MEMORANDUM

TO: FILE

FROM: WILLIAM W. DAVIS

DATE: MAY 19, 1986

RE: AMERICAN LUNG ASSOCIATION/AMERICAN THORACIC SOCIETY ANNUAL MEETING, MAY 11-14, 1986, KANSAS CIY, MISSOURI

II. Community Education Session: Smoking or Health: State and Local Legislation on Involuntary Smoking

This session included a number of presentations by various ALA staff on the future challenges involved in state and local smoking restriction initiatives. Each participant of the conference received a notebook which included copies of the Florida, East Lansing and Contra Costa County "model" nonsmoking ordinances, legislative updates on smoking restrictions and excise tax bills on both the state and local levels, a table of contents from a book written by Hanauer, Barr and Glantz, entitled Legislative Approaches to a Smoke Free Society (American Nonsmokers Rights Foundation) and a list of members of the "Healthy Majority--A Coalition for Clean Indoor Air". (Attachment B)

This session provided several insights. ALA staffpersons are professional and well-organized, and their

strategies are becoming more sophisticated. Their stated aim is to "sandwich" smoking restriction ordinances within Clean Indoor Air Acts, thereby shifting focus and spreading the issue for potential opponents. They view the economic argument against smoking in the workplace as their strongest suit and they are presently working to achieve coalitions with both unions and minority groups. They tend to disassociate themselves with GASP and ASH members (the latter are "a bit too helpful") and their bible is a book entitled Smoke Free Society by Pertschuk et al. (Americans for Nonsmokers Rights).

McDermott distributed a handout which featured testimony by Dr. Philip Witorsch, member of the Indoor Air Pollution Advisory Group, and a rebuttal to that testimony by Repace. McDermott urges the ALA staff to use the Repace rebuttal to counter "industry" claims on "involuntary smoking". (Attachment C)

III. Poster Session - Pulmonary Epidemiology/Scientific Assembly on Environmental and Occupational Health

This session, despite its title, was devoted essentially to the analysis of ETS exposures and potential health effects. Ferris et al. (Harvard University), Tager et al., Lebowitz, Kauffmann et al., Samet et al., Higgens

and Schenker displayed papers related to ETS exposure and health effects. (See Attachment D)

The Harvard group of Ferris et al. evaluated the prevalence of respiratory symptoms for adults in the Six-City Study population. The study was meant to establish baseline for further investigations using smaller groups of nonsmokers. Of interest is the fact that individuals within the Kingston-Harriman area exhibited an exposure-response relationship between fine particulate exposure (outdoors) and incidence of wheeze, cough, chest illness and breathlessness.

Tager et al. presented a predictive model for growth of FEV_1 in children. A field survey of 483 children was compared with the model predictions. Factors such as maternal smoking, etc., when included in the model, predicted with some degree of accuracy the extent of pulmonary function growth reported in the data from the survey.

Paoletti (Lebowitz) et al. presented a paper on the influence of family pulmonary diseases on respiratory symptoms and lung function. The authors concluded that frequent early childhood exposure to familial pulmonary disease is associated with recurrent bronchitis and other pulmonary disease. Such recurrent diseases, prior to the age of 12, in combination with active smoking later in life, were claimed to be risk factors for respiratory disease and symptoms in adulthood.

Kauffmann et al. presented a comparison of respiratory symptoms between smokers and nonsmokers. Symptom prevalence was highest among active smokers and lowest among nonsmokers not exposed to ETS. However, when the results were broken down for individual symptoms (wheeze and breathlessness, for example) or for various flow rates, some of the trends between smokers and nonsmokers were significant, while others where not (See Abstract).

Burchfiel, (Higgens) et al., in a major followup study of 2661 children, found an increase risk of respiratory symptoms and a slower rate of lung growth among children exposed to parental smoking.

Coultas (Samet) et al. reported that cotinine levels in 1286 Hispanic subjects are significantly associated with the number of smokers per household. Salivary cotinine levels were assessed by radioimmunoassay.

Schenker et al., in a study of railroad workers, measured ambient nicotine levels using a personal nicotine monitor. Collection pads were treated with sodium bisulfate and the researchers claimed a 99% retention rate for nicotine. A correlation between respirable nicotine and the number of cigarettes smoked by the workers was reported. It was suggested that nicotine measurements "can be used to validate other measures of passive smoking".

Townsend et al. used data from 12,866 men in MRFIT to evaluate pulmonary function in smokers and ex-smokers.

The researchers reported steeper declines in FEV_1 among smokers compared to ex-smokers. They suggest that a middle-aged, healthy smoker who quits smoking could expect an FEV_1 rate of decline comparable to a never-smoker's.

IV. Joint Scientific/Community Session: Indoor Air Pollution in the Nonindustrial Environment

Jonathan Samet and John Spengler gave presentations on the general problem of indoor air pollution, particulates, combustion products, evaporative products, biological agents and radon. Environmental tobacco smoke was discussed at some length and the following points provide a summary of the discussion highlights:

1. Spengler claimed that while smoking is the major source of respirable particulates in the home, not much is known about the composition or nature of ETS (as distinguished from sidestream smoke). Interestingly, Spengler also discussed the Harvard Six-City Study on kerosene burner emissions (Kingston-Harriman) in which the burners, when lit, forced TSP concentrations to 130ug/m^3 and to an 80ug/m^3 average over the course of 24 hours. Samet similarly discussed a 1985 study by Honicky on the influence of woodburning stoves on the respiratory symptoms of children in Michigan. RSP/TSP concentrations were similar to those noted in the Harvard study. (See Attachment E)

2. Samet spoke directly to the issue of health effects from ETS exposures. He argued that since mainstream and sidestream smoker are similar in composition, one can extrapolate from the health effects of active smoking to the health effects from ETS exposures on the nonsmoker. Samet also claimed that "there is no reason to think ETS is not a carcinogen", that there is no "safe level of exposure" and that the fundamental question is, therefore, "how potent of a carcinogen is ETS and for how many deaths is it responsible?"

3. Samet also suggested, citing his own research, that cotinine is an effective measure of ETS exposure.

4. Samet said that health effects from ETS exposures fall into the following categories:

Upper Respiratory Disease
Lower Respiratory Disease
Respiratory Symptoms
Lung Function
COPD
Cancer, Lung and Other

5. Children, according to Samet, suffer from diseases of the lower respiratory track when exposed to ETS. He cited the work of Harlap (1974), Colley (1974), Rankatullio (1978), and Fergusson (1977). Lung function data, Samet argued, also "consistently" show an effect from ETS exposures

in children. He cited his own study of 1979 (West Pennsylvania) and the 1983 study by Tager et al.

6. Regarding lung cancer and ETS exposures, Samet characterized the Hirayama, Trichopolous, Correa, Knoth and Garfinkel (1985) studies as "positive" and the Garfinkel (1981), Gillis, Koo, Chan and Wu studies as "negative". However, Samet said that even the negative studies showed some positive trends and therefore ought to be interpreted as implicative of the relationship between ETS exposures and lung cancer.

7. Spengler made a few remarks about ETS exposures aboard aircraft. He said that one might find particulate levels in the range of 200-1000ug/m^3 in the smoking sections of L10-11's. Spengler said that he had sampled particulates using the piezobalance aboard flights and had observed levels in the range of 100-500ug/m^3. However, he admitted that there were no complete data regarding aircraft air quality because, in part, earlier studies had used sampling methods which "were not suitable" for low level detection of constituents.

8. Spengler asked Samet during a question and answer period whether there are enough data regarding children's lung function for lawmakers or the courts to declare parental smoking a form of "child abuse." Samet said that regardless of what the data showed, he would "happily testify".

WWD/laa

COMMUNITY EDUCATION SESSIONS

Richard J. O'Brien, M.D., Centers for Disease Control, Division of TB Control, Atlanta, Ga.

For new Lung Association staff, volunteers, and non-TB specialists.

This is a breakfast briefing on current recommendations for the prevention and treatment of tuberculosis. Emphasis is on responses to the questions most often asked of Lung Association staff. Participants are surveyed in advance to determine the most relevant questions for discussion.

(C5) ETHICAL ASPECTS OF PUBLIC POLICY

(Joint Scientific/Community Session)

Tuesday, May 13, 9:00 am - 12:30 pm 204 E (CC)
(No fee)

Kathleen McCormick, Ph.D., National Institute on Aging, Bethesda, Md.

Richard A. Goldberg, Esq., Department of the Public Advocate, Trenton, N.J.

Frank Marsh, J.D., Ph.D., University of Colorado School of Medicine, Denver, Colo.

Michael A. Rie, M.D., Massachusetts General Hospital, Boston, Mass.

Louis R. Sibal M.D., Office of Extramural Research and Training, Bethesda, Md.

For Lung Association staff and volunteers, physicians, nurses, and other health professionals.

Initiating and/or terminating life support systems in patient care and the individual's right-to-know concerning exposure to environmental hazards are two of the topics addressed in this session. Every major program area has a provocative ethical issue associated with it. This symposium focuses on the ethical issues surrounding the Public Policy agenda. Some myths are debunked, arguments buttressed, and new questions asked on thought-provoking topics.

(C6) GROWING OLDER WITH LUNG DISEASE

(Joint Scientific/Community Session)

Tuesday, May 13, 1:30 pm - 4:00 pm 204 E (CC)
(No fee)

Kathleen McCormick, Ph.D., National Institute on Aging, Bethesda, Md.

Eugene R. Bleecker, M.D., Francis Scott Key Medical Center, Johns Hopkins University, Baltimore, Md.

Benjamin Burrows, M.D., University of Arizona College of Medicine, Tucson, Ariz.

Audrey Gift, Ph.D., University of Maryland, Baltimore, Md.

Avram Gold, M.D., Francis Scott Key Medical Center, Johns Hopkins University, Baltimore, Md.

Robert M. Rogers, M.D., University of Pittsburgh College of Medicine, Pittsburgh, Pa.

For Lung Association staff and volunteers, physicians, nurses, and other health profesionals.

Demographic studies show that increasing numbers of individuals are surviving to an advanced age. Pulmonary diseases frequently occur in the elderly population and are an important cause of diminished exercise performance, as well as severe disability. Familiarity with the normal effects of aging on cardiopulmonary function is therefore important to all those who provide educational and health services to older people.

This symposium reviews the physiological changes that occur during the normal aging process and discusses some of the clinical problems unique to the elderly with lung disease and the programs designed to meet their needs.

(C7) RESPIRATORY DISTRESS SYNDROME

(Joint Scientific/Community Session)

Wednesday, May 14, 7:00 am - 8:30 am Count Basie C1
($10 fee, includes continental breakfast) (VH)

Alan H. Jobe, M.D., Harbor UCLA Medical Center, Torrance, Cal.

For Lung Association staff and volunteers, physicians, nurses, and other health professionals.

This session reviews the state-of-the-art in the diagnosis, treatment, and management of patients with respiratory distress syndrome.

D. SMOKING OR HEALTH

(D1) STATE AND LOCAL LEGISLATION ON INVOLUNTARY SMOKING

Monday, May 12, 9:00 am - 12:30 pm Chouteau B (HY)
(No fee)

Shane McDermott, Associate, Smoking or Health, ALA, New York, N.Y.

Fran DuMelle, Associate Director, ALA/ATS Government Relations Office, Washington, D.C.

Angela Mickel, Tri-Agency Tobacco-Free Young America Project, Washington, D.C.

Mark Pertschuk, Americans for Nonsmokers Rights, Berkeley, Cal.

Christine Deputy, Director of Smoking and Health Education, ALA of Florida, Jacksonville, Fla.

Linda Frisch, Associate Director of Program Development, ALA of Maryland, Lutherville, Md.

Karen Krzanowski, Program Director, ALA of Michigan, Lansing, Mich.

For Lung Association staff and volunteers active in seeking state and local legislation on public smoking and workplace policies.

There has been tremendous progress in state and local action against involuntary smoking, particularly in the workplace, in which Lung Associations have played a major role. With success have come new challenges: new tobacco industry tactics and state legislation preempting localities,

KANSAS CITY, MAY 11-14, 1986

(D1) "STATE AND LOCAL LEGISLATION ON INVOLUNTARY SMOKING"

Monday, May 12, 1986
9 am - 12:30 pm

FACULTY

Shane McDermott, Associate, Smoking or Health, ALA National Office
Fran DuMelle, Associate Director, ALA/ATS Government Relations Office
Angela Mickel, Coordinator, Tri-Agency Tobacco-Free Project (Washington, D.C.)
Mark Pertschuk, Associate Director, Americans for Nonsmokers Rights (Berkeley, CA)
Chris Deputy, Director, Smoking and Health Education, ALA of Florida
Karen Krzanowski, Program Director, ALA of Michigan
Linda Frisch, Associate Director for Program Development, ALA of Maryland

AGENDA

	Welcome Overview of Workshop	Shane McDermott
I.	Identifying Key Challenges	
	A. The Florida Experience	Chris Deputy
	B. The Michigan Experience	Karen Krzanowski
	C. Roundtable Discussions/Reports (workshop participants identify key challenges they've faced; a recorder from each roundtable will summarize and report findings)	
	D. The California Experience	Mark Pertschuk
II.	Identifying "Solutions"	
	A. Roundtable Discussions/Reports (workshop participants identify "solutions" found locally and/or suggest potential solutions to key challenges that have been identified; a recorder from each roundtable will summarize and report findings)	
	Available Resources	
	B. Tri-Agency Clearinghouse	Angela Mickel
	C. The Importance of a Legislative Network: the ALA Legislative Network Model	Fran DuMelle
	D. ANR (CNR) Legislative Guide	Mark Pertschuk
	E. The Importance of Coalitions: Maryland's "Healthy Majority"	Linda Frisch
	F. Additional Comments by Faculty Questions/Comments by Participants	

AMERICAN LUNG ASSOCIATION
The Christmas Seal People
AMERICAN THORACIC SOCIETY

EDUCATIONAL OBJECTIVES

1. Identify key challenges (i.e. obstacles, needs) facing us in achieving state and local legislation on involuntary smoking.
2. Identify available resources and potential solutions to use in meeting these challenges.

List of States with Laws Enacted Placing Limitations on Smoking

Alaska	Iowa	New Jersey
Arizona	Kansas	New York
Arkansas	Kentucky	North Dakota
California	Maine	Ohio
Colorado	Maryland	Oklahoma
Connecticut	Massachusetts	Oregon
Delaware	Michigan	Pennsylvania
District of Columbia	Minnesota	Rhode Island
Florida	Montana	South Dakota
Georgia	Nebraska	Texas
Hawaii	Nevada	Utah
Idaho	New Hampshire	Wisconsin
		Washington

States with laws specifically dealing with smoking in the workplace:

Minnesota	(1975)
Utah	(1976)
Nebraska	(1980)
Connecticut	(1983)
Alaska	(1984)
Maine	(1985)
New Jersey	(1985)
Florida	(1985)
Hawaii	(state employees only)
California	(state employees only)

Some cities and towns with ordinances covering smoking in the workplace:

San Marcos (CA) (1983)	Escondido (CA) (1983)	San Jose (CA) (1984)
Santee (CA) 1983)	San Francisco (CA) (1983)	Riverside (CA) (1985)
Vista (CA) (1983)	San Diego (CA) (1984)	Xenia (OH) (1985)
La Mesa (CA) (1984)	Palo Alto (CA) (1983)	Cincinnati (OH) (1985)
Pasadena (CA) (1984)	Ukiah (CA) (1981)	Greeley (CO) (1985)
National City (CA) (1984)	Sacramento (CA) (1985)	Boulder (CO) (1985)
Mountain View (CA) (1984)	San Marcos (CA) (1983)	Loveland (CO) (1985)
Santa Barbara (CA) (1984)	Newton (MA) (1984)	Aspen (CO) (1985)
Coronado (CA) (1984)	Carlsbad (CA) (1983)	Pueblo (CO) (1985)
El Cajon (CA) (1983)	Del Mar (CA) (1983)	Longmont (CO) (1985)
Chula Vista (CA) (1984)	El Cajon (CA) (1983)	Westminster (CO) (1985)
Los Altos (CA) (1979)	Oceanside (CA) (1983)	Grand Junction (CO) (1985)
Los Angeles (1984)	Poway (CA) (1983)	Santa Cruz (CA) (1985)
Los Gatos (CA) (1980)	Fort Collins, (CO) (1984)	Scott's Valley (CA) (1985)
		Topeka (KA) (1986)

Counties with ordinances covering smoking in the workplace:

Sacramento (CA)	(1984)
Suffolk (NY)	(1984)
San Diego (CA)	(1984)
Fresno (CA)	(county employees only) (1983)
Orange (CA)	(county employees only) (1975)
Yolo (CA)	(county employees only) (1984)
Monterey (CA)	(county employees) (1984)
San Mateo (CA)	(county employees) (1984)
Pierce (except Tacoma) (WA)	(1985)
Nassau (NY)	(1985)
Santa Cruz (CA)	(1985)

3/31/86

75. Effect of Steroids and Surfactant on Lung Protein Leak in Ventilated Preterm Rabbits/M. Ikegami, D. Berry, A. Jobe, A. Pettenazzo, T ElKady, S. Seidner, Torrance, Cal., p. A153

76. Alveolar Liquid Pressure Measured By Direct Micropuncture in Isolated Newborn and Adult Rabbit Lungs/C.D. Fike, S.J. Lai-Fook, R.D. Bland, San Francisco, Cal., p. A153

77. Effect of High Frequency Oscillation on Pulmonary Epithelial Permeability During Increased Surface Tension/A.L. Jefferies, R. Burger, T. Kawano, S. Mori, Toronto, ON, Canada, p. A153

78. Synergism of Oxygen Radicals and Elastase in the Pathogenesis of Acute Permeability Pulmonary Edema/R.B. Fox, M.J. Merrigan, Boston. Mass., p. A153

79. The Role of Lung Volume Maintenance During High Frequency Oscillatory Ventilation (HFO-A) in Surfactant Deficient Rabbits/P.R. McCulloch, P G. Forkert, A.B. Froese, Kingston, ON, Canada, p. A154

80. Prevention of Baroinjury with High Frequency Oscillatory Ventilation/R. deLemos, T. Kuehl, D. Null, M. Escobedo, N. Ackerman, J. Coalson, San Antonio, Texas, p. A154

81. Lung Inflammatory Cells Increase During Recovery from Experimental Hyaline Membrane Disease/J C. Jackson, E.Y. Chi, W.E. Truog, W.A. Hodson, Seattle, Wash., p. A154

82. Pulmonary Function in Long Term Survivors of Hyaline Membrane Disease/J. Sheller, W. Lucht, A. Goel, J. Snell, M. Stahlman, Nashville Tenn., p. A154

5P

2:30-5:30 pm — Room 212S (CC)

PULMONARY EPIDEMIOLOGY/Scientific Assembly on Environmental and Occupational Health/J.M. Samet, M.D., Albuquerque, N.M./A.S. Buist, M.D., Portland, Ore./*Cochairing*

Poster Presentations	2:30-4:00
Symposium Discussion	4:00-5:30

1. Prevalence of Respiratory Symptoms in Non-Occupationally Exposed Adults/B.G. Ferris, Jr., D.W. Dockery, F.E. Speizer, J.H. Ware, Boston, Mass., p. A155

2. Asthma, Asthma-Like Symptoms, Chronic Bronchitis and the Degree of Bronchial Hyperresponsiveness in Epidemiologic Surveys/M. Chan-Yeung, S. Vedal, D. Enarson, Vancouver, BC, Canada, p. A155

3. Changes in FEV_1 Across a Workshift Among Unexposed Blue Collar Workers/A.J. Ghio, R.M. Castellan, K.B. Kinsley, Morgantown, W.V., p. A155

4. Investigation of a Longitudinal Model for FEV_1 Growth in Children/J.B. Tager, A. Munoz, S.T. Weiss, F.E. Speizer, San Francisco, Cal., Boston, Mass., p. A155

5. Comparability of Cross-Sectional and Longitudinal Estimates of Annual Loss of FEV_1/D.W. Dockery, T.A. Louis, J.H. Ware, B.G. Ferris, Jr., F.E. Speizer, Boston, Mass., p. A156

6. The Effect of Asthma on Rate of Decline of Lung Function: A Community Study/A.J. Woolcock, J.K. Peat, K. Cullen, Sydney, Australia, p. A156

7. Should Spirometry be Used to Assess Patients with COPD?/C.B. Sherman, Seattle, Wash./J.M. Samet, Albuquerque, N.M., p. A156

8. Effects of Multicollinearity on Inference in Regression Analysis of Occupational Respiratory Epidemiologic Data/N.B. Sussman, M.C. Townsend, S. Mazumdar, Pittsburgh, Pa., p. A156

9. Familial Pulmonary Diseases as a Risk Factor in a General Population/P. Paoletti, G. Viegi, G. Carmignani, F. Di Pede, L. Carrozzi, R Prediletto, G. Pistelli, P. Fazzi, M.D. Lebowitz, C. Giuntini, Pisa, Italy, p. A157

10. Passive Smoking, Respiratory Symptoms and Pulmonary Function: A Longitudinal Study in Children/C.M. Burchfiel, M.W. Higgins, J.B. Keller, W.J. Butler, W.F. Howatt, I.T.T. Higgins, Ann Arbor, Mich., p. A157

11. Respiratory Symptoms and Lung Function in Women with Passive and Active Smoking/F. Kauffmann, D.W. Dockery, F.E. Speizer, B.G. Ferris, Jr., Villejuif, France, Boston, Mass., p. A157

12. Salivary Continine Levels and Passive Tobacco Smoke Exposure in the Home/D.B. Coultas, J M. Samet, C.A. Howard, G.T. Peake, B.J. Skipper, Albuquerque, N.M., p. A157

13. Determinants and Markers of Environmental Tobacco Smoke (ETS) Exposure in an Occupational Setting/M.B. Schenker, S.K. Hammond, S. Woskie, S. Samuels, N. Kado, T. Smith, Davis, Cal., Worchester, Mass., p. A158

14. Pulmonary Function in Relation to Smoking and Smoking Cessation in the Multiple Risk Factor Intervention Trial (MRFIT)/M.C. Townsend, A.G. DuChene, J. Morgan, W. Browner, Pittsburgh, Pa., p. A158

15. Effects of Smoking and Smoking Cessation on the Rate of Decline in FEV_1/A. Camilli, S. Lyle, M. Lebowitz, B. Burrows, Tucson, Ariz., p. A158

PREVALENCE OF RESPIRATORY SYMPTOMS IN NON-OCCUPATIONALLY EXPOSED ADULTS. B.G.Ferris Jr., D.W. Dockery, F.E. Speizer, J.H. Ware. (Departments of Environmental Science & Physiology and Biostatistics, Harvard School of Public Health; Channing Laboratory, Harvard Medical School, Boston MA.)

Trained interviewers administered a standard questionnaire on respiratory symptoms similar to the ATS-DLD questionnaire to a random sample of 8,500 adults aged 25-74 living in six cities. We have previously reported prevalences of chronic phlegm (CP), bouts of cough and phlegm (BCP), persistent wheeze (PW), doctor-diagnosed asthma (A), recurrent chest illness (CI), and grade 2+ breathlessness (B) stratified by sex, age, and smoking-status. From this sample, we selected 477 white male and 1,971 white female non-smokers who had no history of working in dusty environments. Age- and sex-specific illness and symptom rates in this subsample were as follows:

Prevalences of Selected Respiratory Symptoms for Whites, by Age Decade for Males and Females

AGE	25-34		35-44		45-54		55-64		65-74	
SYMPTOM	M	F	M	F	M	F	M	F	M	F
CP	7.4	2.8	2.1	5.5	7.9	5.2	4.3	3.6	6.7	5.9
BCP	5.6	5.8	7.3	5.2	2.3	6.8	2.9	5.1	1.7	4.2
PW	2.5	2.5	1.0	5.2	2.4	2.8	2.9	3.6	0.0	2.6
A	3.1	2.5	1.0	2.2	0.0	2.0	1.4	1.7	1.7	1.2
B	1.2	3.4	2.1	4.8	3.5	5.7	0.0	6.1	1.8	6.8
NUMBER	162	361	96	272	89	499	70	413	60	426

These rates provide reference values for assessing the effects of environmental (occupational and ambient) and personal (cigarette smoking) exposures on respiratory health. As an example, we computed age- and sex-standardized rates for the nonsmoking, nonoccupationally exposed residents of each of the six cities. These rates showed no gradient of CC and CP prevalence across cities, but provided evidence for an exposure-response relationship between the other symptom frequences and level of outdoor particulate air pollutant concentration. Although use of these rates as reference values would not be indicated in studies with large internal control groups, they are of value for investigators who wish to assess respiratory health status in small samples of exposed nonsmokers.

Supported in part by NIEHS grant ES-01108, EPRI Grant RP-1001 and EPA Cooperative Agreement Number CR811650.

CHANGES IN FEV_1 ACROSS A WORKSHIFT AMONG UNEXPOSED BLUE COLLAR WORKERS. A.V. Ohio, R.M. Castellan, P.D. Kinsley. Division of Respiratory Disease Studies, National Institute for Occupational Safety and Health, (NIOSH), Morgantown, WV.

Pre- and post-shift spirometry was obtained on 980 blue collar workers employed at 35 worksites which were judged on the basis of inspection visits and environmental sampling to have no known hazardous occupational respiratory exposures. In addition to the spirometry, a standardized questionnaire was administered by trained NIOSH personnel. A study population of 944 remained following exclusions for incomplete questionnaire data and/or for invalid spirometry. The study group had a mean age of 33 years (range 14-77) and was predominantly male (90%), with 46% current smokers and 13% ex-smokers. Whites comprised 30% and blacks 45% of the population, with the remainder being mainly Hispanic. The majority (76%) worked on the morning shift, and only 3% worked the night shift. The change in FEV_1 across a workshift [ΔFEV_1 = (post-shift FEV_1 - pre-shift FEV_1)/(pre-shift FEV_1) X 100] appeared to be normally distributed with mean = -0.8% and S.D. = 5.8%. Only 2.2% had a shift-related decline of 10% or more in FEV_1, while 13.6% experienced a decline of 5% or more. No significant differences in $\Delta FEV1$ existed when the study population was divided by: age (younger than 40 vs. 40 and older); sex (male vs. female); race (black vs. white); shift (day vs evening); smoking status (current vs. never smokers); or FEV1/FVC ratio (< 70% vs. ≥ 70%). These observations offer a basis for interpreting the results of occupational respiratory morbidity surveys involving shift-related changes in FEV_1. Considering an "abnormal" shift decline in FEV_1 as one which is in the lower 5% of the theoretical distribution of values derived from our data (mean minus 1.65 S.D.), then a shift decline of greater than 10% appears to be a reasonable criterion for an adverse respiratory health effect.

ASTHMA, ASTHMA-LIKE SYMPTOMS, CHRONIC BRONCHITIS AND THE DEGREE OF BRONCHIAL HYPERRESPONSIVENESS IN EPIDEMIOLOGIC SURVEYS. N. Chan-Yeung, S. Vedal, D. Enarson. Dept. of Medicine, Vancouver General Hospital, University of British Columbia.

Measurement of bronchial hyperresponsiveness has been suggested to be a useful test in identifying subjects with asthma in epidemiologic groups. In this presentation we explore the association between the degree of bronchial hyperresponsiveness and physician-diagnosed current asthma, past asthma, asthma-like symptoms such as persistent wheeze and chest tightness without current or past asthma, and chronic bronchitis as reported by questionnaire. We determined bronchial hyperresponsiveness by methacholine inhalation test, administered a standardized respiratory questionnaire and performed spirometry on 1392 male workers in various industries.

Only 694 workers had no respiratory symptoms and normal lung function. The prevalence of reported asthma and symptoms according to degree of hyperresponsiveness relative to the group with no hyperresponsiveness (PC20>8) is shown below:

		Range of PC20			
	No.	<0.5	0.5-<2	>2-<8	>8
Current asthma	25	54.8	20.1	2.4	1
Past asthma	25	9.7	2.7	3.3	1
Persistent wheeze	151	3.1	1.9	2.2	1
Persistent wheeze and chest tightness	30	5.2	1.4	1.5	1
Chronic bronchitis	63	3.4	2.8	2.3	1

With more marked hyperresponsiveness (PC20<0.5 or PC20<2), workers were much more likely to have reported current asthma than workers with PC20>8. This was much higher than analogous ratios for the other symptom groups. However, for the middle range of PC20 (>2 to <8 mg/ml), workers were only 2.4 times as likely to report current asthma compared to workers with less reactivity. This did not differ from that seen with reported past asthma, asthma -like symptoms, or chronic bronchitis. Moreover, 12.1% of subjects with no chest symptoms and normal lung function had PC20<2.

We conclude that the usefulness of measurement of bronchial hyperresponsiveness in the epidemiologic studies is limited.

INVESTIGATION OF A LONGITUDINAL MODEL FOR FEV_1 GROWTH IN CHILDREN. J.B. TAGER, A. MUNOZ, S.T. WEISS, F.E. SPEIZER. VETERANS ADMINISTRATION MEDICAL CENTER, UNIVERSITY OF CALIFORNIA, SAN FRANCISCO, CA: CHANNING LABORATORY, BRIGHAM AND WOMAN'S HOSPITAL, HARVARD MEDICAL SCHOOL, BOSTON, MA

As part of a longitudinal study of early life risk predictors for chronic airflow obstruction, we have published(Am Rev Respir Dis 1985; 131:752) an autoregressive model, based on the 1st 8 years of observation, that describes the growth of FEV_1 and FEF_{25-75} in children and adolescents(age≤20)in terms of previous measurements of lung function, age, sex, height, change in height, interactions between age and change in height , and between sex and height, personal smoking of the children and exposure to maternal smoking. The present investigation was undertaken to determine how this model predicts growth of FEV_1 in subsequent years. 483 children who were part of this study and who provided data during the 9th survey year were evaluated(median age=14yr, range=8-19). A predicted level of FEV_1 for each subject was obtained from the model based on the survey 9 age, height, personal and maternal smoking habits.(Data not used to fit the model.) Residual FEV_1 was computed as survey 9 FEV_1 minus FEV_1 predicted by the model. The mean squared error residual (estimate of fit of the model) was .0057 (median=.0025), which indicates that the model predicted survey 9 FEV_1 very well. The overall mean residual was -.0144(median=-.0148), which indicates that, on average, survey 9 FEV_1 was smaller than predicted by the model. Although the diffefences were not significant, children who were current smokers (n=165) had more negative residuals (mean=-.0156) than never-smokers (n=241, mean=-.0141). Children whose mothers were current smokers (n=165) also had more negative residuals (mean=-.0157) than those whose mothers were never smokers (n=140, mean=-.0097). FEV_1 and FEF_{25-75} % predicted were directly related to the magnitude of the residuals. FEV_1 % predicted at survey 9 (based on a standard nomogram) was 90.06(±12.34) and FEF_{25-75} % predicted was 74.75(±17.48) for the children with the lowest 10% of residuals vs 116.75(±13.95)and 104.02(±24.68), respectively, for the children with the highest 10% of residuals(P<.001, for both).

We conclude that this model is a valuable tool for studying the long-term growth of lung function in children and the factors that influence this growth.

FAMILIAL PULMONARY DISEASES AS A RISK FACTOR IN A GENERAL POPULATION. P. Paoletti, G. Viegi, G. Carmignani, V. Di Pede, L. Carrozzi, N. Fradiletto, G. Pistelli, P. Fazzi, M.D. Labowitz, , C. Giuntini. CNR Institute of Clinical Physiology and 2nd Medical Clinic, University of Pisa, Italy.

Information regarding familial pulmonary diseases (FPD) and respiratory symptoms (by a standardized questionnaire) and lung function were obtained in a general population sample (n=3278), ages 8 to 64 years, living in an unpolluted area of North Italy. There were 279 subjects who reported a family history of chronic bronchitis or emphysema (FCOLD); 442 with a family history of asthma or allergic rhinitis (FASL); 307 with a family history of lung cancer (FLC), and there were 2050 with no FPD (NFPD). Age and smoking did not differ among these groups. Females were significantly less represented in the NFPD group. In smokers (S), respiratory symptoms were more prevalent in subjects with FPD. Significant differences were observed for chronic cough, phlegm, wheeze and attacks of shortness of breath with wheeze (SOBW) in both sexes. Chronic cough and phlegm were associated with FCOLD (21%) and FLC (20%) compared to FASL (16%) and NFPD (14%); wheeze and SOBW were associated with FASL. In non-smokers (NS), no relationships between symptoms and FPD were observed, though wheeze and SOBW were more frequent in FASL. Physician confirmed diagnosis of asthma and COLD were significantly associated with FPD in S, (4-6% vs 2% in NFPD) but only asthma was in NS (7% in FASL). The prevalence rate of respiratory diseases in childhood (recurrent bronchitis, pneumonia or pertussis before 12 years - CRD) investigated was significantly higher in subjects with FPD. Moreover, respiratory symptoms and diseases were significantly more prevalent in subjects with both CRD and FPD. Parameters of the flow volume curve and diffusing capacity, adjusted for sex, age and smoking, did not differ in the 4 groups of FPD. In conclusion, FPD is a risk factor for CRD and, when associated with CRD or with smoking, may increase the susceptibility for respiratory symptoms and diseases in adulthood.

PASSIVE SMOKING, RESPIRATORY SYMPTOMS AND PULMONARY FUNCTION: A LONGITUDINAL STUDY IN CHILDREN. C.M. Burchfiel, M.W. Higgins, J.B. Keller, W.J. Butler, W.F. Mowatt, I.T.T. Higgins. Department of Epidemiology, The University of Michigan, Ann Arbor, MI.

To assess relationships between passive exposure to household cigarette smoke, incidence of respiratory conditions and change in lung function, a longitudinal investigation was conducted using the Tecumseh population. Following baseline examinations of 0-10 year-olds in 1962-65, 2661 (76.4%) completed follow-up questionnaires fifteen years later in 1978-79. Passive smoking indices were defined at baseline and most analyses were restricted to never smokers at follow-up. Age-specific incidence rates of cough, phlegm, bronchitis and cold settling in the chest for males and wheeze for both sexes were higher in offspring of two vs. zero parental smokers. The age-adjusted incidence rate of phlegm in males was significantly related to the number of parental smokers at baseline ($p<.01$, χ^2 test for trend). The relative risk of developing excessive phlegm production was 3.0 and 4.8 for offspring having one and two parental smokers at baseline respectively compared with unexposed offspring. Logistic regression was used to adjust for age, parental education, family size and smoking habits of offspring at follow-up. The odds of being exposed to two vs. zero parental smokers ranged from .90 to 1.96 for six respiratory conditions, with highest values found for asthma in both sexes. Mean annual changes in FEV_1, FVC and $\dot{V}max_{50}$ for males but not females were inversely related to the number of parental smokers at baseline after adjusting for initial height, change in height and baseline lung function using analysis of covariance, although results were not statistically significant. Increases in mean FEV_1 of 82.3, 76.2 and 74.5 ml per year were observed for 10-19 year-old male nonsmokers who had 0, 1 and 2 parental smokers, respectively. Males having 1 and 2 parental smokers experienced increases in lung function which were only 92.6% and 90.5% (respectively) of the magnitude found for unexposed offspring. Thus, an increased risk of developing several chronic respiratory conditions and a slower rate of lung growth may be associated with passive smoking during childhood and adolescence. Additional longitudinal studies are needed.

RESPIRATORY SYMPTOMS AND LUNG FUNCTION IN WOMEN WITH PASSIVE AND ACTIVE SMOKING. F Kauffmann, DW Dockery, FE Speizer, BG Ferris,Jr (INSERM U169, Villejuif, France; Channing Laboratory, Harvard Medical School; Department of Environmental Science & Physiology, Harvard School of Public Health, Boston, MA)

From a random sample of 2220 married women aged 25 to 69 yr from 3 U.S. cities, we identified 275 True Never smokers, i.e. never-smokers married to never smokers, without exposure to smoking at work. There were 407 Ex-Passive, i.e. never smokers married to ex-smokers, 431 Passive, never smokers married to smokers, 331 Ex-smokers, and 756 Active smokers. The prevalences of chronic cough, phlegm, any wheeze and dyspnea (Grade 1+) for the Passives were intermediate between the True Never and the Active. All the differences between the Active and the True Never were significant. Between the Passives and True Never, the difference was significant for dyspnea (OR=1.42) and borderline for wheeze (OR=1.36; $p=.06$). After adjustment for age, city, education level and occupational dust exposure, all the associations remained significant for Active versus True Never. The association between dyspnea and Passive exposure was no longer significant (adj OR=1.13). For wheeze, the adjusted OR were: True Never 1.00; Ex-Passive 1.16($p=.09$); Passive 1.22($p=.07$); ExSmoker 1.26($p=.01$); Active 1.80 ($p<.001$). When Ever Passive (Ex-Passive plus Passive) was compared to True Never, the difference reached significance.

Age, height, and city adjusted FEV_1 and FEV_1/FVC were compared in the 5 groups. Standardized residuals for Passive were intermediate between True Never and Active. For FEV_1/FVC, bigger differences were observed between True Never and the 4 other groups than with FEV_1. For FEV_1/FVC, the trend of True Never>ExPassive>Passive>ExSmoker>Active was more marked in the subgroup of women with wheeze. This trend persisted after exclusion of women with persistent wheeze or asthma. Though not statistically significant, the magnitude of the difference between Passive and True Never suggests that FEV_1/FVC may be decreased in relation to passive smoking among women with a minor degree of wheeze. Further studies are needed to confirm this result and explain why such a trend was not observed for FEV_1. One possible hypothesis is that FEV_1/FVC might be a more sensitive test than FEV_1, especially in the presence of some sort of hyperreactivity.

SALIVARY COTININE LEVELS AND PASSIVE TOBACCO SMOKE EXPOSURE IN THE HOME. D.B. Coultas, J.M. Samet, C.A. Howard, G.T. Peake B.J. Skipper. New Mexico Tumor Registry, University of New Mexico, Albuquerque, NM

Cotinine, the major metabolite of nicotine, can be detected in blood, urine, and saliva of active cigarette smokers and of those passively exposed to environmental tobacco smoke. We have conducted a population-based survey of respiratory diseases among Hispanic residents of Belen, New Mexico. Salivary cotinine levels were measured in 1286 participants by radioimmunoassay; smoking histories were obtained by questionnaire. In adults, salivary cotinine was elevated among current (median=287 ng/ml) versus exsmokers (median=0) and nonsmokers (median=0). In nonsmokers, defined by questionnaire response and a cotinine concentration ≤20 ng/ml, cotinine levels increased with the number of smokers in the home.

COTININE CONCENTRATION* (ng/ml) IN NONSMOKERS

	Number of Smokers per House		
	0	1	≥ 2
Age(years)			
≤ 5	0,1.7 (68)	3.8,4.1 (41)	5.4,5.6 (21)
6-17	0,1.3 (200)	1.8,2.4 (96)	5.3,5.6 (25)
≥ 18	0,1.5 (316)	0.65,2.8 (60)	0,3.7 (12)

*median, mean, and number of subjects in parentheses

Chi-square analysis was performed after categorizing cotinine concentration into three groups: 0-1.0, 1.1-5.0, and 5.1-20.0 (ng/ml). Cotinine levels were significantly associated with the number of smokers per household within each age group ($p<0.01$). We conclude that in the general population, nonsmokers exposed to cigarette smoke in their homes have detectable levels of cotinine and that the levels increased with the number of smokers in the home.

DETERMINANTS AND MARKERS OF ENVIRONMENTAL TOBACCO SMOKE (ETS) EXPOSURE IN AN OCCUPATIONAL SETTING. M.B. Schenker, S.K. Hammond, S. Muakis, S. Samuels, N. Kado, T. Smith. Depts. of Internal Medicine and Environmental Toxicology, Univ. Calif. Davis, CA., and Univ. Mass. Medical School, Worchester, MA.

Passive smoking or ETS exposure in the home and workplace has been associated with a wide spectrum of adverse health effects. However, most epidemiologic studies have relied on self-reported exposure to ETS, which may be very inaccurate due to differences in subjective assessment, in room size and ventilation, and in individual ventilation rates. As part of a study of railroad workers, we measured nicotine on filters from personal sampling pumps collected over a workshift. Nicotine was used because it is a unique and specific marker for tobacco smoke. Respirable nicotine concentrations ($\mu g/m^3$) were highly correlated with the number of cigarettes smoked. Mean (±SEM) respirable nicotine conc. among non-smokers (n=163), 1-14 cigarette (n=39) and 15+ cigarette smokers (n=33) were 2.09 ± 0.41, 9.55 ± 1.36 and 14.13 ± 2.02. Among non-smokers, respirable nicotine concentrations were higher for workers in small volume indoor environments (station, train cab), than for outdoor workers (car repair). Mean resp. nicotine conc. among the non-smoking indoor workers was 6.42 ± 1.20 and among the outdoor workers was 3.35 ± 1.67, despite a higher prevalence of smoking in the outdoor workers. Biologic measures of cigarette smoke exposure (expired CO, urine thiocyanate and urine mutagenicity) were made at the end of the workshifts, and are highly correlated with smoking dose during the shift in active smokers. In a preliminary subset of non-smoking, non-diesel exhaust exposed workers, mean urinary mutagenicity (rev./nmole Cr.) was higher in workers with resp. nicotine 93.0 (n=7, M=2.87 ± 3.08), than in workers with resp. nicotine <3.0 (n=10, M=1.00 ± 0.61), but the difference was not statistically significant. We conclude that respirable nicotine concentration is a useful and specific marker for ETS exposure, and can be used to validate other measures of passive smoking.

Supported by the Health Effects Institute, Cambridge, MA.

EFFECTS OF SMOKING AND SMOKING CESSATION ON THE RATE OF DECLINE IN FEV_1. A. Camilli, S. Lyle, M. Lebowitz, B. Burrows. Department of Internal Medicine, Division of Respiratory Sciences, University of Arizona College of Medicine, Tucson, AZ.

The annual rate of change in FEV_1 (ΔFEV_1) has been determined in 1707 subjects who were at least 20 years of age at the time of their enrollment in a longitudinal study of a general population sample residing in Tucson, Arizona. Subjects had a mean duration of follow-up of 9.4 years and an average of 5.2 spirometric tests. Regardless of smoking status, ΔFEV_1 shows a marked age dependence, and most of the excess decline with smoking appears to occur between 50 and 70 years of age. Smoking effects are greater in men than in women regardless of the numbers of cigarettes smoked. As in other studies, ex-smokers show ΔFEV_1's which are significantly less than smokers and not significantly different from nonsmokers.

Quitting smoking occurred in 26.7% of subjects who were current smokers on entry to the study and was similar in both sexes and age groups. Continuing smokers and quitters had similar levels of initial FEV_1. Female quitters smoked significantly less pack years on entry than continuing smokers. Quitting cigarette smoking during follow-up is associated with an FEV decline which is significantly less than continuing smokers and similar to never and exsmokers. Among subjects less than 35 years of age on entry there is a significant increase in the FEV during follow-up. The ΔFEV's are +21.4 and +26.9 ml/year in males and females respectively. Older subjects (50-70 years of age on entry) who quit smoking during follow-up show ΔFEV's which are lower than continuing smokers and not significantly different from nonsmokers. The effect of smoking cessation in these older subjects is most evident when the ΔFEV is calculated using the last value while the subject smoked as the initial point in the regression. These results suggest that smoking cessation produces an actual improvement in pulmonary function in younger subjects and a relatively prompt return of the ΔFEV to the level of nonsmokers in older subjects.

Supported by SCOR HL 14136.

PULMONARY FUNCTION IN RELATION TO SMOKING AND SMOKING CESSATION IN THE MULTIPLE RISK FACTOR INTERVENTION TRIAL (MRFIT). M.C. Townsend, A.G. DuChesne, J. Morgan, W. Browner. Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, PA.

From 1974-1982, the Multiple Risk Factor Intervention Trial followed 12,866 men, aged 35-57 at entry, at 22 centers in the U.S. 63% of the subjects smoked at baseline and 1,332 stopped smoking permanently during the first year of the study. The primary goal of the present study was to compare the declines in Forced Expiratory Volume in One Second (FEV1) over time in the early permanent quitters and the smokers who continued to smoke throughout the Trial. Secondary goals were to compare levels of FEV1 cross-sectionally and longitudinally in all cigarette smoking groups. Since pulmonary function testing was not well-standardized in MRFIT until the third annual visit cycle, the present study examined changes in FEV1 over the latter half of the Trial and level of FEV1 at the midpoint of the Trial. The present analyses were limited to 6,123 non-users of cigars, cigarillos, and pipes who had 3-5 acceptable FEV1s available in the latter half of the Trial. The major finding was that the Continuing Smokers had significantly steeper FEV1 slopes over the latter half of the Trial than Quitters by First Annual, with -63.8 ml/yr versus -52.5 ml/yr, respectively ($p < 0.01$). In addition, in the cross-sectional comparison of all smoking groups, Never Smokers, Former Smokers, Quitters by First Annual, and Continuing Smokers exhibited a gradient of decreasing FEV1, and all four smoking groups were significantly different from each other ($p < 0.05$). Finally, when mean FEV1 slopes were compared for all smoking groups, Continuing Smokers had significantly steeper slopes than the other smoking groups ($p < 0.05$), but the other groups were not significantly different from each other. Based on the MRFIT cohort, if a middle-aged, healthy smoker stopped smoking permanently, he could expect his FEV1 to deteriorate at the same rate as a never smoker's. If a similar smoker continued to smoke, he could expect his FEV1 to deteriorate at a slightly faster rate.

Source of Iron in Neutrophil-Mediated Killing of Endothelial Cells/D.E. Gannon, J. Varani, G.O. Till, U.S. Ryan, R.H. Simon, P.A. Ward, Ann Arbor, Mich., Miami, Fla., p. A262 *11:45*

Neutrophils Potentiate Endotoxin Induced Prostaglandin E_2 (PGE_2) Production by Cultured Pulmonary Endothelial Cells/C.D. Givens, Jr., K.L. Brigham, B. Meyrick, Nashville, Tenn., p. A262 *12:00*

POSTER SYMPOSIUM

9M

9:15 am-12:15 pm **Room 210S (CC)**

OCCUPATIONAL LUNG DISEASE/Scientific Assembly on Environmental and Occupational Health/J.E. Fish, M.D., Philadelphia, Pa./M.B. Schenker, M.D., Davis, Cal./*Cochairing*

Poster Presentations **9:15-11:00**

Symposium Discussion **11:00-12:15**

103. **Seasonal Variation in Non-Specific Bronchial Hyper-Reactivity in Farmers/M. Hensley, R. Schiccitano, N. Saunders, A. Cripps, J. Ruhno, D. Sutherland, R. Clancy, Newcastle, Australia, p. A262**

104. **Determinants of the Prognosis of Patients with Red Cedar Asthma in a Follow Up Study/M. Chan-Yeung, L. MacLean, P.L. Paggiaro, Vancouver, BC, Canada, p A262**

105. **Does Methacholine Bronchoprovocation Testing Prospectively Identify Trend in FEV_1 in Grainhandlers?/D.A. Enarson, S. Vedal, M. Chan-Yeung, Vancouver, BC, Canada, p. A263**

106. **Non-Specific Bronchial Reactivity in Polyurethane Workers/H.W. Barkman, Jr., J.M. Hughes, D.E. Banks, H.W. Glindmeyer, B.T. Butcher, R.N Jones, H. Weill, D.J. Hendrick, New Orleans, La., p. A263**

107. **Non-Specific Airway Reactivity Increased by Exposure to Cotton Dust/B. Boehlecke, R. Schreiber, J. Fullton, Chapel Hill, N.C., p. A263**

108. **Airway Diameter and the Rate of Pulmonary Function Decline in Grain Workers/S. Vedal, D. Enarson, M. Chan-Yeung, Vancouver. BC, Canada, p. A263**

109. **Relationship Between Dust Exposure and X-Ray Appearance of Pneumoconiosis in Two South African Foundries/A.B. Zwi, M.R. Becklake, A.E. Smit, P.J. Becker, R.E.G. Rendall, Johannesburg, South Africa, p. A264**

110. **Exercise Testing in the Determination of Respiratory Impairment in Pneumoconiosis/S. Vedal, E. Lee-Chuy, R.T. Abboud, Vancouver, BC, Canada, p. A264**

111. **Predictors of Emphysema in S. African Goldminers/M.R. Becklake, L.M. Irwig, D. Kielkowski, I. Webster, M. de Beer, S. Freeman, Johannesburg, South Africa, p. A264**

112. **A Retrospective Cohort Study of Lung Cancer and Diesel Exhaust Exposure in Railroad Workers/E. Garshick, A. Munoz, M.B. Schenker, S. Woskie, T. Smith, F.E. Speizer, Davis, Cal., Boston, Worcester, Mass., p. A264**

113. **Cumulative Effect of Occupational Dust Exposure on Levels of Pulmonary Function and Chronic Respiratory Symptoms/R.J. Korn, D.W. Dockery, F.E. Speizer, J.H. Ware, B.G. Ferris, Jr., Boston, Mass., p. A265**

114. **The Prognostic Significance of Farmer's Lung Disease Antibodies Relative to Measures of Respiratory Disease—A Wisconsin Dairy Farming Population/J.R. Guernsey, D.P. Morgan, J.J. Marx, E.P. Horvath, P. Pierce, J. Merchant, Iowa City, Iowa, p. A265**

115. **Slow Bronchoconstriction After Inhalation of Bacterial Endotoxins/R. Rylander, B. Bake, Gothenburg, Sweden/J. Fischer, Chapel Hill, N.C., p. A265**

JOINT SCIENTIFIC/COMMUNITY SESSION

F2

9:00 am-12:30 pm **Room 204E (CC)**

INDOOR AIR POLLUTION IN THE NON-INDUSTRIAL ENVIRONMENT/ATS Scientific Assembly on Environmental and Occupational Health/ALA National Air Conservation Committee

J.M. Samet, M.D., Albuquerque, N.M.

J.D. Spengler, Ph.D., Boston, Mass.

R. White, M.S.T., New York, N.Y.

This session reviews current information on the sources, personal exposures, health effects, problem investigation and strategies to eliminate indoor air pollution in the home and non-industrial workplace. A discussion of public policy options for controlling indoor air pollution is also presented.

MEET THE PROFESSOR SEMINARS (MP)

12:15-1:30 pm

There is a $15.00 fee per seminar, which includes a box lunch. Advance registration is required. Attendance is limited to 30 per seminar.

501. **Lung Host Defense and Infection (ACI)** **Room 1 (VH)**
Chairman/S. Gryzan, M.D., Pittsburgh, Pa.
Professor/D.C. Zavala, M.D., Iowa City, Iowa

C6

GROWING OLDER WITH LUNG DISEASE/ATS Scientific Assembly on Clinical Problems/Section on Nursing/ALA Lung Disease Committee

Tuesday, May 13
1:30-4:00 pm **204E (CC)**

OBJECTIVES

At the conclusion of this session, the participant will be able to:

1) describe changes in lung function that occur with increasing age;

2) identify measures to prevent functional disabilities of the aged;

3) describe the role of selected modalities (nutrition, exercise) in the care of the aged.

Demographic studies show that increasing numbers of individuals are surviving to an advanced age. Pulmonary diseases frequently occur in the elderly population and are an important cause of diminished exercise performance, as well as severe disability. Familiarity with the normal effects of aging on cardiopulmonary function is therefore important to all those who provide educational and health services to older people. This symposium reviews the physiologic changes that occur during the normal aging process and discusses some of the clinical problems unique to the elderly with lung disease and the programs designed to meet their needs.

F2

INDOOR AIR POLLUTION IN THE NON-INDUSTRIAL ENVIRONMENT/ATS Scientific Assembly on Environmental and Occupational Health/ALA National Air Conservation Committee

Wednesday, May 14
9:00 am-12:30 pm **204E (CC)**

OBJECTIVES

At the conclusion of this session, the participant will be able to:

1) identify sources of lung irritants within the indoor environment;

2) identify mechanisms for controlling exposure to these pollutants;

3) identify actions that will eliminate sources of air pollutions in enclosed environments.

This session reviews current information on the sources, personal exposures, health effects, problem investigation and strategies to eliminate indoor air pollution in the home and non-industrial workplace. A discussion of public policy options for controlling indoor air pollution is also presented.

EFFECTS OF PARENTAL CIGARETTE SMOKING ON YOUNG CHILDREN. E. Galbraith. Johns Hopkins School of Hygiene and Public Health, Baltimore, Md.

This study investigated the relationship between parental cigarette smoking and respiratory illness in a random sample of 795 young urban black children. The purpose was to determine if there are differences in illness in children from birth to three years of age by the amount and source of passive smoke exposure: that is, maternal, paternal, and other household cigarette smoking. Information regarding the child's health history and respiratory illness during the study period of October 1982 to April 1985 was obtained from the computerized record system (CARE) utilized by the Johns Hopkins Children and Youth Program. Information regarding smoking habits of the parents and demographic and confounding variables were obtained through telephone interviews with the parents using a questionnaire based on the pediatric and adult version of the ATS-DLD-78. Results suggest an increased risk of lower respiratory illness as measured by one or more clinic visits for children aged six to 17 months exposed to passive smoking. This risk is strongest in the subgroup of children aged six to 11 months. There is little evidence to support any association between passive smoke exposure and respiratory illness after 18 months of age. Children whose mother smoked during pregnancy had a statistically significant increased risk of lower respiratory illness with a relative risk of 1.6 and double the risk in a subgroup of children six to 11 months of age. Children aged six to 11 months whose mothers smoked more than five cigarettes a day had a relative risk of 1.8 of experiencing at least one episode of lower respiratory illness. There is no evidence in this study of an association between upper respiratory illness in either age group and passive smoke exposure, although clinic visits may not be an accurate reflection of upper respiratory morbidity.

Funded by the American Lung Association of Maryland.

CHRONIC RESPIRATORY SYMPTOMS IN YOUNG CHILDREN AND INDOOR HEATING WITH A WOODBURNING STOVE. J.S. Osborne, III, R.E. Monicky, Michigan State University College of Human Medicine, Department of Pediatrics/Human Development, East Lansing, MI.

A recent report of symptoms of respiratory illness in young children implicated indoor heating with a woodburning stove as the primary etiological factor (Pediatrics 75:587, 1985). We conducted a study to prospectively investigate the occurrence of respiratory symptoms in young children living in homes heated by a woodburning stove (WBS) during the winters of 1982, 1984 and 1985. Thirty-one randomly selected children from WBS-heated homes (study group) in the 5-county Greater Lansing area were matched for age, sex, and residence with 31 children from homes heated by conventional furnaces (control group). Data was collected by interviewing children's parents. Sample attrition during the study was 10% per year. The occurrence of chronic coughing ($p < 0.001$) and wheezing ($p < .05$) was significantly greater in the study group during all three winters. Children in the study group also had greater occurrence of otitis media, bronchitis, and pneumonia than children in the control group. In addition, type of heating changed for 6 children between 1982 and 1985: cough and wheezing symptoms ceased for 5 children no longer exposed to WBS (2 in 1984 and 3 in 1985) while symptoms began and persisted for a child newly exposed to WBS in 1984. Proportions of children manifesting chronic respiratory illness symptoms during respective winters were as follows:

	1982		1984		1985	
	WBS	CONTROL	WBS	CONTROL	WBS	CONTROL
Cough at night	68%	3%	85%	20%	71%	5%
Cough >4d/wk	45%	0%	56%	5%	54%	5%
Wheeze	65%	16%	30%	10%	41%	16%

These differences could not be accounted for by socioeconomic factors, medical histories of children and parents, or exposure to other sources of indoor air pollution associated with respiratory symptoms (i.e., cooking with gas, parental smoking, urea-formaldehyde foam insulation). Findings suggest that indoor heating with a woodburning stove may be a significant factor in the occurrence of chronic respiratory symptoms in young children.

AIRWAY FUNCTION IN INFANTS: SIGNIFICANT ASSOCIATION OF PASSIVE SMOKE EXPOSURE AND RESPIRATORY ILLNESS. V Ackerman, R Tepper, V Long, P Yu. Indiana Univ. School of Medicine, Indianapolis, IN

The development of chronic respiratory disease in older children and adults has been associated with the occurrence of respiratory illness during infancy. This relationship has been primarily based upon the retrospective evaluation of history and lung function many years after infancy. The purpose of our study was to evaluate the association between the lung function of clinically asymptomatic infants and their individual and family respiratory history. We evaluated 58 asymptomatic infants at a mean post-natal age of 10.8 months (Age) who had a mean gestational age at birth of 8.9 months. Eighty percent were Caucasian (code=0) and 20% were Black (code=2). Twenty percent required supplemental oxygen (O_2) and/or mechanical ventilation (MV) as neonates and 25% experienced wheezing (HxAPt) or a lower respiratory tract illness (LRI). Family history was positive for asthma/allergy (HxAFm) in 28% of infants and 55% had exposure to cigarette smoke in their home (Smoke). Pulmonary function tests were performed during sleep including partial flow-volume curves by the rapid chest compression technique $\dot{V}_{maxFRC}$(ml/sec) and mixing index (MI). Analysis included step-wise multiple linear regression with coding yes=1 and no=0 for certain variables (HxAPt, Smoke, HxAFm). Regression equations were:

MI = .0068(Age)-.054(Bwt)-.057(HxAPt)-.035(Smoke)+.56
(R^2=.68), Bwt=birth weight

$\dot{V}_{maxFRC}$ = 19.2(Lt)-35.2(Race)-53.9(Wt)-66.7(HxAFm)-570.5
(R^2=.72), Lt/Wt=Length(cm) and weight(kg) at testing

There was a significant relationship of body size or age with MI and $\dot{V}_{maxFRC}$. MI values were lower in infants with HxAPt ($p<.003$) and in infants with exposure to smoking ($p<.03$). Blacks and infants with HxAFm had significantly lower values for $\dot{V}_{maxFRC}$($p<.03$). Neonatal respiratory history (days of O_2/MV) and gestational age were not statistically significant ($p>.2$). We conclude that passive smoke exposure, respiratory illness and HxAFm may be important determinants of airway function in infants. Prospective studies will be required to evaluate their effects on the growth and development of the lung.

Supported by NIH grants HL01322 and HL29090, Indiana Lung Association, Riley Memorial Association, and ALA-I Fellowship.