Smoking, Secondhand Smoke, and Quitting: A Cessation Program

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Abstract:

This study uses an education program designed to teach young women about the health effects of smoking, secondhand smoke, and the benefits of quitting. We used a PowerPoint presentation and a short questionnaire, administered three times—prior to the presentation, directly after the presentation, and two weeks after the presentation—to measure the effectiveness of the program. Scores on the questionnaire increased greatly directly after the presentation, but decreased on the two-week follow-up, making the program slightly effective. Methods are needed to increase retention. Limitations to this study included difficulty recruiting, leading to a small number of participants. Problems with the Institutional Review Board (IRB) delayed beginning and contributed to the difficulty of recruiting for this study. Only one participant did not complete the two-week follow-up.
Introduction

The harmful health effects of smoking have been known for many years. The first Surgeon General’s Report was published in 1964 based on 7,000 studies available.\(^1\) In this report, the first causal relationship between smoking and health was described: smoking causes lung cancer.\(^2\) This spawned the already great research on smoking and health and gained America’s attention. Since then—with the help of several laws, politicians, private organizations, government organizations, and individuals—the effects of smoking have been decreased.\(^3\) However, more must be done as smoking continues to be responsible for thousands of chronic illnesses and deaths each year.

In 1964, it was established that smoking causes lung cancer. Today, the most serious side effect of smoking continues to be lung cancer, which is the second leading cause of death in the United States and the first among cancers.\(^4\) Ninety percent of lung cancers in women are caused by smoking.\(^5\) More women die from lung cancer than breast cancer.\(^6\) In addition to lung cancer, smoking is also a cause of other cancers including cancer of the mouth, voice box, esophagus, pancreas, stomach, kidneys, bladder, and cervix.\(^7\) Smoking increases the risk of heart disease including stroke, peripheral vascular disease, and abdominal aortic aneurysm.\(^8\) In the United States, coronary heart disease is the leading cause of death among both men and women, whereas stroke is third.\(^9\) Smoking increases the risk of lung disease by ten times, including chronic obstructive pulmonary disease (COPD), emphysema, and chronic bronchitis.\(^10\)

Women are more susceptible to the effects of cigarette smoke than men. Women who smoke have an increased risk of premature death, lower bone density, early menopause, and an increased risk of cancers of the liver, colon, and cervix.\(^11\) In pregnant women, there is an increased risk of pre-term delivery and infant death. Infants of women who smoke are more likely to be stillborn, have a low birth weight, or die from sudden infant death syndrome.
Nicotine in cigarettes reduces the flow of blood through the umbilical cord and reduces the amount of oxygen and nutrients the baby receives. There is also an increased risk of being infertile or having delayed conception for women smokers. Other health effects caused by smoking include periodontitis (gum disease), peptic ulcers, and a lowered immune system that results in frequent hospitalizations, lower rates of survival after surgery, an increased risk of infection, and slower healing. Smokers have higher medical costs than nonsmokers.

Secondhand smoke (SHS), also known as environmental tobacco smoke (ETS) or passive smoking, has harmful health effects on nonsmokers. A mixture of mainstream smoke (smoke breathed out by the smoker) and sidestream smoke (smoke emitted from the burning end of the cigarette), SHS contains over 250 chemicals, 50 of which are known carcinogens. When nonsmokers are exposed to SHS, they are at an increased risk for lung cancer and heart disease. Children exposed to SHS have higher rates of asthma and respiratory illnesses and require hospitalization more often. SHS slows the growth of children’s lungs. Infants exposed to SHS before birth may have reduced birth weights; infants exposed after birth have an increased risk of SIDS.

These harmful effects can be attributed to the dozens of toxic chemicals, poisons, and addictive agents within cigarettes. These chemicals include carbon monoxide, lead, arsenic, polonium, ammonia, and formaldehyde. Smoking has negative health consequences for smokers, and for the unborn children of mothers who smoke and are exposed to secondhand smoke. My research aims to educate young women on campus about the health effects of smoking, secondhand smoke, and quitting through an education program using a PowerPoint presentation.
This literature review is designed to examine prior research on tobacco exposure and infant health. Active smoking is defined as cigarette smoking by the mother. Passive smoking is defined as exposure to SHS, or ETS. Passive smoking that affects an unborn child is referred to as prenatal ETS exposure. Many studies have addressed active smoking and its effects on birth weight, pre-term delivery, SIDS, and other adverse outcomes. These studies have produced similar results and have led to the development of several causal relationships; however, research on passive smoking has been less cohesive and has produced conflicting results. First, I will examine active smoking and adverse health outcomes. Second, I will examine passive smoking and effects related to birth weight, low birth weight (LBW), small-for-gestational-age (SGA), intrauterine growth retardation (IUGR), pre-term delivery, and other effects. Third, I will examine effects of postnatal ETS exposure. Fourth, I will examine the critical time of exposure. Finally, I will examine different methodologies used by the studies.

More research on passive smoking is needed to determine the health effects on infants. The magnitude and the significance of these effects must also be determined. My research purpose is to create a cessation program aimed to educate young female smokers at the University of North Texas (UNT) about the harmful effects of smoking, and to aid them in quitting smoking.

Smoking and Infant Health

Research on the effects of smoking during pregnancy began in 1957, when it was first observed that infants of smokers had lower birth weights than those of nonsmokers. Since then, research has increased greatly, and many causal relationships have been established between smoking and infant health. Maternal smoking during pregnancy has been shown to cause an increased risk of SIDS, decreased head circumference, and delayed cognitive functioning and
language development.\textsuperscript{22} Hanke, Sobala, and Kalinka, as well as Eskenazi, Prehn, and Christianson, showed a decreased birth weight in infants of smokers with a dose-response relationship: the more cigarettes women smoked, the greater the decrease in birth weight.\textsuperscript{23} Difranza, Aligne, and Weitzman also attributed smoking to a decrease in birth weight.\textsuperscript{24} Eskenazi et al. also found that smoking increased the risk of pre-term delivery and an increased risk of LBW.\textsuperscript{25} Hanke et al. observed a decreased bi-parietal diameter (BPD) at 20 to 24 weeks of gestation in infants of smokers.\textsuperscript{26}

\textit{ETS and Infant Health}

Given the effects of maternal smoking and nonsmokers exposed to ETS, questions have arisen about the effects of maternal exposure to ETS during pregnancy. Studies on maternal ETS exposure have produced varying results, from highly significant to no effects.

\textit{ETS and birth weight.} Zhang and Ratcliffe and Eskenazi et al. found statistically significant decreases in birth weights of infants among mothers exposed to ETS.\textsuperscript{27} However, Perera et al. and Hanke et al. showed significant decreases in birth weight of 233 g and 100 g, respectively.\textsuperscript{28} A meta-analysis by Misra and Nguyen showed 10 studies with decreases in birth weight ranging from 25 g to 125 g, but only 4 had significant results.\textsuperscript{29} Decreases in birth weight that are not statistically significant may result in the population’s birth weight decreasing, thereby increasing the risk of LBW.\textsuperscript{30}

\textit{ETS and LBW.} Misra et al. examined six studies related to ETS and LBW.\textsuperscript{31} Five of the six showed significant increases in the risk of LBW among infants of ETS-exposed mothers.\textsuperscript{32} Zhang et al. showed a nonsignificant increased risk of LBW.\textsuperscript{33}

\textit{ETS and IUGR.} Misra et al. compared eight studies that investigated IUGR associated with ETS exposure. Three studies showed a significant increased risk of IUGR with ETS
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exposure.34

ETS and SGA. SGA was examined by Zhang et al., with a nonsignificant increase in risk found.35

Other Effects. A decrease in head circumference and length associated with ETS exposure was observed by Perera et al.36 They also observed that when exposure to ETS is combined with exposure to the pollutant benzo(a)pyrene (BaP), there were greater adverse effects on birth weight and head circumference than with exposure to ETS alone.37 Hanke et al. found that ETS exposure contributed to an insignificant decrease in abdominal circumference and femur length, and a significant decrease in BPD.38

ETS and pre-term delivery. No studies I reviewed examined the risk of premature delivery related to ETS exposure.

Effects of Postnatal ETS

A literature review by DiFranza et al. noted that infants of smoking mothers or ETS-exposed mothers, especially those whose husbands smoked, are usually exposed to ETS themselves after birth.39 Postnatal ETS exposure has been associated with an increased risk of SIDS, frequent respiratory illnesses requiring hospitalization, an increased risk of asthma, and increased ear infections.40

Critical Time of Exposure

DiFranza et al., Misra et al., Hanke et al., and Eskenazi et al. examined critical exposure time. Some believe that ETS exposure may have a greater effect on fetal growth in later pregnancy, with Misra et al. citing the second half of pregnancy and Eskenazi et al. citing the third trimester.41 Hanke et al. believes that critical exposure time may occur in early pregnancy.42 DiFranza et al. states that more evidence is needed to determine if there is a critical time of
Methods Used in Previous Studies

Results of studies vary greatly due to methodology. Some studies use biomarkers, whereas others use self-reporting. There is even variation within these methods due to the different biomarkers used and different ways of assessing self-reporting, or using a combination of the two. Zhang et al. used self-reporting alone, and Eskenazi et al., Hanke et al., and Perera et al. used both self-reporting and a biomarker. The meta-analysis by Misra et al. showed that serum cotinine, serum nicotine, or hair nicotine concentrations can be used as biomarkers, with hair being the best.

Misra et al. states that a biomarker is more accurate than self-reporting for assessing ETS exposure because it leads to less misclassification and accounts for multiple sources and other factors, such as ventilation and room size. However, they also observe that a biomarker is not useful when trying to isolate exposure from one source, such as the home or the workplace; in such cases, a self-report is necessary. Perera et al. concluded that self-reporting may be more accurate than a biomarker because biomarkers only account for exposure within the last few days and do not measure it throughout pregnancy.

Self-reporting produces variation in results due to use of different questionnaires and measurements of exposure. Some studies may consider multiple sources of ETS and some may not, like those that use paternal smoking status only. Variation in results may also be caused by controlling for confounding factors. Misra et al. states that there may be over-controlling in some studies and under-controlling in others.

Summary

Previous research on the effects of exposure to SHS during pregnancy has produced
inconsistent results. Some studies have found significant decreases in birth weight, whereas others have found small reductions. Some studies have found increased risk of IUGR, LBW, and SGA, but this research is not extensive and is limited to few studies. Research is conflicted on a critical time of exposure, with some studies showing the first half of pregnancy and others showing the second half as the most critical time of exposure. The major reason the research is so inconsistent is because of the methodology used. Each study uses a different method of measuring ETS exposure, and this produces varying results. Measuring ETS exposure is difficult using any methodology because exposure may change over time, recall may not be accurate, and biomarkers only show exposure over a few days and may also be inaccurate.

More research is needed to determine if maternal ETS exposure has negative effects on infants and, if so, the magnitude and significance of these effects. Also, a better way of measuring ETS exposure should be developed in addition to determining if there is a critical exposure time and when.

Methodology

Participants in the smoking education program included six women, five nonsmokers, and one smoker. The mean age of participants was 20.83 years. The program was conducted two times with two groups; each group consisted of three women. Participants were recruited through known acquaintances. Recruitment flyers were hung throughout buildings on the UNT at Denton campus. The first group met on Friday, April 4, 2008, at 3:00 p.m., and the second group on Friday, April 11, 2008, at 3:00 p.m. Each group viewed the same PowerPoint presentation containing information on the health effects of smoking, including lung cancer and heart disease, effects of smoking on women, effects of SHS, and information on quitting smoking. Prior to viewing the presentation, participants completed informed consent forms, a personal survey, and
a smoking quiz. The personal survey gathered information about participants such as age, smoking status, and beliefs about smoking. The smoking quiz contained 15 questions, 10 true or false and 5 multiple choice, and was administered three times. The first administration of the quiz was designed to gauge the participants’ knowledge base. After the presentation, participants again completed the smoking quiz. This second administration was compared to the first and used to determine how much participants learned directly after the presentation. Two weeks after the program, participants were emailed the quiz to complete for a third time. This third administration was used to determine how much information was retained by the participants. The percentage correct on each quiz was tracked over the three administrations to measure the effectiveness of the smoking education program.

Results and Conclusions

The average score on the first administration of the quiz was 51. On the second administration, directly after the presentation, the average score was 73.95. The average score on the third administration of the quiz, two weeks after the program, was 58.4. It should be noted that data for one participant was not received for the third quiz. From the first quiz to the second quiz, all participants’ scores increased, corresponding to an average increase of 22.88 points per person. However, from the second quiz to the third quiz, all but one score decreased. This corresponded to an average decrease of 14.34 points between the second and third administrations of the quiz. Between the first quiz and the third quiz, two scores decreased, two scores increased, and one remained the same. This corresponded to an overall average increase of 9.12 points. These results are illustrated in Table 1, and Figures 1 and 2.

These data show that the smoking education program was only effective in the short term, directly after the presentation, but not in the long term. To improve the effectiveness of this
program, methods should be derived to increase the retention of information over a period of time.

Limitations

A major limitation of this study was the low number of participants. There was difficulty in recruiting participants as this study was initially designed for women smokers wanting to quit. This restricted our subject pool significantly. To overcome this, we modified the program and opened it to all UNT female students, improving recruitment only slightly.
Endnotes

2. Ibid.
3. Ibid.
6. Ibid.
9. Ibid.
12. Ibid.
15. Centers for Disease Control and Prevention, “Fact Sheet: Secondhand Smoke.”
16. Ibid.
17. Ibid.
18. Ibid.
19. Ibid.
32. Ibid.
34. Misra and Nguyen. “Environmental Tobacco Smoke and Low Birth Weight” 897-904.
36. Perera et al., “Molecular Evidence” 626-630.
37. Ibid.
40. Ibid.
42. Hanke et al., “Fetal Biometry,” 47-52.
44. Misra and Nguyen. “Environmental Tobacco Smoke and Low Birth Weight” 897-904.
47. Ibid.
48. Ibid.
49. Perera et al., “Molecular Evidence” 626-630.
51. Ibid.
Bibliography

Centers for Disease Control and Prevention. “Cancer.” *Smoking and Tobacco Use*.


Table 1. Individual Scores on Pre-test and Post-test Measures of Knowledge of Smoking Effects

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<th>Quiz 3</th>
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Figure 1. Average Scores on Pre-test and Post-tests

![Average Quiz Scores](image)
Figure 2. Individual Changes in Scores of Pre-test and Post-tests