Physiological Effects of Infant Exposure to Environmental Tobacco Smoke: A Passive Observation Study

Mary Beth Flanders-Stepans, Ph.D., RN
Sara G. Fuller, Ph.D., RN, CS, CPNP, FAAN

MARY BETH FLANDERS-STEPANS is an Assistant Professor in the School of Nursing at the University of Wyoming in Laramie, Wyoming. SARA FULLER is an Associate Professor in the College of Nursing at the University of South Carolina in Columbia, South Carolina.

Abstract

This study explored infant physiologic responses of exposure to environmental tobacco smoke (ETS) using a longitudinal passive observation study with a control group. Fifteen smoking and 15 non-smoking mothers were initially contacted in hospital maternity units, with home visits made when their infants were 2, 4, and 6 weeks old. Exposure to ETS was measured using infant urinary nicotine and cotinine levels. The physiologic effects of infant ETS exposure were measured by rectal temperature, pulse rate, respiratory rate, blood pressure, and oxygen saturation. The smoking mothers in this sample were poorer, had less education, and were less likely to be married than the mothers who did not smoke. At birth, the infants of smoking mothers had higher diastolic blood pressure than infants of non-smoking mothers ($p < .008$). Mothers who smoke cigarettes should be educated that maternal smoking behavior can affect an infant's cardiovascular function. Parents should also be counseled about the risks of smoking in close proximity and/or in an enclosed space with an infant, especially in a motor vehicle.

Journal of Perinatal Education, 8(1), 10–21; environmental tobacco smoke, infant care, perinatal education.

Exposure to environmental tobacco smoke (ETS) poses a significant risk to the health of children (Couriel, 1994;

In the United States, 43% of the children aged 2 months to 11 years live in households with adults who smoke.

In 1987, maternal smoking was associated with \$661 million in medical expenditures for respiratory conditions in children.

U.S. Environmental Protection Agency [EPA], 1992). In the United States, 43% of the children aged 2 months to 11 years live in households with adults who smoke (Pirkle, et al, 1996). This situation may worsen since the prevalence of women smokers increased from 22.8% in 1990 (Morbidity and Mortality Weekly Report, 1994) to 23.1% in 1994 (Kendrick & Merritt, 1996). Childbirth educators are in a unique position to educate women about the consequences of smoking during and after pregnancy.

In children, exposure to ETS increases the mortality from lower respiratory tract illnesses and morbidity from otitis media, bronchitis, pneumonia, tympanotomies, tonsillectomies, and adenoidectomies (DiFranza & Lew, 1996a). ETS exposure also increases the risk of childhood cancer (Pershagen, 1989; Sternfeldt, Berglund, Lindstrom & Ludvigsson, 1986), the development of allergy symptoms (Marini, Agosti, Motta & Mosca, 1996), gastrointestinal reflux (Alaswad, Toubas & Grunow, 1996), Sudden Infant Death Syndrome (SIDS) (DiFranza & Lew, 1996b; Haglund & Cnattinguis, 1990; Schoendorf & Kiely, 1992), laryngospasm (Lakshmipathy, Bokesch, Cowen, Lisman & Schmid, 1996), asthma (Cunningham, O'Connor, Dockery & Speizer, 1996; Ehrlich, et al., 1992), respiratory tract irritations, and lung function impairment (Cunningham, Dockery & Speizer, 1994; EPA, 1992). In addition, there is a positive relationship between maternal smoking and SIDS (Buescher, 1992; EPA, 1992). Estimates in 1987 indicated that exposure from maternal smoking was associated with \$661 million in medical expenditures for respiratory conditions in children (Stoddard & Gray 1997). Even though expo-

Within this sample, diastolic blood pressure at birth was higher for infants of smoking mothers than for infants of non-smoking mothers.

sure to ETS poses a significant risk to children and the mother's smoking pattern is highly related to the child's exposure (EPA, 1992), little is known about the physiologic effects of ETS in children.

Purpose

To investigate the problem of infant exposure to ETS, this study aimed to

1. compare the physiologic effect of exposure to ETS on rectal temperature, pulse, respiration, blood pressure, and oxygen saturation in infants of smoking and non-smoking mothers and
2. explore the relationships among levels of infant exposure to ETS based on infant outcome measures (urinary nicotine and cotinine) and physiologic measures (rectal temperature, pulse, respiration, blood pressure, and oxygen saturation).

The term *infant* used throughout the text of this article refers to those between birth and 6 weeks of age. Neuman's Systems Model was used to guide this study of infant exposure to ETS.

Neuman's Systems Model

Neuman's Model is an open systems model of stress and reaction to stress. Figure 1 provides a wedged view of the concentric circles used by Neuman (1989) to represent an open system in total interface with the environment, including an explanation of an individual's lines of defense. This figure symbolizes the physiologic response of the infant to environmental tobacco smoke. Nicotine and carbon monoxide are considered stressors within this model. The hypothesized "core response" is based upon the literature review.

Review of Literature

ETS is complex. To thoroughly understand the phenomenon of exposure to ETS in children, the human physiologic effects of various components of cigarette smoke and children's health problems related to exposure to ETS are explored.

Human Physiologic Effects. The majority of research to date elucidates the effects of cigarette smoke

Figure 1 Exposure to Environmental Tobacco Smoke Using Neuman's Systems Model

on animal models and adult humans. Cigarette smoke contains thousands of elements comprising more than 4,000 substances (Henningfield, 1986). Cigarette smoke causes the macrophages and polymorphonuclear leukocytes in the lung alveoli to become filled with pigmented residues, thereby reducing the phagocytic action of the macrophages and reducing the antibactericidal activity of these defense cells (Gardner, 1982). This contributes to the increased incidence of bronchitis and pneumonia in those exposed to ETS. In addition, components of tobacco smoke cause tissue activation and play a role in carcinogenesis by attaching to DNA or proteins (Diana, 1992). Studies of the bronchial mucosa indicate that those who smoke cigarettes exhibit epithelial hyperplasia, loss of cilia, mucous gland hypertrophy, and nuclear atypia, as well as enlarged goblet cells (U.S. Center for Disease Control [USCDC], 1986). Two specific components of cigarette smoke—nicotine and carbon monoxide—are known to have direct negative effects on the health of adults.

Nicotine is carried by the circulatory system to the sympathoadrenal system where it causes the release of biogenic amines: epinephrine, norepinephrine, dopamine, and acetylcholine (Summers, Lippiello & Giacobini, 1996). In addition, nicotine stimulates other endocrine glands, causing a sudden release of glycogen (O'Brien & Cohen, 1984). Acute effects of nicotine in adults include a) increased heart rate; b) increased systolic and diastolic blood pressure; c) decreased fingertip temperature (an average reduction of 7 degrees Fahrenheit), reflecting decreased peripheral perfusion; and d) increased heat production, oxygen consumption, basal metabolic rate, and intestinal peristalsis (Dawson, Vestal & Jusko, 1984; Jackson & Holle, 1985).

Cotinine, the major metabolite of nicotine, is pharmacologically inactive (Benowitz, et al., 1983). Cotinine in infants' urine is an accurate measure of chronic exposure to cigarette smoke (Knight, Eliopoulos, Klein, Greenwald & Koren, 1996; Labrecque, Marcoux, Weber, Fabia & Ferron, 1989; Luck & Nau, 1984a,

1984b, Luck & Nau, 1987; Woodward, Grgurinovich & Ryan, 1986). In addition, levels of cotinine in children are positively correlated with maternal smoking and with the number of smokers in the home (U.S. Department of Health and Human Services [USDHHS], 1981; Coultas, Howard, Peake, Skipper & Samet, 1987).

Carbon monoxide (CO) comprises 5% of the content of tobacco smoke (Benowitz, 1990). CO is absorbed into the alveoli through passive diffusion, causing an increase in carboxyhemoglobin in the circulatory system. It competes intensely with oxygen for the heme binding sites since the heme bond with CO is 200 times stronger than the bond formed with oxygen (Marieb, 1992; National Research Council [NRC], 1986). This results in a functional anemia, which decreases the amount of hemoglobin available for oxygen transport (McGrath, 1982). McGrath (1982) reported that exposure to CO resulted in decreased blood pH, increased blood glucose, and increased lactate and pyruvate levels in blood and brain tissue. Since the affinity of heme for CO is so high, CO is not easily eliminated from the body. However, levels of CO are so variable with exposure to tobacco smoke that the NRC (1986) concluded that CO and carboxyhemoglobin are not useful as indicators of exposure to ETS in adults. In addition, levels of CO are affected by other sources of exposure such as automobile exhaust.

Exposure to ETS During Childhood. Studies investigating the effect of cigarette smoking on human infants have tended to be epidemiological rather than physiological in nature. Consequently, the effects of ETS on DNA, bronchial mucosa, macrophages, oxygenation, and the sympathoadrenal system in adults led to speculation about ETS exposure as a cause of health problems in children. Bacteria-induced local deficiencies of the immune barrier of the upper respiratory tract may contribute to the development of atopic diseases (Kilian, Husby, Host & Halken, 1995). Levels of IgA1 protease-producing bacteria were found to be significantly related to passive smoking, "which may lead to allergic sensitization in otherwise predisposed infants" (Kilian, Husby, Host & Halken, 1995, p. 86). Stoddard and Miller (1995) estimated that maternal smoking was responsible for approximately 380,000 cases of childhood asthma/wheezing-induced lower respiratory tract illness (7.5% of the total number) in 1987. Reduced lung function caused by exposure to ETS during the first few years

of life may persist into childhood and may affect the pulmonary function measured throughout life (Cunningham, Dockery & Speizer, 1994). Families must then cope with increased health care costs stemming from the need for medical therapy and emergency department visits (Cunningham, O'Connor, Dockery & Speizer, 1996; Stoddard & Gray, 1997). In a review of the epidemiological evidence, Anderson and Cook (1997) discovered that maternal smoking behavior increases the risk of SIDS twofold.

Preliminary evidence of the physiologic effects of ETS in infants has focused on the effects of maternal smoking on infants who are breastfed. Stepan and Wilkerson (1993) found that infants of smoking breastfeeding mothers had significantly higher oxygen saturation, respiratory rates, and rectal temperatures than did infants of non-smoking mothers. These authors hypothesized that the higher baseline oxygen saturation could be related directly to the higher respiratory rate. However, the presence of carboxyhemoglobin could provide an alternative explanation. Since pulse oximeters measure only hemoglobin saturation, hemoglobin saturated with CO instead of oxygen could produce a falsely elevated reading (Bazeman, Myers & Barish, 1997). Although strong conclusions about causation are not warranted from these data, there is some indication that the components of cigarette smoke in breast milk are stressors to infants.

The increased morbidity and mortality of infants of smoking mothers (American Academy of Pediatrics [AAP], 1997; Anderson & Cook, 1997; Gergen, Fowler, Maurer, Davis & Overpeck, 1998) is alarming. While there is strong agreement in the literature about the significant risks that passive smoking poses for infants, there is limited research exploring infants' actual physiologic responses to ETS.

Research Design

A longitudinal passive observational design with a control group was used. Infants were studied from birth to 6 weeks of age.

Population and Sample. Newborns enrolled in the study were born to women in the western region of the United States who smoked ($n = 17$) or women who did not smoke ($n = 19$). In order to be eligible for

participation in the study, infants must have been a) born in a hospital, b) at least 37 weeks gestational age (according to the Ballard scale), c) discharged from the hospital on the same day their mothers were, d) on oxygen for no more than 24 hours after birth, and e) bottle fed. In addition, they had to weigh at least 2268 grams (5 pounds) at birth, have had a total bilirubin of less than 15 mg/dl in the first week of life, and have had no significant postnatal problems. Mothers were eligible for participation in the study if they had no significant health problems and were discharged with their infants. Mothers were classified as a) "non-smoking" if they had not smoked a cigarette during the previous year or b) "smoking" if they currently smoked five or more cigarettes per day. Women who smoked one to four cigarettes per day were not invited into the study.

Participants were recruited by personal interviews on postpartum units. For each smoking mother recruited, a non-smoking mother was recruited so that the sample size in both groups remained equal at all times. Participants received \$40 upon completion of the study in recognition of the value of their time commitment.

Instrumentation

In congruence with the conceptual model, the mothers' smoking history and the following physiologic variables reflected the infants' core responses to exposure to ETS: a) rectal temperature, b) pulse rate, c) respiratory rate, d) blood pressure, and e) oxygen saturation. Infant urinary nicotine and cotinine levels served as a measure of exposure to ETS.

Maternal Smoking History. The mother's smoking history was collected using the Smoking Habits Questionnaire, which had eight questions that reviewed the number of cigarettes smoked and the number of other smokers in the household, as well as the infant's proximity to the mother when she smoked.

In addition, all mothers were asked to answer five questions on the Generic ETS Exposure Questionnaire so that the investigator became aware of infants in the comparison group who were exposed to ETS. Infants of the non-smoking mothers who answered "yes," "sometimes," or "usually" to any of the questions related to ETS were eliminated from the study. Both questionnaires were evaluated for reading level using RightWriter, ver-

sion 2.1 (1987), and were found to be at grade level 7. After content validity was established by four experts in nursing research, the questionnaires were pilot tested to determine clarity and time required for completion.

A brief infant feeding history was taken so that the investigator was aware of infants who had eaten foods known to contain nicotine. These data were collected even though it would be extremely unusual (but not impossible) for infants up to 6 weeks of age to eat foods such as tomatoes, potatoes, black tea, and cauliflower, which are known to contain nicotine (Davis, Stiles, DeBethizy & Reynolds, 1991).

Core Responses

Physiologic measures were recorded in the same sequence for each infant while the infant was in a quiet state. Pulse rate, oxygen saturation, and blood pressure were recorded first. Multiple measurements were taken so the mean could be entered into the statistical analysis.

Pulse and Oxygen Saturation. The Nellcor N-10 pulse oximeter was used to determine pulse rate and oxygen saturation. This pulse oximeter is accurate within 2% when the saturation is between 70 and 100% (Nellcor Incorporated, 1986). Adhesive sensors suitable for infants weighing 3 to 15 kg were placed on each infant's left great toe. Accuracy of the pulse oximeter in sensing heart rate was validated with the pulse rate from the Critikon's DINAMAP Neonatal Vital Signs Monitor (Critikon, Inc., 1984). Pulse and oxygen saturation rates were measured continuously for 10 minutes. Ten readings were recorded every 60 seconds and were averaged.

Blood Pressure. Blood pressure was taken with the DINAMAP monitor, which measures systolic, diastolic, and mean arterial blood pressure, as well as pulse rate (Critikon, Inc., 1984). The circumference of each infant's arm was measured in order to select an appropriate cuff size. Blood pressure was taken four times every 3 to 5 minutes during each visit. The results were averaged. The monitor was calibrated using the manufacturer's instructions.

Respirations. Respirations were counted for 60 seconds using a stethoscope. While the infant was lying in a supine position, the diaphragm was placed to the right

of the infant's sternum. Respiratory rate was measured 10 times over 10 to 15 minutes and was then averaged.

Rectal Temperature. Rectal temperature was taken as a reflection of core temperature. At the time data were collected, few studies reported that tympanic thermometers measured newborn core temperature accurately (Terndrup, 1992). Therefore, rectal temperature was measured using mercury-in-glass thermometers with bulb ends. Only the bulb of the thermometer was inserted into the rectum and was held in place for at least 1 minute or until the temperature stopped rising (Morley, Hewson, Thornton & Cole, 1992). Since rectal temperatures are not prone to fluctuation, temperature was measured only once. To ensure that thermometers used in the study were accurate, 10 thermometers were placed in the same container of warm water at the same time. Any thermometer having a reading 0.2 degrees Fahrenheit or more different from the other thermometers was not used in the study. Once data collection was completed on an infant, the thermometer was given to the infant's mother.

Urine Nicotine and Cotinine. Urine was collected by placing two cotton balls in the diaper close to the urethral opening. The urine was squeezed out of the cotton balls, placed in polypropylene plastic containers, and frozen at -25 degrees Celsius.

Capillary gas-liquid chromatographic assay was used to determine levels of nicotine and cotinine in urine samples. Analysis of urine samples was done using the model 5890A Hewlett-Packard gas chromatograph equipped with a nitrogen phosphorus detector and a fused-silica capillary column inlet. A model 3393A Hewlett-Packard integrator was utilized for peak area counts. Statistical analysis of the calibration curve revealed an adjusted r^2 of .98 for cotinine before and after freezing the samples.

The analyses of the calibration curves for nicotine revealed an adjusted r^2 of .96 before the samples were frozen but .15 after the samples were frozen. These results are consistent with the findings of Lequang, Rousel, and colleagues (1994) who found cotinine to be much more stable than nicotine. In addition, the test tubes in which the urine samples were stored were contaminated with ambient nicotine. (ETS apparently had seeped into the storage cupboards from cigarettes smoked in an outside smoking area located beneath the

office window.) Evidence of this contamination became apparent when unused test tubes were analyzed for nicotine content after data were collected. Therefore, the results of the nicotine analysis are not reported.

Because the quantity and concentration of urine was different for each collection, creatinine concentration was determined using the Abbott Vision Proclaim System. Results are expressed as a cotinine to creatinine ratio.

Human Subjects. The research proposal and informed consent form were submitted to the University of South Carolina and University of Wyoming Institutional Review Boards, as well as to the review boards at Iverson Memorial Hospital in Laramie, Wyoming, and United Medical Center in Cheyenne, Wyoming. All four review boards approved the research project.

After eligibility was determined using a solicitation guide and informed consent was obtained, baseline data were collected in the hospital by the investigator. Additional data were collected in the homes of the mother-infant pairs at 2, 4, and 6 weeks after delivery.

Data Analysis

Statistical analyses were conducted using the SAS statistical program. The level of significance was set at 0.05 using the Bonferroni adjustment to control for Type I error (Marascuilo & Serlin, 1988). This control was crucial since the same sample was used for all analyses and the variables were related to one another. The analyses included t-tests, Pearson product correlation, and trend analysis.

Description of Population and Sample

The infants enrolled in the study were born to women who smoked ($n = 17$) and women who did not smoke ($n = 19$). However, two of the women who smoked dropped out of the study, while four of the infants of non-smokers were eliminated from the study because they were exposed to ETS. Therefore, there were 15 in each group. The remaining smokers had less education (11.4 years), were less likely to be married (47% married), and had less income (\$10,000–\$19,000 per year) than those remaining in the non-smoking group. All participants in the study were Caucasian. This is a reflec-

tion of limited ethnic diversity in southeastern Wyoming where data were collected. It would be important not to generalize these results because African Americans are thought to metabolize cotinine differently than Caucasians do [Perez-Stable, Herrera, Jacob & Benowitz, 1998]. The infants of smokers and non-smokers in this study did not differ significantly in birthweight, gestational age, or APGAR scores. (See Table 1.)

Physiological Effects. Cotinine levels in the urine of infants of smoking mothers were higher than were levels in urine of infants of non-smoking mothers at all points except when the infants were 4 weeks of age. At this data point, the cotinine levels were higher, but the standard deviation was exceptionally high (1,032.96), which was more than 15 times greater than the means at other time periods due to an outlier in the group of infants of smoking mothers. One infant had a cotinine level of 3,995 ng/mg creatinine. When this outlying value was removed from the analysis, the p value was reduced to 0.0291. The baby having the exceptionally high cotinine level was diagnosed with "smoker's cough" and admitted to the hospital 2 days after the 4-week home visit. The data collector also watched as the mother put her finger into the baby's mouth as a "pacifier" directly after smoking a cigarette and without washing her hands.

Infants of smoking mothers had significantly higher

mean diastolic blood pressures at birth ($p < .008$, Bonferroni corrected) than those whose mothers did not smoke. (See Table 2.) There were no differences in this sample between the infants of smoking mothers and the infants of non-smoking mothers on diastolic blood pressure at 2, 4, and 6 weeks of age or on the other physiologic variables (i.e., temperature, pulse, respiration, systolic blood pressure, and oxygen saturation) at birth, 2, 4, and 6 weeks of age. In addition, there was no relationship between the physiologic measures and cotinine levels.

Discussion

Further evidence that the lines of resistance are activated through exposure to the products of cigarette smoke are found in the difference in urinary cotinine levels between the two groups of infants. Levels of urinary cotinine in the infants of smoking mothers ranged from 0 to 3,995 ng/mg creatinine over 6 weeks, while the range for infants of non-smokers ranged from 0 to 40 ng/mg creatinine. The urine samples of only 2 out of 56 infants of smoking mothers were negative for cotinine, while nearly all urine samples of the infants of non-smoking mothers (47) were negative for cotinine. If the outlying case having a concentration of cotinine of 3,995 ng/mg creatinine is removed, the range narrows (0 to 1,270 ng/mg creatinine). The range in this study is higher than that found

TABLE 1 Birth Information Comparing Infants of Smokers to Infants of Non-smokers

Parameter	M	SD	t	p
BIRTHWEIGHT			-.77 (1, 28)	0.45
Infants of Smokers	2999 g	436.21		
Infants of Non-smokers	3111 g	359.07		
GESTATIONAL AGE			-.49 (1, 27)	0.63
Infants of Smokers	38.67 wk	1.23		
Infants of Non-smokers	38.93 wk	1.64		
ONE-MINUTE APGAR SCORE			.44 (1, 28)	0.66
Infants of Smokers	7.93	0.08		
Infants of Non-smokers	7.73	1.53		
FIVE-MINUTE APGAR SCORE			1.00 (1, 28)	0.33
Infants of Smokers	9.00	0.00		
Infants of Non-smokers	8.93	2.58		

TABLE 2 t-tests Comparing Infant Outcome Measures in Infants of Mothers Who Smoke With Infants of Mothers Who Do Not Smoke

Parameter	M	SD	t	p
COTININE				
<i>Birth</i>		191.24	5.05	.0002
		5.01	(1, 21)	
Infants of Smokers	257.75 ng/mg**			
Infants of Non-smokers	2.78 ng/mg**			
<i>2 Weeks</i>		345.21	2.53	.0179*
		13.30	(1, 28)	
Infants of Smokers	231.47 ng/mg**			
Infants of Non-smokers	7.67 ng/mg**			
<i>4 Weeks</i>		342.2	1.71	.0291
		1.29	(1, 28)	
Infants of Smokers	203.6 ng/mg**			
Infants of Non-smokers	0.33 ng/mg**			
<i>6 Weeks</i>			3.04	.0053**
			(1, 27)	
Infants of Smokers	273.86 ng/mg**			
Infants of Non-smokers	0.33 ng/mg**			
DIASTOLIC BLOODPRESSURE				
<i>Birth</i>			3.1535 (1, 28)***	0.0038***
Infants of Smokers	45.40 mmHG	6.59 mmHG		
Infants of Non-smokers	38.27 mmHG	5.78 mmHG		
<i>2 Weeks</i>			-1.0749 (1, 27)	0.2919
Infants of Smokers	43.14 mmHG	9.43 mmHG		
Infants of Non-smokers	46.67 mmHG	8.22 mmHG		
<i>4 Weeks</i>			1.2381 (1, 28)	0.2259
Infants of Smokers	52.80 mmHG	7.60 mmHG		
Infants of Non-smokers	49.06 mmHG	8.87 mmHG		
<i>6 Weeks</i>			-0.8414 (1, 28)	0.4073
Infants of Smokers	51.47 mmHG	9.25 mmHG		
Infants of Non-smokers	54.20 mmHG	8.53 mmHG		

* p < .05

** Cotinine is expressed in terms of nanograms per milligram of creatinine.

*** p < .008

by Greenberg and colleagues (1991) in 3-week-old infants in North Carolina. Greenberg reported the median cotinine concentration as 79 ng/mg of creatinine with a range of 9 to 643 ng/mg. However, only 38% of the mothers in Greenberg's study smoked.

Mean cotinine levels in infants of smoking mothers were highest at birth (258 ng/mg creatinine) and 6 weeks (274 ng/ml). One would expect the cotinine levels to be highest at birth since all the smoking mothers smoked

during pregnancy. It is not surprising, however, that the cotinine levels at 6 weeks had increased since two thirds of the infants were sleeping in the living room during the day where the exposure was probably higher than in the bedrooms. These results are consistent with those of Greenberg and colleagues (1991) who found an increase in cotinine excretion during the first year of life.

Cotinine is a measure of chronic exposure. This bio-marker can show considerable variability from person

to person due to differences in uptake, distribution, and metabolism (Leaderer, 1990). The results in this study further confirm the considerable variability of cotinine levels even in infants who are the same age. When the outlying value was removed, the standard deviation for cotinine among infants of smoking mothers ranged between 191 ng/mg and 349 ng/mg creatinine. The mother of the infant who had the extremely high level of cotinine was a teenager who had many smokers as friends. When data were collected during the 2-week visit, three friends were present in a very small trailer. They were all smoking as they watched a movie. The infant's cotinine level during this visit was 1,170 ng/mg creatinine, while the level at the 4-week visit was 3,995 ng/mg creatinine, indicating chronically high levels of exposure. The infant's cotinine level dropped dramatically (161 ng/mg creatinine) at the 6-week visit once the mother and infant were taken together into foster care of parents who did not allow smoking in their house.

The infants of non-smoking mothers who had small amounts of cotinine in their urine are of interest. One infant having a urinary cotinine level of 66 ng/mg creatinine at birth was born to a mother who was exposed to ETS in her work place. (She did not return to work until after data collection was complete.) The mothers of three other infants with small levels of cotinine (trace, trace, and 24 ng/mg creatinine) in their urine reported at the time of data collection that a grandparent who smoked had cared for the infants for a short period of time in the infant's own home. These infants were retained in the study because the mothers stated that the grandparent had definitely not smoked around the baby at all or in the same house with the baby even though the grandparents did have the odor of cigarette smoke on their clothing. Thus, it appears that babies can get some nicotine exposure by merely being around those with nicotine on their clothing.

When the data were analyzed for each individual data collection point (birth, 2, 4, and 6 weeks), diastolic blood pressure at birth was higher for infants of smoking mothers than for infants of non-smoking mothers. (See Table 2.) The higher mean diastolic blood pressure (45.4 mmHg) at birth in infants of smokers when compared to the diastolic blood pressure of infants of non-smokers (38.27) cannot be attributed to passive exposure to ETS but rather to *in utero* exposure. However, this finding supports Beratis, Panagoulas and Varvarigou (1996)

who determined that there was a significant positive correlation between the number of cigarettes smoked by mothers during pregnancy and the blood pressure of the neonates in their sample. Speaking in Neuman's terms, the difference in diastolic blood pressure among infants of smoking mothers is an indication that the products of cigarette smoke do penetrate the flexible line of defense (placenta) and the normal line of defense (cardiovascular system), causing the lines of resistance to be activated through an increase in blood pressure. (See Figure 1.)

There were no consistent results across the first 6 weeks to support the research hypothesis that there was a relationship between infant physiologic measures and infant urinary cotinine levels. When the data were analyzed for each data collection point, the results varied from week to week. Thus, while there may well be physiologic changes in infants exposed to ETS that account for their higher levels of illness, those measures have not been captured in this study.

Implications for Perinatal Education

Smoking behavior during pregnancy may cause changes in the cardiovascular functioning of infants, while exposure to ETS is associated with illness. Therefore, the following recommendations are made to those who counsel smoking mothers:

1. Encourage pregnant women to stop smoking. According to Windsor and colleagues (1993), the most effective smoking cessation methods for pregnant women include a health education counseling session at the first prenatal visit followed by use of a self-help manual. In addition, reinforcement at follow-up visits with social support in the form of a "buddy letter, a buddy contract, and a buddy tip sheet," as well as a newsletter with testimonials from successful quitters, has been successful in increasing the unit rate among pregnant women (Windsor, Lowe, et al., 1993, p. 202).
2. If pregnant women cannot quit, provide them with strategies to cut down since the negative outcomes for newborns seem to be dose related (Eskenazi, Prehn & Christianson, 1995). If a new mother cannot quit smoking after her infant is born, encourage her to smoke outside away from the baby.

One woman in this study who faithfully smoked outside reduced her infant's cotinine level from 238 ng/mg creatinine at birth to just a trace during the following weeks.

3. Caution mothers never to smoke in the car, an enclosed space. One of the infants in this study had cotinine levels 5 times higher than the two previous levels after accompanying her parents on an 8-hour car trip.
4. If smoking is restricted to one room in the house, mothers should know that restricting smoking to one room having a door that can be closed may not be as effective as they might think. The young woman who restricted smoking to the garage lived in a basement apartment and did not protect her infant from the exposure to ETS. Her baby's mean cotinine level was 632 ng/mg creatinine, while the mean for all babies of smoking mothers across 6 weeks was 304 ng/mg creatinine.

Recommendations for Future Research

Generalizability of the results of this study to other populations of infants is limited because of the small sample size and data collection at high altitude (>5000 feet). Studies should be conducted to determine whether infants born to smoking mothers at lower altitudes have elevated diastolic blood pressure or whether this finding is limited to infants born at high altitudes. Studies should be conducted to determine the extent to which chronic changes in blood pressure of infants of smoking mothers occur later and have an effect on cardiovascular functioning later in life. Intervention studies can be conducted determining whether it is possible to reduce infant exposure to ETS by restricting smoking behavior to one room the infant does not occupy. Maternal protective behaviors could be explored. For example, if smoking occurs in one room only, does opening a window a crack significantly lower ETS exposure as measured by urinary cotinine? The use of Neuman's Systems Model to organize the study of the interaction of the infant, the mother, her smoking behavior, and the environment would provide nurses with more strategies to protect infants from ETS.

Summary

The problem of infant exposure to ETS is complex. There are many environmental conditions that have an effect

upon the level of exposure. For example, maternal smoking in an enclosed area (small trailer or car) immensely increases infant exposure. In addition, smoking during pregnancy may affect infant cardiovascular functioning (increased diastolic blood pressure). Efforts to educate women about the possible health consequences their infants may endure if exposed to ETS and provide them with strategies to decrease exposure must continue.

Acknowledgment

This research was funded in part by the National Institute of Nursing Research, fellowship number 1F31 NR06990-01. The Sigma Theta Tau Chapter, Alpha Pi, also provided a nursing research grant. In addition, Johnson & Johnson Medical Incorporated loaned a DINAMAP 8100 blood pressure monitor.

References

- Alaswad, B., Toubas, P. & Grunow, J. (1996). Environmental tobacco smoke exposure and gastroesophageal reflux in infants with apparent life-threatening events. *Journal of the Oklahoma State Medical Association (FH3)*, 89(7), 233-237.
- Benowitz, N. (1990). Pharmacokinetic considerations in understanding nicotine dependence. In Ciba Foundation Symposium 152. *The biology of nicotine dependence*. New York: John Wiley & Sons.
- Benowitz, N. & Jacob, P. (1984). Daily intake of nicotine during cigarette smoking. *Clinical Pharmacology and Therapeutics*, 35, 499-504.
- Benowitz, N., Jacob, P., Jones, R. & Rosenberg, J. (1982). Interindividual variability in the metabolism and cardiovascular effects of nicotine in man. *The Journal of Pharmacology and Experimental Therapeutics*, 221, 368-372.
- Benowitz, N., Kuyt, F., Jacob, P., Jones, R. & Osman, A. (1983). Cotinine disposition and effects. *Clinical Pharmacology Therapeutics*, 34, 604-611.
- Beratis, N., Panagoulas, D. & Varvarigou, A. (1996). Increased blood pressure in neonates in infants whose mothers smoked during pregnancy. *Journal of Pediatrics*, 128(6), 806-12.
- Buescher, P. (1992). CHES analysis links smoking and infant mortality. *Infant mortality monitor: North Carolina governor's commission on reduction of infant mortality*, 2(2), 1.
- Coultas, D., Howard, C., Peake, G., Skipper, B. & Samet, J. (1987). Salivary cotinine levels and involuntary tobacco smoke exposure in children and adults in New Mexico. *American Review of Respiratory Disease*, 136, 305-309.

Infant Exposure to Environmental Tobacco Smoke: A Passive Observation Study

- Coultas, D., Samet, J., McCarthy, J. & Spengler, J. (1990). Variability of measures of exposure to environmental tobacco smoke the home. *American Review of Respiratory Disease*, 142, 60-2606.
- Couriel, J. (1994). Passive smoking and the health of children. *Thorax: The Journal of the British Thoracic Society*, 49(8), 731-734.
- Critikon, Inc. (1984). *Dinamap adult/pediatric and neonatal vital signs monitor model 1846 operation manual*. Tampa, Florida: Critikon, Inc.
- Cunningham, J., Dockery, D. & Speizer, F. (1994). Maternal smoking during pregnancy as a predictor of lung function in children. *American Journal of Epidemiology*, 139(12), 1139-1152.
- Cunningham, J., O'Connor, G., Dockery, D. & Speizer, F. (1996). Environmental tobacco smoke, wheezing, and asthma in children in 24 communities. *American Journal of Respiratory Critical Care Medicine*, 153, 218-224.
- Davis, R., Stiles, M., DeBethizy, J. & Reynolds, J. (1991). Dietary nicotine: A source of urinary cotinine. *Food and Chemical Toxicology*, 29(12), 821-827.
- Dawson, G., Vestal, R. & Jusko, W. (1984). In D. J. Balfour (Ed.), *Nicotine and the tobacco smoking habit*. New York: Pergamon Press.
- Diana, J. (1992). Tobacco smoking and nutrition. In J. N. Diana & W. R. Pryor (Eds.), *Tobacco smoking and nutrition: Influence of nutrition on tobacco associated health risks*. *Annals of the New York Academy of Sciences*, 686, 1-11.
- DiFranza, J. & Lew, R. (1996a). Morbidity and mortality in children associated with the use of tobacco products by other people. *Pediatrics*, 97(4), 560-567.
- DiFranza, J. & Lew, R. (1996b). Effect of maternal cigarette smoking on pregnancy complications and sudden infant death syndrome. *Journal of Family Practice*, 40(4), 385-394.
- Ehrlich, R., Dattan, M., Godbold, J., Saltzberg, D., Grimm, K., Landrigan, P. & Lilienfeld, D. (1992). Childhood asthma and passive smoking. *American Review of Respiratory Disease*, 145, 594-599.
- Eskenazi, B., Prehn, A. & Christianson, R. (1995). Passive and active maternal smoking as measured by serum cotinine: The effect on birthweight. *American Journal of Public Health*, 85(3), 395-398.
- Gardner, D. (1982). Effects of gases and airborne particles on lung infections. In J. J. McGrath & C. D. Barnes (Eds.), *Air pollution—physiological effects*. New York: Academic Press.
- Greenberg, R., Bauman, K., Strecher, V., Keyes, L., Glover, L., Haley, N., Stedman, H. & Loda, F. (1991). Passive smoking during the first year of life. *American Journal of Public Health*, 81(7), 850-853.
- Haglund, B. & Cnattinguis, S. (1990). Cigarette smoking as a risk factor for sudden infant death syndrome: A population-based study. *American Journal of Public Health*, 80, 29-32.
- Henningfield, J. (1986). Nicotine an old-fashioned addiction. *The Encyclopedia of Psychoactive Drugs*. New York: Chelsea House Publishers.
- Jackson, F. & Holle, R. (1985). Smoking perspectives 1985. *Primary Care*, 2, 197-216.
- Kilian, M., Husby, S., Host, A. & Halken, S. (1995). Increased proportions of bacteria capable of cleaving IgA1 in the pharynx of infants with atopic disease. *Pediatric Research*, 38, 182-186.
- Kendrick, J. & Merritt, R. (1996). Women and smoking: An update for the 1990s. *American Journal of Obstetrics and Gynecology*, 175(3), 528-535.
- Knight, J., Eliopoulos, C., Klein, J., Greenwald, M. & Koren, G. (1996). Passive smoking in children: Racial differences in systemic exposure to cotinine by hair and urine analysis. *Chest*, 109(2), 446-450.
- Labrecque, M., Marcoux, S., Weber, J., Fabia, J. & Ferron, L. (1989). Feeding and urine cotinine values in babies whose mothers smoke. *Pediatrics*, 83(1), 93-9.
- Lakshmipathy, N., Bokesch, P., Cowen, D., Lisman, S. & Schmid, C. (1996). Environmental tobacco smoke: A risk factor for pediatric laryngospasm. *Anesthesia and Analgesia*, 82(4), 724-727.
- Leaderer, B. (1990). Assessing exposures to environmental tobacco smoke. *Risk Analysis*, 10(1), 19-26.
- Luck, W. & Nau, H. (1984a). Exposure of the fetus, neonate, and nursed infant to nicotine and cotinine from maternal smoking. *New England Journal of Medicine*, 311, 672.
- Luck, W. & Nau, H. (1984b). Nicotine and cotinine concentration in serum and milk of nursing mothers. *British Journal of Clinical Pharmacology*, 118, 9-15.
- Luck, W. & Nau, H. (1987). Nicotine and cotinine concentrations in the milk of smoking mothers: Influence of cigarette consumption and diurnal variation. *European Journal of Pediatrics*, 146, 21-26.
- Marascuilo, L. A. & Serlin, R. C. (1988). *Statistical methods for the social and behavioral sciences*. New York City, NY: W. H. Freeman and Company.
- Marieb, E. (1992). *Human anatomy and physiology*. Redwood City, CA: Benjamin/Cummings Publishing.
- Marini, A., Agosti, M., Motta, G. & Mosca, F. (1996). Effects of dietary and environmental prevention programme on the incidence of allergic symptoms in high atopic risk infants: Three years follow-up. *Acta Paediatrica Sinica Supplement (BD5)*, 414, 1-21.
- McGrath, J. (1982). Physiological effects of carbon monoxide. In J. J. McGrath & C. D. Barnes (Eds.), *Research topics in physiology: Air pollution—physiological effects*. New York: Academic Press.

- Morbidity and Mortality Weekly Report. (1994). Surveillance for selected tobacco-use behaviors—United States, 1900-1994. 43(SS-3), 1-42.
- Morley, C., Hewson, P., Thornton, A. & Cole, T. (1992). Axillary and rectal temperature measurements in infants. *Archives of Disease in Childhood*, 67(1), 122-125.
- National Research Council. (1986). *Environmental tobacco smoke: Measuring exposures and assessing health effects*. Washington, DC: National Academy Press.
- Nellcor Incorporated (1986). *User's manual: Nellcor N-10 Portable Pulse Oximeter*. Hayward, CA: Nellcor Incorporated.
- Neuman, B. (1989). *Neuman systems model*. Norwalk, CT: Appleton & Lange.
- O'Brien, R. & Cohen, S. (1984). *The encyclopedia of drug abuse*. New York: Facts on File Inc.
- Pershagen, G. (1989). Childhood cancer and malignancies other than lung cancer related to passive smoking. *Mutation Research*, 222, 129-135.
- Pirkle, J., Flegal, K., Bernert, J., Brody, D., Etzel, R. & Maurer, K. (1996). Exposure of the US population to environmental tobacco smoke: The third national health and nutrition examination survey, 1988 to 1991. *Journal of the American Medical Association*, 275(16), 1233-1240.
- Schoendorf, K. & Kiely, J. (1992). Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. *Pediatrics*, 90(6), 905-908.
- Stepans, M. & Wilkerson, N. (1993). Physiologic effects of maternal smoking on breastfeeding infants. *American Journal of Nurse Practitioners*, 5(3), 105-113.
- Sternfeldt, M., Berglund, K., Lindstrom, J. & Ludvigsson, J. (1986). *Lancet*, 1350-1352.
- Stoddard, J. & Gray, B. (1997). Maternal smoking and medical expenditures for childhood respiratory illness. *American Journal of Public Health*, 87(2), 205-209.
- Stoddard, J. & Miller, T. (1995). Impact of parental smoking on the prevalence of wheezing respiratory illness in children. *American Journal of Epidemiology*, 141(2), 96-102.
- Su, C. (1984). Actions of nicotine and smoking on circulation. In D. J. Balfour (Ed.), *Nicotine and the tobacco smoking habit*. New York: Pergamon Press.
- Terndrup, T. (1992). An appraisal of temperature assessment by infrared emission detection tympanic thermometry. *Annals of Emergency Medicine*, 21(12), 1483-1492.
- U.S. Center for Disease Control. (1986). *Health consequences of involuntary smoking: A report of the surgeon general*. Rockville, MD: U.S. Department of Health and Human Services. Office of Smoking and Health. DHHS(CDC) 87-8398.
- U.S. Department of Health and Human Services. (1981). *The health consequences of smoking: The changing cigarette. A report of the surgeon general*. Rockville, MD: Office on Smoking and Health. DHHS(PHS), 815-856.
- U.S. Environmental Protection Agency. (1992). *Respiratory health effects of passive smoking: Lung cancer and other disorders*. Washington, DC: Office of Health and Environmental Assessment Office of Research and Development.
- Windsor, R., Lowe, J., Perkins, L., Smith-Yoder, D., Artz, L., Crawford, M., Amburgy, K. & Boyd, N. (1993). Health education for pregnant smokers: Its behavioral impact and cost benefit. *American Journal of Public Health*, 83(2), 201-205.
- Windsor, R., Qing Li, C., Lowe, J., Perkins, L., Ershoff, D., & Glynn, T. (1993). The dissemination of smoking cessation methods for pregnant women: Achieving the year 2000 objectives. *American Journal of Public Health*, 83(2), 173-178.
- Woodward, A., Grgurinovich, N. & Ryan, P. (1986). Breastfeeding and smoking hygiene: Major influence on cotinine in urine of smokers' infants. *Journal of Epidemiology and Community Health*, 309-315.

The Search for Truth

All truth goes through three stages. First it is ridiculed, then it is violently opposed. Finally, it is accepted as self-evident.

—Schoepenhauer

Do not expect to arrive at certainty in every subject which you pursue. There are a hundred things wherein we mortals . . . must be content with probability, where our best light and reasoning will reach no farther.

—Issac Watts

A great deal of intelligence can be invested in ignorance when the need for illusion is deep.

—Saul Bellow