THE PREVALENCE OF SYMPTOMATIC CHRONIC BRONCHITIS IN TWO IRON ORE PROCESSING PLANTS

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THE PREVALENCE OF SYMPTOMATIC CHRONIC BRONCHITIS

IN TWO IRON ORE PROCESSING PLANTS

by

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ABSTRACT

This research was undertaken to study the prevalence of symptomatic chronic bronchitis in a group of mining and mill workers to determine if there was an association between symptomatic chronic bronchitis and tobacco smoking, length of dust exposure or age, individually or synergistically.

Analyses were done by two statistical methods. First, bivariate analysis done by cross tabulations using Chi-square as a test of significance gave the following results.

(a) There was a significant association between tobacco smoking and symptomatic chronic bronchitis. This association held when controlled by age and length of dust exposure.

(b) Age was not significantly associated to symptomatic chronic bronchitis.

(c) Length of dust exposure was not significantly associated to symptomatic chronic bronchitis.

Second, a log-linear analysis was used to test different models for associations among the variables. The results confirmed the association between symptomatic chronic bronchitis and smoking, and showed that there were some interactive effects between the independent variables, smoking, age and length of exposure which may be explained by the correlation between age and length of exposure and by some differences in smoking habits by age.

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CHAPTER I

GENERAL INTRODUCTION

Today chronic bronchitis is a widespread problem causing disease, disability and mortality. The economic cost of chronic bronchitis is reflected in hospital beds and physicians' services, nursing services, drugs, disability compensation and loss of productivity due to morbidity and mortality.

In the United States for the year 1977, the cost of Chronic Obstructive Pulmonary Disease (COPD) was estimated to be $1.0 billion for direct cost of treatment, $3.8 billion for costs due to morbidity and $900 million for costs due to mortality. These estimates were thought to be too low due to COPD being underdiagnosed and underreported and these estimates do not account for costs in terms of suffering (Hinshaw & Murrey, 1980).

This research involved the study of symptomatic chronic bronchitis in miners and mill workers in two iron ore processing plants in Eastern Canada.

Research Question

The objective of this thesis was to study the prevalence of symptomatic chronic bronchitis and determine if there was a relationship between symptomatic chronic bronchitis and tobacco smoking, length of dust exposure and age in 2,504 male miners and mill workers, employed at two iron ore processing plants. The number of female workers in the plant was too small to yield significant results so to most other regions in Canada. The mean annual temperature is below freezing, sometimes -22° C. The first snow fall usually occurs late September, and the last snow fall in late May. Summer is basically two months of the year, July and August, with an average temperature of 13° C. The two mining towns have developed since 1962 and 1965 respectively, with the majority of the working population employed directly with these industries.

Plant A and Plant B are located in separate communities several kilometers apart. The ore content of both mines is similar in that it is basically 35-38% iron with dust mixtures of silica. Both operations involve open-pit mining, ore concentration and transportation of ore through the various steps in their respective process.

Plant A differs from Plant B in the process of concentration of ore: Plant A uses a dry grinding process, whereas Plant B has a wet grinding process. Plant A also has a pellet plant operation.

Limitations

Since this is a cross-sectional study, the selective removal of workers from dusty jobs due to ill health may lead to an underestimation of respiratory symptoms and thus reduce the prevalence of symptomatic chronic bronchitis. Selective removal of workers from jobs is also known as the "healthy worker effect." This selection process can be seen more clearly before a person begins work, shortly after work and when the person reaches 50-54 years of age (Koskela, Luoma & Hernberg, 1976).

Hernberg (1980) found the healthy worker effect to vary, depending on certain factors. He identified five factors important to the healthy worker effect: (a) The younger the age group, the more marked was the healthy worker effect; (b) the identification of cohorts over time decreases the healthy worker effect, the health status of workers began to approach that of the general population; (c) the healthy worker effect was greater for non-whites than for whites in a mixed labour force; (d) diseases with silent early stages and rapid fatal course do not cause a healthy worker effect; (e) other factors such as labour shortage, unemployment, company hiring policy, physical demanding jobs, and certain occupations have an effect on the healthy worker effect.

Many factors, most unquantifiable, confuse and complicate the theory of the healthy worker effect. However, all researchers must be aware of the methodologic problems of the healthy worker effect when occupational groups are being studied.

Poor recall of symptoms of chronic bronchitis and tobacco smoking by the workers may be a source of error in this study.

The group may not be a truly representative sample of the population as a whole because of the demands and requirements of the mining industry.

Secondary analysis of data collected for other purposes means that the researcher faces restrictions in the method, amount and type of data to be analyzed.

Chapter Outline

Chapter I General Introduction

(a) Research Question
(b) Definition of Key Terms
(c) General Background Information
(d) Limitations of the Study
(e) Chapter Outline

Chapter II Review of Related Literature

(a) Literature Review
(b) Etiologic Factors Affecting Symptomatic Chronic Bronchitis
(c) State of the Art
(d) Anticipated Contribution

Chapter III Conceptual Framework and Methodology

(a) Statement of Hypothesis
(b) Models
(c) Definition and Measurement of Variables
(d) Discussion of Statistical Analyses

Chapter IV Descriptive and Comparative Analyses

Chapter V Multivariate Contingency Tables

Chapter VI Log-Linear Analyses

Chapter VII General Conclusion

(a) Restatement of Objectives
(b) Conclusion and Discussion with respect to support or rejection of hypothesis

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CHAPTER II

REVIEW OF RELATED RESEARCH

Literature Review

In 1805, Badham first made the diagnosis of chronic bronchitis, which he applied to chronic cough, breathlessness and recurrent exacerbations. Not much attention was given this disease until 1952. In December, 1952, a cold fog hung over London for five days. During this time the death rate rose by 4,000; the people mainly affected were those suffering from respiratory disease.

The following year the Medical Research Council of Britain set up a committee to advise and encourage research into chronic bronchitis. At this time confusion prevailed over a definition for symptomatic chronic bronchitis due to the poorly understood process of chronic bronchitis and emphysema.

In 1959, at the Ciba Guest Symposium, clinicians, epidemiologists and patholgists together agreed that emphysema would be defined on an anatomical basis as enlargement of air spaces with destructive changes in their walls, chronic bronchitis would be defined on a clinical basis as chronic expectoration, and asthma on a functional basis, as reversible airflow obstruction. The term chronic obstructive lung disease (COLD) was to be used for all irreversible obstruction.

The belief held at that time was that chronic bronchitis and emphysema was a single disease, chronic bronchitis was the middle stage and emphysema the latter stage. This idea was reinforced by the Medical

Research Council of Britain (1965) when they suggested that chronic bronchitis be classified into simple, mucopurulent and obstructive.

Today some researchers hold the belief that asthma, chronic bronchitis and emphysema is a disease spectrum. Fishman (1980) referred to chronic bronchitis and emphysema in terms of a spectrum. He felt it was not realistic, although possible, to try and account for the separate effects of the two diseases. Crofton and Douglas (1969) suggested the concept of a spectrum when they suggested that chronic bronchitis developed into primary emphysema. The American Lung Association (1977, p. 11) stated:

> The disease spectrum ranges from pure obstructive airway disease with chronic bronchitis but no emphysema through various combinations to severe emphysema without significant bronchitis.

Other researchers question this theory and support the theory that asthma, chronic bronchitis and emphysema have separate natural histories and are distinct separate diseases. Fletcher, Elmes, Fairbain and Wood (1959) viewed chronic bronchitis and emphysema as a single disease; however, their recent research supports the theory of two distinct diseases (Fletcher, Petro, Tinker & Speizer, 1976). Hinshaw and Murrey (1980) gave distinct pathologic entities for chronic bronchitis and emphysema with a common causal factor, tobacco smoke. Mitchell, Vincent, Ryan and Filley (1964) felt it was necessary to discriminate between chronic bronchitis and emphysema in order to improve the therapy for each, thus preventing or arresting their individual courses of development.

Thurlbeck (1977, p. 344) puts forth the argument that if chronic mucous hypersecretion (chronic bronchitis) precedes emphysema then

> . . . the frequency of chronic mucous hypersecretion would increase steadily with age, paralleling the increase in the frequency of emphysema. This does not happen: chronic mucous

hypersecretion is common before the age of 40 years in smokers, and its frequency does not increase as much as the frequency and severity of emphysema does with age. . . .

Due to existing overlap of symptoms of asthma, chronic bronchitis and emphysema an umbrella term Chronic Obstructive Lung Disease (COLD) was coined by the Ciba Guest Symposium (1959) to combine the reporting of these diseases. This term has become overused, and now is used synonymously with chronic obstructive pulmonary or lung disease (COPD, COLD), and chronic airway disease (CAD) (Thurlbeck, 1977). In 1969 the National Centre for Health Statistics added a new code number, ICDA No. 519.3 to cover the family of Chronic Obstructive Lung Disease (COLD) (American Lung Association, 1977).

Hinshaw and Murrey (1980) express the opinion that this term leaves much to be desired because it included several specific disorders with different clinical manifestations, pathologic features, requirements for therapy and prognosis.

<u>Etiologic Factors Affecting Symptomatic Chronic Bronchitis</u>

The factors that are suspected of having a causal role in symptomatic chronic bronchitis are varied. It is not known to what extent certain factors influence the development of symptomatic chronic bronchitis, whether they act alone or in synergism and to what extent.

Three suspected etiologic factors--tobacco smoking, age and length of dust exposure--will be studied in this research and their relationship, if any, to symptomatic chronic bronchitis.

<u>Tobacco smoking</u>. In the literature reviewed cigarette smoking is the one factor that consistently rates the highest as a causative

factor in symptomatic chronic bronchitis. The risk appears to increase as the number of cigarettes increase. Pipe and cigar smokers tend to show a lower rate of symptoms than cigarette smokers but higher than non-smokers. Ex-smokers show an improvement in their carbon monoxide level within hours of stopping, and sputum production, cough and breathlessness are reduced within weeks. Smoking and dust exposure seem to have an interacting effect and produce a higher rate of symptoms (Sleuis-Creamer, Walters & Sichel, 1967; Thurlbeck, 1977). A comprehensive overview of the literature reviewing the evidence linking tobacco smoking with chronic obstructive lung disease and respiratory symptoms may be found in The Surgeon General's Report, 1979A, "Smoking and Health."

Age. The American Lung Association (1977) acknowledged that ventilatory lung function deteriorated with age; however, they did not feel that COPD was primarily caused by aging. Higgins (1980) found the mortality rates in both sexes increased with age for chronic bronchitis, with females lower than males. Manfreda, Nelson and Cherniack (1978) found that low function lung tests were significantly relating to aging. Buist, Ghezzo, Anthonisen, Cherniack, Ducis, Macklem, Manfreda, Martin, McCarthy and Ross (1979) found that aging had a greater effect on various measures of lung function than smoking. Mittman, Pedersen and Barbela (1974) found that once length of exposure to dust and smoking had been taken into account, age by itself did not play an independent role in the symptoms of chronic bronchitis.

Throughout the literature, chronic bronchitis has been associated with increasing age, an upward trend at age 40 years and the peak age group being 50-65 years (Crofton & Douglas, 1969; The College of

General Practitioners, 1961; Gregory, 1961; Oswald, Harold & Martin, 1953).

Occupational factors (dust and fumes). Through the years symptomatic chronic bronchitis has been generally seen to be prevalent in men from low socioeconomic class, living in urban areas and working in a dusty environment. In the United States today, mortality rates for bronchitis are twice as high among semi-skilled workers and labourers as among professional workers (Higgins, 1980). The Medical Research Council of Britain (1966) published a report "Chronic Bronchitis and Occupation." This report was in response to the concern of the Minister of Pensions and National Insurance over persons eligible for insurance benefits. Persons with a clear chest film were not eligible while persons with pneumoconiosis were eligible for pensions. The Council's final report stated:

> However, on present evidence, intensity of dust exposures does not appear to be a very significant factor in determining the presence of bronchitis in this group of workers. (p. 102)

This report held far-reaching implications and was refuted by many people at that time. However, little scientific evidence was available to support the rebuttals.

Some studies done with the intent of measuring the relationship between dust exposure alone and respiratory symptoms have not been very successful, as can be seen from the following studies. Lowe, Campbell and Khosla (1970), in their study in South Wales, concluded that, if there was any relation between respiratory disability and atmospheric pollution in the two steel works, it is too slight to be detected, and that cigarette smoking was the most important factor in the etiology of SCB. Sleuis-Creamer et al. (1967) in Transvaal found chronic bronchitis to be significantly more common in miners than in non-miners for every age and smoking category except in non-smokers. In non-smoking miners and non-miners, no significant difference was found in the prevalence of chronic bronchitis. Clark, Harrington, Asta, Morgan and Sargent (1980) found that bronchitis symptoms and impairment of expiratory flow did not correlate significantly with exposure to dust, in their study of 249 men with 20 or more years of exposure to taconite dust in north eastern Minnesota, but was significantly related to cigarette smoking. Gregory (1971) found no evidence of the influence of dusty work on chronic bronchitis in his study of 340 steel-workers in Sheffield, England. Chan-Yeung, Wong, MacLean, Tan, Dorken, Schulzer, Dennis and Grzybowski (1980), in their study at Powell River, B.C., were unable to demonstrate an increased prevalence of respiratory symptoms among workers exposed to gases and chemicals in the Craft Mill. However, workers exposed to wood dust had slightly but significantly lower pulmonary function compared to other groups. The American Lung Association (1977) sees cigarette smoking as a much more important cause of COPD than occupational exposure.

Epidemiological studies done since 1966, supporting the view that prolonged inhalation of dust leads to increased respiratory symptoms are: Pratt, Vollmer and Miller (1980A) in their study of 565 unselected inflation fixed lungs found an association between cotton dust exposure and bronchitis. Musk, Peters, Wegman and Fine (1977) concluded in their study of 974 granite dust workers in Vermont, that present dust concentrations in the granite sheds caused excessive deterioration of lung capacity, however, Graham, O'Grady and Dubuc (1981) found conflicting results and questioned the measurement of the pulmonary function tests. Valic and Eugenijazuskin (1971), in their study of 60 cotton and 90 jute non-smoking females in Yugoslavia, found that exposure to cotton and jute dust caused significant reduction of FEV, FVC and PEF, over the first working shift in the week, cotton dust causing more effect than jute. Walker, Archibald and Allfield (1971), in their epidemiological survey of 881 men working in the coke industry, suggested that the combination of smoking, pollution from the coke-ovens and previous occupation, appears to be an important factor in the cause of bronchitis and reduced ventilatory capacity. Fairman, O'Brian, Swecker, Amandus and Shoub (1977), in their study of 1,438 surface coal miners in the U.S.A., found exposure to surface mine dust increased the prevalence of chronic bronchitis, but cigarette smoking was a more common factor in the prevalence of chronic bronchitis. Karava, Hernberg, Kaskela and Luoma (1976), in their study of 1,000 foundry workers in Finland, found that those classified as in dusty occupations had a higher frequency of chronic bronchitis. Smoking strongly increased the prevalence of chronic bronchitis and a combination of both exposures produced a stronger effect. Rogan, Attfield, Jacobsen, Roe, Walker and Walton (1973), found a progressive reduction in $FEV_{1.0}$ with increasing cumulative exposure to airborne dust. Kibelstis, Morgan, Keger, Lapp, Seaton and Morgan (1973), in their epidemiologic study of 8,555 bituminous coal miners in the U.S.A., found that smokers had a significantly higher prevalence of chronic bronchitis than their non-smoking or ex-smoking co-workers. Face workers had more bronchitis than surface workers, reflecting their lower dust exposure. The effect of smoking was found to be five times greater than the effect of coal dust. Dosman, Cotton, Graham, Robert, Froh and Barnett (1980), in their study of 180 non-smoking workers in Saskatchewan, concluded that exposure to grain dust is associated with an increased prevalence of chronic bronchitis and evidence of airflow obstruction. McDonald, Becklake, Fournier-Massey and Rossiter (1972), in their study of 1,015 male, chrysotile asbestos mine and mill workers in Quebec, found that age and smoking were related to bronchitis, dust exposure may have had an effect on non-smokers and light smokers. Pemberton (1968) reported that the survey done in Northern Ireland with flax spinners indicates that chronic bronchitis can be caused from flax dust. Mikov (1974), in his study of 779 foundry workers in Yugoslavia, concluded that tobacco smoking and air pollution at workplace were significant in the incidence and prevalence of chronic bronchitis.

Other etiologic factors that are suspected in the development of symptomatic chronic bronchitis are sex, infection, socioeconomic factors, heredity, air pollution and alcohol. These factors will be briefly looked at in the following studies.

<u>Sex</u>. The prevalence of symptoms of chronic bronchitis was found to be higher in males than females. This may be due to differences in smoking habits (Crofton, 1969). Bouhuys, Beck and Schoenberg (1979) found chronic bronchitis to be more common in men than women.

<u>Infection</u>. Throughout the years, respiratory infections were thought to play an important part in the development of chronic lung disease. In recent years, with more sophisticated research being carried out, there appears to be very little epidemiological or clinical evidence to support infection as a causal factor in the development of chronic lung disease (Fletcher, 1979; Crofton, 1969). The American

Lung Association (1977), in its overview of the available evidence, could not find a specific viral or bacterial agent as a causative agent in the development of chronic lung disease. Monti and Ross (1978) concluded in the Tecumset study that acute infection may play a role in the development of chronic lung disease.

Socioeconomic factors. Holland, Halil, Bennett and Elliott (1969) found that social class, area of residence, family size and past history of respiratory infections were associated with chronic respiratory disease. Lebowitz (1977) found that education and income were inversely related to respiratory problems. Fletcher (1979) gave the ratio of bronchitis mortality in unskilled labourers to that of professional classes as 5:1 in 1971; however, it is still unclear what aspects of low socioeconomic status are more clearly responsible for the symptoms. Higgins, Keller and Metzner (1977) found that respiratory disease was related to occupation, education and income, in their population study of Tecumset, Michigan.

Heredity. Like many other suspect factors in the development of symptomatic chronic bronchitis, heredity is a factor which seems to play a part in its development (American Lung Association, 1977). However, strong evidence is not available to support this theory (Crofton, 1969). Hinshaw and Murrey (1980) stated that Erickson (1965) discovered a specific type of emphysema related to the absence of serum alpha-1-antitrypsin. Mittman et al. (1974) concluded that at least part of symptomatic chronic bronchitis is genetically determined. Familial tendencies were found to be considered risk factors in chronic lung disease by Cohen, Ball, Brashears, Diamond, Kreiss, Levy, Menkes, Permutt and Tackman (1977); Cohen (1980).

Air pollution. Atmospheric pollution has been associated with high episodes of respiratory mortality and morbidity. The most well known incidences are the two smogs which hung over London in 1952 and 1962. The particulates that make up the air pollution, causing the most harm is hard to pinpoint; however, SO_2 and H_2SO_4 in combination is highly suspect.

Bouhuys, Beck and Schoenberg (1978) found no significant difference in the presence of chronic bronchitis between urban and rural residents.

The American Lung Association (1977) suggested a relationship between urban air pollution and chronic bronchitis. Hinshaw and Murrey (1980) have stated that although epidemiologic studies indicate an association between symptomatic chronic bronchitis and air pollution, the most harmful is personal pollution, tobacco smoking. Holland, Bennett, Cameron, Flarey, Leeder, Schilling, Swan and Waller (1979) give one of the most up-to-date overviews of the available evidence relating to air pollution and respiratory symptoms. Fletcher (1979) sees air pollution as much less important than cigarette smoking.

Alcohol. Recent research has given conflicting findings regarding alcohol consumption as a factor in the development of respiratory symptoms. Alcohol consumption is most often associated with tobacco smoking, and the effects of tobacco smoking are well documented. Lebowitz (1981) found that alcohol consumption was a significant risk factor in the development of respiratory symptoms. Chan-Yeung et al. (1980) found that alcohol consumption affected pulmonary function but was not significant.

State of the Art

Today chronic obstructive pulmonary disease is the most common chronic pulmonary disease. In recent years its prevalence has reached epidemic proportions, being the sixth leading cause of death (American Lung Association, 1977).

Throughout the reviewed literature there appears to be two theories on the history and course of chronic bronchitis. One area of thought views symptomatic chronic bronchitis as a spectrum preceded by asthma and followed by emphysema. This spectrum of disease is thus classified as chronic obstructive lung disease (Fishman, 1980; Crofton, 1969; Ciba Guest Symposium, 1959; Medical Research Council of Britain, 1965).

The other theory held by other researchers is that asthma, chronic bronchitis and emphysema are distinct diseases and have separate natural histories. Classifying these three diseases under one umbrella term, chronic obstructive lung disease, presents difficulties when one tries to compare morbidity rates of these distinct diseases (Fletcher et al., 1976; Hinshaw & Murrey, 1980; Huhti, 1965).

As one can see there is no agreement at present on the history or course of symptomatic chronic bronchitis. The definition of these diseases in most literature vary, if it is dealt with at all. The definitions most accepted are those set forth by the Ciba Guest Symposium (1959) and approved by the American Thoracic Society (1962). These definitions of asthma, chronic bronchitis and emphysema are as follows:

Asthma. Intermitten or reversible obstructive disease. Asthma refers to the condition of subjects with widespread narrowing of the bronchial airways, which changes its severity over short periods of time either spontaneously or under treatment, and is not due to cardiovascular disease.

Chronic bronchitis refers to the condition of subjects with chronic or recurrent excessive mucous secretion in the bronchial tree. The words "chronic or recurrent" may be defined as occurring on most days for at least three months in the year during at least two years.

Emphysema is a condition of the lung characterized by increase beyond the normal in the size of air spaces distal to the terminal bronchiole either from dilation or from destruction of their walls.

Many etiological factors are suspected of having a causal role in the development of symptomatic chronic bronchitis, which was noted in the literature review. The three factors studied in this research are tobacco smoking, age and length of dust exposure.

Tobacco smoking has been generally agreed upon as being one of the most important and causal factors in the development of symptomatic chronic bronchitis.

Aging is often shown to be an important factor in the development of symptomatic chronic bronchitis. The age group, 40 years and upward, is often associated with an upward trend of symptoms of chronic bronchitis. However, other studies show that aging by itself is not a primary cause of COPD or plays an independent role in the development of symptomatic chronic bronchitis.

Dust exposure is a very controversial factor in the etiology of symptomatic chronic bronchitis. Mineral dust has been suspected for a long time, however the scientific evidence is still questionable.

Textile dust would seem to produce stronger evidence as a factor in the etiology of symptomatic chronic bronchitis (Bouhuys et al., 1979; Pratt, Vollmer & Miller, 1980A).

Thirteen years ago, Gilson (1970) assessed the available evidence using the test suggested by Hill (1965). His assessment of the available evidence at that time would appear to hold true for today. A summary of his assessment is summarized in the following:

Strength:	The rating is low.
Consistency:	The rating is high.
Specificity:	The rating clearly low.
Temporality:	Questionable.
Biological gradient:	Questionable for mineral dust, the biological gradient is better established for textile dusts producing byssinosis.
Plausibility:	Rated very high.
Coherence:	The answer is no. The evidence is strong for cigarette smoking causing chronic bronchitis; other fumes and dust may also be a causative factor.
Experiment:	There is good experimental evidence indicating cigarette smoking as a causative agent. The evidence is not so good for mineral dust but stronger for textile dusts.

Due to the interchangeable diagnosis used in defining symptomatic chronic bronchitis, it seemed preferable to study the prevalence of symptomatic chronic bronchitis as defined by the Ciba Guest Symposium (1959), which has been used by most researchers in this area since that time.

Anticipated Contribution

As Morgan (1978) has pointed out, most preventive measures and in particular dust standards, are directed at the control of pneumoconiosis rather than of industrial bronchitis. If the latter indeed has a significant effect on symptomatic chronic bronchitis, present dust control methods might well prove to be ineffective or deficient.

Karava et al. (1976) found that in spite of the effect of selection, excess bronchitis could be demonstrated in workers from dusty environments. Therefore effective dust control must be initiated not only with regard to silica dust, but also with respect to total dust.

If the relationship between chronic bronchitis, tobacco smoking, length of dust exposure and age proves to be significant, one would expect that more effective methods of dust control, safety procedures and health examinations would be implemented, and workers made aware of the increased hazards to their health by tobacco smoking and the additional danger with dust effect.

CHAPTER III

CONCEPTUAL FRAMEWORK AND METHODOLOGY

Statement of Hypothesis

The objective of this research is to determine if there is an association between symptomatic chronic bronchitis and tobacco smoking, length of dust exposure and age, individually or synergistically (see Fig. 1).

The null hypothesis is stated as follows:

> There is no relationship between chronic bronchitis and age, dust exposure and tobacco smoking, individually or synergistically.

The data to be analyzed were obtained from the American Thoracic Questionnaire as published by the American Thoracic Society, Epidemiological Project (1978) with minor modifications for application by interviewer. The questionnaire was administered to all male workers employed as miners and mill workers in two mining operations in Eastern Canada.

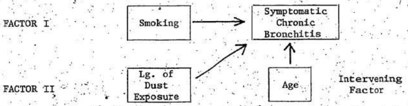
The questionnaire information was obtained by self-reported symptoms through personal interview by trained interviewers.

Definitions and Measurement of Variables

The variables to be used: symptomatic chronic bronchitis, tobacco smoking, length of dust exposure and age will be defined and measured in the following manner:

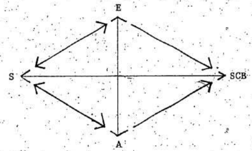
Model A

Simplest model showing direct effects of the independent variables on SCB individually.



Model B

A more complex model showing indirect effects of the independent variables on SCB syngeristically.



S = Tobacco smoking
E = Length of dust exposure
A = Age
SCB = Symptomatic chronic bronchitis.

Figure 1. Models.

Symptomatic Chronic Bronchitis. "Cough and phlegm during most days for a minimum of three months in a year and for two successive years" (American Thoracic Society, 1962).

Symptomatic chronic bronchitis will be determined by information collected on the ATS Questionnaire based on the following respiratory symptoms: cough, phlegm and time.

The specific questions analyzed to determine chronic bronchitis were (see Appendix A for complete derivation):

Question 7(E): Do you usually cough like this on most days for 6 consecutive months or more during the year?

Question 7(F): For how many years have you had this cough?

Question 8(E): Do you bring up phlegm like this on most days for 3 consecutive months or more during the year?

Question 8(F): For how many years have you had trouble with phlegm?

Tobacco smoking. Tobacco smoking was determined from information obtained from the ATS Questionnaire. The specific questions analyzed for tobacco smoking were (see Appendix A for complete derivation):

Question 28(B): Do you now smoke cigarettes as of one month ago?

Question 28(C): How many cigarettes do you smoke per day?

Question 29(B): Do you now smoke a pipe as of one month ago?

Question 30(B): Do you now smoke cigars as of one month ago?

Length of dust exposure. The occupational history of the workers was taken and used to determine the length of time employed in the mining plants, thus giving length of time exposed to dust.

Years of exposure was calculated by taking the total number of days worked (a day consisted of eight hours) and dividing by 220, which is the average number of days worked in a year. The following formula is an example:

$$\text{Yrs. of Exposure} = \frac{\text{Total number of days worked}}{220}$$

It was decided to use length of employment as a proxy measurement for dust exposure for the following reasons: First, historical measurements of dust within both plants had significant validity and reliability problems. This was due to (1) the type of instruments used, midget impinger, (2) the location of the instruments, area sampling instead of personal sampling. Second, most workers had many changes in their positions and locations within the plant. The relationship between the name of a given job and its possible location within the plant was frequently not known. Since dust exposure indices were calculated by combinations of area readings of dust and probable location of the worker within each job category, the degree of inaccuracy in placing the employee was a very important factor in the validity of its dust exposure.

These two factors--problems with actual measurement of dust and difficulty in locating the employees in specific areas through their employment history--were important enough to make a decision not to use them. In fact, the use of dust exposure indices for each worker calculated by a very complex algorithm which included many assumptions and "averaging" operations using dust data which were suspected in its accuracy was considered to be a deceptive method, which would assume a quality of data that was not supported by the evidence.

The use of length of employment as a proxy for dust exposure has obvious problems, the most important being that it assumes a relationship between years of employment and dust exposure which in fact has not been demonstrated. However, it was felt that the decision to use length of employment as an indirect measure of dust exposure was a reasonable and justifiable one. Other researchers have used this method in the past (Dosman et al., 1980; Fairman et al., 1977; Mikov, 1974).

Age. Workers were divided into four age groups: ≤ 29 years, 30–39 years, 40–49 years, ≥ 50 years for the first analyses.

The question used was Number 1, date of birth, by year, month, day (see Appendix A for complete derivation).

Statistical Analyses

This research was done using the following methods of analyses.

Chapter IV gives a descriptive and comparative analyses of Plant A and Plant B. The plants were examined separately and together by age, smoking habits and length of dust exposure, with descriptive and comparative statistics.

Chapter V gives an analysis of the total work force, using cross tabulation, multivariate analyses. Tests for significance were done by a non-parametric method, Chi-square test.

In Chapter VI, the data are analyzed by a log-linear model. The purpose of using this model is to determine if there is a relationship between the variables chronic bronchitis and age, length of dust exposure and tobacco smoking, individually or synergistically. This analysis is achieved by testing and ordering the importance of the effects among the factors, and forming a model of best fit for the data sample.

The log-linear analysis is applicable for this data because of the small frequencies in some cells, and the ability to test for significant interactions among the variables.

CHAPTER IV

DESCRIPTIVE AND COMPARATIVE ANALYSES

Chapter IV will give a descriptive and comparative analyses of each plant. The independent variables, age, smoking and length of exposure, will be analyzed by bivariate analyses.

Plant A had a population of 2,206 workers; 1,973 (89.4%) workers answered the ATS Questionnaire and had work histories completed giving length of exposure. In Plant A, the youngest age was 20 years; the oldest age was 66 years; the mean age was 35 years with a standard deviation of 9.45.

Plant B had a population of 586 workers; 531 (90.6%) workers answered the ATS Questionnaire and had work histories completed giving length of exposure. In Plant B, the youngest age was 20 years; the oldest age was 66 years; the mean age was 37.39 with a standard deviation of 8.99.

Plants A and B together had a population of 2,792 workers; 2,504 (89.7%) workers answered the ATS Questionnaire and had work histories completed giving length of exposure.

Plants A and B together had a maximum length of exposure of 31.31 years, mean length of exposure of 8.51 years with a standard deviation of 5.99 years.

The treatment of the variable smoking was done after a careful study of the results of the appropriate questions in the ATS Questionnaire (Questions 28, 29 and 30, Appendix A). Appendix B shows the results of this analysis. Taking into account the frequencies for never smoked, ex-smokers and present smokers, of cigarettes, pipe and cigar, alone or in combination, it was decided:

- To place only pipe and/or cigar smokers in the light smokers' category.
- To classify all combinations of present smokers according to the number of cigarettes smoked.

Tobacco smokers were classified into three categories: never smoked, ex-smokers and present smokers. Present smokers were subdivided into light smokers (≤ 14 cigarettes per day), moderate smokers (15-24 cigarettes per day), and heavy smokers (≥ 25 cigarettes per day). This classification of smoking has been used by researchers in the past (Mikov, 1974; Lowe, Campbell & Khasla, 1970; Doll & Hill, 1964; Karava et al., 1976; Crofton & Douglas, 1969; Ashley, 1980).

Light smokers were the subjects that presently smoke ≤ 14 cigarettes a day. There were 42 subjects that only smoked pipe, cigar or their combination, and were included with the light smokers' category.

The specific questions and answers that were examined were (see Appendix A for complete derivation):

Question 28(B): Do you now smoke cigarettes as of one month ago?

Answer: (1) Yes

Question 28(C): How many cigarettes do you smoke per day?

Answer: $\leq$ 14 cigarettes

Question 29(B): Do you now smoke a pipe as of one month ago?

Answer: (1) Yes

Question 30(B): Do you now smoke cigars as of one month ago?

Answer: (1) Yes

Medium smokers were subjects that presently smoke 15-24 cigarettes per day with any combination of other smoking. The specific questions examined were (see Appendix A for complete derivation):

Question 28(B): Do you now smoke cigarettes as of one month ago?

Answer: (1) Yes

Question 28(C): How many cigarettes do you smoke per day?

Answer: 15-24 cigarettes

Question 29(B): Do you now smoke a pipe as of one month ago?

Answer: (1) Yes

Question 30(B): Do you now smoke cigars as of one month ago?

Answer: (1) Yes

Heavy smokers were subjects that presently smoke $\geq$ 25 cigarettes per day, with any combination of other smoking.

The specific questions examined were (see Appendix A for complete derivation):

Question 28(B): Do you now smoke cigarettes as of one month ago?

Answer: (1) Yes

Question 28(C): How many cigarettes do you smoke per day?

Answer: $\geq$ 25 cigarettes

Question 29(B): Do you now smoke a pipe as of one month ago?

Answer: (1) Yes

Question 30(B): Do you now smoke cigars as of one month ago?

Answer: (1) Yes

Never smoked--are the subjects that never smoked any tobacco. The specific questions were (see Appendix A for complete derivation):

Question 28(B): Do you now smoke cigarettes as of one month ago?

Question 29(B): Do you now smoke a pipe as of one month ago?

Question 30(B): Do you now smoke cigars as of one month ago?

Answers to these questions (8) — Non-applicable

Ex-smokers--are the subjects that had quit smoking as of one month prior to the study. The specific questions were (see Appendix A for complete derivation):

Question 28(B): Do you now smoke cigarettes as of one month ago?

Question 29(B): Do you now smoke a pipe as of one month ago?

Question 30(B): Do you now smoke cigars as of one month ago?

Answers to these questions (2) — No

Table 1

Age by Plants A and B

Age	Plant A		Plant B		Total	
	No.	%	No.	%	No.	%
≤ 29	647	32.8	73	13.7	720	28.8
30–39	765	38.8	257	48.4	1,022	40.8
40–49	359	18.2	133	25.0	492	19.6
≥ 50	202	10.2	68	12.8	270	10.8
TOTAL	1,973	100	531	100	2,504	100

$\chi^2_3 = 74.83$, $p < .001$.

Table 1 shows workers in Plants A and B by four Age Categories, who answered the ATS Questionnaire and had work histories completed. χ^2 shows a significant difference in ages between the two plants, $\chi^2_3 = 74.83$, $p < .001$.

Table 1A

Age by Plants A and B

Age	Plant A		Plant B		Total	
	No.	%	No.	%	No.	%
≤ 39	1,412	71.57	330	62.15	1,742	69.57
≥ 40	561	28.43	201	37.85	762	30.43
TOTAL	1,973	79.79	531	21.21	2,504	100

$\chi_1^2 = 17.53,\ p < .001$

Table 1A shows workers in Plants A and B by two age categories, ≤ 39 years and ≥ 40 years, who answered the ATS Questionnaire and had work histories completed. χ^2 shows a significant difference in ages between the two plants, $\chi_1^2 = 17.53$, $p < .001$

Tables 1 and 1A show the population of both plants. Plant A has the majority of the workers, 79.79%; Plant B has 21.21% of the target population. There is a significant difference in the ages between Plants A and B. Plant A has a younger population than Plant B, especially in age group ≤ 29 years. Plant B has has an older population in age groups 30-39 years and 40-49 years. The majority of workers in both plants are in the age group ≤ 39 years. Plant A has 71.57%, Plant B has 62.15%. Overall, 69.57% of the target population is in this age group.

Table 2

Length of Exposure by Plants A and B

Length of Exposure	Plant A		Plant B		Total	
	No.	%	No.	%	No.	%
$\leq$ 5 yrs.	738	37.40	193	36.35	931	37.18
6–10 yrs.	609	30.87	105	19.77	714	28.51
$\geq$ 11 yrs.	626	31.73	233	43.88	859	34.31
TOTAL	1,973	100	531	100	2,504	100

$\chi^2_2 = 36.19, \ p < .001$

Table 2 shows Plants A and B by lengths of exposure, $\leq$ 5 years, 6–10 years, $\geq$ 11 years. There is a significant difference between lengths of exposure for Plants A and B, $\chi^2_2 = 36.19$, $p < .001$. Both Plants have approximately the same percent of workers in the $\leq$ 5 year exposure. Plant A has a significantly higher percent of workers in the 6–10 year exposure category whereas Plant B has a significantly higher percent of workers in the $\geq$ 11 year exposure category.

For the overall target population, the highest percent of workers is in the $\leq$ 5 year exposure category, 37.18%, and the lowest percent of workers in the 6–10 year exposure category, 28.51%.

Table 3

Smoking by Plants A and B

Smoking Group	Plant A		Plant B		Total	
	No.	%	No.	%	No.	%
Never Smoked	408	20.68	102	19.21	510	20.36
Ex Smokers	608	30.82	167	31.45	775	30.95
Smokers	957	48.50	262	49.34	1,219	48.68
TOTAL	1,973	100	531	100	2,504	100

$\chi_2^2 = 0.56$, $p > 70$

Table 3A

Present Smokers by Plants A and B

Present Smokers	Plant A		Plant B		Total	
	No.	%	No.	%	No.	%
Light	272	28.42	67	25.57	339	27.81
Mod.	334	34.90	98	37.40	432	35.44
Heavy	351	36.68	97	37.02	448	36.75
TOTAL	957	100	262	100	1,219	100

$\chi_2^2 = .97$, $p > .50$

Tables 3 and 3A show Plants A and B by smoking categories. There is no significant difference between Plants A and B for smoking categories. For the target population, 20.36% never smoked, 30.95% are ex-smokers, and 48.68% are smokers. When the present smoking categories are sub-divided into light, moderate and heavy smokers, both plants have approximately the same percent in each category.

Table 4

Age by Length of Exposure, Plant A

	Length of Exposure							
Age	≤ 5 yr.		6-10 yr.		≥ 11 yr.		Total	
	No.	%	No.	%	No.	%	No.	%
≤ 39	646	45.7	514	36.4	252	17.8	1,412	100
≥ 40	92	16.4	95	16.9	374	66.7	561	100
TOTAL	738	37.4	609	30.9	626	31.7	1,973	100

Table 5

Age by Length of Exposure, Plant B

	Length of Exposure							
Age	≤ 5 yr.		6-10 yr.		≥ 11 yr.		Total	
	No.	%	No.	%	No.	%	No.	%
≤ 39	141	42.73	84	25.45	105	31.82	330	100
≥ 40	52	25.87	21	10.45	128	63.68	201	100
TOTAL	193	36.35	105	19.77	233	43.88	531	100

Table 6

Age by Length of Exposure, Target Population

	Length of Exposure							
Age	≤ 5 yr.		6-10 yr.		≥ 11 yr.		Total	
	No.	%	No.	%	No.	%	No.	%
≤ 39	787	45.18	598	34.33	357	20.49	1,742	100
≥ 40	144	18.90	116	15.22	502	65.88	762	100
TOTAL	931	37.18	714	28.51	857	34.23	2,504	100

Table 4 (Plant A), Table 5 (Plant B) and Table 6 (Target Population) show the relationship between age and length of exposure; as expected, both variables are correlated.

Table 7

Age by Smoking, Plant A

Age	Never		Ex		Light		Moderate		Heavy		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
≤ 29	180	27.82	142	21.95	97	14.99	121	18.70	107	16.54	647	100
30–39	151	19.74	247	32.29	101	13.20	132	17.25	134	17.52	765	100
40–49	55	15.32	138	38.44	43	11.98	51	14.21	72	20.06	359	100
≥ 50	22	10.89	81	40.10	31	15.35	30	14.85	38	18.81	202	100
TOTAL	408	20.68	608	30.82	272	13.79	334	16.93	351	17.79	1,973	100

Table 8

Age by Smoking, Plant B

Age	Never		Ex		Light		Moderate		Heavy		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
≤ 29	26	35.62	12	16.44	14	19.18	13	17.81	8	10.96	73	100
30–39	50	19.46	78	30.35	34	13.23	42	16.34	53	20.62	257	100
40–49	21	15.79	52	39.10	14	10.53	22	16.54	24	18.05	133	100
≥ 50	5	7.35	25	36.76	5	7.35	21	30.88	12	17.65	68	100
TOTAL	102	19.21	167	31.45	67	12.62	98	18.46	97	18.27	531	100

Table 9

Age by Smoking, Target Population

Age	Never		Ex		Light		Moderate		Heavy		Total	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
≤ 29	206	28.61	154	21.39	111	15.42	134	18.61	115	15.97	720	100
30–39	201	19.67	325	31.80	135	13.21	174	17.03	187	18.30	1,022	100
40–49	76	15.45	190	38.62	57	11.59	73	14.84	96	19.51	492	100
≥ 50	27	10.0	106	39.26	36	13.33	51	18.89	50	18.52	270	100
TOTAL	510	20.37	775	30.95	339	13.54	432	17.25	448	17.89	2,504	100

Table 7 (Plant A), Table 8 (Plant B), and Table 9 (Target Population) show age by smoking categories. The percent of workers in the smoking categories does not differ in the age groups.

The percent of workers who never smoked is higher for the younger age group, while the percent of ex-smokers is higher in the older age group.

Table 10

Age by Smoking, Plant A

Age	Non-Smokers		Smokers		Total	
	No.	%	No.	%	No.	%
≤ 39	720	51	692	49	1,412	100
≥ 40	296	52.76	265	47.24	561	100
TOTAL	1,016	51.50	957	48.50	1,973	100

$X_1^2 = 0.50, p < .30$

Table 11

Age by Smoking, Plant B

Age	Non-Smokers		Smokers		Total	
	No.	%	No.	%	No.	%
≤ 39	166	50.30	164	49.70	330	100
≥ 40	103	51.24	98	48.76	201	100
TOTAL	269	50.66	262	49.34	531	100

$X_1^2 = 0.04, p > 80$

Table 12

Age by Smoking, Target Population

Age	Non-Smokers		Smokers		Total	
	No.	%	No.	%	No.	%
≤ 39	886	50.86	856	49.14	1,742	100
≥ 40	399	52.36	363	47.64	762	100
TOTAL	1,285	51.32	1,219	48.68	2,504	100

$\chi_1^2 = 0.48, \; p > .30$

Table 10 (Plant A), Table 11 (Plant B), and Table 12 (Target Population) show smoking categories collapsed into non-smokers and present smokers, for age groups collapsed into two categories, ≤ 39 years and ≥ 40 years. There is no significant difference between age categories or the non-smokers and present smokers. For both age groups, the percent of non-smokers is slightly higher than the percent for present smokers.

Summary

In the descriptive and comparative analyses for Plants A and B, there was found to be a significant difference in the ages of both plants. Plant A had a significantly younger population than Plant B, especially in the ≤ 29 year age group, with 32.8% as compared to 13.7% in Plant B.

For both plants, the majority of workers are in the age group ≤ 39 years, Plant A has 71.45%, Plant B has 62.15%. For the overall target population, 69.57% is in the age group ≤ 39 years.

When comparing Plants A and B for length of exposure, there showed a significant difference for length of exposure. For the ≤ 5 year exposure group, both plants had approximately the same percent. For the 6-10 years exposure group, Plant A had a significantly higher percent of workers than Plant B. For the ≥ 11 years exposure group, Plant B had a significantly higher percent of workers than Plant A. For the overall target population, the ≤ 39 year age group had 45.18% with ≤ 5 years exposure and 20.49% with ≥ 11 years' exposure. The reverse held true for the ≥ 40 year age group, having 18.90% with ≤ 5 years' exposure and 65.88% with ≥ 11 years' exposure.

When comparing smoking by Plants A and B, there was no significant difference in smoking patterns between Plants A and B.

In comparing length of exposure by age for each plant separately and together, as would be expected, there is a significant difference with the younger age group, ≤ 39 years, having less years of exposure than the older age group, ≥ 40 years, which has more years of exposure, showing an intercorrelation between these two variables.

Analyses by smoking categories by age for Plants A, B and the target population were done. Plant A and the target population had a significantly higher percent of workers in the ex-smoking category. In the present smoking category, there was no significant difference among the sub-division categories: light, moderate and heavy. For Plant B, the numbers were too small to draw any conclusions. For this reason, it was decided to do further analyses by Plants A and B combined, the target population.

CHAPTER V

MULTIVARIATE CONTINGENCY TABLES

Chapter V gives a statistical analyses by the target population using multivariate cross tabulation analysis. Significance is tested by a non-parametric method, Chi-square test.

Symptomatic chronic bronchitis will be shown by each plant for age and length of dust exposure to clearly show that the small numbers of subjects having symptomatic chronic bronchitis, and the small total numbers in each classification for Plant B makes Chi-square testing unreliable, therefore Plants A and B will be combined (target population) for most tables and final analysis.

Symptomatic chronic bronchitis will be determined by information collected on the questionnaire based on the following respiratory symptoms: cough, phlegm and time.

The specific questions and answers to determine chronic bronchitis were (see Appendix A for complete derivation):

Question 7(E): Do you usually cough like this on most days for 6 consecutive months or more during the year?

Answer: (1) Yes

Question 7(F): For how many years have you had this cough?

Answer: $\geq$ 2 years

Question 8(E): Do you bring up phlegm like this on most days for 3 consecutive months or more during the year?

Answer: (1) Yes

Question 8(F): For how many years have you had trouble with phlegm?

Answer: $\geq$ 2 years

TABLE 13

SCB by Age, Plant A

Age	No		Yes		Total	
	No.	%	No.	%	No.	%
≤ 29	599	92.6	48	7.4	647	100
30-39	718	93.9	47	6.1	765	100
40-49	331	92.2	28	7.8	359	100
≥ 50	182	90.1	20	9.9	202	100
TOTAL	1,830	92.7	143	7.3	1,973	100

$\chi^2_3 = 3.69,\ p > .20$

Table 13 shows the relationship between SCB and four age categories in Plant A. There is a slight increase in SCB for the older age group but this difference is not significant, $\chi^2_3 = 2.59$, $p > .20$.

Table 14

SCB by Age, Plant B

Age	No		Yes		Total	
	No.	%	No.	%	No.	%
≤ 29	69	94.5	4	5.5	73	100
30–39	230	89.5	27	10.5	257	100
40–49	122	91.7	11	8.3	133	100
≥ 50	61	89.7	7	10.3	68	100
TOTAL	482	90.8	49	9.2	531	100

$\chi^2_3 = 1.96, \; p > .50$

Table 14 shows the relationship between SCB and four age categories in Plant B. Due to the small number of SCB in Age category ≤ 29 years, and the small totals for Age ≤ 29 years and ≤ 50 years, it is not feasible to draw any reliable conclusions from Plant B. In Table 15, which follows, both plants will be analyzed together.

Table 15

SCB by Age, Target Population

Age	No		Yes		Total	
	No.	%	No.	%	No.	%
≤ 29	668	92.78	52	7.22	720	28.75
30–39	948	92.76	74	7.24	1,022	40.76
40–49	453	92.27	39	7.72	492	19.63
≥ 50	243	90.00	27	10.00	270	10.77
TOTAL	2,312	92.37	192	7.63	2,504	100

$\chi^2_3 = 2.59,\ p > .30$

Table 15 shows the relation between SCB and age groups in four classes. There is a slight increase in SCB through the age groups, with a maximum difference of 2.78% between the youngest and oldest age groups. This difference is not significant, $\chi^2_3 = 2.59$, $p > .30$.

Table 16

SCB by Length of Exposure, Plant A

Exposure Groups	No		Yes		Total	
	No.	%	No.	%	No.	%
≤ 5 yr.	681	92.3	57	7.7	738	100
6-10 yr.	572	93.9	37	6.1	609	100
≥ 11 yr.	577	92.2	49	7.8	626	100
TOTAL	1,830	92.8	413	7.2	1,973	100

Table 16 shows length of exposure by SCB for Plant A. There is no ~~significant~~ difference in SCB for exposure groups in Plant A.

Table 17

SCB by Length of Exposure, Plant B

Length of Exposure	No		Yes		Total	
	No.	%	No.	%	No.	%
≤ 5 yr.	172	89.1	21	10.9	193	100
6-10 yr.	91	86.7	14	13.3	105	100
≥ 11 yr.	219	94.0	14	6.	233	100
TOTAL	482	91.8	49	9.2	531	100

Table 17 shows length of exposure by SCB for Plant B. The totals for exposure categories in Plant B do not have large enough numbers from which to draw any reliable conclusions. Table 18 will show length of exposure by SCB for both plants.

Table 18

SCB by Length of Exposure, Target Population

Length of Exposure	No		Yes		Total	
	No	%	No	%	No	%
≤ 5 yr.	853	91.62	78	8.38	931	100
6-10 yr.	663	92.86	51	7.14	714	100
≥ 11 yr.	796	92.67	63	7.33	859	100
TOTAL	2,312	92.34	192	7.66	2,504	100

$\chi^2_2 = 1.08$, $p > .50$

Table 18 shows length of exposure by SCB for the target population. There is a slightly higher rate of SCB in the group with less exposure, but the difference is not significative.

Table 18A

SCB by Length of Exposure for Non-Smokers, Target Population

Length of Exposure	No		Yes		Total	
	No	%	No	%	No	%
≤ 5 yr.	439	97.77	10	2.23	449	100
6-10 yr.	353	98.33	6	1.67	359	100
≥ 11 yr.	460	96.44	17	3.56	477	100
TOTAL	1,252	97.43	33	2.57	1,285	100

$\chi^2_2 = 3.25$, $p > .10$

Table 18A shows length of exposure by SCB for the non-smokers in the target population, as a further check to show that length of exposure is not significant in the non-smokers for this target group.

Table 19

SCB by Smoking for Target Population

Smoking	No		Yes		Total	
	No	%	No	%	No	%
Non-Smokers	1,252	97.43	33	2.57	1,285	51.32
Light	317	93.51	22	6.49	339	13.54
Moderate	381	88.19	51	11.81	432	17.25
Heavy	362	80.80	86	19.20	448	17.89
TOTAL	2,312	92.34	192	7.66	2,504	100

$\chi^2_3 = 142.42,\ p < .001$

Table 19 shows smoking categories by SCB for the target population. The difference in SCB by smoking groups is highly significant, $\chi^2_3 = 142.42$, $p < .001$.

Table 20

Smoking by SCB for ≤ 39 Years of Age, Target Population

Smoking	No		Yes		Total	
	No.	%	No.	%	No.	%
Non-Smokers	871	98.31	15	1.69	886	50.86
Light	231	0.41	15	6.10	246	14.12
Moderate	272	88.31	36	11.69	308	17.68
Heavy	242	80.13	60	19.87	302	17.34
TOTAL	1,616	92.76	126	7.24	1,742	100

$\chi^2_3 = 121.96$, $p < .001$

Table 21

Smoking by SCB for ≥ 40 Years of Age, Target Population

Smoking	No		Yes		Total	
	No.	%	No.	%	No.	%
Non-Smokers	381	95.49	18	4.51	399	52.36
Light	86	92.47	7	7.53	93	12.21
Moderate	109	87.90	15	12.10	124	16.27
Heavy	120	82.19	26	17.81	146	19.16
TOTAL	696	91.34	66	8.66	762	100

$\chi^2_3 = 26.13$, $p < .001$

Tables 20 and 21 show the association between SCB and smoking controlling for age, ≤ 39 years (Table 20) and ≥ 40 years (Table 21). The rate of SCB remains significantly associated with smoking, the younger age group, ≤ 39 years, shows a higher significance than the ≥ 40 years age group.

Table 22

Smoking by SCB for ≤ 5 Year Exposure Group, Target Population

Smoking	No		Yes		Total	
	No.	%	No.	%	No.	%
Non-Smokers	439	97.77	10	2.23	449	100
Light	124	93.94	8	6.06	132	100
Moderate	155	89.08	19	10.92	174	100
Heavy	135	76.70	41	32.30	176	100
TOTAL	853	91.62	78	8.38	931	100

$\chi^2_3 = 75.54, p < .001$

Table 23

Smoking by SCB for 6-10 Year Exposure Group, Target Population

Smoking	No		Yes		Total	
	No.	%	No.	%	No.	%
Non-Smokers	353	97.68	6	2.32	359	100
Light	98	94.23	6	5.77	104	100
Moderate	110	87.30	16	12.70	126	100
Heavy	102	81.60	23	18.40	125	100
TOTAL	663	91.69	51	8.31	714	100

$\chi^2_3 = 32.99, p < .001$

Table 24

Smoking by SCB for ≥ 11 Year Exposure Group, Target Population

Smoking	No		Yes		Total	
	No.	%	No.	%	No.	%
Non-Smokers	460	96.44	17	3.56	477	100
Light	95	92.23	8	7.77	103	100
Moderate	116	87.88	16	12.12	132	100
Heavy	125	85.03	22	14.97	147	100
TOTAL	796	92.67	63	7.33	859	100

$\chi^2_3 = 27.05,\ p < .001$

Tables 22, 23 and 24 show SCB by smoking controlling for length of exposure, ≤ 5 years, 6-10 years and ≥ 11 years. The association remains significant, although it diminishes as length of exposure increases.

Summary

Analyses of Plants A and B by multivariate contingency tables were carried out by Plants A and B combined, target population, due to the small number of cases of symptomatic chronic bronchitis in Plant B.

Analyses showed that there was no significant difference in symptomatic chronic bronchitis when examined by age groups or length of exposure to dust.

Analyses of SCB by smoking categories controlling for age groups $\leq$ 39 years and $\geq$ 40 years, show SCB is highly significant for $\leq$ 39 year age group, very significant for $\geq$ 40 year age group, but not as high as for the $\leq$ 39 year age group. This indicates that in older subjects, smoking is less important as a factor in SCB.

Analyses of SCB by smoking categories controlling for lengths of exposure ($\leq$ 5 years, 6-10 years and $\geq$ 11 years), show SCB is highly significant for $\leq$ 5 year exposure group. SCB remains significant for the 6-10 year and $\geq$ 11 year age group, but decreases as exposure increases. As it is the case with age, smoking is a less important factor in explaining SCB when length of exposure increases and the number of workers with SCB decreases with exposure.

CHAPTER VI

LOG-LINEAR ANALYSES

Because of the limitations of analysis by contingency tables, it was decided to use a log-linear model to test for the interactions of variables: smoking, age, and length of exposure on SCB. This was done using the P3F Program of the BMDP (Biomedical Data Package) (1977).

The log-linear model operates upon the expected cell frequencies under the hypothesis that a particular model adequately represents the data. The expected cell frequencies Fijkl, are estimated as a function of a multiplicative set of parameters, γ's, for main and interaction effects of the variables in the model. For the "saturated" model, the expected cell frequencies are:

$$F_{ijkl} = N\gamma_i^E \gamma_j^A \gamma_k^C \gamma_l^S \gamma_{ij}^{CA} \cdots \gamma_{ijkl}^{EACS}$$

This model can be replaced by an equivalent model, in which the expected cell frequencies and parameters are transformed to their natural logs, yielding an additive model:

$$G_{ijkl} = \theta + \lambda_i^E + \lambda_j^A + \lambda_k^C + \lambda_l^S \cdots \lambda_{ijkl}^{EACS}$$

The analysis is based on fitting a (hierarchical) log-linear model to the cell frequencies; that is the logarithm of the expected cell frequency is written as an additive function of main effects and interactions in a manner similar to the usual analysis of variance model.

The program tests the appropriateness of models by the likelihood $\chi^2(G^2)$ and by the usual χ^2 goodness-of-fit.

The question of interest is whether some of the parameters of the saturated model can be deleted, by setting a given tau or lambda equal to 1 or 0 respectively, and still generate expected cell frequences, F_{ijkl}, close to the observed frequences, fijkl. By deleting certain γ or λ effect parameters from the model, the assertion is made that these effects or "interactions" are absent in the data and that only the marginal tables corresponding to the remaining parameters in the model are required to represent adequately relationships among the variables.

The data will first be printed in a multiway frequency table (Table 25). Table 26 will test for marginal and partial association to screen the various interactions to determine whether they are necessary in the model for the data being used, whether they are not necessary, or questionable. The next step will be to use a step-wise search procedure to determine the appropriate model to best fit the data.

From Table 26, we see that the fourth order interaction [SCAE] is not significant when tested for either partial or marginal association and therefore is not needed in the model. In the third order interaction [SAE] is moderately significant for both marginal and partial association and will probably be needed in the model. The interaction [SCA] does not show a significant association for either test, but is questionable, as it has the next highest likelihood ratio $X^2(G^2)$. It is doubtful if it is needed in the model, however it will be tried in the model. Both tests are non-significant for the remaining third order effects so they will not be used in testing for models of best fit. In the two-way interactions both partial and marginal associations are highly significant for [SC], [SA] and [AE], therefore they will be used to test for best fit in the model. One test, marginal association, is significant for [SE], and the other test, partial association, is moderately significant. So [SE] will be used to test for best fit in the model, however it is doubtful if it will be needed. Both tests are non-significant for interactions [CA] and [CE], so they will not be used for best fit in the model.

Since the model is hierarchical the main effects S, C, A, and E are implicitly specified and will be used for testing model of best fit.

Using the guidelines from Table 26 and the above analyses, the main models selected to be fitted to the data are the following:

Model 1 [A, E, S, C]

Model 2 [AE, SC]

Model 3 [SCA, AE, SE]

Model 4 [SAE, SC]

Model 5 [ASC, AES]

By using a step-wise search procedure with add and delete, the following models were found. The add command forms models by starting with the minimal set of effects and then fitting one effect at a time. Delete command forms models that are included by the model and differ from it by only one effect. In testing for the model of best fit, likelihood ratio χ^2 (G^2) will be used. In the following models, the four main variables A, E, S, and C, are automatically included in each model by the BMDP Program.

Table 25

Multiway Frequency Table

Observed cross-classification of four variables:
1) Length of Exposure to dust
2) Age
3) Symptomatic Chronic Bronchitis
4) Smoking Habits

(E)	(A)	(C)	(S)		SMOKERS		
EXPOSURE	AGE	CB	NEVER	EX	LIGHT	MOD.	HEAVY
≤ 5 yr.	≤39 yr.	Yes	1	6	7	15	30
		No	197	171	109	138	113
	≥40 yr.	Yes	1	2	1	4	11
		No	12	59	15	17	22
6-10 yr.	≤39 yr.	Yes	1	4	4	14	20
		No	134	166	87	90	78
	≥40 yr.	Yes	1	0	2	2	3
		No	17	36	11	20	24
≥11 yr.	≤39 yr.	Yes	1	2	4	7	10
		No	73	130	35	44	51
	≥40 yr.	Yes	2	12	4	9	12
		No	70	187	60	72	74

(A) The observed frequencies in the above table includes the target population from both Plant A and Plant B.

(B) Variables: E = Length of exposure to dust
A = Age
C = Symptomatic Chronic Bronchitis
S = Smoking Habits

Table 26

A Test of <u>Partial Association</u> of the Factors	A Test of <u>Marginal Association</u> of the Factors
It is calculated as the difference between the Full K-th Order Model and that which excludes only the specified effect. K is the number of Factors in the effect.	The table is summed over the unspecified indices and then the effects are tested to be zero.

	VARIABLES	DEGREE OF FREEDOM	LR X^2	PROB.	LR X^2	PROB.
*	S	4	201.01	0.00	--	--
*	C	1	2079.26	0.00	--	--
*	A	1	389.07	0.00	--	--
*	E	2	29.41	0.0000	--	--
*	SC	4	129.31	0.0	131.19	0.0
*	SA	4	27.77	0.0	50.19	0.0
+	SE	8	16.51	0.0356	38.81	0.0
	CA	1	2.43	0.1193	2.43	0.1194
	CE	2	0.93	0.6195	0.84	0.6562
*	AE	2	441.24	0.0	467.67	0.0
‡	SCA	4	5.71	0.2216	7.60	0.1076
	SCE	8	3.61	0.8909	6.21	0.6240
*	SAE	8	20.27	0.0094	19.77	0.0112
	CAE	2	3.39	0.1832	2.66	0.2647
	SCAE	8	9.49	0.3029	--	--

* Variables showing significant effect most likely to be used to identify best fitting model.

\+ Variables showing moderate significance. Probably belong in the model.

‡ Variables showing questionable significance.

S = Smoking
C = Symptomatic Chronic Bronchitis
A = Age
E = Length of Exposure to Dust

MODEL 1		D.F.	LR χ^2	PROB.
[A, E, S, C]		51	713.57	0.0

Delete - the following models are included in the above model and differ from it by only one effect.

MODEL	EFFECT	D.F.	LR χ^2	PROB.
(a) [E, S, C]	A	52	1102.63	0.0
Difference due to	A	1	389.06	0.0
(b) [A, S, C]	E	53	742.98	0.0
Difference due to	E	2	29.41	0.0
(c) [A, E, C]	S	55	914.58	0.0
Difference due to	S	4	201.01	0.0
(d) [A, E, S]	C	52	2792.83	0.0
Difference due to	C	1	2079.25	0.0

Add - two factor interaction terms - the following models include the above and differ from it by only one "effect."

MODEL	EFFECT	D.F.	LR χ^2	PROB.
(e) [SC, A, E]	SC	47	582.38	0.0
Difference due to	SC	4	131.19	0.0
(f) [SA, E, C]	SA	47	663.39	0.0
Difference due to	SA	4	50.18	0.0
(g) [SE, A, C]	SE	43	674.76	0.0
Difference due to	SE	8	38.81	0.0
(h) [CA, E, S]	CA	50	711.15	0.0
Difference due to	CA	1	2.43	0.1194

	EFFECT	D.F.	LR χ^2	PROB.
(i) [CE, A, S]	CE	49	712.73	0.0000
Difference due to	CE	2	0.84	0.6570
(j) [AE, S,C]	AE	49	245.91	0.0000
Difference due to	AE	2	467.66	0.0

MODEL 2	D.F.	LR χ^2	PROB.
[AE, SC]	45	114.72	0.0

Delete - the following models are included in the above model and differ from it by only one "effect."

MODEL	EFFECT	D.F.	LR χ^2	PROB.
(a) [A, E, SC]	AE	47	582.39	0.0
Difference due to	AE	2	467.67	0.0
(b) [S, C, AE]	SC	49	245.91	0.0
Difference due to	SC	4	131.19	0.0

Add - the following models include the above model and differ from it by only one "effect."

MODEL	EFFECT	D.F.	LR χ^2	PROB.
(c) [SA, AE, SC]	SA	41	64.53	0.0109
Difference due to	SA	4	50.19	0.0000
(d) [SE, AE, SC]	SE	37	75.90	0.002
Difference due to	SE	8	38.82	0.0000
(e) [CA, AE, SC]	CA	44	112.29	0.0000
Difference due to	CA	1	2.43	0.1191
(f) [CE, AE, SC]	CE	43	113.88	0.0000
Difference due to	CE	2	0.84	0.6559

MODEL 3	D.F.	LR X^2	PROB.
[SCA, AE, SE]	28	37.26	0.1132

Delete - the following models are included in the above model and differ from it by only one "effect."

MODEL	EFFECT	D.F.	LR X^2	PROB.
(a) [SC, SA, CA, AE, SE]	SCA	32	44.86	0.0652
Difference due to	SCA	4	7.60	0.1075
(b) [SCA, SE]	AE	30	483.94	0.0
Difference due to	AE	2	446.68	0.0
(c) [SCA, AE]	SE	36	55.08	0.0218
Difference due to	SE	8	17.82	0.0226

Add - the following models include the above model and differ from it by only one "effect."

MODEL	EFFECT	D.F.	LR X^2	PROB.
(d) [CE, SCA, AE, SE]	CE	26	36.30	0.0863
Difference due to	CE	2	0.96	0.6176
(e) [SAE, SCA]	SAE	20	17.48	0.6214
Difference due to	SAE	8	19.78	0.0112

MODEL 4	D.F.	LR X^2	PROB.
[SAE, SC]	25	26.93	0.3592

Delete - the following models are included in the above model and differ from it by only one "effect."

MODEL	EFFECT	D.F.	LR X^2	PROB.
(a) [SA, SE, AE, SC]	SAE	33	46.71	0.0573
Difference due to	SAE	8	19.78	0.0112
(b) [C, SAE]	SC	29	158.13	0.0
Difference due to	SC	4	131.19	0.0

Add - the following models include the above model and differ from it by only one "effect."

MODEL	EFFECT	D.F.	LR X^2	PROB.
(c) [CA, SAE, SC]	CA	24	25.00	0.4016
Difference due to	CA	1	1.86	0.1731
(d) [CE, SAE, SC]	CE	23	26.55	0.2758
Difference due to	CE	2	0.39	0.8241

MODEL 5	D.F.	LR X^2	PROB.
[ASC, AES]	20	17.48	0.6214

Delete – the following models are included in the above model and differ from it by only one "effect."

MODEL	EFFECT	D.F.	LR X^2	PROB.
(a) [SC, CA, SAE]	SCA	24	25.08	0.4014
Difference due to	SCA	4	7.60	0.1075
(b) [SE, AE, SCA]	SAE	28	37.26	0.1132
Difference due to	SAE	8	19.78	0.0112

Add – the following models include the above model and differ from it by only one "effect."

MODEL	EFFECT	D.F.	LR X^2	PROB.
(c) [CE, SCA, SAE]	CE	18	16.46	0.5607
Difference due to	CE	2	1.03	0.5982

Starting with the simplest model 1, [A, E, S, C], consisting of only main effects we have:

D.F.	LR χ^2	PROB.
51	713.57	0.0

The LR χ^2 is highly significant and the model gives a very poor fit to the data. We also find by deleting each variable one at a time that no main effect can be dropped from the model.

Next we add 2-factor interaction terms, by adding each one separately to the model we find that only effects CA (1, h) and CE (1, i) are non-significant with probability values respectively of 0.1194 and 0.6570. We therefore proceed to model 2, with the 2-way interaction terms that appear to be most significant AE and SC. Model 2 [AE, SC, A, E, S, C] still provides a poor fit to the data so we add a further 2-way interaction term. We add the next most significant term, namely [SA] and obtain model 2(c) [SA, AE, SC, A, E, S, C] with a probability value of .01, indicating still a poor fit, but better than model [AE, SC]. As predicted, we find that SE and the remaining 2-way interaction terms do not make a significant improvement to the model.

Although model 2(c) [SA, AE, SC] is a poor fit having:

D.F.	LR χ^2	PROB. Value
41	64.53	0.0109

we now take it as our best model of fit and see if we can improve on it. We now consider adding a 3-way interaction. We found in Table 26 the most possible significant effects, of the four, was the 3-way interaction effect SAE with the effect of SCA questionable.

In model 3 we add SCA, which also includes effect CA, to obtain model 3(c) [SCA, AE], which has:

D.F.	LR X^2	PROB. Value
36	55.08	.0218

This gives a difference of:

D.F.	LR X^2	PROB. Value
5	9.45	.0924

from model 2(c)[SA, AE, SC], which is non-significant.

In model 4 we add SAE, which also includes effect SE, to obtain model 4 [SAE, SC] which has:

D.F.	LR X^2	PROB. Value
25	26.43	.3592

This gives a difference of:

D.F.	LR X^2	PROB. Value
16	37.6	.0001

from model 2(c) [SA, AE, SC], which is significant and makes for a good fit.

The difference between model 2(c) [SA, AE, SC] and model 4 [SAE, SC] is highly significant.

In adding the term SAE, however, we have also added term SE. To find out how significant term SE is, we look at model 4(a) [SA, AE, SC, SE], and find that it has:

D.F.	LR X^2	PROB. Value
33	46.71	0.0573

This gives a difference of:

D.F.	LR X^2	PROB. Value
8	17.82	0.0226

from model 2(c) [SA, AE, SC], which is a significant improvement and makes the model nearly a good fit.

Also, if we compare model 4 [SAE, SC] to model 4(a) [SA, AE, SC, SE], we find a difference of:

D.F.	LR χ^2	PROB. Value
8	19.78	0.01120

so addition of SAE interaction to model 4(a) [SA, AE, SC, SE] is significant.

Our model of best fit now becomes model 4 [SAE, SC] with:

D.F.	LR χ^2	PROB. Value
25	26.93	.3592

We know now that we cannot improve the fit of this model by deleting effects SC or adding effects CA or CE.

Since the other third-order interactions were not significant in Table 23, we can predict that they will not add significance to the model.

In model 5, we test to see if the two, 3-way interactions will give a better fit, model 5 [ASC, AES] with:

D.F.	LR χ^2	PROB. Value
20	17.48	0.6214

This gives a difference of:

D.F.	LR χ^2	PROB. Value
5	9.45	0.0924

from model 4 [SAE, SC], which is not significant. Therefore, adding the interaction SCA and CA does not give a significant improved fit so we are led to consider model 4 [SAE, SC] to be the model of best fit.

For the data, this model indicates an interaction between S, A, E (Smoking, Age and Length of Exposure) which is to be expected since age would link all three together.

It also indicates no interaction between C, A, E (Symptomatic Chronic Bronchitis, Age and Length of Exposure). However, smoking and symptomatic chronic bronchitis are interacting for this data.

The best model to fit the data is [AES, SC], a 3-way interaction and a 2-way interaction. The 3-way interaction, Age, Dust Exposure and Smoking, and the 2-way interaction, Smoking and Chronic Bronchitis, as well as the "main effects" of the four variables, smoking, age, dust exposure and chronic bronchitis, are the only ones required to account for the variation in cell frequencies observed in Table 22.

From model 4, [SAE, SC] we can obtain the logit model

$$\begin{matrix} A & E & S & C \\ i & j & k & l \end{matrix} \quad \text{logit}_k = W + W_{(k)}$$

$$\log Mijkl = \theta + \mu_2^A + \mu_j^E + \mu_k^S + \mu_1^C$$

$$+ \mu_{ij}^{AE} + \mu_{ik}^{AS} + \mu_{jk}^{ES} + \mu_{kl}^{SC} + \mu_{ijk}^{AES}$$

If we consider a logit model, i.e., we look at the Symptomatic Chronic Bronchitis rate for each combination of factors i j k,

$$\frac{M\,i\,j\,k_1}{M\,i\,j\,k_2}$$

$$\text{then } \text{logit}_k = \log \frac{M\,i\,j\,k_1}{M\,i\,j\,k_2} = (\mu_1^c - \mu_2^c) + (\mu_{k1}^{SC} - \mu_{k2}^{SC})$$

$$= W + W_{s(k)}$$

which says that the log of symptomatic chronic bronchitis rate depends only on an additive effect due to smoking for this data.

Summary

These data were analyzed by log-linear method to determine the relationship, if any, of tobacco smoking, age and length of dust exposure to SCB.

From the log-linear analyses we found that age, length of dust exposure and tobacco smoking were related to each other but tobacco smoking was the only additive effect that affected symptomatic chronic bronchitis.

CHAPTER VII

CONCLUSIONS

Conclusion

This research was undertaken to study the prevalence of symptomatic chronic bronchitis in a group of mining and mill workers, to determine if there was an association between symptomatic chronic bronchitis, and tobacco smoking, length of dust exposure or age, individually or synergistically.

The null hypothesis was stated as:

> There is no relationship between symptomatic chronic bronchitis and tobacco smoking, dust exposure and age, individually or synergistically.

Analyses were done by two statistical methods. First, bivariate analysis done by cross tabulations, using Chi-square as a test of significance, gave the following results.

(a) There was a significant association between tobacco smoking and symptomatic chronic bronchitis as shown by Table 19 ($\chi_3^2 = 142.42$, $p >$ .001). This association held when controlled by age (Tables 20 and 21) and by length of dust exposure (Tables 22 to 24).

(b) Age was not significantly associated to symptomatic chronic bronchitis (Table 15).

(c) Length of dust exposure was not significantly associated to symptomatic chronic bronchitis (Tables 16 to 18).

There were some differences in the age and length of exposure distributions of these variables in both plants; to correct for these, separate tables were prepared. Due to the small frequencies in some cells, no conclusions can be derived from Plant B. The analysis for Plant A confirms the results shown for the target population.

Second, a log-linear analysis was used to test different models for associations among the variables under study. The results showed that the following model had the best fit [SAE, SC].

This model confirmed the association between symptomatic chronic bronchitis and smoking, [the SC term], and showed that there were some interactive effects between the independent variables [the SAE term], which could be explained by the correlation between age and length of exposure, and by some differences in smoking habits by age.

Discussion

In this study of 2,504 male miners and mill workers, the mean age for the target population was 37.39 years with a standard deviation of 8.99 years. The mean length of employment was 8.51 years with a standard deviation of 5.99 years.

The majority of workers were in the age category ≤ 39 years, 69.6%, a relatively young population.

In the length of employment group, for the largest population the highest percent of workers were in the ≤ 5 year exposure group, 37.18%. The next highest percent of workers were in the ≥ 11 years' exposure group, 34.31%. Therefore the majority of workers, 65.69% were in the ≤ 10 years' exposure group.

In the tobacco smoking categories of never smoked, ex-smokers, light, moderate and heavy, the highest percent of workers were in the ex-smokers' category, the majority of these were in the older age group.

The next highest percent was in the never smoked group, the majority of these were in the young age group. Present smokers made up approximately 50% of the workers. When they were sub-divided into light, moderate and heavy there was no overall difference between the subdivisions.

Comparisons of prevalence of symptomatic chronic bronchitis with other studies is very difficult due to different criteria used for defining symptomatic chronic bronchitis, differences in populations studied, their occupations, age, sex and smoking patterns. However, general comparisons may be made keeping in mind the results may have been achieved by different criteria and methods.

For example, one recent Canadian study by Neri et al. (1975) gave somewhat similar results to the present study. The prevalence of SCB in the male population of Ottawa, a non-industrial town, was 7.2%, while the male population of Sudbury, a nickel and copper mining town, gave 11.27%. However, Neri used a less restrictive definition of chronic bronchitis than the one used in this study, "the production of phlegm on most days for at least three months in each year" (Fletcher et al., 1959).

Tobacco smoking was found significantly associated to symptomatic chronic bronchitis, increasing in significance as tobacco smoking increased ($\chi^2_3 = 142.42$, $p > .001$, Table 19). The same result holds true when symptomatic chronic bronchitis was analyzed by tobacco smoking controlling for age and by length of exposure.

For the analyses in Chapter V, the categories Never Smoked and Ex-Smokers are combined into non-smokers due to small numbers in the cells. In looking at these tables, one must keep in mind that the category of non-smokers has a much higher percent of ex-smokers as age increases, Table 9. The combination of these two categories may account for the higher prevalence rate of symtomatic chronic bronchitis in the ≥ 40 year category for the non-smoking group, as given in Table 21.

The result that tobacco smoking is significantly associated with symptomatic chronic bronchitis is supported by most of the following literature reviewed, including Clark et al (1980), Huhti (1965), Karava et al. (1976), Neri et al. (1975).

One of the best overviews of the existing literature showing the strong evidence relating tobacco smoking to respiratory disease is Smoking and Health (1979A), a report to the Surgeon General.

Length of exposure was not found significantly associated to symptomatic chronic bronchitis in these data. This result is in agreement with other researchers who have found that dust exposure was not significantly related to symptomatic chronic bronchitis, such as Chan-Yeung et al. (1980), Clark et al. (1980), Lowe et al. (1970).

However, before we can make any conclusions regarding exposure to dust, we have to keep in mind the proxy measurement which was used in the measuring of exposure to dust, the length of employment averaged the amount of dust over the target population, thus making a possible association between dust exposure and symptomatic chronic bronchitis.

Another factor to be considered is that the composition of the dust, iron and silica may not lend itself specifically to producing respiratory symptoms of the large airways but to other diseases of the lung, i.e., pneumoconiosis and small airway disease. This factor may be well worth questioning when one looks at the overall rate of symptomatic chronic bronchitis for the target population of 7.7%, Plant A 7.3% and

Plant B 9.2%. There is a slight but not significant higher rate of symptomatic chronic bronchitis in Plant B.

This is interesting due to the fact that Plant B has a wet grinding process and does not have a pellet plant, as opposed to Plant A. These two factors would ordinarily indicate that Plant A would have more dust and combustion gases in the work place. Plant B does have a significantly older population than Plant A. However, age was not significantly associated to symptomatic chronic bronchitis, but it does appear to have a slight effect.

Age was not found significantly associated to symptomatic chronic bronchitis, however there was a slightly higher rate of symptomatic chronic bronchitis as age increased (Table 12). This result is in compliance with the American Lung Association (1977) which stated that aging by itself was not a primary cause of COPD. Huhti (1965) also found that the effect of age on respiratory symptoms was only slight.

Age may also be related to the healthy worker effect. Due to the harsh climate and hard work of mining, the selection process in seeking employment would most likely favour the strong, young, and healthy. The selective removal of workers from the mines if they develop respiratory symptoms, as they advance in age, would also contribute to age not being a significant factor in this target population.

From this research and literature reviewed it is very convincing that tobacco smoking is a major cause of respiratory problems, more so than any other factor. Other factors such as occupation, age, sex, social class, etc. all may have an indirect or synergistic effect on respiratory diseases, but none shows up with as much consistency and in such magnitude as the effects of personal pollution--tobacco smoking.

This is not to say that industrial pollution is any less important, however, with legislation such as the Clean Air Act and Threshold Level Values of known pollutants, persons are being protected against excessive exposure to particulates and gaseous substances more so than years ago.

It would seem that reduction in tobacco smoking would not only greatly reduce the prevalence of symptomatic chronic bronchitis, but would allow other factors to be investigated without obscuring their effects on chronic bronchitis symptoms.

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APPENDIX A

Following are the excerpts from the ATS Questionnaire used for this research. The questions analyzed were questions related to:

No. 1	Birth date
No. 3	Male/Female
No. 7	Cough
No. 8	Phlegm
No. 28	Cigarette smoking
No. 29	Pipe smoking
No. 30	Cigar smoking

1. What is your date of birth? $\overline{Y\ Y}$ $\overline{M\ M}$ $\overline{D\ D}$

3. Are you male or female?
   Male ____ (1)
   Female ____ (2)

COUGH

7. A. Do you usually have a cough? For example, a cough with your first smoke or when first going out-of-doors, but not just clearing your throat?

Yes ____ (1)
No ____ (2)

IF YES TO 7A, ASK THE FOLLOWING QUESTION
IF NO TO 7A, CHECK N/A AND SKIP TO 7C

B. Do you usually cough at all as much as 4 to 6 times a day, 4 or more days out of the week?

Yes ____ (1)
No ____ (2)
N/A ____ (8)

C. Do you usually cough at all on getting up or first thing in the morning?

Yes ____ (1)
No ____ (2)

(PROBE: DO YOU USUALLY COUGH WHEN YOU GET UP OR DO YOU COUGH FIRST THING IN THE MORNING?)

D. Do you usually cough at all during the rest of the day or at night?

Yes ____ (1)
No ____ (2)

IF YES TO ANY OF THE ABOVE QUESTIONS, ASK THE FOLLOWING:

E. Do you usually cough like this on most days for 6 consecutive months or more during the year?

Yes ____ (1)
No ____ (2)
N/A ____ (8)

F. For how many years have you had this cough? (CODE 88 FOR N/A AND 99 FOR DK)

____ No.Yrs.

PHLEGM

8. A. Do you usually bring up phlegm from your chest? You should count phlegm with the first smoke or on first going out of doors and count swallowed phlegm. Exclude phlegm from your nose.

Yes ____ (1)
No ____ (2)

(PROBE: COUNT PHLEGM IF YOU RAISE IT UP FROM YOUR LUNGS, BUT NOT IF YOU MERELY CLEAR IT FROM YOUR THROAT).

IF YES TO 8A, ASK THE FOLLOWING QUESTION:

B. Do you usually bring up phlegm like this as much as twice a day, 4 or more days a week?

Yes ____ (1)
No ____ (2)
N/A ____ (8)

C. Do you usually bring up phlegm at all on getting up, or first thing in the morning?

Yes ____ (1)
No ____ (2)

(PROBE: DO YOU USUALLY BRING UP ANY PHLEGM?)

D. Do you usually bring up phlegm at all during the rest of the day or at night?

Yes ____ (1)
No ____ (2)

IF YES TO ANY OF THE ABOVE, ASK THE FOLLOWING:

E. Do you bring up phlegm like this on most days for 3 consecutive months or more during the year?

Yes ____ (1)
No ____ (2)
N/A ____ (8)

F. For how many years have you had trouble with phlegm?

____
No.Yrs.

(PROBE: "TROUBLE" MEANS CONSTANT CLEARING OF THROAT AND BRINGING UP PHLEGM).
(CODE 88 FOR N/A AND 99 FOR DK).

TOBACCO SMOKING

28. A. Have you ever smoked cigarettes? Yes means more than 20 packs of cigarettes or 12 oz. of tobacco in a lifetime or more than 1 cigarette a day for 1 year.
Yes ____ (1)
No ____ (2)

IF YES TO 28A:

B. Do you now smoke cigarettes as of 1 month ago? Yes ____ (1)
No ____ (2)
N/A ____ (8)

IF YES TO 28B:

C. How many cigarettes do you smoke per day? (CODE 88 FOR N/A AND 99 FOR DK).
____ No.Cigarettes

IF NO TO 28B:

D. How old were you when you stopped? (CODE 88 FOR N/A AND 99 FOR DK).
____ Age

E. How old were you when you first started regular cigarette smoking? (CODE 88 FOR N/A AND 99 FOR DK).
____ Age

F. On the average over the entire time you smoked, how many cigarettes did you smoke per day?
____ No. Cigarettes

G. (Do/Did) you inhale the cigarette smoke? Not at all ____ (1)
Slightly ____ (2)
Moderately ____ (3)
Deeply ____ (4)
N/A ____ (8)

29. A. Have you ever smoked a pipe?
Yes means more than 12 oz. of tobacco in a lifetime.

Yes ____ (1)
No ____ (2)

IF YES TO 29A

B. Do you now smoke a pipe as of 1 month ago?

Yes ____ (1)
No ____ (2)
N/A ____ (8)

IF YES to 29B

C. How much pipe tobacco do you smoke per week?
(PROBE: A STANDARD POUCH OF TOBACCO CONTAINS 1 1/2 oz.)
(CODE 88 FOR N/A AND 99 FOR DK)

____ Oz/wk.

IF NO to 29B

D. How old were you when you stopped?
(CODE 88 FOR N/A AND 99 FOR DK).

____ Age

E. How old were you when you started regular pipe smoking?
(CODE 88 FOR N/A AND 99 FOR DK)

____ Age

F. On the average over the entire time you smoked a pipe how much pipe tobacco did you smoke per week?
(CODE 88 FOR N/A AND 99 FOR DK).

____ Oz/wk.

G. (Do/Did) you inhale the pipe smoke?

Not at all ____ (1)
Slightly ____ (2)
Moderately ____ (3)
Deeply ____ (4)
N/A ____ (8)

30. A. Have you ever smoked cigars?
Yes means more than 1 cigar a week for a year. Yes ___ (1)
No ___ (2)

IF YES TO 30A:

B. Do you now smoke cigars as of 1 month ago? Yes ___ (1)
No ___ (2)
N/A ___ (8)

IF YES TO 30B:

C. How many cigars do you smoke per week?
(CODE 88 FOR N/A AND 99 FOR DK). ___ Cigars per wk.

IF NO TO 30B:

D. How old were you when you stopped?
(CODE 88 FOR N/A AND 99 FOR DK). ___ Age

E. How old were you when you started regular cigar smoking?
(CODE 88 FOR N/A AND 99 FOR DK). ___ Age

F. On the average, over the entire time you smoked cigars how many cigars did you smoke per week?
(CODE 88 FOR N/A AND 99 for DK). ___ Cigars per wk.

G. (Do/Did) you inhale the cigar smoke? Not at all ___ (1)
Slightly ___ (2)
Moderately ___ (3)
Deeply ___ (4)
N/A ___ (8)

APPENDIX B

ANALYSIS OF SMOKING PATTERNS

As shown in Chapter III, when explaining the method used to measure tobacco smoking, the ATS Questionnaire has three major questions related to smoking, one code for cigarettes, cigar and pipe, and each divided into seven different sections, plus one question for the number of cigarettes smoked. For each method of smoking there are three "pure" states: Smokers, ex-smokers and never smoked and, in addition, all possible combinations, for a total of 27 combinations. Figures 2 to 4 show this in a three-dimensional tabulation, prepared from data for each plant separately, and for both plants (target population).

Smoking Patterns

In order to classify tobacco smoking, the smoking patterns of the communities were analyzed to show the smoking combinations and frequencies. For each method of smoking--cigarette, pipe and cigar--there are three possible pure states: smoker, ex-smoker and never smoked, however there are 27 theoretical combinations. All combinations and the frequency of workers in each of them were analyzed to study the smoking pattern in the population under study. The following figures give the findings of this analyses of smoking patterns.

Never Smoked Pipe

CIGARETTES	Cigar S	Cigar Ex	Cigar N
S	10 1	10 2	776 3
Ex	6 4	22 5	465 6
N	4 7	1 8	$\boxed{410}$ 9

Ex-Pipe Smokers

CIGARETTES	Cigar S	Cigar Ex	Cigar N
S	9 10	10 11	78 12
Ex	5 13	31 14	92 15
N	1 16	1 17	3 18

Present Pipe Smokers

CIGARETTES	Cigar S	Cigar Ex	Cigar N
S	5 19	2 20	32 21
Ex	1 22	0 23	11 24
N	1 25	1 26	3 27

= Never Smoked = N

= Ex-Smokers = Ex

= Present Smokers = S

Figure 2. Smoking Combinations, Plant A.

Never Smoked Pipe

CIGARETTES	Cigar S	Cigar Ex	Cigar N
S	2 (1)	6 (2)	233 (3)
Ex	1 (4)	3 (5)	139 (6)
N	0 (7)	2 (8)	$\boxed{119}$ (9)

Ex-Pipe Smokers

CIGARETTES	Cigar S	Cigar Ex	Cigar N
S	3 (10)	4 (11)	44 (12)
Ex	1 (13)	9 (14)	35 (15)
N	0 (16)	0 (17)	1 (18)

Present Pipe Smokers

CIGARETTES	Cigar S	Cigar Ex	Cigar N
S	1 (19)	1 (20)	7 (21)
Ex	0 (22)	0 (23)	6 (24)
N	0 (25)	0 (26)	1 (27)

= Never Smoked = N

= Ex-Smokers = Ex

= Present Smokers = S

Figure 3. Smoking Combinations, Plant B.

Never Smoked Pipe

CIGARETTES	Cigar S	Cigar Ex	Cigar N
S	12 1	16 2	1,009 3
Ex	7 4	25 5	604 6
N	4 7	3 8	$\boxed{529}$ 9

Ex-Pipe Smokers

CIGARETTES	Cigar S	Cigar Ex	Cigar N
S	12 10	14 11	122 12
Ex	6 13	40 14	127 15
N	1 16	1 17	4 18

Present Pipe Smokers

CIGARETTES	Cigar S	Cigar Ex	Cigar N
S	6 19	3 20	39 21
Ex	1 22	0 23	17 24
N	1 25	1 26	4 27

= Never Smoked = N

= Ex-Smokers = Ex

= Present Smokers = S

Figure 4. Smoking Combinations, Target Population.

From Figure 3, we obtain three large groups:

	Plant A		Plant B		Total	
	No.	%	No.	%	No.	%
Never Smoked (1)	410	20.6	119	19.3	529	20.5
Ex-Smokers (2)	615	30.9	189	30.6	804	31.2
Current Smokers (3)	965	48.5	310	50.2	1,275	49.4
Total	1,990	100	618	100	2,579	100

(1) Cell No. 9

(2) Cells Nos. 5, 14, 6, 8, 15, 17, 18

(3) Cells Nos. 1, 2, 3, 4, 7, 10, 11, 12, 13, 16, 19, 20, 21, 22, 23, 24, 25, 26, 27

This analysis shows that for both plants there are 529 (20.5%) subjects who never smoked, one possible combination; 804 (31.2%) ex-smokers, seven possible combinations; and 1,275 (49.4%) current smokers, 19 possible combinations.

Further analyses of this last group, current smokers shows:

	Plant A		Plant B		Total	
	No.	%	No.	%	No.	%
Only cigarettes (cell 3)	776	80.4	233	75.2	1,009	79.1
Only cigar (cell 7)	4	0.4	0	0.0	4	0.3
Only pipe (cell 27)	3	0.3	1	0.3	4	0.3
Smoking Combinations (16 cells)	182	18.9	76	24.5	258	20.2
Total	965	100	310	100	1,275	100

This analysis shows that pure pipe and cigar smokers are a very small number, eight (0.62%) of the current smoking population. Pure cigarette smokers number 1,009 (79%), and mixed types number 258 (20%).

Further analyses of this last group, smoking combinations show:

	Plant A		Plant B		Total	
	No.	%	No.	%	No.	%
Cigarette Smokers (1) plus other combinations	58	31.9	14	18.4	72	27.9
Cigarette and ex-smokers (2)	98	53.8	54	71.1	152	58.9
Other Smokers (3) (pipe and cigar)	26	14.3	8	10.5	34	13.2
Total	182	100	76	100	258	100

(1) Cell Nos. 19, 20, 21, 10, 1

(2) Cell Nos. 11, 12, 2

(3) Cell Nos. 25, 26, 27, 22, ~~16~~, 13, 7, 4

This analysis shows that for 258 combination smokers, 224 (87%) include cigarettes and their combinations, and only 34 (13%) smoke combinations that do not include cigarettes.

Therefore it is possible to conclude that 1,233 subjects smoke cigarettes alone or in combination, that represents 47.8% of the target population under study. The relatively small number of pure cigar and pipe smokers, and of combination pipe and cigar smokers (42 or 1.6%) of the target population, justifies their inclusion in the light smokers category. Other researchers have included pipe and cigar smokers with the light classification of smokers (Karava et al., 1976; Bouhuys et al. 1979).