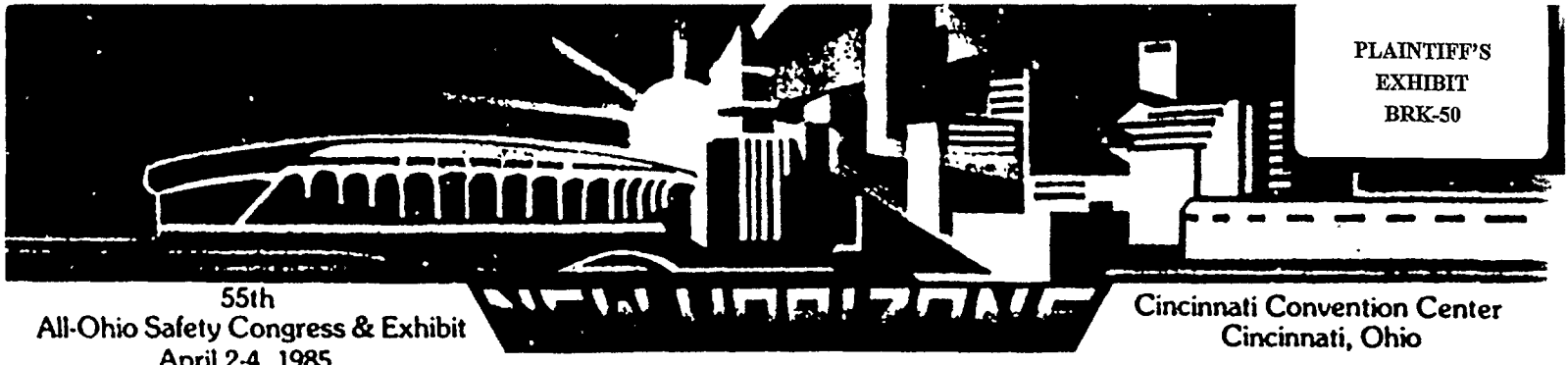

PLAINTIFF'S
EXHIBIT
BRK-50

55th
All-Ohio Safety Congress & Exhibit
April 2-4, 1985

Cincinnati Convention Center
Cincinnati, Ohio

**IN SAFETY AND HEALTH**

February 5, 1985

Legal Section
Industrial Commission of Ohio
246 North High Street
Columbus, Ohio 43215

RE: OD-20000-22
Wylman Johnson
U.S. Steel Corporation

Attention: Ms. Judy Spencer

Dear Ms. Spencer:

On November 5, 1984, the U.S. Steel Corporation, Lorain, was visited to gather information on the above claim. Don Girolkmo, Manager, Personnel Safety and Health, was interviewed.

The claimant's medical background is per the facsimile file. Fairly heavy smoking was noted in the August 9, 1982, hospital admitting diagnosis (one pack per day).

The claimant's work history is from U.S. Steel and the facsimile file.

| | |
|---|---|
| 4 to 9, 1947 | Pittsburgh and Lake Erie Railroad - Laborer |
| 1947-1955 | Worked coal mine for five years |
| 1955-1982 | U.S. Steel, Lorain - various jobs, see facsimile. |

U.S. Steel

The claimant had various jobs at the Lorain facility.

Most jobs involved the foundry which is permanently closed. The open hearth is also permanently closed.

2

The majority of the claimant's work at U.S. Steel was ingot mold chipping. By using a pneumatic chipping hammer, the sand lining of the mold was removed. The sand kept the molten steel from the mold. The cooled ingots were removed and an employee crawled into the mold and removed the sand lining.

The Lorain operation is markedly different now as compared to the claimant's work history. This would not allow a recreation of the claimant's job or exposure picture.

Some prior Commission occupational disease investigations and sampling information from U.S. Steel were obtained. The claimant and other ingot mold chippers exposures are on the enclosed table.

The exposure data indicate elevated levels of respirable dust containing crystalline silica. The company required respiratory protection after 1973.

Prior to 1973, unprotected exposures to elevated levels of silica laden dust is reasonable. The 18 years prior to U.S. Steel's respiratory protection program, the claimant was exposed to elevated levels of silica dust.

**Health Effects (crystalline silica)**

Classical silicosis is a chronic disease characterized by formation of scattered rounded silica containing nodules of scar tissue. Continued exposure to crystalline silica can produce complicated silicoses. This can lead to respiratory disability even after exposure ceases. The scar tissue cannot function as normal lung tissue (transmit gases).

**Coal Mines**

The claimant could have been exposed to unknown concentrations of coal dust. The probability of developing pneumoconiosis from coal dust is a function of the exposure concentration and quartz content. Both of these factors are unknown as the specific relation to the claimant's condition and coal dust would be speculation.

Reports of coal dust exposures and lung carcinoma are contradictory. Scarano et al. (1972) found an excess of lung cancer deaths in the U.S. anthracite mines. Rook et al. (1979) studied Lancashire mines and concluded there was no positive association of carcinoma of the lung and pneumoconiosis.[2]

## Conclusions

Considering individual susceptability, a cause-and-effect relation to silica exposures and the silicosis diagnosis is reasonable. Adenocarcinoma and pure work exposures are suspect as a good cause and effect.

Respectfully submitted,

Richard Wilder

Richard Wilder
Industrial Hygienist

RW/klp

[1]Documentation of TLV's, ACGIH, 1983.

[2]Industrial Toxicology, Hamilton and Hardy, 1983.

THE INDUSTRIAL COMMISSION OF OHIO

Richard F. Celeste
Governor

Division of Safety and Hygiene

One Government Center
Suite 1173
Jackson and Erie Streets
Toledo, Ohio 43604

PLAINTIFF'S
EXHIBIT
BRK-49

August 2, 1989

Legal Section
Industrial Commission of Ohio
246 North High Street
Columbus, Ohio 43215

Re: OD 34609-22
Albert Dublin
U.S. Steel, Lorain Works
Lorain, Ohio 44052

Attn.: Judy Spencer

Risk Number SI 0002

Dear Ms. Spencer,

Enclosed is the report of the industrial hygiene investigation for the occupational disease claim number 34609-22, filed by Albert Dublin, against the United States Steel Corporation, Lorain-Cuyahoga Works, Lorain, Ohio.

For ease of reading the report has been broken into the following sections:

1. Introduction
2. Work History
3. Literature Review
4. Discussion
5. Closing
6. References

Respectfully submitted

W. J. Brutsche
Industrial Hygienist

WJB

enc: Claimant's work history

OD # 34609-22
Albert Dublin
United States Steel Corporation
Lorain-Cuyahoga Works
Risk Number SI 0002

## INTRODUCTION

On June 15, 1989 a visit was made to the Lorain-Cuyahoga Works of the United States Steel Corporation in Lorain Ohio in an attempt to gather information relative to the above cited occupational disease claim. The claimant alleges that his medically diagnosed condition of "mixed pneumoconiosis with marked acute and chronic bronchitis..." appears to be the result of exposure dust from slag crushing operations.

## WORK HISTORY

The claimant's work history with was provided during the visit and has been attached to this report. From this sheet it was determined that the claimant worked in the slag crushing operations for approximately 26 years. During this time the claimant worked as both a general laborer and millwright. As a general laborer the claimant could have worked any number of odd jobs. As a millwright the claimant would have worked on equipment maintenance and repair.

## LITERATURE REVIEW

### Pneumoconiosis

The terminology used in describing dust induced diseases needs to be understood. The generic term for these diseases is "pneumoconiosis". The pneumoconiosis diseases are generally classified into the more specific categories of benign, fibrogenic, mixed dust, man made fiber, and organic materials pneumoconiosis. The benign category include, but are not necessarily limited to the dusts of iron oxide, barium, tin, perlite, and carbon black. Fibrogenic dusts includes, but is not necessarily limited to, free crystalline silica, diatomaceous earth, asbestos, and talc.

Mineral pneumoconiosis is rarely caused by the inhalation of a single dust and should, therefore, be referred to as mixed dust pneumoconiosis. Mixed dust pneumoconiosis agents include graphite, coal, clays, fullers earth, sillimanite, granite, mica, slate, calcium dusts, and amorphous silica. The man made mineral fibers of rock wool or fiberous glass most generally make up this category. Finally, organic dusts include certain plant dusts, grain dusts, bird droppings, wood dusts, cotton dusts, and proteolytic enzymes. (1)

Cor pumonale, enlargement and failure of the right ventricle, often accompanies chronic pneumoconiosis, resulting from disorders of the lungs, pulmonary vessels or chest wall which involve an increase in pulmonary arterial pressure. (2)

Chronic Bronchitis

The characteristic pathological features of bronchitis were first described by Reid and consist of an increase in the mucous-secreting glands of the airways. In the human it is the mucous secreting glands of the large airways that are mostly responsible for mucous secretion and that are predominantly involved in bronchitis. It is now clear that mucous gland hypertrophy occurs as a result of any chronic irritant effect on the airways and that cigarette smoke, dust, and air pollutants, such as sulfur dioxide, ozone, and ammonia can all act as the inciting factor. The heavier and the longer the period of exposure, the greater the likelihood of developing bronchitis. In trying to study the effects and frequency of bronchitis in a working population, it becomes evident that there are two important confounding factors, namely, cigarette smoking and age. In smokers, cigarette smoking completely overshadows the effects of dust and air pollutants.

Numerous investigations have shown that the continued inhalation of dust leads to the development of bronchitis as manifested by chronic cough and sputum. Over the past several years a definite relationship between the prevalence of these symptoms and the level of dust exposure has been demonstrated both in coal miners and in South African gold miners. It becomes difficult, however, to separate the effects of cigarette smoking and dust in cigarette smokers, since the effects of smoking overwhelm those of dust. For the most part it is possible to detect the consequences of dust exposure only in nonsmokers, although in some studies cigarette smoking and dust appear to have had a synergistic effect. (3)

**DISCUSSION**

Presence of dust in workplace

Relative to the claimant's exposure to dust, the company has provided the results of a 1980 industrial hygiene survey for the slag crushing operations. Please note that Tables 1 and 2 serve to establish the presence of silica and ranges of total dust for the survey period. It is literally impossible to infer much else from these tables because length of sample periods, number of samples, and respirable dust sample information is lacking. As stated previously the information contained in this table merely establishes the presence of silica and total dust in the claimant's work environment.

Medical records

In discussions with a company nurse, through Mr. Clark, pulmonary function tests were administered approximately every two years and date back to 1975. Chest x-rays appear to date back to 1968. Along with these x-rays there appear to be various medical interpretations.

Pulmonary function, height differences

Within the facsimile file there are two different sets of pulmonary function studies. Attention is drawn to the fact that each of these sets of data lists a different height for the claimant (66 and 67 inches). Company medical records indicate a third height of 67.75 inches. "For any given subject, age, height, sex and race influence his or her ventilatory capacity and lung volumes. Weight and other factors make a far lesser contribution." "....Black subjects have lung volumes and flow rates that are about 12 to 15 per cent less than white subjects of the same age and height. Other racial differences have been reported in Eskimos, Chinese, and East Indians." (4)

Most pulmonary function tests show wide intersubject variability which is in part related to sex, age, body size, and race. These factors must be taken into account before attempting to interpret a test result. One method of reducing variability is to calculate the ration of two measurements which normally bear a relatively fixed relationship to one another. Common examples include the $FEV_1/FVC$, CV/VC, and RV/TLC ratios... While relating two measurements reduces the range of values and the coefficient of variation, it does not necessarily fully account for sex, age, or even body size relationships.(5) Thus the significance of illustrating the differences in the listed height of the claimant may warrant further investigation.

Claimant's smoking history

As illustrated in the "Literature Review" section of this report, smoking habits can interfere with an accurate diagnosis. To this end, information found in the facsimile file there are conflicts. The time frames for when the claimant supposedly quit smoking are at issue. A doctor's notes (The Cleveland Clinic Foundation letter of January 27, 1987) indicates quit approximately ten years ago and the District Hearing Officer's notes cites 1962.

CLOSING

In general, according to the literature the present evidence for the existence of industrial bronchitis as a disease is overwhelming, and it is equally clear that it is an entity distinct from pneumoconiosis. Industrial bronchitis is a nonspecific response to a variety of agents, including inert dusts and various gases and vapors. The predominant symptoms are those of cough and sputum, and for the most part these symptoms are of diagnostic import only in nonsmokers. The condition is due to the deposition of particles that are mostly between three and ten microns and are thus larger than the usual respirable fraction. Since such particles are removed by the mucocilliary escalator, they leave no radiographic stigmata.

Industrial bronchitis is associated with decreased flow in the large airways and occasionally with a slight increase in residual. volume. The small airways are affected but to a lesser extent. The decrement in ventilatory capacity that occurs in this condition are of the most part larger than those that cause the pneumoconiosises, there is often only a fair correlation between respirable dust measurements and the prevalence of bronchitis. At the present time, there is no evidence that industrial bronchitis leads to emphysema. The prevalence and incidence of industrial bronchitis are likely to be better related to measurements of total dust or to measurement of those particles that range from 0.5 to 20 microns than to respirable dust.

One of the more important questions that remains unanswered is whether the reduction in ventilatory capacity induced by industrial bronchitis is reversible. It is well recognized that the bronchitis of cigarette smokers clears up a few months after the subject stops smoking, but this is not true of the impairment associated with concomitant emphysema. Whether there is an improvement in symptoms and ventilatory capacity in subjects with industrial bronchitis when dust exposure terminates is as yet unknown.(6)

The claimant worked 40 years in the Steel Mill with 26 of those years in the slag crushing operations. During this time the claimant was probably exposed to varying concentrations of quartz containing dust. What remains unclear is what effect the discrepancy in the claimant's height could have on the results of the pulmonary function studies and the degree to which the claimant's smoking habit may have contributed to his condition.

In conclusion, the determination of whether or not the claimant's condition work related is best left to the opinion of competent medical authorities.

REFERENCES

1. Hamilton and Hardy's Industrial Toxicology, Fourth Edition, 1983, 337-392.

2. A Guide to the Work-Relatedness of Disease, DHEW (NIOSH) Publication No. 79-116, p. 246.

3. Occupational Lung Diseases, Morgan and Seaton, Second Edition, W.B. Saunders Company, 1984. Pages 521-537.

4. Occupational Lung Diseases, Morgan and Seaton, Second Edition, W.B. Saunders Company, 1984. Pages 107-128.

5. Occupational Respiratory Diseases, U.S. Department of Health and Human Services,Public Health Service, Centers for disease Control, National Institute for Occupational Safety and Health, Publication No. 86-102, September 1986, 155-170.

6. Occupational Lung Diseases, Morgan and Seaton, Second Edition, W.B. Saunders Company, 1984. Pages 533-535.

# RECORDS CERTIFICATION

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