

A TWO PART THESIS:  
ASSESSMENT OF EXPOSURE TO SECONDHAND SMOKE AT OUTDOOR BARS AND  
RESTAURANTS IN ATHENS, GA, USING SALIVARY COTININE  
AND  
MEASURING PM<sub>2.5</sub> AND CARBON MONOXIDE IN REAL-TIME FROM SECONDHAND  
SMOKE AT OUTDOOR BARS AND FAMILY RESTAURANTS IN ATHENS, GA

by

JONATHON CHASE HALL

(Under the Direction of Luke P. Naeher)

ABSTRACT

The first study is an assessment of exposure to secondhand smoke (SHS) at outdoor bars and restaurants using salivary cotinine as a biological marker. Saliva samples from non-smokers, age 21-30, were analyzed for cotinine both pre- and post-exposure to SHS. Salivary cotinine from subjects at the bar sites increased by an average ( $\pm$  std. dev.) of  $0.114 \pm 0.163$  ng/ml compared to an average increase of  $0.039 \pm 0.049$  ng/ml for subjects at the restaurant sites. Subjects at the control site had an average increase of  $0.006 \pm 0.016$  ng/ml.

The second study measures, in real-time, levels of particulate matter  $\leq 2.5$  microns in diameter (PM<sub>2.5</sub>) and carbon monoxide (CO) at outdoor bars and family restaurants in Athens, GA. The average PM<sub>2.5</sub> and CO levels at the bar were  $123.2 \mu\text{g}/\text{m}^3$  and 1.46 ppm, respectively. The restaurant recorded average PM<sub>2.5</sub> and CO readings of  $59.2 \mu\text{g}/\text{m}^3$  and 1.48 ppm, respectively. A Dustrak aerosol monitor measured PM<sub>2.5</sub>.

INDEX WORDS: secondhand smoke, salivary cotinine, personal exposure, environmental tobacco smoke, particulate matter, carbon monoxide

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B.S., Biology, Georgia Southern University, 2004

A Thesis Submitted to the Graduate Faculty of The University of Georgia in Partial Fulfillment  
of the Requirements for the Degree

MASTER OF SCIENCE

ATHENS, GEORGIA

2008

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August 2008

## ACKNOWLEDGEMENTS

A special thank you to the following people:

- Dr. Luke Naehar, for his support and encouragement throughout my graduate studies which enabled me to accomplish my ultimate goal of acceptance to dental school.
- Dr. Erin Lipp and Dr. Dan Hall, for serving on my Thesis committee and all of their advice and guidance along the way (or something like this).
- Dr. J. Tom Bernert, at the Centers for Disease Control and Prevention in Atlanta, GA, for his full support of this study and his willingness to offer advice.
- Dr. Lou Kudon of the Northeast Health District for his collaboration on this project.
- The Northeast Georgia Health District, Athens Community Wellness Council, and the Athens Tobacco Prevention Coalition for funding of the study.
- All members of Dr. Naehar's Laboratory, for their help and encouragement during this study.
- My parents, Wayne and Nancy Hall, for their full support throughout my academic career. They have given me so many great opportunities throughout life and I cannot thank them enough.
- Kristin Rulison, for her support over the past two years at the University of Georgia. She has inspired me on so many levels and I will be forever grateful for her encouragement in the pursuit of my personal goals.
- Adam Gray, for all his help inside and outside of the lab. He has not only been a great work partner, but also a great friend.

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CHAPTER 1  
INTRODUCTION

Chapter 1 outlines the information presented in each chapter. Chapter 2 presents a review of relevant literature related to exposure to secondhand smoke completed during the author's graduate studies. Chapter 3 contains a manuscript to be submitted for publication in the Journal of Occupational and Environmental Health (JOEH) in July 2008. This study assesses exposure to secondhand smoke at outdoor bars and family restaurants in Athens, GA, during the summer and fall of 2007, using salivary cotinine as a biological marker. Saliva samples were collected from non-smokers, age 21-30, both pre and post exposure to secondhand smoke and analyzed for cotinine. Chapter 4 reports data that discusses the levels of particulate matter <2.5  $\mu\text{m}$  aerodynamic diameter ( $\text{PM}_{2.5}$ ) and carbon monoxide (CO) at outdoor bars and family restaurants in Athens, GA, as well as a corresponding count of smokers in the area of the air monitoring. Real-time breathing-level height measurements of  $\text{PM}_{2.5}$  and CO were taken during four weekend nights in summer 2007. Statistical analysis was performed to determine the correlation between the air pollutant levels and the number of smokers located at the study sites. Chapter 5 contains discussion and conclusions based on the findings of the previous chapters.

CHAPTER 2  
SELECTED LITERATURE REVIEW OF SECONDHAND SMOKE AND EXPOSURE TO  
SECONDHAND SMOKE

Secondhand smoke (SHS), also referred to as environmental tobacco smoke, is a mixture of sidestream cigarette smoke and mainstream cigarette smoke consisting of almost 5000 chemical compounds, of which 43 are known human and animal carcinogens (Witschi et al., 1997; Brownson et al., 2002). The majority of involuntary exposure to SHS is attributed to sidestream smoke, which is defined as the smoke curling off the end of a lit cigarette while mainstream smoke is defined as the smoke inhaled directly from a lit cigarette (Witschi et al., 1997). The known carcinogens include polyaromatic hydrocarbons, 4-aminobiphenyl (4-ABP), 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), formaldehyde, benzene, lead, chromium, and cadmium (Hecht, 2004). In addition to these known carcinogens, some other compounds present in SHS are carbon monoxide, ammonia, nicotine, hydrogen cyanide, and sulfur dioxide (IARC, 1987; USEPA, 1993; Repace 1998; CalEPA 1997). Due to the harmful components and adverse health effects, SHS is considered to be the most important contaminant of indoor air (Law and Hackshaw, 1996).

### **Health Effects Associated With SHS Exposure**

Exposure to SHS was the focus of the 2006 U.S Surgeon Generals Report (US Surgeon General, 2006). The report cited SHS as being the cause of many adverse health effects in adults and children. In this report, the Surgeon General stated that due to increasing scientific evidence, there is no level of exposure to SHS that is considered safe and even breathing a small amount can be harmful. The USEPA concluded in a 1992 report, that SHS was indeed a carcinogen and estimated that as many as 3,000 lung cancer deaths each year can be associated with SHS exposure (USEPA, 1992). In a 1998 report, OSHA estimated that 21 million Americans are exposed to SHS at the workplace and that the number of lung cancer cases each year, of non-smoking American workers, would increase to 700 (OSHA, 1998). Repace and

colleagues developed models that suggest that 400 lung cancer deaths each year and 4000 heart disease death each year occur in office workers that are exposed to SHS (Repace et al., 1998) In the United Kingdom each year, exposure to SHS in the workplace may contribute up to one-fifth of all deaths in the general population, aged 20-64 years, and exposure to SHS at home may account for 2700 deaths in people aged 20-64 and 8000 deaths among people greater than 65 years of age (Jamrozik, 2005).

Non-smokers exposed to SHS demonstrate similar physiological reactions as current smokers. Panagiotakos et al., (2004) found that white blood cells, C-reactive proteins, homocysteine, fibrinogen, and oxidized low-density lipoprotein (LDL) cholesterol levels all increase in non-smokers exposed to SHS (Panagiotakos et al., 2004). These increases were also found in smokers. Another study by Otsuka et al., (2001) reported that after breathing SHS for 30 minutes, non-smokers had the endothelial function of coronary arteries compromised to a level that was similar to smokers (Otsuka et al., 2001). This rapid response to SHS has also been shown in platelet function and oxidation of LDL cholesterol (Glantz and Parmley, 1991; Valkonen and Kuusi, 1998).

Non-smoking adults are more prone to developing coronary heart disease, lung cancer, and acute respiratory symptoms when exposed to SHS, than when not exposed (U.S. Surgeon General's Report 2006). It has been estimated that a non-smoker exposed to SHS is equivalent to smoking 0.5 to 2 cigarettes per day (USEPA 1992). An analysis of 19 published studies of relative risk of ischemic heart disease in non-smokers, determined that the relative risk of ischemic heart disease developing in a 65 year-old non-smoker exposed to SHS was 1.30 (95% confidence interval 1.22 to 1.38), while the relative risk of a person the same age that smokes one cigarette a day, was 1.39 (1.18 to 1.64) (Law et al., 1997). For comparison, someone age 65,

who smokes 20 cigarettes a day, has an estimated relative risk of 1.78 (1.31 to 2.44) of developing ischemic heart disease (Law et al., 1997).

Children that are exposed to SHS on a regular basis have a higher risk of sudden infant death syndrome, acute respiratory infections, and asthma, than do children not exposed to SHS (U.S. Surgeon General's Report 2006). Involuntary exposure to secondhand smoke can also have harmful effects during fetal development. The Fourth Report of the Independent Scientific Committee on Smoking and Health determined the difference in birth weight of babies born to mothers exposed to SHS during pregnancy, and mothers not exposed during pregnancy, is 24g (Law and Hackshaw, 1996). Another study by Hegaard et al. (2006) found that non-smoking women exposed to SHS, both inside the home and outside the home, during pregnancy gave birth to children with a 79 g birth weight reduction compared to children of mothers that were not exposed to SHS (Hegaard et al., 2006). There is also evidence that high exposure to SHS as a child is the cause of 17% of lung cancer cases in non-smokers each year (Janerich, 1990). Tang and colleagues (1999) determined, by measuring 4-ABP-hemoglobin adducts and PAH albumin, that genetic damage can occur in minority children exposed to relatively low levels of household SHS (Tang et al., 1999).

California's EPA published a report in 1997 discussing SHS and the health effects associated with child exposure (CalEPA, 1997). In Californians, there is an estimated relative risk of 1.2 to 1.4, between low birth weight and pre-natal exposure to SHS. Post-natal exposure to SHS has an estimated relative risk of 1.62 for middle ear infection, and an estimated relative risk of 1.5 to 2 for lower respiratory disease (CalEPA, 1997). Non-smokers in California exposed to SHS, have a lifetime lung cancer risk of 0.7 %, with a relative risk of 1.2 (CalEPA, 1997).

The large volume of scientific evidence linking SHS exposure to adverse health effects has resulted in a world-wide reaction to reduce involuntary exposure. Indoor smoking bans have been enforced in many countries to reduce employee and customer exposure to SHS and as a result many scientific studies were performed at workplaces to determine the difference in indoor SHS levels by analyzing exposure and pollutant levels, both pre- and post-smoking ban (Valente et al., 2007; Johnsson et al., 2006; Gallus et al., 2006; Chapman et al., 2001; Mulcahy et al., 2005; Barone-Adesi et al., 2006; Juster et al., 2007; Semple et al., 2007; Allwright et al., 2005; Waring and Siegel, 2006; Bolte et al., 2007).

### **Global concern**

Ireland introduced a complete ban on indoor workplace smoking which included bars and restaurants in March 2004 – it was the first country to enforce a ban of this magnitude (Mulcahy et al., 2005; Semple et al., 2007). Some of the most notable effects of the ban were observed at the Irish pubs with a recorded 75-96% post-ban drop in  $PM_{2.5}$  in pubs and a 47-74% post-ban drop in  $PM_{10}$ , both relative to pre-ban levels (Mulcahy, 2005). Similar results were found by Semple et al., 2007, inside Scottish pubs after an indoor smoking ban in 2005 (Semple et al., 2007). The average  $PM_{2.5}$  level of  $246 \mu\text{g}/\text{m}^3$  was reduced to an average of  $20 \mu\text{g}/\text{m}^3$  after implementation of the ban with an average reduction of 86%.  $PM_{2.5}$  is emitted at high levels from tobacco combustion and has been associated with many of the same health effects as SHS (Leaderer, 1990; Pope et al., 2001). Therefore,  $PM_{2.5}$  is a reliable indicator of SHS exposure. Carbon monoxide (CO) is also a product of combustion and can be used as a marker of secondhand smoke exposure. It is important physiologically because CO inhibits the blood's ability to deliver oxygen to the heart (Glantz, 1995).

Following Ireland, Italy banned all indoor smoking in January 2005 (Gallus et al., 2006). This ban resulted in a decrease in hospital admissions for acute myocardial infarction from 922 cases in February-June 2004 (pre-ban) to 832 cases in February-June 2005 (post-ban) for people under the age of 60 in the region of Piedmont, Italy (Barone-Adesi, 2006). Gallus and colleagues (2006) discovered through analysis of official legal sales, that cigarette sales in Italy declined by 8.9% after the ban was implemented, which suggests the ban even had a positive effect on the health of smokers, in addition to non-smokers, by reducing their smoking frequency (Gallus et al., 2006). Based on a study of 40 hospitality venues in Italy, which included restaurants, bars, and game rooms, average PM<sub>2.5</sub> levels reduce from 119.3 µg/m<sup>3</sup> to 43.3 µg/m<sup>3</sup> a year after enforcement of the new ban (Valente et al., 2007). Similar bans on indoor smoking have been enforced in Finland, Australia, and Norway, with studies reporting observed positive health effects occurring post-smoking ban (Johnsson et al., 2005; Chapman et al., 2001; Eagen et al., 2006).

### **Concern in the United States**

San Francisco and New York are two major cities in the United States that have enacted bans on indoor workplace smoking, similar to the European cities mentioned previously (Eisner, 1998; Farrelly, 2005). Bartenders in San Francisco reported decreased respiratory illnesses and an increased lung forced vital capacity (FVC) that corresponds with their reported decrease in SHS exposure, after implementation of the smoking ban (Eisner, 1998). New York banned all smoking inside of bars, restaurants, and bowling alleys (Farrelly, 2005). Hospitality workers at these establishments presented an average decrease in respiratory symptoms of 88% and an average decrease in salivary cotinine from 3.6 ng/ml to 0.8 ng/ml, from their baseline level (pre-ban) to the twelve-month follow-up (Farrelly, 2005). In September 2005, Austin Texas enforced

an indoor smoking ban that resulted in a 70-99% decrease in PM<sub>2.5</sub> and a significant decrease in CO inside of bars (Waring and Siegel, 2006).

### **Cotinine as a biological marker**

Cotinine, the major metabolite of nicotine, is considered the best biomarker of exposure to SHS in both active smokers and non-smokers due to its high specificity to tobacco (CDC, 2005; Jarvis, 1985). Cotinine has a relatively long half-life of about 17 hours and can be measured by sampling serum, saliva, urine, or hair (Etter et al., 2000). The National Health and Nutrition Examination Survey (NHANES) 2001-2002, reports the average serum cotinine level of all non-smokers as 0.034 ng/ml, with salivary cotinine levels being typically 15% greater in value than serum values (CDC, 2005; Bernert et al., 2000).

Due to the non-invasive method of saliva collection, many scientific studies conducted in the field setting have been successful using salivary cotinine as an SHS exposure tool (Abrams, 1987). Children in Scotland with an average age of 11.4, displayed a salivary cotinine decrease from 0.36 ng/ml to 0.22 ng/ml after an indoor smoking ban was enforced at most public places (Akhtar, 2007). A study conducted in Liverpool used salivary cotinine levels to determine the association between child exposure to secondhand smoke and socioeconomic status (Delpisheh, 2005). Allwright et al., 2005, found that salivary cotinine levels decreased by 80%, from median 29.0 nmol/L to 5.1 nmol/L, in non-smoking bar workers along with a decrease in self-reported respiratory irritations, after implementation of the smoking ban in Ireland (Allwright et al., 2005). Hotel hospitality workers in Ireland also exhibited a 69% post-ban decrease, 1.6 ng/ml to 0.5 ng/ml, in salivary cotinine (Mulcahay, 2005).

## **Outdoor Exposure to SHS**

With indoor smoking bans being enforced world-wide in restaurants and bars, all the past indoor smoking has now moved outside leading to the question of what level of SHS are people exposed to while outside of restaurants and bars. In a recent study at the University of Georgia, Naeher and colleagues (2007) determined that PM<sub>2.5</sub> and CO levels at outdoor bars and restaurants were positively correlated with SHS, and not with vehicle traffic (Naeher et al., 2007). There is a paucity of data in the literature looking at personal SHS exposure levels for non-smokers in outdoor settings where heavy smoking is occurring, like in smoking areas outside of bars and restaurants.

The objective of this study was to assess personal exposure to SHS at outdoor family restaurants and bars in Athens, GA, a city of 102,000 people, using salivary cotinine as a biological marker. In 2006, Athens enforced a complete indoor smoking ban at bars and restaurants, making Athens an excellent study site. During summer and fall 2007, saliva samples were obtained from non-smokers, age 21-30, both pre- and post-exposure to SHS, and analyzed for salivary cotinine. In addition, in summer 2007 only, smoker count and ambient concentrations of PM<sub>2.5</sub> and CO were measured in real-time at breathing height level.

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## CHAPTER 3

# ASSESSMENT OF EXPOSURE TO SECONDHAND SMOKE AT OUTDOOR BARS AND FAMILY RESTAURANTS IN ATHENS, GA, USING SALIVARY COTININE<sup>1</sup>

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<sup>1</sup>Hall, J.C., Bernert, J.T., Hall, D.B., St.Helen, G., Kudon, L.H., Naeher, L.P. To be submitted to Journal of Occupational and Environmental Hygiene.

## **Abstract**

**Introduction:** Exposure to secondhand smoke (SHS) in the outdoor setting is a growing public health concern due to recent indoor smoking bans. The objective of this study was to measure salivary cotinine, a metabolic product of nicotine, in subjects age 21-30, at outdoor bars and restaurants in Athens, GA.

**Methods:** Non-smokers participated during 6-hr periods at outdoor bars and restaurants over six weekends in 2007. One non-smoker control was included each study day for comparison. Pre- and post-exposure saliva samples (N=29 person-days at the bar type site, N=28 person-days at the restaurant type site, and N=11 person-days for the control) were collected and analyzed for cotinine.

**Results:** The mean change in the response, ( $\ln(\text{post}) - \ln(\text{pre})$ ) salivary cotinine levels, was significantly impacted by the type of site (bar, restaurant, control) ( $F = 5.09$ ; d.f. = 2, 6.7;  $p = 0.0455$ ). The mean change in response at the bar sites was 0.9645 ng/ml and was significantly different from zero ( $t = 4.63$ ; d.f. = 9.24;  $p = 0.0011$ ). The mean change in the response at the restaurant sites was 0.7042 ng/ml and was significantly different from zero ( $t = 4.33$ ; d.f. = 4.47;  $p = 0.0097$ ). The mean change in response of the control was 0.1478 ng/ml and was not significantly different from zero ( $t = 0.86$ ; d.f. = 3.87;  $p = 0.4397$ ). Salivary cotinine from subjects at the bar sites increased by an average of  $0.114 \pm 0.163$  ng/ml (increase  $\pm$  std. dev.) compared to an average increase of  $0.039 \pm 0.049$  ng/ml for subjects at the restaurant sites. Subjects at the control site had an average increase of  $0.006 \pm 0.016$  ng/ml.

**Conclusion:** Non-smokers exposed to SHS at outdoor bars and restaurants have a measurable increase in salivary cotinine when comparing pre- and post- SHS exposure salivary cotinine levels.

## **Introduction**

Secondhand smoke (SHS), also referred to as environmental tobacco smoke (ETS), is a mixture of sidestream cigarette smoke and mainstream cigarette smoke consisting of almost 5000 chemical compounds, of which 43 are known human and animal carcinogens.<sup>(1,2)</sup> SHS consists primarily of sidestream smoke, the smoke emitted from the smoldering tip of a cigarette between puffs, with additional contribution from mainstream smoke exhaled by the smoker.<sup>(2)</sup> In addition to the known carcinogens, other compounds present in SHS include carbon monoxide (CO), ammonia, nicotine, hydrogen cyanide, and sulfur dioxide.<sup>(3-6)</sup> Due to the harmful components and adverse health effects due to exposure, SHS is considered to be the most important contaminant of indoor air.<sup>(7)</sup>

Exposure to SHS causes lung and nasal sinus cancer, heart disease, and sudden infant death syndrome (SIDS).<sup>(8)</sup> Serious impacts of SHS on children include both the induction and exacerbation of asthma, bronchitis and pneumonia, middle ear infection, chronic respiratory symptoms, and low birth weight.<sup>(4,9)</sup> In 2006, the United States Surgeon General's Report stated that SHS contains many chemicals that can quickly irritate and damage the lining of the airways. Even brief exposure can result in upper airway changes in healthy persons and can lead to additional and more frequent asthma attacks in children who already have asthma. The Surgeon General also reported that due to increasing scientific evidence, there is no level of exposure to SHS that is considered safe.<sup>(8)</sup>

Due to the increasing scientific evidence associating adverse health effects with exposure to SHS, indoor smoking bans have been enforced in many countries. Ireland, Italy, Norway, Scotland, Finland, Australia, New Zealand and the United States have all passed laws banning indoor smoking either on a local or national level.<sup>(10)</sup> Studies have been conducted in these

countries prior to and following imposition of smoking bans to monitor select indoor air pollutants associated with SHS, most notably PM<sub>2.5</sub> and CO, with the results showing a significant decrease in indoor levels of these pollutants.<sup>(11,12)</sup> Bar workers in San Francisco and Ireland reported significantly decreased respiratory illnesses after implementation of indoor smoking bans and a decrease in the rate of hospital admissions for acute myocardial infarction was observed in New York state and Italy after implementation of smoking bans.<sup>(12-15)</sup> These studies provide evidence that SHS is harmful and that decreased exposure can result in positive health outcomes.

To determine personal SHS exposure, air contaminants that are associated with SHS such as PM<sub>2.5</sub> and CO can be measured, but use of a biological marker of exposure may provide a stronger representation of the actual SHS personal dose. Cotinine, the primary proximate metabolite of nicotine, is considered to be the best biomarker of exposure to tobacco smoke in both active smokers and non-smokers due to its high specificity for tobacco.<sup>(16,17)</sup> Cotinine has a relatively long half life of 17-hours and can be measured by sampling serum, saliva, urine, or hair.<sup>(18)</sup> Due to the non-invasive method of saliva collection, many studies conducted in field settings have used salivary cotinine as an SHS exposure tool.<sup>(19)</sup> Studies that include both children and adults have reported decreased salivary cotinine levels after exposure to SHS has been either lowered or eliminated.<sup>(11,20,21)</sup>

The objective of this study was to assess exposure to SHS in a group of non-smokers, age 21-30, at outdoor bars and family restaurants in Athens, GA using salivary cotinine as a biological marker.

## **Methods**

### *Study Locations*

This study was conducted in the summer and fall of 2007 at outdoor bars and family restaurants where smoking was permitted in Athens, GA, a city with a population of approximately 102,000 people. Verbal approval to conduct the study was obtained from individual restaurant and bar managers and owners prior to the first day of sampling. Descriptions of the study sites and the dates they were sampled are displayed in Table 3-1. During the summer, two bars (Site A and Site C) and one family restaurant (Site B) were chosen as study sites. All locations were sampled for six-hours each on four weekend nights (Week 1-4) in June, with the exceptions of site A which was only sampled during weeks 1-3 and site C which was only sampled during week 4.

During the fall, two family restaurants and three bars were chosen as study sites. In week 5, two family restaurants (Site B and Site D) and one bar (Site A) were utilized. Week 6 consisted of three bars (Site A, Site E, and Site F) and one family restaurant (Site D). Sites A, B, and D were sampled on October 6, 2007 from 2PM to 8PM; Site D on November 2, 2007 from 4PM-10PM; Sites A, E, and F on November 3, 2007, from 7PM to 1AM. The two fall study dates were chosen by anticipation of large crowds, and related high outdoor SHS exposures, at the study sites due to popular athletic events on television and in Athens, GA.

For both summer and fall sessions, a control site (Site G) was located on the north campus of the University of Georgia (UGA). This area was located outdoors, with no walls or roof, and had very little human traffic or environmental tobacco smoke (SHS) exposure.

### *Subject Selection*

Subjects used in the study were all non-smoking college students between the ages of 21 and 30, who volunteered to participate. Eleven subjects participated in the summer, with a total of 64 person-days and 20 subjects participated in the fall, with a total of 64 subject days (n=64). A total of five subjects participated in both the summer and fall study dates. Subjects were not exposed to SHS on consecutive days of the study because salivary cotinine levels would still be elevated from exposure on the previous day.

Questionnaires were verbally administered to potential study participants to determine the persons' eligibility for the study. The questionnaire included questions on current and past smoking status, and current SHS exposure. If the person did not smoke, did not live with a smoker, and did not use nicotine in any other form (i.e., smokeless tobacco, nicotine replacement therapy), they were considered eligible to participate in the study. The study protocol was reviewed and approved by Institutional Review Boards at the University of Georgia and the Centers for Disease Control and Prevention. The sample population was not random, rather subjects were chosen on availability and were all students at the University of Georgia.

### *Exposure assessment*

Human subjects provided saliva samples, both pre- and post-exposure to SHS, using Salivettes (Sarstedt, Newton, NC). The samples were collected at UGA's Environmental Health Science (EHS) building within 30-minutes before, and after, exposure to SHS at the study sites. The EHS laboratory is located approximately one-mile from the study sites and subjects were transported in vehicles to ensure no additional SHS exposure. Subjects fully saturated the cotton swab portion of the salivette with saliva; usually taking about 2-minutes. The subjects were

instructed to avoid SHS exposure as much as possible 48-hours prior to providing the saliva sample to minimize the pre-exposure salivary cotinine concentration.

The salivette cases contained a label that presented: the week number of the study, the subject ID number, and either PRE or POST (to distinguish pre- or post-SHS exposure). The samples were collected by the investigators and immediately placed in plastic storage bags and then in a freezer (- 20 °C) to preserve the saliva until analysis at the Centers for Disease Control and Prevention (CDC) in Atlanta, GA. The samples were shipped on dry ice overnight to CDC each week of the study.

#### *Salivary Cotinine Analysis*

Salivary cotinine analysis was performed by a method previously described in detail <sup>(22)</sup>. Briefly, samples were spiked with a cotinine-D<sub>3</sub> internal standard, extracted with methylene chloride, dried and reconstituted in water, and analyzed by LC APCI tandem mass spectrometry. Cotinine concentrations were quantified by comparison with standards using least squares linear regression. All analytical runs included a blank and two QC pools, and all results are from runs that were confirmed to be in statistical control.

#### *Statistical Analysis*

Cotinine differences (post – pre) were calculated for each subject and an average change in cotinine was calculated for all three site types (bar, restaurant, and control). A linear mixed effects model was used to fit the data and determine if the mean response is different for each study site type. The response was analyzed as  $\ln(\text{post}) - \ln(\text{pre})$ . Random subject and visit effects were included to account for the variability between visits and subjects. Estimates for the mean response at each study site were calculated along with statistics to test if the mean response was zero. Estimates of the differences between the bar and control and the restaurant and the

control were calculated with t-tests to determine if the difference is significantly different from zero. Due to the multiple comparisons with the control site, adjusted p-values were calculated to determine the significance of the results using Dunnett's method. A significance level of  $p < 0.05$  was used for all statistical tests.

## Results

Subjects were studied at seven locations over the duration of the study period. Table 3-1 displays descriptions of the study sites and the dates each site was sampled over the six-week study period. During the summer, site A was cancelled after the third sampling period for logistical reasons, and replaced with site C. Site A was used again for the fifth and sixth sampling days. Site G (control) was the same location for all six sampling periods.

Calculated by post- minus pre-SHS exposure salivary cotinine levels, the subjects at the bar sites had the largest increase in salivary cotinine after the exposure period, followed by subjects at the restaurant sites, and lastly by subjects at the control sites (see Figure 3-1 and Table 3-2). Average differences for all subjects located at the bar, restaurant and control site were 0.114 ng/ml, 0.039 ng/ml, and 0.006 ng/ml, respectively.

Using the model,  $y_{ijk} = \mu_k + s_i + v_{j(k)} + e_{ijk}$ , (Model 1.1)

in which the response is given as,  $(\ln(\text{post}) - \ln(\text{pre}))$ , it was determined that mean response was not the same for all site types ( $F=5.09$ ,  $d.f.=2, 6.7$ ,  $p\text{-value}=0.045$ ). The t-values demonstrate there was a significant increase in  $\ln(\text{cotinine})$  from pretest to posttest for both the bar and restaurant, but not the control site. The p-values for the bar, restaurant, and control were 0.0011, 0.0097, and 0.4397, respectively, demonstrating that the control was the only site where the mean response was not significantly different than zero. These data are represented with the corresponding t-values in Table 3-3.

Table 3-4 displays the estimates of the difference between the bar and the control in the mean response, and the difference between the restaurant and the control in the mean response. This is mathematically represented as (bar mean – control mean) and (restaurant mean – control mean). Adjusted p-values demonstrated that the estimate values were significantly different from zero for the bar and restaurant (0.0176 and 0.0461, respectively). Unadjusted p-values are presented, but due to the multiple comparisons with the control, adjusted p-values were also calculated using Dunnett's method presented in Table 3-4.

## **Discussion**

Many studies have shown an association between indoor exposure to SHS and an increase in salivary cotinine concentration.<sup>(11,22)</sup> However, we are not aware of any prior studies that have documented exposure to SHS in outdoor settings using salivary cotinine measurements. This exposure warrants attention because the many indoor smoking bans established across the world have encouraged the movement of previous indoor smoking outdoors, and thus estimating the extent of outdoor SHS exposure of nonsmokers is a potentially significant public health issue. Many previous studies have utilized questionnaires to determine the amount of SHS exposure, but objective and quantitative methods such as biomarker measurements can be particularly beneficial in this area.

Salivary cotinine was chosen as the biological marker for this study because of the non-invasive saliva collection and ease of collection in the field setting. Cotinine is the major metabolic product of nicotine with a half-life of about 17-hours, so it was appropriate for this study design.<sup>(11,20,21)</sup> The most recent National Health and Nutrition Examination Survey (NHANES) indicates a national average for serum cotinine among non-smokers of 0.034 ng/ml; salivary cotinine levels are typically about 15% - 30% higher.<sup>(16,23)</sup> Previous studies have shown

that serum and salivary levels of cotinine are highly correlated with a 1.1-1.4 saliva to blood ratio.<sup>(24,25)</sup>

Subjects located at the bar type site had higher salivary cotinine level differences (post-exposure level – pre-exposure level) than subjects at the restaurant or control site, as presented in table II. This is to be expected, as the amount of smokers is typically higher at bars than restaurants and the amount of smoke exposure at the control site was minimal, if not completely zero.<sup>(26)</sup> Subjects located at the bar and restaurant sites had mean response (ln(post) – ln(pre)) that was significantly different than zero, while the control subjects did not have a mean response significantly different than zero. This was expected due to the very low level of SHS exposure at the study areas.<sup>(26)</sup> The mean response for all sites was also determined to vary, with larger mean responses among the bar and restaurant participants than in those at the control site.

To compare the results of this study with NHANES 2001-2002 background serum cotinine levels, we applied the regression equation,

$$\log_{10}(\text{salivary}) = 0.962817 * \log_{10}(\text{serum}) + 0.127478, \quad (\text{Equation 1.1})$$

in order to obtain projected serum cotinine levels from our salivary cotinine levels.<sup>(22)</sup> After applying the equation, the average pre-exposure for the bar, restaurant, and control were 0.046 ng/ml, 0.023 ng/ml, and 0.028 ng/ml, respectively. Post exposure levels for the bar, restaurant, and control were 0.126 ng/ml, 0.050 ng/ml, and 0.032 ng/ml, respectively. NHANES 2001-2002 reports serum cotinine background levels of nonsmokers, age >20, as 0.034 ng/ml. The pre-exposure salivary cotinine levels from this study, after adjustment, are consistent with the NHANES background levels.

The variation in pre-exposure cotinine levels for subjects at the bar type sites can be attributed to subjects who had pre-exposure salivary cotinine levels much higher than the

majority of participating subjects. An example is subject 009, whom only participated during weeks one to four, with a geometric mean pre-exposure cotinine value of 0.604 ng/ml (N=4). The geometric mean pre-exposure cotinine values for all subjects during the first four-weeks, excluding subject 009, was 0.0483 ng/ml (N=31). Subject 009 reported that his roommate smokes cigarettes, but not inside the home, so he did not believe that he was directly exposed to any SHS. This demonstrates the sensitivity of using salivary cotinine as a biological marker of SHS exposure. Etter et al., (2000) found that non-smokers who lived with smokers or had friends that smoke, had salivary cotinine levels 1.5 times higher than non-smokers who do not live with smokers or do not have friends that smoke.<sup>(27)</sup>

Another example of increased pre-exposure cotinine levels was subject 008, who was an active smoker who did not report this information at the beginning of the study, and thus was excluded from all calculation presented in this report. Salivary cotinine analysis was performed for the first three dates of the study for subject 008, with a mean pre-exposure value of 46.6 ng/ml (N=3). For comparison, the geometric mean pre-exposure salivary cotinine level for all participating non-smoking subjects during first three weeks of the study, was 0.06 ng/ml (N=25). The subject later reported that he/she did not smoke cigarettes 48-hours prior to the study date, thus further demonstrating the sensitivity of salivary cotinine and the importance of non-smokers avoiding all SHS to the extent possible before participation studies of this design.

Comparing summer to fall pre-exposure salivary cotinine levels (see Table 3-5), the bar pre-exposure level was 0.094 ng/ml in the summer and was 0.054 ng/ml in the fall. This difference can be attributed to subject 009, mentioned in the previous paragraph, who did not participate during the two fall study dates. A pre-exposure salivary cotinine value difference was also seen at the restaurant site with levels for the summer and fall being 0.050 ng/ml and 0.023,

respectively. The increased levels during the summer is due to subject 004, who was exposed to low levels of SHS during the week leading up to the study period, but tried to prevent exposure at least 48-hours before study participation. Subject 004 had an average pre-exposure cotinine level of 0.135 ng/ml (N=4) and the other subjects at the restaurant site had an average pre-exposure cotinine level of 0.036 ng/ml (N=12). Again, this illustrates the sensitivity of cotinine as a biological marker of SHS exposure.

Post-exposure salivary cotinine levels also differed between summer and fall (see Table 3-5). The bar type site had an average summer post-exposure cotinine level of 0.300 ng/ml (N=11) and an average fall post-exposure level of 0.123 ng/ml (N=14). The restaurant type site displayed the same trend in results. Summer and fall post-exposure cotinine levels were 0.107 ng/ml (N=16) and 0.047 ng/ml (N=12), respectively. Due to increased crowds during the fall for school and athletic events in Athens, we anticipated higher levels of smokers along with higher salivary cotinine levels. Week 5 of the study was conducted during an away University of Georgia football game that resulted in small crowds at the study sites, which can explain for the lower than expected fall cotinine levels.

Non-smoking hotel workers in Ireland, who were exposed to SHS during their 8-hour work shift, had a mean post-work salivary cotinine level of 2.86 ng/ml. This level decreased to 1.29 ng/ml, after a smoking ban was put into effect in Ireland.<sup>(11)</sup> A study of non-smoking hospitality workers in New Zealand found a mean salivary cotinine difference (post – pre) of 1.11 ng/ml in workers of smoking-allowed workplaces and 0.02 ng/ml in workers of non-smoking workplaces.<sup>(28)</sup> This difference in cotinine concentrations observed in workers not exposed to SHS in the workplace is below the mean difference for both the bar and restaurant

type sites in our study (0.116 ng/ml and 0.05 ng/ml, respectively), but similar to what we found at the control site

Repace and colleagues (1998) determined that an average salivary cotinine level of 0.40 ng/ml corresponds to an increased lifetime mortality risk of 1/1000 for lung cancer and 1/100 for heart disease.<sup>(29)</sup> The average salivary cotinine levels for all six study dates in our study did not reach this level reported by Repace et al. that are potentially harmful. Although, the average post-exposure salivary cotinine level for the bar site in the summer was 0.30 ng/ml, which is close in magnitude to the levels reported by Repace and colleagues. Further studies should be conducted to determine if workers repeatedly exposed to SHS at outdoor bars have sustained salivary cotinine levels in the range of 0.30 ng/ml to 0.40 ng/ml. From those results, conclusions could be made regarding if this poses a concern to the health of workers and patrons at bars.

This study had several limitations. For example, an accurate count of the total number of cigarettes lit during a study period, for both summer and fall dates, might have provided an independent estimate of the amount of smoke the subjects were exposed to while at the sites. Another interesting aspect would be to measure the air nicotine present at the sites in comparison to the amount of salivary cotinine reported. This could be accomplished by using air nicotine monitors. The study population consisted of healthy college students between the ages of 21 and 30. Different age groups, such as children and the elderly, could have different nicotine metabolism rates which would result in either decreased or increased mean cotinine changes. Race of the subjects should also be taken into consideration of future studies to obtain a more diverse study population. This study consisted of fifteen Caucasians, five African-Americans, one Hispanic, and two Indians. Finally, a larger study population and more study dates could be expected to strengthen the statistical analyses.

## **Conclusions**

Subjects who spend several hours in areas outside of bars and restaurants where people smoke have higher concentrations of the nicotine metabolite, cotinine, in their saliva compared to subjects congregating where people do not smoke, indicating higher SHS exposure in the former group. Although the increment in cotinine concentrations and thus the SHS exposure levels were low, the current view is that there is no level of customary exposure to SHS that can be regarded as safe<sup>(8)</sup>. This study demonstrates that non-smokers outside of bars and restaurants exposed to SHS have a measurable difference in the pre and post-exposure salivary cotinine levels.

## **Acknowledgements**

The authors wish to thank all of the participating establishments and study participants that participated in the study. Thank you to Dr. Thomas Bernert and his laboratory at the Centers for Disease Control and Prevention in Atlanta, GA, for the salivary cotinine analysis and support of the project. This work was funded in part by the Northeast Health District, Athens Community Wellness Council, and the Athens Tobacco Prevention Coalition.

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Table 3-1: Descriptions of all sites utilized in the study.

Site ID	Site type	Week sampled	Tables for customers (n=)	Roof at site	Outdoor patio area (ft <sup>2</sup> )
A	Bar with outdoor patio	1 to 6	6	None	576
B	Family restaurant outdoor patio	1 to 5	8	None	1984
C	Bar with outdoor patio	4	12	None	896
D	Family restaurant with outdoor patio	5 to 6	17	None	1800
E	Bar with outdoor patio	6	10	None	684
G	Open air area on north campus of UGA	1 to 6	0	None	3690

Table 3-2: Descriptive statistics of the salivary cotinine data.

Site	Statistics	Pre-exposure cotinine level	Post-exposure cotinine level	Cotinine level difference (post - pre)
Bar (N=25)	Geo Mean	0.069	0.182	0.114
	Minimum	0.011	0.040	-0.275
	Maximum	0.959	1.056	0.559
	Median	0.048	0.128	0.070
	Std. Dev.	0.268	0.296	0.163
Restaurant (N=28)	Geo Mean	0.036	0.075	0.039
	Minimum	0.011	0.025	-0.010
	Maximum	0.218	0.404	0.186
	Median	0.036	0.072	0.037
	Std. Dev.	0.048	0.078	0.049
Control (N=11)	Geo Mean	0.043	0.049	0.006
	Minimum	0.030	0.027	-0.006
	Maximum	0.081	0.094	0.039
	Median	0.041	0.051	0.001
	Std. Dev.	0.015	0.023	0.016

Table 3-3: Estimates of the mean response at each site type.

Site	Parameter Estimate	t-value	p-value
Bar	0.9645	4.63	0.0011
Restaurant	0.7042	4.33	0.0097
Control	0.1478	0.86	0.4397

Table 3-4: Estimates of the difference in response between the site and control.

Site	Parameter Estimate	t-value	p-value	Adj. p-value
Bar	0.8167	3.05	0.0062	0.0176
Restaurant	0.5564	2.35	0.0321	0.0461

Table 3-5: Average salivary cotinine levels for each site by season.

Site	Pre (summer)	Post (summer)	Pre (fall)	Post (fall)	Post - Pre (summer)	Post - Pre (fall)
Bar	0.094	0.300	0.054	0.123	0.208	0.044
Restaurant	0.050	0.107	0.023	0.047	0.063	0.024
Control	0.041	0.048	0.050	0.053	0.009	0.005

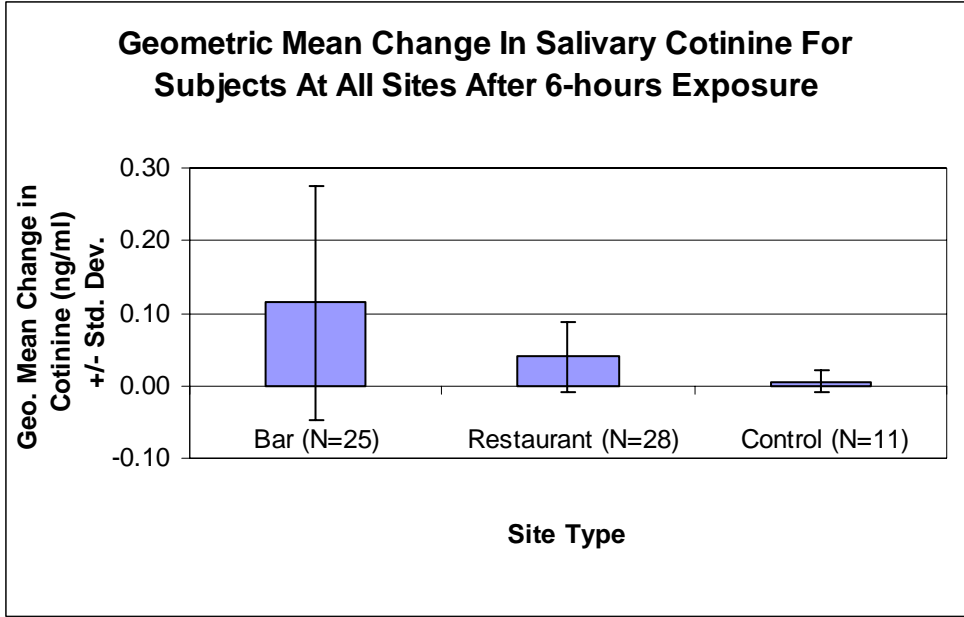


Figure 3-1: Mean change in salivary cotinine for all sites with standard deviation bars.

CHAPTER FOUR  
MEASURING PM<sub>2.5</sub> AND CARBON MONOXIDE IN REAL-TIME FROM  
SECONDHAND SMOKE AT OUTDOOR BARS AND FAMILY RESTAURANTS  
IN ATHENS, GA

**Abstract**

Exposure to secondhand smoke is associated with adverse health effects. The objectives of this study were to measure real-time levels of PM<sub>2.5</sub> and CO at outdoor bars and family restaurants where smoking is permitted in Athens, GA, during four weekend nights in June 2007. In addition, smokers and pedestrians were counted at each site. A Dustrak aerosol monitor and Langan CO monitor were used to measure the pollutants at each site. The average PM<sub>2.5</sub> and CO levels recorded at the bar site were 123.2 µg/m<sup>3</sup> and 1.46 ppm, respectively. The restaurant recorded average PM<sub>2.5</sub> and CO readings of 59.2 µg/m<sup>3</sup> and 1.48 ppm, respectively. The control sites for the bar and restaurant had average PM<sub>2.5</sub> levels of 61.9 µg/m<sup>3</sup> and 48.2 µg/m<sup>3</sup>, respectively, and average CO levels of 1.42 ppm and 1.08 ppm, respectively. The results demonstrate that areas with a higher average number of smokers have elevated levels of PM<sub>2.5</sub> and CO.

Key Words: secondhand smoke, particulate matter, carbon monoxide

**Introduction**

According to the 2006 Surgeon General's Report, there is no level of exposure to secondhand smoke (SHS) that is considered safe. The report also lists the increased health risks to children exposed to SHS, such as more severe asthma, ear problems, acute respiratory

infections, and sudden infant death syndrome (SIDS). Adults exposed to SHS at home or work are at a 25-30% increased risk of developing heart disease and a 20-30% increased risk of developing lung cancer.<sup>1</sup> Reports such as this have received attention from the public and as a result many states have banned indoor smoking at workplaces, including restaurants and bars.<sup>2-5</sup>

Recent studies have been conducted at outdoor bars and restaurants to measure levels of PM<sub>2.5</sub> and carbon monoxide (CO) to determine if the levels are associated with cigarette smoking in the area.<sup>6,7</sup> These studies have reported that SHS levels outside of bars and restaurants are increased in the presence of cigarette smoking and are not correlated with vehicle traffic, which can be a source of PM<sub>2.5</sub> and CO.<sup>8</sup> The interest in outdoor exposure to SHS is driven by the concept that since indoor smoking bans have been put into effect, the amount of outdoor smoking has increased.

This current study was conducted at an outdoor bar, an outdoor family restaurant, and a control site during four weekends in June of 2007 in Athens, GA, a city of 102,000 people that banned indoor smoking at bars and restaurants in 2005. The objectives were to measure breathing-level height PM<sub>2.5</sub> and CO and determine if the recorded concentrations are correlated to the number of smokers and the number of pedestrians in the area.

## **Methods**

### *Study Locations*

This study was conducted over four weekends in June 2007, in downtown Athens, GA, a city of approximately 102,000 people. Two of the locations (site A and site B) were bars, one location (site C) was a family restaurant, and one location (site D) was a control site with little to no smoking. All sites were open-air type areas with no roofs. The restaurant site was sampled for 6-hours on four Friday nights with a corresponding control site and the bar site was sampled

on four Saturday nights with a corresponding control site. Site A was sampled for the first three study dates, but due to logistical reasons, was not available for the fourth study date. Site B was sampled only on the fourth study day.

#### *Air Pollutant Sampling*

PM<sub>2.5</sub> (real-time) and CO (real-time) were measured at the bar, restaurant, and control sites during the study. PM<sub>2.5</sub> was logged every 5-minutes for 6-hours using a DustTrak 8520 aerosol monitor with data logger and CO was logged every 5-minutes for 6-hours using a Langan CO monitor. Both pieces of equipment were located near the subjects at breathing level height. After completion of the sampling period each day, the data was downloaded onto computers at UGA's Environmental Health Science (EHS) laboratory for analysis.

Both the DustTrak and Langan were calibrated at the EHS laboratory at the beginning and end of each study day. The DustTrak monitor was calibrated using a HEPA zeroing filter and the Langan CO monitor was calibrated by using a >99.999% N<sub>2</sub> (zero gas) and a 50 ppm CO gas. Batteries were also fully charged for both pieces of equipment prior to use.

#### *Counting Smokers*

Investigators located at the bar, restaurant, and control sites counted smokers and pedestrians in 5-minute intervals to correlate with the data obtained from the air sampling equipment. A smoker was defined as anyone who was smoking a cigarette within the site's patio area and a pedestrian was anyone located on the patio area, regardless of their smoking status. Both values included anyone walking through the patio area, even if they were not a patron of the establishment. At the end of the 5-minute interval, a new smoker count would begin and included smokers from the previous count, if they were still smoking a cigarette. A new pedestrian count was also conducted every 5-minutes.

To ensure an accurate count of smokers and pedestrians, the investigators used hand-held counters and recorded their results in notebooks that contained logging sheets. Stop watches were used to accurately measure 5-minute intervals. After the sampling day was complete, the results were transferred to an Excel spreadsheet.

## **Results and Discussion**

The average PM<sub>2.5</sub> and CO levels recorded at the bar-type sites, A and B, for all four sampling days, were 123.2 µg/m<sup>3</sup> and 1.46 ppm, respectively. The restaurant, site C, recorded average PM<sub>2.5</sub> and CO readings of 59.2 µg/m<sup>3</sup> and 1.48 ppm, respectively. The control-bar and control-restaurant sites recorded an average PM<sub>2.5</sub> level of 61.9 µg/m<sup>3</sup> and 48.2 µg/m<sup>3</sup>, respectively and average CO readings of 1.42 ppm and 1.08 ppm, respectively. Both control sites had zero average number of smokers, while the bar and restaurant had fifteen and eight, respectively.

The bar site had a higher average number of smokers than the control-bar site and this resulted in higher levels of PM<sub>2.5</sub> and CO at the bar site. The restaurant site had a higher number of smokers than the control-restaurant site, also resulting in higher levels of PM<sub>2.5</sub> and CO at the restaurant site. Comparing the bar site to restaurant site, there is again a connection with greater number of smokers and higher pollutant levels at the site. The descriptive statistics are displayed in Table 4-1. Figures 4-1 and 4-2 show the graphs of PM<sub>2.5</sub> and CO versus the number of smokers for all sites, excluding the control sites. A general linear increase can be seen in both graphs with the liner trend for PM<sub>2.5</sub> being more significant than CO.

The original data were severely skewed, so the log transformation and SAS procedure, CORR, were applied for statistical analysis to determine the correlation between pollutant levels and number of smokers and number of pedestrians at the study sites (see Table 4-2 and Table 4-

3). There was significant correlation found between  $PM_{2.5}$  and the number of smokers and the number of pedestrians for all study sites ( $p < 0.001$  and  $p < 0.001$ , respectively). The same was found for the correlation between number of smokers and number of pedestrians and CO level at all study sites ( $p = 0.0002$  and  $p < 0.0001$ , respectively). Therefore, more smokers and more pedestrians result in higher pollutant levels at the study sites. Breaking the results down by site, the bar had significant correlation between the number of smokers and number of pedestrians and  $PM_{2.5}$  ( $p < 0.0001$  and  $p < 0.001$ , respectively) and CO ( $p = 0.0002$  and  $p < 0.0001$ ). The trends were that same at the restaurant site for  $PM_{2.5}$  ( $p < 0.0001$  and  $p < 0.0001$ ) and CO ( $p < 0.0001$  and  $p = 0.0049$ , respectively). Both control sites (control-bar and control-restaurant) did not have significant correlation between the number of smokers and the number of pedestrians and pollutant levels. This is to be expected because the control sites were chosen due to their lack of smokers and pedestrian traffic.

$PM_{2.5}$  measurements were taken using a DusTrak aerosol monitor, which are factory calibrated using Arizona Test Dust which has an aerodynamic diameter of  $4.4 \mu m$ . Studies have shown combustion aerosols, such as cigarette smoke, often scatter two to three times more light per unit mass than the dust used during calibration.<sup>9,10</sup> Therefore, we estimate that the  $PM_{2.5}$  values reported in this study are over estimated by 1.5x to 2x.

Although the data set is small, the findings of this study are similar to results in other published studies.<sup>6,7</sup> A limitation of this study was the number of study days and equipment malfunction ( $n = 2$  sample days for  $PM_{2.5}$  readings at the control-bar site), so it is difficult to make broad generalizations. Another limitation is that human exposure is not measured, but this could be accomplished by using human subjects and incorporating a biological marker into the study design.

## Conclusions

The findings of this study show that PM<sub>2.5</sub> and CO concentrations are elevated at outdoor bars and restaurants where smoking is permitted, but not at areas where smoking is minimal.

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Table 4-1: Descriptive statistics of the PM<sub>2.5</sub> and CO data.

Site	Statistics	PM <sub>2.5</sub> (µg/m <sup>3</sup> )	CO (ppm)	smokers	pedestrians
Bar	Mean	123.2	1.46	8	48
	Maximum	662.2	2.64	45	164
	Minimum	31.0	1.12	0	1
	Median	97.6	1.40	7	32
	Std. Dev.	84.9	0.28	7	38
Control-Bar	Mean	61.9	1.42	0	3
	Maximum	83.9	1.83	2	17
	Minimum	42.6	1.13	0	0
	Median	63.2	1.45	0	3
	Std. Dev.	11.0	0.18	0	3
Restaurant	Mean	59.2	1.48	2	31
	Maximum	726.8	3.36	15	187
	Minimum	20.3	0.94	0	1
	Median	43.6	1.50	1	25
	Std. Dev.	67.6	0.34	3	32
Control-Rest	Mean	48.2	1.08	0	1
	Maximum	128.9	2.05	1	16
	Minimum	30.4	0.73	0	0
	Median	44.8	1.05	0	0
	Std. Dev.	12.1	0.26	0	2

Table 4-2: Correlation between smokers and pedestrians and PM<sub>2.5</sub> at each site.

Site	Smokers	Pedestrians
All	<0.0001	<0.0001
Bar	<0.0001	<0.0001
Control-Bar	none	<0.0996
Restaurant	<0.0001	<0.0001
Control-Rest	none	0.1504

Table 4-3: Correlation between smokers and pedestrians and CO at each site.

Site	Smokers	Pedestrians
All	<0.0001	<0.0001
Bar	0.0002	<0.0001
Control-Bar	none	<0.0001
Restaurant	<0.0001	0.0049
Control-Rest	none	0.1813

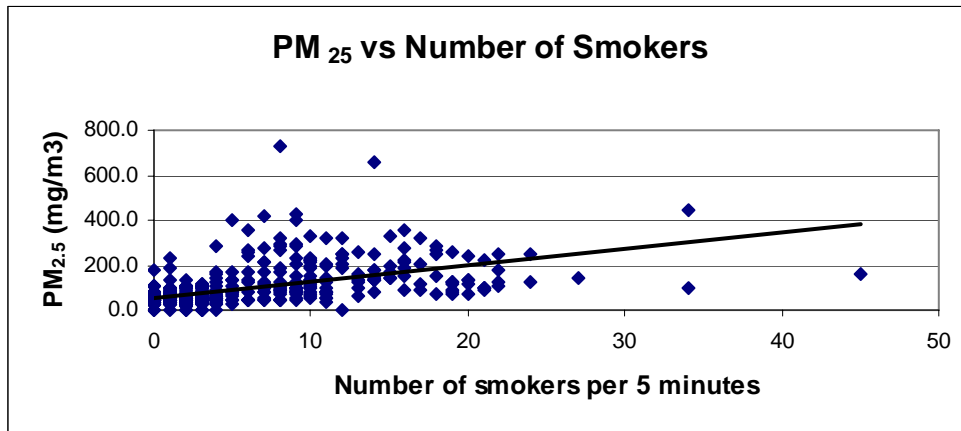


Figure 4-1: PM<sub>2.5</sub> vs. smokers for all sites other than control sites.

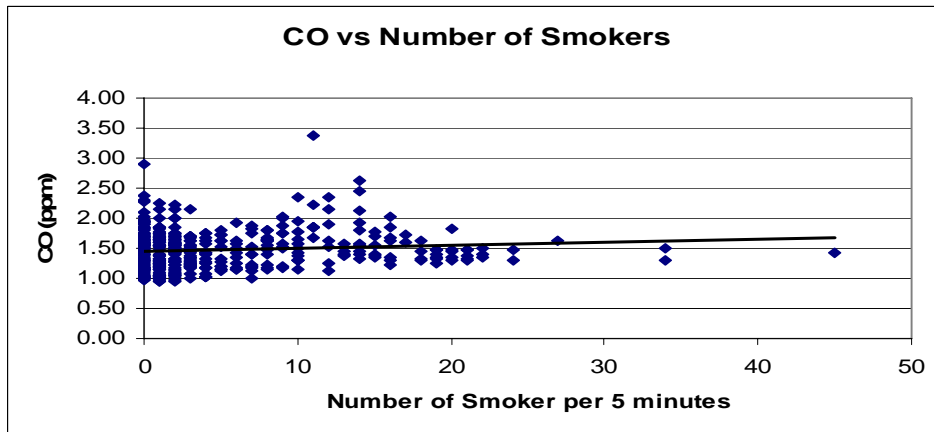


Figure 4-2: CO vs. smokers for all sites other than control sites.

CHAPTER 5  
CONCLUSIONS

### **Assessment of exposure to secondhand smoke at outdoor bars and family restaurants in Athens, GA, using salivary cotinine.**

The results of this study demonstrate that nonsmokers, age 21-30, exposed to SHS at outdoor bars and family restaurants have increased levels of the nicotine metabolite, cotinine, in their saliva compared to nonsmokers congregating where people do not smoke, indicating higher SHS exposure in the former group. The mean change in salivary cotinine (post-pre) for subjects at the bars and restaurants were higher than the mean change in salivary cotinine for subjects at the control site, after an exposure period of several hours.

The post salivary cotinine levels were low in value, but were elevated after exposure and as reported by the United States Surgeon General in 2006, no exposure level to SHS can be considered safe. The post exposure cotinine levels were also higher than the national nonsmoker cotinine levels given in NHANES III, developed by the Centers for Disease Control and Prevention. Statistical analysis yielded evidence of a significant increase in  $\ln(\text{cotinine})$  from pretest to posttest for both the bar and restaurant but not the control site, demonstrating that the control was the only site where the mean response was not significantly different than zero

The findings in this study show that nonsmokers exposed to secondhand smoke at outdoor bars and restaurants have measurable differences in the pre and post-exposure salivary cotinine levels. The results can add valuable information to the current literature concerning exposure to SHS outdoors.

### **Measuring PM<sub>2.5</sub> and carbon monoxide in real-time from secondhand smoke at outdoor bars and family restaurants in Athens, GA**

The results of this study demonstrate that PM<sub>2.5</sub> and CO, both markers of SHS, are higher in magnitude at outdoor areas where cigarette smoking is actively occurring compared to outdoor

areas with little to no active cigarette smoking. Mean pollutant levels were higher at the bar and restaurant site than the corresponding control sites over the 6-hour study period. This corresponds to a higher number of smokers at the bar and restaurant sites than at the control sites. The bar site also had a higher average number of smokers than the restaurant resulting in a higher level of pollutants than the restaurant site. These trends indicate that outdoor cigarette smoking results in elevated levels of SHS related pollutants and areas with higher numbers of smokers also have elevated levels of pollutants.

The findings of this study show that  $PM_{2.5}$  and CO concentrations are elevated at outdoor bars and restaurants where smoking is permitted.

### **Thesis Summary**

Exposure to SHS has harmful effects on children and adults and has been deemed unsafe at any level by the United States Surgeon General. Due to the published health effects, indoor smoking bans at bars and restaurants have been enforced which has resulted in an increased frequency of outdoor smoking. This raises the question of nonsmoker exposure in the outdoor environment.

By using a biological marker, investigators can obtain a quantitative and subjective measurement of exposure to SHS. In the case of cigarette smoke, cotinine is a metabolic product of nicotine and can be measured by sampling blood, saliva, or urine. Cotinine has a relatively long half-life of 17-hours making it an excellent tool for determining exposure to SHS. Air pollutants, such as  $PM_{2.5}$  and CO, have been found to be associated with cigarette smoking and can also be measured to determine the amount of SHS in an area.

Interest in SHS exposure has become a growing concern to the public and also public health officials. It has global, national, and local implications and has an effect on all members

of society. Through the development and execution of scientific studies, scientists will be able to determine if exposure to SHS in the outdoor environment poses any health risks to the general public.

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