Respiratory Symptoms and Pulmonary Function in Chicken Catchers in Poultry Confinement Units

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To evaluate the respiratory consequences of working in poultry confinement units, we completed a cross-sectional epidemiologic study of respiratory symptoms and pulmonary function in 59 chicken catchers. The results were compared to a published reference standard of nonexposed blue-collar workers. Chicken catchers reported a high rate of acute symptoms associated with work in poultry houses. They also reported statistically significant higher rates for chronic phlegm (39.0%) and chronic wheezing (27.1%) than nonexposed blue-collar workers. Chicken catchers had significant decrements over a work shift in forced vital capacity (-2.2%) and forced expiratory volume in 1 sec (-3.4%), and there was suggestive evidence that they had decreased preshift pulmonary function compared with nonexposed blue-collar workers. These results indicate that chicken catchers are at risk for respiratory dysfunction and emphasize the need to develop measures to minimize their exposure to respiratory toxicants in poultry confinement units.

Key words: respiratory dysfunction, chronic bronchitis, pulmonary function tests, respiratory toxicants, organic dust, endotoxins, ammonia

INTRODUCTION

In the chicken processing industry, poultry confinement workers include growers and catchers who work in the poultry houses where birds are raised as well as hangers who work in the receiving areas of processing plants. Airborne contaminants in poultry confinement units include the mixture of agents comprising organic poultry dust—skin debris, broken feather barbules, insect parts, aerosolized feed, and poultry excreta—and a variety of immunogenic agents, such as viable bacteria and Gramnegative bacterial endotoxins [Lenhart and Olenchock, 1984]. Industrial hygiene surveys in the chicken processing industry have demonstrated that poultry confine-

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ment workers are exposed to high concentrations of such respiratory toxicants [Lenhart et al., 1982, in press; Olenchock et al., 1982]. In poultry houses, ammonia may also be an important respiratory toxicant [Manninen, 1989; Lenhart et al., in press].

Several studies suggest that poultry growers have an increased risk of respiratory dysfunction [Stahuljak-Beritic et al., 1977; Petro et al., 1978; Muller et al., 1986; Williams et al., 1984]. Whereas poultry growers spend a comparatively short time in confinement units feeding and watering birds [Lenhart and Olenchock, 1984], chicken catchers and hangers are exposed to aerosolized organic dust and other respiratory toxicants throughout most of their work shift and may be expected to have a greater risk of developing respiratory dysfunction. However, the respiratory health status of these workers has received relatively little attention. Therefore, we completed a cross-sectional epidemiologic study of respiratory symptoms and pulmonary function in a small group of chicken catchers from two different North Carolina poultry processing plants.

MATERIALS AND METHODS The Poultry Plants

At both of the poultry plants studied, the processing procedures are similar. Once chickens are ready for processing, a crew of chicken catchers consisting of six to ten catchers and one loader operator travel to a poultry house. Chickens are caught by hand and loaded onto trucks for transport to the processing plant. Each chicken catcher grabs about eight to 15 birds and places them in a crate. When a pallet of crates is full, the loader operator transports the pallet by forklift to a truck. A crew usually loads about 35,000 birds during an average 6 hr work shift. At the receiving area of the processing plant, chicken hangers shackle the birds to an overhead conveyor, which transports them to the slaughtering area.

The Study Populations

All 53 chicken catchers and six loader operators from the two plants were invited to participate in the study; all these workers were male, and all agreed to participate. For a control group, we had planned to recruit nonexposed poultry processing workers, who did not work in confinement units, but were not allowed access to these workers because of logistic problems at both plants. We then contacted other industries in the area but were unsuccessful in finding a suitable control group. Therefore, we used a 1985 reference standard from a National Institute for Occupational Safety and Health (NIOSH) study of North Carolina nonexposed blue-collar workers [Petersen and Castellan, 1984; Petersen and Hankinson, 1985]. This study selected approximately 1,400 workers from three major industry groups, food and kindred products, synthetic textile mills, and electrical equipment and supplies. Workers were eliminated from the analysis if they currently worked in dusty areas or with known respiratory irritants or if they had worked previously in occupations with suspected respiratory hazards for a total of 5 years or more. Occupational settings with suspected respiratory hazards included cotton, flax, hemp, jute, paper, and pulp mills; sawmills; mines; farms; grain elevators; mixing areas in bakeries; and receiving areas in poultry plants. There were 1,372 and 1,279 workers included in the respiratory symptoms and pulmonary function analyses, respectively.

Questionnaire

The same modified version of the respiratory symptoms questionnaire developed by the Medical Research Council (MRC) of Great Britain [MRC Committee on the Etiology of Chronic Bronchitis, 1960] was used for chicken catchers as that used in the referenced nonexposed blue-collar workers. The questionnaire was supplemented with questions concerning demographic information, occupational history, past medical history, and smoking habits. Specific chronic respiratory symptoms were defined as follows: chronic cough, cough on most days for at least 3 months each year; chronic phlegm, phlegm production on most days for at least 3 months year; episodic cough and phlegm, periods of cough and phlegm lasting at least 3 weeks in the past 3 years; grade 2 dyspnea, shortness of breath when hurrying on level ground or walking up a slight hill; grade 3 dyspnea, shortness of breath when walking with other people of same age on level ground, grade 4 dyspnea, shortness of breath when walking at own pace on level ground; chronic wheezing, wheezing on most days or nights; dyspnea and wheezing, attacks of shortness of breath with wheezing with normal breathing between attacks; and chest illness, chest illness with excess phlegm preventing usual activities for at least a week in the past three years. We also recorded acute symptoms that chicken catchers attributed to work in poultry houses and that occurred either during or shortly after completing a work shift, such as cough, wheezing, dyspnea, chest pain, chest tightness, eye irritation, nasal irritation, throat irritation, sneezing, headache, malaise, nausea, and fever.

Pulmonary Function Tests

Chicken catchers performed at least three maximal forced expiratory maneuvers on a waterless rolling-sealed spirometer before and after a work shift. In the blue-collar study, workers performed at least five maximal forced expiratory maneuvers on the same type of spirometer either before, during, or after a work shift. In both studies, the largest forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV₁) were used regardless of the curves on which they occurred. The ratio of FEV₁ and FVC (FEV₁/FVC%) was then calculated from these values. The spirometer and methods used in both studies met the minimum spirometry standards recommended by the American Thoracic Society [1979].

Occupational Exposure Measurements

To estimate occupational exposures, personal sampling equipment for inhalable and respirable dust and ammonia was randomly allocated to the chicken catchers. In general, each worker wore only one type of sampling apparatus. Equipment for inhalable dust sampling consisted of a three-piece, closed-face, 37 mm cassette containing a tared 5-µm-pore-size polyvinyl chloride (PVC) filter and a cellulose supporting pad. Each cassette was connected by tubing to a sampling pump operated at a flow rate of 1.5 liters/min. Equipment for respirable dust sampling consisted of a 10 mm Dorr-Oliver nylon cyclone connected by tubing to a sampling pump operated at a flow rate of 1.7 liters/min. All respirable dust particles separated by the cyclone were collected on a tared 5-µm-pore-size filter supported by a cellulose pad and contained in a two-piece, closed-face, 37 mm cassette. The mass of inhalable and respirable dust collected on each filter was determined gravimetrically [Department of Health and Human Services, 1984]. After gravimetric analysis, each dust sample was

analyzed for its bacterial endotoxin content by the quantitative chromogenic *Limulus* amebocyte lysate test [Jacobs, 1989]. Ammonia was measured using a Draeger long-duration ammonia detector tube connected by tubing to a sampling pump operated at a flow rate of 15–20 cc/min.

Analysis

In the nonexposed blue-collar study, linear logistic prediction equations were developed for chronic respiratory symptoms [Petersen and Castellan, 1984]. For current cigarette smokers (smokers), ex-cigarette smokers (ex-smokers), and those who had never smoked tobacco (nonsmokers), equations were fit separately using age, sex, race, height, weight, and education (years of school completed) as independent variables. For each chicken catcher, the blue-collar study prediction equations were used to calculate the predicted probability of having each symptom. The individual probabilities were then summed to give an expected number of subjects with each symptom. This procedure adjusts for the independent variables considered in the development of each equation. Expected prevalence rates for each symptom were thus calculated and compared to the exposed workers' observed rates.

For each pulmonary function variable, chicken catchers' preshift values were subtracted from postshift values, and the mean of the differences was calculated. Also, each exposed worker's change in a pulmonary function value was expressed as a percentage of the preshift value. The group mean percentage change in FVC, FEV $_1$, and FEV $_1$ /FVC% was then calculated.

In the nonexposed blue-collar study, prediction equations were also developed for the pulmonary function indices measured [Petersen and Hankinson, 1985]. Equations were fit using age, sex, race, height, weight, education (years of school completed), and smoking status (current cigarette smokers, ex-cigarette smokers, nonsmokers, and pipe- or cigars-only smokers). For each chicken catcher, these equations were used to calculate the predicted values for FVC, FEV₁, and FEV₁/FVC%. Exposed workers' predicted values for a pulmonary function variable were then subtracted from observed values, and the mean of the differences was calculated.

Statistical Tests

The Yates corrected χ^2 test [Rosner, 1982a] and the Fisher exact test [Rosner, 1982a] were used to compare differences in chronic respiratory symptom prevalence rates between chicken catchers and nonexposed blue-collar workers. The results of pulmonary function measurements (both acute effects across the work shift and exposed workers' preshift values compared to nonexposed blue-collar workers' predicted values) were analyzed by using the test for differences of paired variables [Rosner, 1982b].

RESULTS

We completed questionnaires on all 59 chicken catchers; however, spirometry was performed on only 43 workers. The 16 workers who did not have spirometry were similar to the 43 workers who did in terms of age, race, education, height, cigarette smoking status, pack-years of cigarette smoking, past occupational exposure to suspected respiratory hazards, and years of work exposure.

Fifty-one (86.4%) of the 59 chicken catchers complained of at least one acute

	Chicken catchers (N = 59)		Predicted Values ^a		Prevalence	
Symptom	No.	Percent	No.	Percent	odds ratio	p value
Chronic cough	19	32.2	9	15.3	2.64	0.05
Chronic phlegm	23	39.0	8	13.6	4.07	< 0.01
Episodic cough and phlegm	9	15.3	3	5.1	3.36	0.13
Grade 2 dyspnea	22	37.3	14	23.7	1.91	0.16
Grade 3 dyspnea	12	20.3	4	6.8	3.54	0.06
Grade 4 dyspnea	3	5.1	1	1.7	3.11	0.62
Chronic wheezing	16	27.1	4	6.8	5.12	0.01
Dyspnea and wheezing	10	16.9	7	11.9	1.52	0.60
Chest illness	3	5.1	1	1.7	3.11	0.62

TABLE I. Prevalence of Chronic Respiratory Symptoms in Chicken Catchers Compared to Nonexposed Blue-Collar Workers' Predicted Values

symptom associated with work in poultry houses. The highest prevalence rate was found for rhinitis (69.5%), followed by cough (61.0%), eye irritation (44.1%), sneezing (39.0%), nasal irritation (33.9%), wheezing (32.2%), headache (28.8%), dyspnea (22.0%), malaise (18.6%), nausea (16.9%), throat irritation (15.3%), chest pain (13.6%), chest tightness (11.9%), and fever (8.5%). Chicken catchers also reported high rates of chronic respiratory symptoms (Table I). Compared to nonexposed blue-collar workers, exposed workers had higher rates for chronic phlegm and chronic wheezing. Exposed workers also had higher rates of chronic cough, chronic episodic cough and phlegm, all three grades of dyspnea, dyspnea and wheezing, and chest illness, but these differences were not significant at the 5% level. In addition, 17 (28.8%) of the chicken catchers reported both chronic cough and chronic phlegm (not shown in Table I).

Table II shows the mean and percentage change over a work shift in FVC, FEV₁, and FEV₁/FVC% in 39 workers from whom we obtained both pre- and post-shift information. All three pulmonary function indices were decreased over the work shift; the decreases in FVC and FEV₁ were statistically significant. Ten (26%) of the workers had a decrement of >5% in FEV₁, and four (10%) had a decrement of >10% in FEV₁.

Table III shows the mean of the differences in FVC, FEV₁, and FEV₁/FVC% between chicken catchers and nonexposed blue-collar workers. All three preshift indices were decreased in the exposed workers; the difference of 0.23 liters for FVC was statistically significant, whereas the differences of 0.21 liters for FEV₁ and 2.12% for FEV₁/FVC% approached statistical significance.

Chicken catchers with ≥ 5 years of consecutive work exposure reported chronic respiratory symptoms more frequently than chicken catchers with < 5 years of consecutive work exposure. Prevalence rates for four of the chronic respiratory symptoms, stratified by years of consecutive work exposure, are detailed in Table IV. For all four symptoms, prevalence rates were higher for the ≥ 5 years of work exposure category. Similarly, when comparing chicken catchers and nonexposed blue-collar workers, pulmonary function deficits were generally confined to exposed workers in the ≥ 5 years of work exposure category (Table V).

^aBased on nonexposed blue-collar workers.

TABLE II. Pulmonary Function Values in Chicken Catchers Measured Across the Workshift

Test variables	Preshift (N = 39)	Across shift difference (post pre-)		
	$(mean \pm SD)$	Mean	Percent	p value
FVC (liters)	4.60 ± 0.88	-0.10	2.2	0.04
FEV ₁ (liters)	3.54 ± 0.80	-0.12	3.4	0.01
FEV ₁ /FVC (%)	76.78 ± 8.23	-0.94	1.2	0.10

TABLE III. Preshift Pulmonary Function Values in Chicken Catchers Compared to Nonexposed Blue-Collar Workers' Predicted Values

Test variables	Chicken catchers (N = 43) $(mean \pm SD)$	Mean of the differences	p value
FVC (liters)	4.60 ± 0.88	-0.23	0.04
FEV ₁ (liters)	3.54 ± 0.80	-0.21	0.05
FEV ₁ /FVC (%)	76.78 ± 8.23	-2.12	0.06

TABLE IV. Prevalence of Chronic Respiratory Symptoms in Chicken Catchers Compared to Nonexposed Blue-Collar Workers' Predicted Values (by Duration of Work Exposure)

Symptom	Chicken catchers (N = 59)		Predicted values ^a		
	No.	Percent	No.	Percent	p value
Chronic cough					
<5 Years ⁵	7	20.6	5	14.7	0.90
≥5 Years ^c	12	48.0	4	16.0	0.03
Chronic phlegm					
<5 Years	10	29.4	4	11.8	0.13
≥5 Years	13	52.0	4	16.0	0.02
Grade 3 dyspnea					
<5 Years	4	11.8	2	5.9	0.67
≥5 Years	8	32.0	2	8.0	0.08
Chronic wheezing					
<5 Years	7	20.6	2	5.9	0.09
≥5 Years	9	36.0	2	8.0	0.04

^aBased on nonexposed blue-collar workers.

Results of industrial hygiene sampling to estimate the exposures of chicken catchers have been reported elsewhere [Lenhart et al., in press]. The geometric mean 8 hr time-weighted average (TWA) concentration of inhalable dust for 17 chicken catchers was 20.2 mg/m³ (range 5.74–39.8). Thirteen (76.5%) of the individual samples taken exceeded 15 mg/m³, and 15 samples (88.2%) exceeded 10 mg/m³. The geometric mean 8 hr TWA concentration of respirable dust for 19 workers was 1.8 mg/m³ (range 0.6–4.2). The geometric mean 8 hr TWA inhalable concentration of bacterial endotoxins for 17 workers was 250 ng/m³ (range 27–700). Furthermore, 14 (82.4%) of the individual samples taken exceeded 100 ng/m³, whereas all 17 (100%)

 $^{{}^{}b}N = 34.$

 $^{^{}c}N = 25.$

Test variables	Chicken catchers (N = 43) $(mean \pm SD)$	Mean of the differences	p value
FVC (liters)			
<5 Years ^a	4.91 ± 0.72	+0.09	0.50
≥5 Years ^b	4.30 ± 0.92	-0.57	< 0.01
FEV ₁ (liters)			
<5 Years	3.95 ± 0.55	+0.09	0.34
≥5 Years	3.13 ± 0.79	-0.52	< 0.01
FEV ₁ /FVC (%)			
<5 Years	80.77 ± 5.72	-0.36	0.74
≥5 Years	73.01 ± 9.60	-3.95	0.05

TABLE V. Preshift Pulmonary Function Values in Chicken Catchers Compared to Nonexposed Blue-Collar Workers' Predicted Values (by Duration of Work Exposure)

of the samples exceeded 10 ng/m³. Finally, the geometric mean 8 hr TWA concentration of ammonia for 16 workers was 6 ppm (range 2–26). Two of the 16 (12.5%) individual samples equaled or exceeded 25 ppm.

DISCUSSION

The acute symptoms reported by chicken catchers suggest that several different clinical syndromes may be related to work exposures in poultry confinement units, including acute airways inflammation (rhinitis, upper airway irritation, and cough), bronchitis (cough, wheezing, and dyspnea), and reactive airway disease (wheezing, dyspnea, and chest tightness) [Rylander, 1986]. Some of the symptoms may represent acute effects to low levels of ammonia exposure (nasal and eye irritation) [Department of Health and Human Services, 1974; Manninen et al., 1989] or to high levels of bacterial endotoxins (eye irritation, rhinitis, cough, chest tightness, headache, malaise, nausea, and fever) [Lenhart and Olenchock, 1984].

Chicken catchers reported high rates of chronic respiratory symptoms. The symptom complex of cough, phlegm, wheezing, and dyspnea suggests an airway abnormality such as chronic bronchitis [Rylander, 1986]. A substantial proportion of exposed workers reported both chronic cough and chronic phlegm, which also suggests chronic bronchitis [Kelsey et al., 1986].

The significant decrement in FEV₁ across the work shift indicates that chicken catchers may be at increased risk for work-related acute reductions in lung function, which, with continued exposure, may result in chronic lung disease [Hankinson, 1986]. The reduction in preshift pulmonary function compared to nonexposed blue-collar workers, particularly in chicken catchers with ≥ 5 years of work exposure, suggests that such a chronic effect may be present.

Our data are consistent with limited information from other studies of poultry workers exposed to similar work environments. A NIOSH investigation used the MRC questionnaire to evaluate 25 black male chicken hangers in a North Carolina poultry processing plant [Richards et al., 1985]. Hangers reported higher prevalence

 $^{^{}a}N = 22.$

 $^{^{}b}N = 21.$

rates of chronic cough, phlegm, and wheezing than nonexposed black males in other departments of the processing plant. Two of the three personal dust samples collected exceeded 15 mg/m 3 . Investigators in Sweden studied 47 poultry workers involved in egg production and chicken raising [Thelin et al., 1984]. The workers reported high rates of acute respiratory symptoms associated with work, and there was a statistically significant average decrease in FEV $_1$ across the work shift. Organic dust and bacterial endotoxin levels were elevated. Donham and coworkers [1990] recently evaluated 257 Midwestern poultry workers involved in turkey production, egg production, broiler production, loading, and poultry processing (hangers) and 150 nonpoultry, nonexposed, blue-collar controls. They found that poultry work was associated with a significant decrease in FEV $_1$ and FEF $_{25-75}$ over the workshift. However, preshift pulmonary function values did not differ significantly between exposed and nonexposed workers.

Several studies of poultry farmers also suggest that exposure to poultry dust may result in respiratory dysfunction. A study of 61 poultry farm workers in Yugoslavia documented pulmonary function values significantly lower than predicted normal values [Stahuljak-Beritic et al., 1977]. A study of 42 poultry farmers in Germany showed mild, but significant, reductions in FVC, indicating a combined obstructive and restrictive defect of ventilation [Petro et al., 1978]. A second investigation in Germany involved 339 poultry farmers [Muller et al., 1986]. A longitudinal evaluation of 25 of the farmers demonstrated changes in the small airways after 5 years, indicating early impairment of pulmonary function. Finally, in a study of 25 North Carolina poultry farmers, investigators reported that growers with >1 hr of work exposure had a larger decrement in FEV₁ than growers with <1 hr of work exposure; however, this difference did not reach statistical significance [Williams et al., 1984].

There are several limitations to our study that should be considered when interpreting the results. First, exposed subjects were aware that this was an evaluation of respiratory function and work exposures; therefore, they may have been biased toward answering questions about respiratory symptoms positively and/or making submaximal forced expiratory maneuvers. We attempted to minimize such response bias by using standardized instruments and procedures for each study subject, the MRC questionnaire [MRC Committee on the Etiology of Chronic Bronchitis, 1960] for determining chronic respiratory symptoms and ATS recommendations [American Thoracic Society, 1979] for obtaining pulmonary function values.

A second limitation is the use of historical data for a control group; this could have resulted in many biases, not the least of which is measurement differences between exposed and nonexposed subjects. Such measurement bias is of particular concern when considering pulmonary function testing. Even though the same type of spirometer and the same general techniques were used in both studies, subtle procedural differences could have had substantial effects on the results [Hankinson, 1986].

A third limitation concerns possible inadequate control of confounding, particularly for two variables not included in the development of the prediction equations [Petersen and Castellan, 1984; Petersen and Hankinson, 1985], some measure of total cigarette smoke exposure such as pack-years of cigarette smoking and past occupational exposure to potential respiratory hazards. To explore the possibility of confounding further, we stratified by each of these variables separately and found high rates of respiratory symptoms and pulmonary function deficits in most strata. This indicates that confounding due to these variables may not have been responsible for

all positive effects; however, simultaneous control of both variables is needed to help verify such an assumption.

In spite of these limitations, there are several arguments against bias as an explanation for all of the positive findings in this study. These include the magnitude of the differences in symptom rates and pulmonary function values, the increase in measures of respiratory dysfunction in chicken catchers with ≥5 years of consecutive work exposure, and results of other studies demonstrating respiratory impairment in workers with similar exposures [Richards et al., 1985; Thelin et al., 1984; Donham et al., 1990; Stahuljak-Beritic et al., 1977; Petro et al., 1978; Muller et al., 1986; Williams et al., 1984]. Larger, more well-controlled studies of this specific work group are needed to help verify the present findings.

Even without substantial chronic effects on pulmonary function, the levels of organic dust and bacterial endotoxins in this work environment present an acute health hazard and should be reduced. Engineering controls to decrease the amount of respiratory toxicants should be developed and evaluated. Until engineering controls that prove beneficial can be implemented, NIOSH/MSHA-approved respirators [Lenhart et al., in press] offer an immediate method of exposure reduction and should therefore be evaluated in this occupational setting as well.

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REFERENCES

American Thoracic Society (1979): ATS statement—Snowbird workshop on standardization of spirometry. Am Rev Respir Dis 119:831–838.

Department of Health and Human Services (1974): "Criteria for a Recommended Standard: Occupational Exposure to Ammonia." Washington, DC: U.S. Government Printing Office.

Department of Health and Human Services (1984): NIOSH Manual of Analytical Methods, Third Edition, Volume 1." Washington DC: U.S. Government Printing Office.

Donham KJ, Leistikow B, Merchant JP, Leonard S (1990): Assessment of US poultry worker respiratory risks. Am J Ind Med 17:73-74.

Hankinson JL (1986): Pulmonary function testing in the screening of workers: Guidelines for instrumentation, performance, and interpretation. J Occup Med 28:1081–1092.

Jacobs RR (1989): Airborne endotoxins: An association with occupational lung disease. Appl Indust Hyg 4:50-56.

Kelsey JL, Thompson WD, Evans AS (1986): Cross-sectional and other types of studies. In "Methods in Observational Epidemiology." New York: Oxford University Press, pp 187–211.

Lenhart SW, Morris PD, Akin RE, Olenchock SA, Service WS, Boone WP (1990): Organic dust, endotoxin, and ammonia exposures in the North Carolina poultry processing industry. Appl Occup Environ Hyg 5:611-618.

Lenhart SW, Olenchock SA (1984): Sources of respiratory insult in the poultry processing industry. Am J Indust Med 6:89-96.

- Lenhart SW, Olenchock SA, Cole EC (1982): Viable sampling for airborne bacteria in a poultry processing plant. J Toxicol Environ Health 10:613-619.
- Manninen A, Kangas J, Linnainmaa M, Savolainen (1989): Ammonia in Finnish poultry houses: Effects of litter on ammonia levels and their reduction by technical binding agents. Am Indust Hyg Assoc J 50:210-215.
- MRC Committee on the Etiology of Chronic Bronchitis (1960): Standardized questionnaire on respiratory symptoms. Br Med J 2:1665.
- Muller S, Bergmann KC, Kramer H, Wuthe H (1986): Sensitization, clinical symptoms, and lung function disturbances among poultry farm workers in the German Democratic Republic. Am J Ind Med 10:281-282.
- Olenchock SA, Lenhart SW, Mull JC (1982): Occupational exposure to airborne endotoxins during poultry processing. J Toxicol Environ Health 9:339-349.
- Petersen M, Castellan RM (1984): Prevalence of chest symptoms in nonexposed blue-collar workers. J Occup Med 26:367-374.
- Petersen M, Hankinson J (1985): Spirometry reference values for nonexposed blue-collar workers. J Occup Med 27:644-650.
- Petro W, Bergmann K-C, Heinze R, Muller E, Wuthe H, Vogel J (1978): Long-term occupational inhalation of organic dust-Effect on pulmonary function. Int Arch Occup Environ Health 42: 119-127.
- Richards T, Morring K, Gamble J, Petersen M (1985): HETA 84-280, Perdue, Inc., Lewiston, NC, available from National Technical Information Service, Port Royal Road, Springfield, VA 22161.
- Rosner B (1982a): Hypothesis testing: Categorical data. In: "Fundamentals of Biostatistics." Boston: Duxbury Press, pp 288-343.
- Rosner B (1982b): Hypothesis testing: One-sample inference. In: "Fundamentals of Biostatistics." Boston: Duxbury Press, pp 171–224.
- Rylander R (1986): Lung diseases caused by organic dusts in the farm environment. Am J Ind Med
- Stahuljak-Beritic D, Dimov D, Buthovic D, Stilinovic L (1977): Lung function and immunological changes in poultry breeders. Int Arch Occup Environ Health 40:131-139.
- Thelin A, Tegler O, Rylander R (1984): Lung reactions during poultry handling related to dust and bacterial endotoxin levels. Eur J Respir Dis 65:266-271.
- Williams TM, Hickey JLS, Boehlecke BA, Jones WG (1984): HETA 83-195, David Mayer Poultry Farm, Hobgood, NC, available from National Technical Information Service, Port Royal Road, Springfield, VA 22161.