>
<tr>
<td>1 Month P.Q.D. (N)</td>
<td>66.7 (21)</td>
<td>50.0 (18)</td>
<td>15.0 (20)</td>
<td>11.46**</td>
<td>a,c</td>
</tr>
<tr>
<td>2 Months P.Q.D. (N)</td>
<td>70.0 (20)</td>
<td>40.0 (15)</td>
<td>12.5 (18)</td>
<td>9.32**</td>
<td>a</td>
</tr>
<tr>
<td>3 Months P.Q.D. (N)</td>
<td>63.2 (19)</td>
<td>38.5 (13)</td>
<td>11.8 (17)</td>
<td>9.98**</td>
<td>a</td>
</tr>
<tr>
<td>4 Months P.Q.D. (N)</td>
<td>63.2 (19)</td>
<td>38.5 (13)</td>
<td>12.5 (16)</td>
<td>9.32**</td>
<td>a</td>
</tr>
<tr>
<td>5 Months P.Q.D. (N)</td>
<td>63.2 (19)</td>
<td>41.7 (12)</td>
<td>13.3 (15)</td>
<td>8.58*</td>
<td>a</td>
</tr>
<tr>
<td>6 Months P.Q.D. (N)</td>
<td>63.2 (19)</td>
<td>41.7 (12)</td>
<td>13.3 (15)</td>
<td>8.58*</td>
<td>a</td>
</tr>
<tr>
<td>1 Year P.Q.D. (N)</td>
<td>29.4 (17)</td>
<td>13.3 (15)</td>
<td></td>
<td>1.20</td>
<td></td>
</tr>
</tbody>
</table>

** indicates p<.01
* indicates p<.05

a=SI group significantly different from no treatment control group
b=SI group significantly different from LA group
c=LA group significantly different from no treatment control group
P.Q.D.=post quit day
FIGURE 4-1  The Percentage of Subjects Quitting in Each Group

Time Post Quit

- SI Group
- LA Group
- Control Group
successfully quit smoking and later relapsed, $\chi^2(2) = 2.16$, n.s..

4.1.3 Hypothesis 3: Number of cigarettes smoked post quit

Table 4-2 contains the means and the standard deviations for the number of cigarettes smoked at each of the seven post quit assessment periods. One way analysis of variance, followed by Newman Keuls comparisons, indicate that the SI group and the LA group smoked significantly fewer cigarettes at the one month post quit assessment period than did the no treatment control group $F(56)=8.79$, $p<.01$. At the six month post quit assessment period, the SI group smoked significantly fewer cigarettes than did those in the LA group and the no treatment control group $F(2,43)=6.40$, $p<.01$. As well, at the one year post quit follow up, the SI group smoked significantly fewer cigarettes than did those in the LA group $F(1,30)=4.6$, $p<.05$.

With respect to change in smoking behaviour, those individuals in the SI group smoked significantly fewer cigarettes at the six month assessment period $t(11)=3.52$ and at the one year assessment period $t(14)=3.04$, $p<.01$ when compared to the baseline smoking rates (Figure 4-2). There was no significant change in smoking behaviour by those individuals in the no treatment control group at the six month assessment period $t(14)=1.61$, n.s.

4.1.4 Hypothesis 4: Predictors of Smoking Cessation

All participants were required to complete the Profile of Moods States questionnaire, the Anger Expression Scale and the Smoking Confidence Questionnaire prior to the commencement of treatment. None of the psychometric
Table 4-2

Group Comparisons for Cigarettes Smoked at Post Quit Assessment Periods

<table>
<thead>
<tr>
<th>Time</th>
<th>SI Group M</th>
<th>SI Group SD</th>
<th>LA Group M</th>
<th>LA Group SD</th>
<th>Control Group M</th>
<th>Control Group SD</th>
<th>F Value</th>
<th>Newman Keuls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>25.52</td>
<td>(9.02)</td>
<td>26.50</td>
<td>(17.45)</td>
<td>22.60</td>
<td>(8.67)</td>
<td>.54</td>
<td></td>
</tr>
<tr>
<td>1 Month</td>
<td>2.23</td>
<td>(4.31)</td>
<td>9.33</td>
<td>(19.87)</td>
<td>19.55</td>
<td>(12.06)</td>
<td>8.79**</td>
<td>a,c</td>
</tr>
<tr>
<td>2 Months</td>
<td>2.45</td>
<td>(4.31)</td>
<td>8.00</td>
<td>(10.96)</td>
<td>20.05</td>
<td>(10.76)</td>
<td>18.84**</td>
<td>a,c</td>
</tr>
<tr>
<td>3 Months</td>
<td>3.21</td>
<td>(5.74)</td>
<td>11.53</td>
<td>(11.53)</td>
<td>18.94</td>
<td>(18.94)</td>
<td>9.90**</td>
<td>a,b</td>
</tr>
<tr>
<td>4 Months</td>
<td>4.31</td>
<td>(6.77)</td>
<td>11.38</td>
<td>(15.37)</td>
<td>18.56</td>
<td>(10.34)</td>
<td>7.54**</td>
<td>a</td>
</tr>
<tr>
<td>5 Months</td>
<td>4.73</td>
<td>(7.60)</td>
<td>13.41</td>
<td>(17.32)</td>
<td>18.26</td>
<td>(18.26)</td>
<td>5.72**</td>
<td>a</td>
</tr>
<tr>
<td>6 Months</td>
<td>4.63</td>
<td>(7.31)</td>
<td>13.66</td>
<td>(17.28)</td>
<td>18.86</td>
<td>(10.95)</td>
<td>6.40**</td>
<td>a,b</td>
</tr>
<tr>
<td>1 Year</td>
<td>12.82</td>
<td>(10.24)</td>
<td>19.60</td>
<td>(17.27)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

** indicates p<.01
* indicates p<.05
a=SI Group significantly different from Control Group
b=SI Group significantly different from LA Group
c=LA Group significantly different from Control Group
FIGURE 4-2  Mean Number of Cigarettes Smoked Per Group Post Cessation

Mean Number of Cigarettes Smoked

Post Cessation

- SI Group
- LA Group
- Control Group
tests were able to differentiate between those individuals who successfully quit smoking and those who did not. The findings for the psychometric tests were:

(F2,56)=.881, n.s. - Profile of Mood States; F(2,56)=.638, n.s. - Anger Expression Scale; and F(2,56)=.559, n.s.

4.2 Daily Measures Post Quit

Subjects in all three groups were required to monitor and record their symptoms (on the Self-reported Symptom Checklist) and their mood state (as measured by the POMS) daily, for a continuous 30 day assessment period, post designated quit day. Figure 4-3 and Figure 4-4 present the means for the Self-reported Symptom Checklist and the POMS for all of the participants in the three groups during the assessment period.

In order to attempt to capture any differences between the groups over the 30 day period on the daily records of the Self-report Symptom Checklist and the POMS, the data were broken into five segments, each segment consisting of the average for a consecutive six day period. A repeated measures anova, contrasting symptoms by groups over time was performed. Table 4-3 contains the means and the standard deviations of the five segments for the Symptom Check List and Table 4-4 contains the means and the standard deviations of the five segments for the POMS.

A two way repeated measures ANOVA was performed, comparing Symptom segment scores by group. There was no significant difference between the groups F(2,56)=.481, n.s.. However, there was a significant group by time
FIGURE 4-3 Group Comparisons on Symptom Ratings

Mean Symptom Score

Days Post Designated Quit Day

- SI Group  - LA Group  - Control Group
Figure 4-4 Group Comparisons on POMS Scores

Mean POMS Scores

Days Post Designated Quit Day

SI Group - LA Group

Control Group
Table 4-3

Symptom Segment Scores By Group (n=59)

<table>
<thead>
<tr>
<th>Time</th>
<th>SI Group</th>
<th>LA Group</th>
<th>Control Group</th>
<th>F(2,56)</th>
<th>Newman Keuls</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>95.42</td>
<td>88.38</td>
<td>53.00</td>
<td>6.77**</td>
<td>a,c</td>
</tr>
<tr>
<td>SD</td>
<td>(44.99)</td>
<td>(40.86)</td>
<td>(29.87)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>73.09</td>
<td>77.22</td>
<td>53.30</td>
<td>2.48</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>(42.45)</td>
<td>(38.65)</td>
<td>(23.60)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>58.47</td>
<td>66.94</td>
<td>46.70</td>
<td>1.43</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>(48.08)</td>
<td>(33.70)</td>
<td>(24.570)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>42.14</td>
<td>53.33</td>
<td>49.25</td>
<td>.55</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>(37.03)</td>
<td>(36.11)</td>
<td>(27.07)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>40.28</td>
<td>47.27</td>
<td>67.90</td>
<td>1.90</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>(38.55)</td>
<td>(39.52)</td>
<td>(59.13)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

** indicates p<.01
a=SI Group significantly different from the Control Group
b=LA Group significantly different from the Control Group

Segment 1 = Days 1 - 6
Segment 2 = Days 7 - 12
Segment 3 = Days 13 - 18
Segment 4 = Days 19 - 24
Segment 5 = Days 25 - 30
Table 4-4

POMS Segment Scores By Group (n=59)

<table>
<thead>
<tr>
<th>Time</th>
<th>SI Group</th>
<th>LA Group</th>
<th>Control Group</th>
<th>F(2,56)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segment 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>188.90</td>
<td>195.33</td>
<td>180.05</td>
<td>.05</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(124.15)</td>
<td>(172.37)</td>
<td>(151.10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segment 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>176.38</td>
<td>188.83</td>
<td>170.65</td>
<td>.079</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(122.35)</td>
<td>(156.29)</td>
<td>(150.86)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segment 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>176.76</td>
<td>172.83</td>
<td>171.10</td>
<td>.007</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(162.17)</td>
<td>(145.08)</td>
<td>(149.73)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segment 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>155.87</td>
<td>124.22</td>
<td>171.00</td>
<td>.63</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(130.13)</td>
<td>(105.13)</td>
<td>(149.42)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segment 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>133.04</td>
<td>113.5</td>
<td>165.15</td>
<td>.92</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(115.76)</td>
<td>(96.83)</td>
<td>(140.96)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Segment 1 = Days 1 - 6
Segment 2 = Days 7 - 12
Segment 3 = Days 13 - 18
Segment 4 = Days 19 - 24
Segment 5 = Days 25 - 30
interaction, $F(4, 228) = 7.30, p<.001$. To examine this interaction, a series of one-way ANOVAS were performed comparing the means of the five symptom segment scores. As shown in Table 4-3, the groups differed only during the first six days. Subsequent Newman Keuls comparisons showed that the no treatment control group displayed significantly fewer symptoms than did those individuals in either treatment group.

A two way repeated measures ANOVA comparing Self-reported Symptom Checklist segment scores by group (quitters vs. non quitters), showed a significant difference between those who quit and those who did not quit, with respect to symptom of withdrawal scores $F(1, 57) = 8.78, p<.01$. Those who quit smoking scored significantly higher on the Self-reported Symptom Checklist (see Figure 4-5). As well, there was a significant group by time interaction showing that the two groups changed differently over time, $F(4, 228) = 4.28, p<.0001$. Due to the fact that the Mauchly sphericity test was significant, at $p<.001$, the Greenhouse Geisser epsilon correction factor was applied. The interaction was still found to be significant, $F(2.67, 152) = 4.28, p<.01$.

A two way repeated measures ANOVA was also performed comparing POMS scores by group (quitters vs. non quitters). It was found that the groups both changed significantly over time, in that their POMS scores decreased over time $F(1, 57) = 5.26, p<.05$. However, there was no significant group by time interaction, indicating that the groups did not change differently over time, $F(4, 228) = 1.52, n.s.$ (see Figure 4-6).
Figure 4-5  Symptom Ratings for Quitters and Non-Quitters

Mean Symptom Rating

Days Post Designated Quit Day

--- Quit  --- Non-Quit
Figure 4-6 POMS Ratings for Quitters and Non-Quitters
Participants also completed the Smoking Confidence Questionnaire at the end of each of the five weeks of the 30 day post quit assessment period. The means and the standard deviations are presented in Table 4-5. The groups did not differ during any of the post cessation assessment periods with respect to smoking confidence.

Those individuals in the SI group were required to record their urge/desire to have a cigarette on a daily basis for the first 30 consecutive days post quit day. In order to obtain a better estimate of trend with respect to the urge ratings, the scores were compiled into five, six day segments. A repeated measures ANOVA was performed in order to investigate urge ratings by group (quitters vs. non quitters). As shown in Table 4-6, no significant differences were found between those individuals who did not quit, relative to their self-reported urge ratings, F(1,19)=.00, n.s. There was a statistically significant decrease in urge rating over time, F(4,76)=5.39, p<.001. However, there was no significant group by time interaction F(4,7)=.20, n.s.

4.2.1 Personality Variables

Two by two ANOVAS were performed for each assessment period on the two treatment groups to examine the relationship of each of the personality variables relative to whether the subjects quit smoking or not. Only vigor, which is a subscale of the POMS, was significantly related to whether subjects quit smoking or not. This was only significant at one month post quit. Subjects who quit smoking had significantly lower mean vigor scores (M=10.0) than subjects who
<table>
<thead>
<tr>
<th>Time</th>
<th>SI Group M</th>
<th>SI Group SD</th>
<th>LA Group M</th>
<th>LA Group SD</th>
<th>Control Group M</th>
<th>Control Group SD</th>
<th>F(2,56)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week One</td>
<td>227.14</td>
<td>(50.28)</td>
<td>239.05</td>
<td>(67.91)</td>
<td>240.75</td>
<td>(37.02)</td>
<td>.668</td>
<td>n.s.</td>
</tr>
<tr>
<td>Week Two</td>
<td>236.76</td>
<td>(56.59)</td>
<td>246.94</td>
<td>(59.10)</td>
<td>242.65</td>
<td>(34.35)</td>
<td>.822</td>
<td>n.s.</td>
</tr>
<tr>
<td>Week Three</td>
<td>251.80</td>
<td>(76.73)</td>
<td>248.66</td>
<td>(66.23)</td>
<td>248.15</td>
<td>(38.70)</td>
<td>.979</td>
<td>n.s.</td>
</tr>
<tr>
<td>Week Four</td>
<td>268.76</td>
<td>(73.97)</td>
<td>239.72</td>
<td>(55.80)</td>
<td>249.20</td>
<td>(48.35)</td>
<td>.316</td>
<td>n.s.</td>
</tr>
<tr>
<td>Week Five</td>
<td>272.38</td>
<td>(71.13)</td>
<td>247.05</td>
<td>(57.73)</td>
<td>244.50</td>
<td>(38.08)</td>
<td>.216</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

Table 4-5

Post Cessation Smoking Confidence Questionnaire Scores (n=59)
Table 4-6

Urge Ratings 30 Day Post Cessation for SI Group (n=21)

<table>
<thead>
<tr>
<th>Segment</th>
<th>Quitters (N=13)</th>
<th>Non Quitters (N=8)</th>
<th>F(1,19)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>One (days 1 - 6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>37.69</td>
<td>35.12</td>
<td>.487</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(9.23)</td>
<td>(5.48)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Two (days 7 - 12)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>34.92</td>
<td>37.75</td>
<td>.535</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(9.42)</td>
<td>(10.84)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Three (days 13 - 18)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>33.83</td>
<td>34.00</td>
<td>.881</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(8.46)</td>
<td>(9.98)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Four (days 19 - 24)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>27.00</td>
<td>27.62</td>
<td>.881</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(7.87)</td>
<td>(11.13)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Five (days 25 - 30)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>28.07</td>
<td>26.37</td>
<td>.711</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(9.97)</td>
<td>(10.28)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
did not quit (M=13.8), as demonstrated by the Newman Keuls contrasts, F(1,34)=6.25, p=.017. None of the other analysis showed any significant differences.

4.3 Relapse Data

Analysis of variance was performed to examine the relationship of each of the personality variables to time of relapse. A significant difference was obtained with Newman Keuls contrast on the depression variable, a subscale of the POMS F(2,35)=4.53, p=.0087. Subsequent Newman Keuls analysis indicated that those individuals who relapsed during the first week post quit, scored significantly higher on the depression scale than did those individuals who relapsed during the second week, and those individuals who did not relapse. No other significant differences were found relative to the personality variables and time of relapse. However, the vigor subscale of the POMS was found to be significantly correlated r=.46, p<.05 with non relapse.

Individuals who relapsed were requested to complete a reason for relapse questionnaire (see Appendix L). For those individuals who quit smoking by the designated quit day in the SI group, 25% relapsed due to peer pressure, 25% relapsed due to depression, 12.5% due to symptoms and 12.5% due to consuming alcohol. In the LA group 50% relapsed due to peer pressure and 50% due to symptoms.
The present study was designed to evaluate the effectiveness of the Christensen and Pass (1983) social interactional coping skills program (Social Interactional) in a smoking cessation and relapse prevention study. At the designated quit day, the Social Interaction group had significantly more quitters than the comparative Lung Association group and the control group. The Social Interaction program was very effective as a mechanism for achieving smoking abstinence for the duration of the active intervention process. These strong clinical results are comparable to the more effective smoking cessation programs cited in literature reviews (Brandon, 1994; Carmody, 1992; Halpern & Warner, 1993).

5.1 Smoking Cessation

The Social Interaction group was able to maintain an abstinence status of 63.2% at the six month follow-up assessment period, compared to the abstinence rate of 41.7% for the Lung Association group and 13.3% for the no treatment control group. At the one year follow-up, 29.4% of individuals in the Social Interaction group had abstained from smoking, relative to 13.3% of individuals in the Lung Association group. The one year abstinence rate of the Social
Interaction group is in keeping with the outcome success rates of more effective smoking cessation clinics (Leventhal and Cleary, 1980). Although not statistically significant relative to the Lung Association group at the six month post cessation assessment period, the Social Interaction program was statistically more effective than the no treatment control group, whereas the Lung Association program was not. The Social Interaction program was more effective than most smoking cessation programs, which on average, demonstrate that only about 30% of individuals who reach abstinence, maintain abstinence for three months (Brandon, Zelman, Baker, 1987; Danaher, 1977; Hunt & Bespelac, 1974).

The findings support the first hypothesis that the Social Interaction program would significantly increase the success of smoking cessation and significantly reduce the relapse rate of smokers. Although successful in achieving smoking cessation, the high abstinence rate could not be maintained after six months. However, the Social Interaction program was very effective in reducing the number of relapses within the first six months. Research has demonstrated that most relapses occur within the first 90 days post cessation (Hunt, Barnett and Brand, 1971; Le Houezec, 1998; Marlatt and Gordon, 1985). The second hypothesis, that those individuals assigned to the Social Interaction group would relapse later than those in the Lung Association group was supported. Within the first six months the Social Interaction group had fewer relapses when compared to the Lung Association group and when compared to the findings of other smoking cessation studies (Le Houezec, 1998, Marlatt and Gordon, 1985; Mudde et al., 1995).
The high abstinence rate was not maintained at the one year follow-up, but those who relapsed in the Social Interaction group were smoking significantly fewer cigarettes than those who relapsed in the Lung Association group. This finding supports the third hypothesis of the study which predicted that those individuals who relapsed in the Social Interaction group would smoke fewer cigarettes on average than those individuals who relapsed in the Lung Association group. The significant decrement in amount of smoking does have positive health implications in terms of risk for cardiovascular disease and cancer. It could be postulated that an individual's quality of life could be dramatically improved if they successfully decreased their rate of smoking. As well, the decrease in smoking rate would eventually benefit the health care system in terms of current bed shortages and funding decreases.

5.2 Demographic and Personality Variables

The only psychometric test which differentiated between the three groups prior to the onset of treatment was the Smoking Confidence Questionnaire. Those individuals who were assigned to the SI group had significantly more confidence that they could resist smoking in various situations, relative to those individuals who were assigned to the No Treatment group. However, this difference disappeared once the test was re-administered at each of the five one week assessment periods post cessation. It is possible that those individuals who were assigned to the SI group felt more confident that their treatment would be effective due to the fact that it was a new program. This explanation would be in keeping
with Brownell's incremental learning model (1986) which proposes not only that individuals' self-efficacy increases at each attempt at quitting, but it also increases with each new method utilized.

There were no significant differences on any of the demographic or personality variables between those individuals who quit smoking and those who did not quit smoking. The Smoking Confidence Questionnaire and the Anger Expression Questionnaire were also unable to differentiate between quitters and non-quitters. Nor was there any predictive validity for relapse with either test. Of interest was the finding that smoking confidence did not change over time in either the quit or non-quit groups. This may suggest that those individuals who were not successful during this cessation attempt were still hopeful that they had the ability to quit in the future. This would be in keeping with Brownell's (1986) incremental-learning model, which suggests that each attempt at quitting, increases the smoker's knowledge of the process which is needed for achieving abstinence. Brownell's finding contrasts Hughes' (1988) hypothesis that individuals who fail to quit smoking have lower self-efficacy than those individuals who successfully quit smoking. The non-quitters were not different from the quitters in terms of self-efficacy as measured by the Smoking Confidence Questionnaire.

All subjects were required on a daily basis to complete a Self-report Symptom Checklist and the Profile of Mood States for the first 30 days after the designated quit day. Those individuals who quit smoking reported significantly more symptoms than the non-quitters. They consistently indicated that they were
experiencing more anxiety and irritability, as well as poor concentration and restlessness. Hughes et al. (1991) found that self-reported withdrawal symptom ratings provided by individuals in their cessation study were consistently supported by a secondary observer (usually a family member) who knew the smoker very well. As in the Hughes (1991) study, the quitter's symptom scores from this study changed over time, with the quitters demonstrating fewer symptoms over time. Those individuals in the Social Interaction group and the Lung Association group reported more withdrawal symptoms than did those individuals in the no treatment control group during the first six day segment. Those in the Social Interaction group did not report significantly more or less symptoms than those in the Lung Association group, even though they were taught to focus on physiological cues as part of their treatment program. This may suggest that although those individuals in the Social Interaction group were to focus on body sensations, that this aspect of treatment and relapse prevention did not create a state of hyper vigilance to body sensations and symptoms of withdrawal. The lack of a relationship between severity of symptom rating and relapse is also consistent with several studies (Hatsukami, Hughes, and Pickens; 1985; Hughes et al., 1991; Hughes et al., 1986; Gross and Stitzer, 1989; Klesges, Meyers, and Winders, 1989; Shiffman, 1979).

No significant difference with respect to Profile Of Mood States scores between quitters and non-quitters was demonstrated. The Profile Of Mood States scores significantly decreased over the 30 day evaluation period in both treatment
groups. However, those individuals who quit smoking reported significantly lower scores on the Vigor subscale of the Profile Of Mood States at the one-month post quit date. This significant difference disappeared at the subsequent follow-up assessments. This finding may be reflective of the decrease in energy which is often reported post cessation (Pomerleau et al., 1983, 1995; Hughes et al., 1988; Hatsukami, 1984). It is also possible that the lower scores on the Vigor subscale reflect vegetative signs of depression.

Members of the Social Interaction group were required to report their desire (urge) to smoke for 30 days post quit. There were no significant differences in the urge ratings between quitters and non-quitters, however the urge scores decreased over time for both the quitters and non-quitters. The lack of statistical difference between the groups suggests that a majority of participants in the Social Interaction group were able to effectively cope with their urge to smoke, despite a strong desire to smoke.

5.3 Relapse

Although studying smoking abstinence rates following two different treatment models was the main goal of the study, relapse prevention was also of interest. Those individuals who relapsed within the first week post quit, scored significantly higher on the Depression subscale of the Profile Of Mood States, than did those who relapsed during the second week and those who did not relapse at all. The investigation of a relationship between depression and relapse is a growing area of research (Brandon, 1994; Carmody, 1992; Carton, Jouvent &
Chronic smoking has recently been shown to have antidepressive effects. According to Le Houezec (1998) nicotine inhibits monoamine oxidase, the enzyme responsible for the degradation of serotonin and norepinephrine. As a result, more serotonin and norepinephrine are left in the system. The increase of serotonin and norepinephrine result in a decrease in depression symptomatology.

Le Houezec (1998) reviewed a sample of 1200 young adult smokers and found that their smoking dependence was related to their history of major depression. Twenty-seven percent of the nicotine dependent smokers had a history of major depression, while only 12 percent of the nondependent smokers had a history of major depression. The percentage of depression in the nonsmokers was slightly above nine percent. Similarly, Le Houezec's review suggests that there is a higher smoking prevalence in depressed people (46%), whereas in the general population smoking prevalence is 15 percent.

Several studies (Breslau, Kilbery & Andreski, 1991; Carton, Jouvent & Widlocher, 1994; Glassman and Stetner, 1990; Hall, Munoz, Reus & Sees, 1993; Pomerleau et al., 1984) have shown that those individuals who indicated that they smoked in response to depressed mood states had significantly lower abstinence rates at 12 months posttreatment. Similarly, Brandon (1994) found that those individuals who reported negative affect before quitting had poorer long term success with cessation than those individuals who did not report negative affect. Both Marlatt and Gordon (1985) and Shiffman (1985) found that negative affect
was deemed the precipitant for relapse in a majority of ex-smokers. Le Houezec (1998) investigated abstinence rates for individuals who were provided bupropion, an antidepressant drug, post smoking cessation. After one year post quit, it was found of individuals who were given a placebo only 12.4% had not relapsed. However, for those individuals who received doses of 100 mg. per day, 150 mg. per day, and 300 mg. per day, the abstinence rates at one year were 19.6%, 22.9% and 23.1%, respectively. This high success rate, which occurred without adjunct therapy suggests a possible significant role for addressing depression as a component in standard smoking cessation programs and for relapse prevention.

Participants in the study were required to record their reasons for relapse. It was found that in the Social Interaction group 25% of the subjects relapsed due to peer pressure, 25% due to depression, 12.5% due to symptoms and 12.5% due to consuming alcohol. The reasons for relapse were found to be moderately different in the Lung Association group where 50% of the participants reported that they relapsed due to peer pressure and the other 50% of participants reported that they relapsed due to their symptoms. The findings for the Social Interaction group are comparable to those of Marlatt and Gordon (1985) who found that 37% of individuals relapsed due to negative emotional states and 32% relapsed in response to social pressure. However, the results of those in the Lung Association group do not parallel the findings of Marlatt and Gordon as closely. It is possible that the individuals in the Social Interaction group were more cognizant of their emotional states and physical states due to the content of the Social Interaction
program, which placed significant emphasis on understanding and coping with both emotional and physiological cues and stimuli. Baer and Lichtenstein (1988) found that 31% of the subjects in their study could not find an attribution for their relapse. Reviewing the reported reasons for relapse from the individuals in the Lung Association group, one may query if they were able to differentiate between their emotional and physical states. If they could not, it is possible that they generalized both their emotional and physical states to that of the general category of symptoms.

Peer pressure played a significant role in both the Social Interaction group and the Lung Association group in terms of reason for relapse. Although, peer pressure accounted for 25% of the relapse rate in the Social Interaction group, peer pressure as well as depression (also 25%) were the primary reasons for relapse. In comparison in the Lung Association group, 50% of subjects stated that they relapsed due to peer pressure. The Social Interaction program taught reappraisal techniques and used role play to assist in the acquisition of coping skills to deal specifically with high risk social situations. Those in the Social Interaction group demonstrated more success than did those in the Lung Association group in maintaining abstinence in high risk social situations. This may suggest that the role playing did contribute to maintaining abstinence, as the high abstinence rate was maintained for six months.

12.5% of those who relapsed in the Social Interaction group indicated the reason for relapse was their difficulty coping with symptoms, whereas 50% of
those who relapsed in the Lung Association group stated that they relapsed due to difficulty coping with symptoms. Although those in the Social Interaction group were not reporting more or less symptoms than others, they were less likely to relapse as a result of their symptoms. Body sensations (urges, cravings, withdrawal symptoms) and coping mechanisms were a focus of the relapse prevention sessions in the Social Interaction program. Participants were taught the technique of reappraisal as a means of dealing with the potentially threatening internal and external physical stimuli. Similarly, participants in the Social Interaction group were taught to recognize and validate their physical sensations instead of using avoidance techniques (such as smoking to alleviate nervousness or boredom). The techniques taught in the Social Interaction group for coping with symptoms (i.e. withdrawal and craving) were successful, as fewer individuals in the Social Interaction group relapsed due to symptoms than in the Lung Association group.

5.4 Limitations of the Present Study and Suggestions for Future Research

Prior to considering the potential clinical and community implications of the study, the limitations of the study must be addressed. The Social Interaction program demonstrated clinical success for obtaining smoking cessation. However, it was less successful in relapse prevention after six months post cessation. The high rate of abstinence at the six month post cessation assessment suggests that the program is successful in the short term. Overall the study suggests the potential benefit of including various components from the Social Interaction
program into the common existing approaches of smoking cessation programs. Future studies should attempt to incorporate several booster sessions between six months and one year post cessation.

A potential limitation of the study is that depression was not addressed during the initial assessment. Although the Profile Of Mood States was used during the initial gathering of information, the intention was not to use the tool as a diagnostic screen for identification and modification of treatment. As well, the Profile Of Mood States may not have been sensitive enough to measure the vegetative signs and symptoms of depression. In the future, the program would need to be modified to assess mental health issues, such as depression. Several studies (Abrams et al, 1987; Carmody, 1992; Covey et al. 1990; Lerman, Audrain, Orleans, Boyd, Gold, Main and Caporaso, 1996) found that depressed individuals were less likely to quit smoking successfully. As well, they are also more likely to join smoking cessation programs than individuals without a history of depression. Covey et al. (1990) also found in a survey of 1,004 children aged 15 to 16 years, who were currently smoking, had displayed depressive symptoms when they had been interviewed nine years earlier. Future research in smoking cessation should include a mechanism to screen for depression. The level of depression should be addressed and treated concurrently with smoking dependence.

The small sample size also limits the generalizability of the study. Although, the small sample size and the large number of variables increases the probability of Type I errors in the data, the significant findings were consistent with recent
smoking cessation studies. In order to thoroughly investigate the efficacy of the Social Interaction Program, a much larger sample size would be required. This would improve the statistical validity of the findings and allow for broader interpretation of the results.

The repeated administration of the psychometric tests (Profile Of Mood States, Smoking Confidence Questionnaire, and Self-report Symptoms Checklist) although essential in terms of investigating a possible relationship with relapse, the repeated testing may have made the tools less clinically sensitive. Gilbert et al. (1998) found that self-reported negative mood states decrease overtime, not necessarily as a function of actual mood change, but due to the repeating of the tests in a short period of time. Similarly, Sharpe and Gilbert (1998) stated testing effects of repeated administration of mood and depression measures should be addressed as they have implications regarding the perceived effectiveness of treatment programs. Therefore, the changes in mood scores may not be a reflection of actual state change, but a confounding effect of repeated testing. Although a considerable number of current and recent smoking cessation studies have also used the same psychometric tests, the test re-test reliability and validity of the frequent use has not been addressed in the majority of smoking cessation literature. This leads one to question the validity of research investigating the relationship between mood states and smoking cessation self-efficacy.

The fact that the psychometric and demographic tests were administered after the individuals were assigned to groups may also be a limitation to the study.
Although the subjects did not know who the treatment facilitators were at the time of filling out the questionnaires, they were cognizant as to which group they were assigned. If this study were to be replicated, it would be recommended that the initial psychometric testing be completed prior to random group assignment.

Different therapists were used to provide the two treatment programs. The therapist working for the Lung Association had eight years of experience in teaching and facilitating the Lung Association Countdown Programme. She was also an ex-smoker. The therapist facilitating the Social Interaction Program was the author of the study, a doctoral student at the Ontario Institute of Education, University of Toronto. The author had never facilitated a smoking cessation program and was a non-smoker. Due to the fact that there were two different facilitators, it is possible that some of the positive outcomes of the study were in fact attributable to the facilitators' personal characteristics, rather than the programs themselves. Similarly, the facilitator of the Social Interaction Program had a vested interest in ensuring the program's success due to the fact that she created the treatment program.

Although the community where the study was conducted has significant ethno-cultural diversity, the participants in the study were primarily Caucasian and English speaking. The lack of ethno-cultural diversity in the sample of participants for the study needs to be taken into account when attempting to generalize the results to a more diversified population. The largest minority group in the community where the study was conducted is Aboriginal. The majority of
individuals of aboriginal heritage are very reluctant to participate in research studies if asked and rarely partake in group treatment programs, with the exception of healing circles and sweat lodges (Nishnawbe-Aski, 1996). Attempts should be made in the future to broaden the demographic diversity of the sample size so that it is more reflective of the ethno-cultural diversity in Canada.

5.5 Implications of the Present Study

5.5.1 Research

The results of the study suggest that a high rate of abstinence can be achieved and maintained for at least six months. Many studies have attempted to find the elusive variables that predict success and relapse. However, most research is assuming the existence of a heterogeneous population. The diversity of smokers should be taken into consideration when attempting to introduce models for smoking cessation. The Social Interaction model can accommodate the entho-diversity of participants as it teaches the basics of reappraisal which is individualized to the specific individual. Understanding the diversity in smokers may be more effective in promoting change than using personality and mood tests to categorize individuals into pre-existing groups.

5.5.2 Clinical Practice and Prevention

The Social Interaction program also has significant potential to be utilized as a smoking prevention program. Recognizing that 95% of smoking cessation attempts are self initiated (Carmody, 1992) and recognizing that smoking cessation programs have had poor success in maintaining long term abstinence,
it may be wiser to look at providing extensive prevention programming to youth, starting in primary school. Because the Social Interaction program teaches interactional skills, it may assist the adolescent to deal with the peer pressure, which is the major contributing factor to starting smoking.

Smoking prevention, especially in the child and adolescent domain, has recently acquired a larger profile in health organizations and provincial and state governments (Pederson et. al, 1994; World Health Organization 1998; Xie et al. 1992). The World Health Organization (1995) has noted the relationship between the increase in adolescent smoking and the increase in the advertising trend of the tobacco industry, which is targeting the younger populations. McNeil (1991) suggests that between 33% and 50% of those youth who experiment with smoking, become regular smokers. On the other hand, the most successful smoking cessation and relapse prevention programs have demonstrated better success rates for long term abstinence with the older, male smoker, who has smoked for a number of years (Pomerleau, et al, 1993; Lichtenstein et al 1996; Shiffman, 1993). The fastest growing increase in smoking rates is in the adolescent age group. At present, there appears to be a large gap in service which is specifically targeting the younger population. Similarly, it would appear that increasing smoking rates are reflective of prevention programs which are not effective.

Recent research by King et al. (1996) suggests that adolescent smoking is a collective, peer behaviour. Those individuals who have lower self-esteem and
possible academic related difficulties are more likely to seek acceptance through an affiliation with a similar peer group. Once the youth is integrated into the smoking peer group, the pressure to smoke becomes overwhelming (King et. al., 1996). Therefore, it is appropriate to target this age group with a smoking prevention program which not only reinforces the negative health consequences of smoking (which is the primary focus of most prevention programs), but incorporates an interpersonal skills component, as offered in the Social Interaction model.

The use of the Social Interaction model for prevention has the potential to teach youth the skills to deal with the social situations in which they are subjected to peer pressure and have a desire to conform. Similarly, the Social Interaction model can teach the skills of reappraisal which would hopefully generalize to other equally unhealthy social situations (i.e. drug use, sexual behaviour). It is proposed that the Social Interaction model, as demonstrated in the smoking cessation program, be incorporated into school smoking prevention program in order to teach youth to effectively cope with the pressure to start smoking. Although the “Just Say No” campaign regarding drug use has been in place for over ten years, it is essential that youth are taught health prevention skills which are generalizable.

It must be recognized that any program, whether it is treatment or prevention oriented will have great difficulty succeeding in isolation. Changes need to occur at a larger systemic level. The government needs to reinstate the previous taxation levels, which were reduced due to consumer and lobbyist
demands. The laws regarding tobacco advertisement and sponsorship need to be made effective at present, not in two years time. Similarly, existing laws, such as legal age for tobacco purchase, need to be strictly enforced.

The Social Interaction smoking cessation and relapse prevention model has been successfully modified as a prevention and treatment program for adolescent, First Nation and Inuit solvent abusers (Minthorn-Biggs, 1998). The Social Interaction approach for inhalation abuse treatment is currently in use at the Ka-Na-Chi-Hih Specialized Solvent Abuse Treatment Centre, in Thunder Bay, Ontario and the Whiskyjack Treatment Centre, in Thompson, Manitoba. Ka-Na-Chi-Hih and Whiskyjack have been funded since 1996, by Health Canada (First Nation and Inuit Branch), to provide community prevention and treatment for chronic adolescent and adult solvent abusers from across Canada. The basic model of the smoking cessation and relapse prevention presented in this study was broken down into two components; 1) community prevention and 2) achieving abstinence. The core of both programs is the implementation of the social interactional skills, as described by Christensen and Pass (1983). The abstinence program is considerably longer than the program which was initially offered for smoking cessation. At present, adult solvent abusers may stay at Ka-Na-Chi-Hih for up to two years, while adolescent solvent abusers can stay at Whiskyjack for six months (with the possibility of a six month extension). However, the length of the program allows for more emphasis on social skill practice and relapse prevention. To date at the Thunder Bay program site, sixteen adults have been successfully treated
and re-integrated into their home communities, with a success rate of 75% at six month follow-up (Minthorn-Biggs, 1998). This abstinence rate is significantly higher than the average of 6%, which has been demonstrated by other treatment programs in Canada (Nishnawbe-Aski, 1996).

5.6 Conclusion

The Social Interaction program was very successful in achieving initial abstinence. The high abstinence rate was maintained for at least six months. Although the Social Interaction program was not statistically superior in terms of abstinent rates at the one year assessment relative to the Lung Association group, those individuals in the Social Interaction group were smoking significantly fewer cigarettes than those in the Lung Association group at one year post quit. This would suggest that the coping skills introduced in the program were successful in assisting those who continued to smoke to moderate their cigarette intake. The benefit of smoking less has significant impact both on the individual and the community as a whole. The effectiveness of the Interpersonal Coping Skills Program as a mechanism to achieve high initial rates of abstinence suggests that future treatment programs should be skilled based in orientation. That is, participants should be educated as to why they smoke and how to modify their existing coping skills.
References


Paxton, R. (1980). The effects of a deposit contract as a component in a behavioral program for stopping smoking. *Behavior Research and Therapy*. 18, 45-


and Clinical Psychology. 38, 105-111.


Appendix A

Categories for Classification of Relapse Episodes

1. Intrapersonal-Environmental Determinants. Includes all determinants that are primarily associated with intrapersonal factors (within the individual), and/or reactions to non-personal environmental events. Includes reactions to interpersonal events in the relatively distant past (i.e., in which the interaction is no longer of significant impact).

A). Coping with Negative Emotional States. Determinant involves coping with a negative (unpleasant) emotional state, mood, or feeling.
1). Coping with Frustration and/or Anger. Determinant involves an experience of frustration (reaction to a blocked goal-directed activity) and/or anger (hostility, aggression) in terms of the self or some nonpersonal environmental event. Includes all reference to guilt, and responses to demands ("hassles") from environmental sources or from within the self that are likely to produce feelings of anger.
2). Coping with Other Negative Emotional States: Determinant involves coping with emotional states other than frustration/anger that are unpleasant or aversive including feelings of fear, anxiety, tension, depression, loneliness, sadness, boredom, worry, apprehension, grief, loss, and other similar dysphoric states. Includes reactions to evaluation stress (examinations, promotions, public speaking, etc.), employment and financial difficulties, and personal misfortune or accident.

B). Coping with Negative Physical-Physiological States. Determinant involves coping with unpleasant or painful physical or physiological reactions.
1). Coping with Physical States Associated with Prior Substance Use. Coping with physical states that are specifically associated with prior use of drug or substance, such as "withdrawal agony" or "physical craving" associated with withdrawal. (Note: References to "craving" in the absence of withdrawal are classified under Section E below).
2). Coping with Other Negative Physical States. Coping with pain, illness, injury, fatigue, and specific disorders (e.g., headache, menstrual cramps, etc.) that are not associated with prior substance use.

C). Enhancement of Positive Emotional States. Use of substance to increase feelings of pleasure, joy, freedom, celebration, and so on (e.g., when traveling, or on vacation). Includes use of substance for primarily positive effects - to "get high" or to experience the enhancing effects of a drug.

D). Testing Personal Control. Use of substance to "test" one's ability to engage in controlled or moderate use; to "just try it once" to see what happens; or in cases in which the individual is testing the effects of treatment or a commitment to abstinence (including tests of "willpower").

E). Giving in to Temptations or Urges. Substance use in response to "internal" urges, temptations, or other promptings. Includes references to "craving" or intense subjective desire, in the absence of interpersonal factors. (Note: References to "craving" which are associated with prior drug use or withdrawal are classified under Section B-1 above).
1). In the Presence of Substance Cues. Use occurs in the presence of cues associated with substance of use (e.g., running across a hidden bottle or pack of cigarettes, passing by a bar, seeing an ad for cigarettes). (Note: Where other individuals are using the substance, refer to Category 11-B below).
2). In the Absence of Substance Cues. Here, the urge or temptation comes "out of the blue" and is followed by the individual's attempt to procure the substance.

2. Interpersonal Determinants. Includes determinants that are primarily associated with interpersonal factors: reference is made to the presence or influence of other individuals as part of the precipitating event. Implies the influence of present or recent interaction with another person or persons, who exert some influence on the user (reactions to events that occurred in the relatively distant past are classified in Category 1). Just being in the presence of others at the time of the relapse does not justify an interpersonal classification, unless some mention is made or implied that these people had some influence or were somehow involved in the event.
Appendix A continued

A). Coping with Interpersonal Conflict. Coping with a current or relatively recent conflict associated with employer-employee relations.
   1). Coping with Frustration and/or Anger. Determinant involves frustration (reaction to blocked goal-directed activity), and/or anger (hostility, aggression) stemming from an interpersonal source. Emphasis is on a situation in which the person feels frustrated or angry with someone and includes involvement in arguments, disagreements, fights, jealousy, discord, hassles, guilt, and so on.
   2). Coping with Other Interpersonal Conflict. Determinant involves coping with conflicts other than frustration and anger stemming from an interpersonal source. Feelings such as anxiety, fear, tension, worry, concern, apprehension, etc., which are associated with interpersonal conflict, are examples. Evaluation stress in which another person or group is specifically mentioned would be included.

B). Social Pressure. Determinant involves responding to the influence of another individual or group of individuals who exert pressure (either direct or indirect) on the individual to use the substance.
   1). Direct Social Pressure. There is direct contact (usually with verbal interaction) with another person or group who puts pressure on the user or who supplies the substance to the user (e.g., being offered a drug by someone, or being urged to use a drug by someone else). Distinguish from situations in which the substance is obtained from someone else at the request of the user (who has already decided to use).
   2). Indirect Social Pressure. Responding to the observation of another person or group that is using the substance or serves as a model of substance use for the user. If the model puts any direct pressure on the individual to use the substance, then the lapse should be categorized under 11-B1, above.

C). Enhancement of Positive Emotional States. Use of substance in a primarily interpersonal situation to increase feelings of pleasure, celebration, sexual excitement, freedom, and the like. Distinguish from situations in which the other person(s) is using the substance prior to the individual's first use (classify these under Section II-B above).

Categories for Classification of Relapse Episodes (from Marlatt and Gordon, 1985, pp. 80-81)
Appendix B

Social Interactional Coping Skills Program for Smoking Cessation and Relapse Prevention

Week One:

Session One:
- An introduction of the clients to each other
- A brief overview of the cessation program
- A discussion of the health hazards of smoking
- Explanation of the use of the diary
- Introduction to social stimuli
homework: smoking diary

Session Two:
- Discuss homework
- Review concept of social stimuli
- Discuss differences between observations, inference, and evaluation
- Review language usage pertaining to emotions and inferences
- Identification of aversive social stimuli
homework: smoking diary with the identification of aversive social stimuli possibly related to smoking behaviour

Week Two:

Session Three:
- Review social interactional program to date
- Review concept of aversive social stimuli
- Discuss smoking journal in detail, i.e. attempting to identify the aversive social stimuli in the situations related to smoking behaviour
- Examine socialization experience
homework: smoking diary

Session Four:
- Review diary and identify aversive social stimuli and troublesome social stimuli
- Introduce the concept of emotions and actions and self-produced controlling stimuli and response tendencies.
- Review diary identifying external and internal troublesome stimuli
homework: smoking diary

Week Three:

Session Five:
- Review program to date with special concentration on emotions, actions, and self-produced controlling stimuli and response tendencies
- Begin the introduction of coping skills for modifying reactions to aversive stimuli whether external or internal
- Review diary and identify the external and internal stimuli which cue smoking behaviour
- Discuss and identify high risk situations

**Homework:** smoking diary and practicing of coping skills

### Session Six:
- Review coping skills methods
- Review diary and identify personal high risk situations
- Demonstrate the desensitization procedure with volunteer
- Organize group into pairs to practice coping techniques
- Coping with negative mood states
- What is relapse prevention
- Prepare to cut down smoking by 1/3 (have clients review diaries in order to decide which situations would be easiest to eliminate the smoking behaviour).

**Homework:** continue smoking diary, practice coping skills and lengthen the exposure time of the client to the controlling stimulus and prepare client to reduce cigarette consumption on the day of session seven.

### Week Four:

#### Session Seven:
- Have clients monitor their diary and cigarette count for the day
- Discuss the possible withdrawal symptoms which may be experienced with cutting down and the eventual cessation of smoking
- Introduce the coping skill of rational analysis
- Use role play to demonstrate the irrational nature of appraisals
- Coping with physical cues
- How to cope with a lapse

**Homework:** continue smoking diary and practice coping skills; have clients start charting their cigarette consumption.

#### Session Eight:
- Review program to date
- Discuss difficulties in cutting down cigarette consumption
- Have clients re-identify high risk situations and offer possible coping skills which may reduce the potency of the situation
- Divide group into pairs to practice coping skills with respect to personal high risk situations (i.e. those situations in which the aversive stimulus leads the individual to smoke)
- Prepare to cut down smoking consumption by a further 25%
- Introduce behaviour variation
- Coping with conflict
- Helping a lapse from turning into relapse

**Homework:** continue smoking diary and write possible behaviour variations for those smoking situations; continue charting of cigarette smoking
Appendix B continued

Week Five:

Session Nine:
- Discuss how clients are coping with the reduction in smoking
- Organize the telephone help list, those clients who are willing to offer their names, and phone numbers so that those co-members who are in need of a pep talk may call for assistance.
- Review the three coping skills used to date and link to relapse prevention
- Role play with volunteer demonstrating the integration of the coping skills
- Divide group into pairs for further role playing
- Discuss Q-Day, which is on session eleven, the quit day
- Help prepare for Q-Day

homework: continue smoking diary and practice coping skills and continue charting smoking behaviour

Session Ten:
- Discuss difficulties to date
- Review diaries
- Further role playing
- Discuss client expectations of Q-Day and the physical benefits of quitting smoking
- What do I do if I lapse
- Reviewing the categories of potential relapse

homework: continue smoking diary and practicing coping skills and continue charting smoking behaviour

Week Six:

Session Eleven:
- Q-Day
- Review the benefits of quitting smoking
- Hand out the POMS and the withdrawal symptom questionnaires
- Have each individual volunteer a high risk situation and the coping skill they intend to utilize
- Coping with a lapse

homework: daily responding to the withdrawal symptom questionnaire, POMS, urge questionnaire, and record daily cigarette consumption

Session Twelve:
- Review program to date
- Discuss problems with effective coping
- Further role playing
- Prepare for weekly meetings, encourage use of telephone for pep talks

homework: daily responding to the withdrawal symptom questionnaire, POMS, urge questionnaire, and record daily cigarette consumption
Week Seven:
Session Thirteen:
- Discuss personal difficulties
- Identify personal high risk situations and why you may lapse
- Role playing
homework: continue with withdrawal symptom questionnaire, urge questionnaire, POMS, and record daily cigarette consumption.

Week Eight:
Session Fourteen:
- Discuss personal difficulties
- Review the identification of troublesome stimuli
- Discuss the personal successful coping situations and relation to relapse prevention
- Brief role play
homework: continue with withdrawal symptom questionnaire, urge questionnaire, POMS, and record daily cigarette consumption.

Week Nine:
Session Fifteen:
- Discuss personal difficulties
- Identify and discuss personal high risk situations and offer effective coping skills for cessation maintenance and relapse prevention
- Brief role play
homework: continue with withdrawal symptom questionnaires, urge questionnaires, POMS, and record daily cigarette consumption.

Week Ten:
Session Sixteen:
- Fill out the last of the questionnaires
- Record average daily cigarette smoked since Q-Day
- Review successful coping skills, linked with relapse prevention
- Inform clients of the three, four, five, six month and 1 year follow-ups
- Review relapse prevention strategies
**Appendix C**  
**Lung Association Countdown Calendar**

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
<th>Day 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Session 1&lt;br&gt;Date: Monitor</td>
<td>Butt Jar Tests</td>
<td>Session 2&lt;br&gt;Date: Eat Breakfast</td>
<td>Deep Breathe</td>
<td>Drink Water</td>
<td>Journal</td>
<td>Delay</td>
</tr>
<tr>
<td>Day 3</td>
<td>Day 9</td>
<td>Day 10</td>
<td>Day 11</td>
<td>Day 12</td>
<td>Day 13</td>
<td>Day 14</td>
</tr>
<tr>
<td>Walk</td>
<td>Tape</td>
<td>Session 3&lt;br&gt;Date: Plan Strategy</td>
<td>New Clean Zone</td>
<td>Walk</td>
<td>Coping Technique</td>
<td>Slim Snacks</td>
</tr>
<tr>
<td>Non Smoking Day 2</td>
<td>Non Smoking Day 3</td>
<td>Non Smoking Day 4</td>
<td>Non Smoking Day 5</td>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
</tr>
<tr>
<td>Non Smoking Day 2</td>
<td>Non Smoking Day 3</td>
<td>Non Smoking Day 4</td>
<td>Non Smoking Day 5</td>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
</tr>
<tr>
<td>Non Smoking Day 3</td>
<td>Non Smoking Day 4</td>
<td>Non Smoking Day 5</td>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
</tr>
<tr>
<td>Non Smoking Day 3</td>
<td>Non Smoking Day 4</td>
<td>Non Smoking Day 5</td>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
</tr>
<tr>
<td>Non Smoking Day 4</td>
<td>Non Smoking Day 5</td>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
</tr>
<tr>
<td>Non Smoking Day 4</td>
<td>Non Smoking Day 5</td>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
</tr>
<tr>
<td>Non Smoking Day 5</td>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
</tr>
<tr>
<td>Non Smoking Day 5</td>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
</tr>
<tr>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
</tr>
<tr>
<td>Non Smoking Day 6</td>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
</tr>
<tr>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
</tr>
<tr>
<td>Non Smoking Day 7</td>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
</tr>
<tr>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
</tr>
<tr>
<td>Non Smoking Day 8</td>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
</tr>
<tr>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
</tr>
<tr>
<td>Non Smoking Day 9</td>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
</tr>
<tr>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
</tr>
<tr>
<td>Non Smoking Day 10</td>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
</tr>
<tr>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
<td>Non Smoking Day 17</td>
</tr>
<tr>
<td>Non Smoking Day 11</td>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
<td>Non Smoking Day 17</td>
</tr>
<tr>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
<td>Non Smoking Day 17</td>
<td>Non Smoking Day 18</td>
</tr>
<tr>
<td>Non Smoking Day 12</td>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
<td>Non Smoking Day 17</td>
<td>Non Smoking Day 18</td>
</tr>
<tr>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
<td>Non Smoking Day 17</td>
<td>Non Smoking Day 18</td>
<td>Non Smoking Day 19</td>
</tr>
<tr>
<td>Non Smoking Day 13</td>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
<td>Non Smoking Day 17</td>
<td>Non Smoking Day 18</td>
<td>Non Smoking Day 19</td>
</tr>
<tr>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
<td>Non Smoking Day 17</td>
<td>Non Smoking Day 18</td>
<td>Non Smoking Day 19</td>
<td>Non Smoking Day 20</td>
</tr>
<tr>
<td>Non Smoking Day 14</td>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
<td>Non Smoking Day 17</td>
<td>Non Smoking Day 18</td>
<td>Non Smoking Day 19</td>
<td>Non Smoking Day 20</td>
</tr>
<tr>
<td>Non Smoking Day 15</td>
<td>Non Smoking Day 16</td>
<td>Non Smoking Day 17</td>
<td>Non Smoking Day 18</td>
<td>Non Smoking Day 19</td>
<td>Non Smoking Day 20</td>
<td>Non Smoking Day 21+</td>
</tr>
</tbody>
</table>

Non Smoking Day 16 Alternatives My Goal Be Aware Can The Beginning
Appendix D

Personal Consent Form

I (print name in full) ___________________________ agree to participate in the smoking cessation study offered to me by Mary-Beth Minthorn-Biggs. The nature of the study has been explained to my satisfaction, and I understand that the study can not guarantee that I will quit smoking. All questionnaires have been explained to me and I have been informed that all information obtained is confidential. I am free to withdraw from the study at any time I should so desire.

_________________________  ___________________________
Date                        Signature
Appendix F

Smoking Confidence Questionnaire

Please indicate, using the scale of 1 to 10 (a score of 1 indicates very unlikely and a score of 10 indicates very likely) the probability that you would be able to resist the urge to smoke in the following situations:

1: very unlikely.................................................................10: very likely

1. When you feel impatient.
2. When you are waiting for someone or something.
3. When you feel restless.
4. When you want to take a break from work or some other activity.
5. When you want something to do with your hands.
6. When you want to relax.
7. When you want to sit back and enjoy a cigarette.
8. When you want to concentrate.
9. When you are overly excited.
10. When you see others smoking.
11. When you have finished a meal or a snack.
12. When you light up a cigarette to go along with some activity you are doing (for example, fixing a bicycle, writing a letter, doing housework).
13. When you feel frustrated.
14. When you are worried.
15. When you feel upset.
16. When you feel tense.
17. When you feel nervous.
18. When you feel depressed.
19. When you feel annoyed.
20. When you feel oversensitive.
21. When you feel angry with yourself.
22. When you feel anxious.
23. When you want something in your mouth.
24. When you want to cheer up.
25. When you feel you need more energy.
26. When you want to keep slim.
27. When you want to avoid eating sweets.
28. When you feel tired.
29. When you want to reward yourself for something you’ve done or tell yourself that you can have a cigarette if you complete some task.
30. When you want to have time to think in a conversation.
31. When you are resting.
32. When you want to keep yourself busy.
33. When you are trying to pass the time.
34. When you feel bored.
35. When you simply become aware of the fact that you are not smoking.
36. When someone offers you a cigarette.
37. When you are drinking an alcoholic beverage.
38. When you are drinking coffee or tea.
39. When you feel uncomfortable.
40. When you feel embarrassed.
41. When you are in a situation in which you feel smoking is a part of your self-image.
42. When you want to feel more mature and sophisticated.
43. When you want to feel more attractive.
Appendix G
Profile of Mood States
Below is a list of words that describe feelings people have. Please read each one carefully. Then fill in ONE circle under the answer to the right which best describes HOW YOU HAVE BEEN FEELING DURING THE PAST WEEK INCLUDING TODAY.

The numbers refer to these phrases.
0 = Not at all
1 = A little
2 = Moderately
3 = Quite a bit
4 = Extremely

<table>
<thead>
<tr>
<th>No.</th>
<th>Word</th>
<th>Col</th>
<th>O.P.</th>
<th>NOT AT ALL</th>
<th>A LITTLE</th>
<th>MODERATELY</th>
<th>QUITE A BIT</th>
<th>EXTREMELY</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Friendly</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>2</td>
<td>Tense</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>3</td>
<td>Angry</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>4</td>
<td>Worn out</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>5</td>
<td>Unhappy</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>6</td>
<td>Clear-headed</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>7</td>
<td>Lively</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>8</td>
<td>Confused</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>9</td>
<td>Sorry for things done</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>10</td>
<td>Shaky</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>11</td>
<td>Listless</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>12</td>
<td>Peved</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>13</td>
<td>Considerate</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>14</td>
<td>Sad</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>15</td>
<td>Active</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>16</td>
<td>On edge</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>17</td>
<td>Grouchy</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>18</td>
<td>Blue</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>19</td>
<td>Energetic</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>20</td>
<td>Panicky</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>21</td>
<td>Hopeless</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>22</td>
<td>Relaxed</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>23</td>
<td>Unworthy</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>24</td>
<td>Spiteful</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>25</td>
<td>Sympathetic</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>26</td>
<td>Uneasy</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>27</td>
<td>Restless</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>28</td>
<td>Unable to concentrate</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>29</td>
<td>Fatigued</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>30</td>
<td>Helpful</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>31</td>
<td>Annoyed</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>32</td>
<td>Discouraged</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>33</td>
<td>Resentful</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>34</td>
<td>Nervous</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>35</td>
<td>Lonely</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>36</td>
<td>Miserable</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>37</td>
<td>Muddled</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>38</td>
<td>Cheerful</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>39</td>
<td>Bitter</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>40</td>
<td>Exhausted</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>41</td>
<td>Anxious</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>42</td>
<td>Ready to fight</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>43</td>
<td>Good natured</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
<tr>
<td>44</td>
<td>Gloomy</td>
<td>O</td>
<td>O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O O O</td>
</tr>
</tbody>
</table>

*MAKE SURE YOU HAVE ANSWERED EVERY ITEM.*
Appendix H
Anger Expression Scale
Self-Rating Questionnaire

STAXI Item Booklet (Form HS)

Name ____________________________ Sex _______ Age _______ Date __________

Education ____________________ Occupation ___________________________ Marital Status _______

Instructions

In addition to this Item Booklet you should have a STAXI Rating Sheet. Before beginning, enter your name, sex, age, the date, your education and occupation, and your marital status in the spaces provided on this booklet and at the top of the Rating Sheet.

This booklet is divided into three Parts. Each Part contains a number of statements that people use to describe their feelings and behavior. Please note that each Part has different directions. Carefully read the directions for each Part before recording your responses on the Rating Sheet.

There are no right or wrong answers. In responding to each statement, give the answer that describes you best. DO NOT ERASE! If you need to change your answer, make an “X” through the incorrect response and then fill in the correct one.

Examples

1.  ①  ☒  ④
2.  ①  ③  ④
Part 1 Directions

A number of statements that people use to describe themselves are given below. Read each statement and then fill in the circle with the number which indicates how you feel right now. Remember that there are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to best describe your present feelings.

<table>
<thead>
<tr>
<th>Fill in 1 for Not at all</th>
<th>Fill in 3 for Moderately so</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fill in 2 for Somewhat</td>
<td>Fill in 4 for Very much so</td>
</tr>
</tbody>
</table>

How I Feel Right Now

1. I am furious.
2. I feel irritated.
3. I feel angry.
4. I feel like yelling at somebody.
5. I feel like breaking things.
6. I am mad.
7. I feel like banging on the table.
8. I feel like hitting someone.
9. I am burned up.
10. I feel like swearing.

Part 2 Directions

A number of statements that people use to describe themselves are given below. Read each statement and then fill in the circle with the number which indicates how you generally feel. Remember that there are no right or wrong answers. Do not spend too much time on any one statement, but give the answer which seems to best describe how you generally feel.

<table>
<thead>
<tr>
<th>Fill in 1 for Almost never</th>
<th>Fill in 3 for Often</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fill in 2 for Sometimes</td>
<td>Fill in 4 for Almost always</td>
</tr>
</tbody>
</table>

How I Generally Feel

11. I am quick tempered.
12. I have a fiery temper.
13. I am a hotheaded person.
14. I get angry when I'm slowed down by others' mistakes.
15. I feel annoyed when I am not given recognition for doing good work.
16. I fly off the handle.
17. When I get mad, I say nasty things.
18. It makes me furious when I am criticized in front of others.
19. When I get frustrated, I feel like hitting someone.
20. I feel infuriated when I do a good job and get a poor evaluation.

Continued
Part 3 Directions

Everyone feels angry or furious from time to time, but people differ in the ways that they react when they are angry. A number of statements are listed below which people use to describe their reactions when they feel angry or furious. Read each statement and then fill in the circle with the number which indicates how often you generally react or behave in the manner described when you are feeling angry or furious. Remember that there are no right or wrong answers. Do not spend too much time on any one statement.

<table>
<thead>
<tr>
<th>Fill in 1 for Almost never</th>
<th>Fill in 3 for Often</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fill in 2 for Sometimes</td>
<td>Fill in 9 for Almost always</td>
</tr>
</tbody>
</table>

When Angry or Furious...

21. I control my temper.
22. I express my anger.
23. I keep things in.
24. I am patient with others.
25. I pout or sulk.
26. I withdraw from people.
27. I make sarcastic remarks to others.
28. I keep my cool.
29. I do things like slam doors.
30. I boil inside, but I don't show it.
31. I control my behavior.
32. I argue with others.
33. I tend to harbor grudges that I don't tell anyone about.
34. I strike out at whatever infuriates me.
35. I can stop myself from losing my temper.
36. I am secretly quite critical of others.
37. I am angrier than I am willing to admit.
38. I calm down faster than most other people.
39. I say nasty things.
40. I try to be tolerant and understanding.
41. I'm irritated a great deal more than people are aware of.
42. I lose my temper.
43. If someone annoys me, I'm apt to tell him or her how I feel.
44. I control my angry feelings.
Appendix I
Self-report Symptom Checklist

Day Number

Please rate, using a scale of 0 (not present) to 5 (very strong), the degree to which you experienced any of the following:
0 = not present
5 = very strong

Circle the most appropriate rating

<table>
<thead>
<tr>
<th></th>
<th>Weight gain</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Irritability</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>2.</td>
<td>Anxiety</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>3.</td>
<td>Sleeplessness</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>4.</td>
<td>Poor concentration</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>5.</td>
<td>Restlessness</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>6.</td>
<td>Anger</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>7.</td>
<td>Nausea</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>8.</td>
<td>Depression</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>9.</td>
<td>Urge to have a cigarette</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>
Appendix J

Personal Information

Name__________________________

Age__________________________

Sex__________________________

Marital Status__________________

Education_____________________ 

Age when started smoking________

Number of years smoking________

Number of cigarettes presently smoking per day____________

Number of times you have attempted to quit smoking________

List the different methods used to assist you quit smoking

______________________________________________________________________________

______________________________________________________________________________

______________________________________________________________________________

How long was the longest period of time you remained cigarette free

________(days or months or years)

Do any other members of your family smoke?____________

If yes, please indicate which family members

______________________________________________________________________________

Are any of your family members attempting to quit smoking at present?____

If yes, which members____________________________________
Appendix K

The following questions deal with the circumstances in which you were recently tempted to smoke or did smoke. Please answer each one.

Where were you?
____ home  ____ work  ____ someone else's home  ____ restaurant or bar  ____ vehicle  ____ other

What were you doing?
____ working  ____ eating or drinking  ____ socializing  ____ relaxing  ____ other

Had you been consuming coffee?  ____ yes  ____ no

Had you been consuming alcohol?  ____ yes  ____ no

Were other people with you?  ____ yes  ____ no

How many of them were smoking? ____

Were cigarettes available?  ____ yes  ____ no

From what source? ______________________

If you smoked, how did you get the cigarette(s)?
____ I asked someone for it
____ It was offered to me
____ I bought it
____ I found it
____ Other

How were you feeling:
____ Happy  ____ Relaxed  ____ Neutral  ____ Angry  ____ Anxious  ____ Depressed

What single thing contributed most to your being tempted to smoke?
____ How you were feeling
____ Seeing cigarettes or people smoking
____ Habit of smoking along with food, coffee, or relaxation
____ Symptoms you were having
____ Other

How did you feel after the episode was over? (Check all that apply)
____ Relieved  ____ Hopeful  ____ Successful  ____ Worried  ____ Disappointed
____ Guilty  ____ Like a failure  ____ Hopeless  ____ Other

Did you anticipate that this situation would tempt you to smoke:
____ Yes  ____ No

How often do you encounter situations like this one?
____ More than once per day  ____ About once per day
____ A few times per week  ____ About once per week
____ A few times per month  ____ Less than twice a month

When you're in situations like this, are you always tempted to smoke?
____ Always  ____ Usually
____ Rarely  ____ Never
Appendix L

Reason for Relapse

Date of relapse

Day post quit

Choose the primary reason why you smoked (only check one).

____ Peer pressure
____ Consuming alcohol
____ Celebration
____ Depressed mood
____ Anxious mood
____ Stressed
____ Watching others
____ Symptoms
____ Argument with significant other (partner, employer, co-worker)
____ Craving or urge
____ Post meal
____ Other (please describe)
Appendix M
Comparison between Quitters and Non-Quitters on Pre-Treatment Variables (n=59)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Quitter (n=19)</th>
<th>Non-Quitter (n=40)</th>
<th>t(57)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>41.94</td>
<td>41.00</td>
<td>.31</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(12.61)</td>
<td>(10.31)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age Started</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>16.63</td>
<td>16.60</td>
<td>.04</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(3.28)</td>
<td>(2.83)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Years Smoked</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>24.89</td>
<td>24.15</td>
<td>.23</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(13.42)</td>
<td>(10.66)</td>
<td></td>
<td></td>
</tr>
<tr>
<td># Cigs Smoked Per Day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>23.21</td>
<td>25.60</td>
<td>-.71</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(9.71)</td>
<td>(13.03)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Times Quit</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>3.78</td>
<td>3.32</td>
<td>.71</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(2.25)</td>
<td>(2.36)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Max. Length of Time Quit (days)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>239.9</td>
<td>120.67</td>
<td>1.89</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(322.33)</td>
<td>(164.70)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking Confidence</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>199.21</td>
<td>215.27</td>
<td>-.95</td>
<td>n.s.</td>
</tr>
<tr>
<td>SD</td>
<td>(64.08)</td>
<td>(58.62)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Appendix M Continued
<table>
<thead>
<tr>
<th></th>
<th>M</th>
<th>SD</th>
<th>AngerOut</th>
<th>M</th>
<th>SD</th>
<th>AngerIn</th>
<th>M</th>
<th>SD</th>
<th>AngerCon</th>
<th>M</th>
<th>SD</th>
<th>AngerExpression</th>
</tr>
</thead>
<tbody>
<tr>
<td>POMS (Pre)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>24.73</td>
<td>26.71</td>
<td>-1.20</td>
<td>14.36</td>
<td>4.54</td>
<td>-0.75</td>
<td>18.05</td>
<td>4.57</td>
<td>0.83</td>
<td>23.47</td>
<td>5.56</td>
<td>-0.01</td>
</tr>
<tr>
<td></td>
<td>34.32</td>
<td>29.52</td>
<td>n.s.</td>
<td>15.20</td>
<td>5.64</td>
<td>n.s.</td>
<td>16.82</td>
<td>564</td>
<td>n.s.</td>
<td>23.50</td>
<td>9.75</td>
<td>n.s.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>