# *Dust, Vapors, and More Dust Some Suggestive Data and Case Studies on the Subtlety of Occupational Lung Disease in Early 20th Century Lancaster*

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The industrial revolution that so dramatically altered the landscape of 19th century America provided this nation with many benefits. Perhaps the most obvious was a higher standard of living — made possible by mass production and the enhanced purchasing power of the worker. A somewhat less obvious benefit, seen throughout the western world, was a sharp reduction in mortality rates. As René Dubos recently noted, mortality rates of roughly 500/100,000 in 1850 declined to approximately 50/100,000 by 1950.[1]

This decline in mortality was a product of a better diet, warmer clothing, better and warmer homes, a purer water supply, improved sanitation, and better medical care. Paradoxically, the industrialization that led to lower mortality rates also created new work environments with a variety of health hazards — hazards not particularly well understood as late as the 20th century. The purpose of this essay is to analyze some data on mortality, disease, and occupation — together with some case studies — for early 20th century Lancaster in an effort to identify subtle connections. This task involves a plunge into "occupational medicine" several decades before such a specialty existed.

## *Leading Causes of Mortality In Turn-Of-The-Century Lancaster*

Data published by the Board of Health of the City of Lancaster for the years 1891-1908 reveal that consumption (tuberculosis) was the leading cause of death with 937 victims. Heart disease (the leading killer nationally) was a close second with 913, pneumonia a close third with 896, and apoplexy a distant fourth with 683 victims.[2] Furthermore, data from the same reports made it quite clear that the highest mortality rates — regardless of disease — were consistently found in the cotton mill district located in the southwest quadrant of town.[3] (Significantly, the southwest quadrant was not necessarily the bottom of the socio-economic scale as the southeast quadrant, housing the local black community, was at least as poor.) The prominance of consumption coupled with high mortality rates in the mill district led this author to begin to think about cause-and-effect. Was it possible, for example, that the health of mill worker's lungs had been impaired which, in turn, left them unusually susceptible to lung disease? And, if this was a distinct possibility, what kind of evidence existed to shed additional light on the matter?

## *Rossmere Sanatorium Patient Records*

Arguably, the best source of information with the potential to link the Lancaster work involvement to occupational disease is found in the records of the Rossmere Sanatorium, currently in the possession of the American Lung Association of Lancaster County. The Rossmere Sanatorium, located immediately northeast of the city, was established in 1925 by the Tuberculosis Society of Lancaster County to provide a verdant and bucolic setting in which local victims of consumption might rest, enjoy fresh air, and recover, if possible. The local sanatorium was a convenient alternative to state-run facilities at Mt. Alto and Hamburg, though chronic cases continued to gravitate to state facilities. (Patients from outside Lancaster County were required to place their names on a state facility waiting list.)[4]

Rossmere Sanatorium operated for about 32 years, closing its doors in 1957 when the need for housing TB patients had severely diminished. During this time patient record cards were kept, and these 5 x 8 cards contain a great deal of information including: (1) the patient's occupation, (2) the nature of the work, and (3) the employer. The cards also reveal date of entry and release, and a prognosis — frequently a date of death as well. What can reasonably be inferred from this information?

**Table 1**
**LEADING OCCUPATIONS OF ROSSMERE PATIENTS, 1925-1945**

| | | | |
|---|---|---|---|
| laborers | 169* | engineers | 18 |
| iron and steel workers | 80* | carpenters | 17* |
| sales | 80 | waiters & waitresses | 17 |
| textile workers | 77* | painters | 16* |
| clerical workers | 65 | railroad workers | 16 |
| factory workers | 53* | barbers | 13* |
| nurses | 41 | maids | 11* |
| farmers | 34* | machinists | 10* |
| stenographers | 32 | teachers | 9 |
| mechanics | 31* | attendants | 9 |
| bus and truck drivers | 27 | cement workers | 8* |
| tobacco workers | 26 | janitors | 8* |
| bakers and cooks | 26 | plumbers | 8 |
| linoleum workers | 25* | asbestos workers | 7* |
| machine operators | 22 | lock workers | 6* |
| electricians | 21 | laundry workers | 4 |
| watchworkers | 18* | welders | 3* |

*Indicates those occupations now understood to be hazardous to the lungs.

Note: Patients also included 423 housewives, 141 in housework, 87 school children, 12 pre-school children, and 74 no occupation.

Source: Patient Record Cards, Rossmere Sanatorium, Lancaster, Pennsylania. Data previously compiled for a report that appears to have been unpublished but circulated sometime in 1945 or 46.

The date for the years 1925-45 reveal that many workers who came to Rossmere were previously engaged in jobs now known to be harmful to the lungs. Some 18 of 34 occupations listed or 586 of 1,007 patients (58.2%) fall into this category. Admittedly, the statistics are rather crude. Such broad categories as "laborer" and "factory worker" undoubtedly conceal as much as they reveal, and surely some included under such headings labored in benign environments. Even a few narrower job classifications could profit from additional refinement. For example, a lockworker engaged in grinding might be distinguished from someone assembling locks. While these adjustments would tend to reduce the number of patients "previously exposed to a hazardous environment," other refinements might inflate the figures. For example, another researcher might want to include bus and truck drivers (exposed to fumes) or laundry workers (exposed to chemicals and their vapors). In any event, regardless of the exact number, it remains reasonable to infer from the data that many of Rossmere's patients were likely to have suffered some previous degree of lung impairment which, in turn, rendered them particularly vulnerable to contracting tuberculosis, a communicable disease.

## *Additional Statistical Information*

Prior to stumbling upon the aforementioned compilation of data for all patients at Rossmere between 1925 and 1945, this author took a random sample of patients treated between 1925 and 1934 who had previously been part of the labor force. It was determined that 94 of 205 (46%) had been part of a work environment generally understood (today) to be harmful to the lungs. Some of the potentially more harmful work experiences included:

**Table 2**

**POTENTIALLY HARMFUL OCCUPATIONS OF A RANDOM SAMPLE* OFF ROSSMERE PATIENTS, 1925-1934 (205 Patients)**

| | | | |
|---|---|---|---|
| silk mill hand | 12 | hauler of ashes | 3 |
| farmer | 9 | carpenter | 3 |
| moulder-foundry | 8 | plater, brusher, and | |
| cotton mill hand | 5 | polisher — watch parts | 3 |
| machinist | 4 | stone mason | 3 |
| linoleum worker | 4 | barber | 3 |
| asbestos worker | 3 | painter | 2 |

Source: Patient Record Cards, Rossmere Sanatorium, Lancaster, Pennsylvania.

*Random sample of workers

The data from a random sample essentially reinforce the preceding compilation. The major differences appear to be in labeling. That is, where Table 1 lumped all textile workers together, Table 2 makes a distinction between silk and cotton mill hands. Or, those Table 1 labels "iron and steel workers" appear in Table 2 as "moulders-foundry." Asbestos workers appear in both listings in modest numbers; however, there is reason to believe that additional cases are buried under such job titles as "factory hand" and "laborer." In any event, Table 2 also leads a researcher to suspect that occupational damage to the lungs left a worker unusually predisposed to consumption.

An additional question asked of the random sample of 205 concerned their prognosis on leaving Rossmere. Here again, previous work experience proved to be important. That is, of the 94 workers treated at Rossmere — previously engaged in work harmful to the lungs — 58.4% had a negative prognosis or died within a year of leaving Rossmere. In contrast, only 48.5% of the rest of the workers had a negative prognosis or died within a year of release.[5] This suggests that any prior impairment of the lungs may also have influenced the chances for improvement or recovery.

## *Other Possibilities*

Remaining mindful throughout that correlations do not prove cause-and-effect, the historian as detective can readily see still other *suggestive* connections between occupations and disease/mortality data — again related to the lungs. The existence of over a thousand mill hands in early 20th century Lancaster caused the author to wonder about the presence of byssinosis — commonly known today as "brown lung."[6]

Once considered a form of asthma, byssinosis is contracted through extended exposure to cotton dust particles.[7] In light of the fact that the federal government did not "recognize" byssinosis as a separate disease until 1968,[8] there is no reason to search for turn-of-the-century data on the ailment. It can be noted, however, based on modern research, that a mill hand with byssinosis who continues working in the cotton mill can expect to see the problem develop into bronchitis, and data on bronchitis is available.[9] The *Select* and *Common Council Minutes* for the City of Lancaster indentify bronchitis as the 11th leading cause of death in 1898 and the 8th most significant cause of death in 1903.[10] Thus, the brown lung that is singled out for special attention in cotton mill communities today was most likely enveloped in statistics on bronchitis in an earlier day.

Again on the subject of mill hands, it is equally provocative to think about a connection between mill hands working long hours in damp mills (damp to facilitate spinning) and the incidence of pneumonia in the mill district.

One final general observation on the problem of lung ailments is the possibility that their full impact may not be accurately measured in mortality data if victims of consumption, bronchitis, etc. happened to die of heart failure — at which point a victim of a lung ailment became a statistic in the column under heart disease.

## *A Few Case Studies*

Reiterating the notion that what is statistically suggestive falls short of conclusive scientific proof, the reader may be wondering at this point about specific concrete examples of the way in which dusts and vapors in the workplace actually harmed particular workers. To that end, three brief case studies are offered. They are taken from the pages of the *Lancaster Law Review* for the period 1928-41, and all involve litigation stemming from decisions handed down by the Workman's Compensation Board. (Beginning in 1915, Pennsylvania employers had to carry compensation insurance for worker injuries. In 1937 Workmen's Compensation expanded to include occupational diseases such as asbestosis and silicosis.)

The three cases represent three different kinds of problems that injured the lungs. The first stems from an industrial accident releasing deadly fumes, the second deals with the immediate perils of silica, and the third involves the contraction of asbestosis and the complications that followed.

(1) Morris S. Hollinger was a mixer of benzol (a solvent) for the United States Asbestos Company of Manheim. It was his duty to, "pump benzol from building #35 to building #32 into a covered, elevated storage tank from which it passed by gravity into an open vat on the floor of the same building."[11] On the evening of August 13, 1928 benzol overflowed and was found on the floor of building #32. It is not known what action Hollinger took in response to this accident, but at some point he exited the building. His body was found at 9:30 p.m. approximately 10-12 feet from building #32. (Hollinger had last been seen alive at 6:30 p.m.) His body was warm and not stiff. He had a cut on his nose.

Dr. P.F. Guie, a local physician called to the scene, smelled Hollinger's breath and noted the presence of benzol fumes. He diagnosed the problem as benzol poisoning and called the County Coroner. Dr. J.D. Hershey, Lancaster County Coroner, and Dr. R.N. Klemmer of Lancaster City performed the autopsy and concluded that Hollinger's death was caused by "acute benzol poisoning."

The Workmen's Compensation Board found in favor of the widow Lizzie Hollinger, and the U.S. Asbestos Company and their insurer went into Lancaster County Common Please Court to appeal. The central question was, "Can the ultimate conclusion that the decedent was *not* killed in the course of his employment be fairly inferred from the basic facts?" The court said no — there was "no other cause of death except that possibly it was from chronic instead of acute benzol poisoning." Thus, a judgment was entered against U.S. Asbestos in 1930 for $2,691 with interest from August 20, 1928.[12]

(2) Howard R. Stauffer worked for 25 years (1916-41) as a moulder in the iron foundry of the Hubley Manufacturing Company of Lancaster. Silica was used in the foundry and thus he was exposed to silica dust. Sometime during 1939 a Dr. H.R. Bryson examined Stauffer and diagnosed the presence of silicosis. Despite the diagnosis, the moulder continued to work until January 24, 1941 at which point he was declared totally disabled. He died March 17, 1941.

The Workmen's Compensation Act, expanded in 1937 to include occupational diseases such as silicosis, was narrowed a bit in 1939 by the requirement that, "disability or death must be caused solely, instead of primarily, by silicosis, etc. to make compensation possible."[13] Even in the light of this more stringent definition, the Workmen's Compensation Board found in favor of widow Susan W. Stauffer which, in turn, led the Hubley Manufacturing Company to appeal in Common Pleas Court.

Dr. Thelma G. Boughton pathologist at Lancaster General Hospital testified that, "The primary cause of death is pulmonary silicosis, the immediate cause of death was infection."[14] Had testimony demonstrated that the primary cause was infection, compounded by silicosis, compensation would have been denied. The appeal was dismissed and widow Stauffer awarded $879.16.

(3) Dawson M. Rhoads began working as a spinner for the United States

Asbestos Division of Raybestos Manhatten in 1915. After 5 years he left and held another job for 6 years, and then returned to Raybestos Manhatten for the next 13 years in the mule spinning department. He stated the source of his physical ailment in his own words: ". . .you put the twist into the roving and while these spindles are turning that is what puts a twist in it and that, of course, throws off dust and you inhale it."[15]

Some time in the mid 1930s a Dr. Hershey of Manheim examined Dawson, told him he was totally disabled — suffering from asbestosis, and advised him to stop working. Complications ensued. In January of 1938 a Dr. Paul Snoke diagnosed asbestosis "with early pulmonary tuberculosis of the chronic fibrous type and a constricted pericardium."[16] In a hearing before the Workmen's Compensation Board Dr. Snoke testified that he believed "asbestosis played a definite part in reducing resistence and allowing TB to develop."[17] Dr. Hershey testified that Dawson's heart trouble resulted from constricted lungs that put an added burden on the heart. The Workmen's Compensation Board concluded that asbestosis was, "the cause of tuberculosis of the lung; that tuberculosis had spread and affected the pericardium."[18] Put another way, these connections were medically, legally, and financially meaningful.

When Raybestos Manhatten went into court to appeal in 1939 there was no challenge to the earlier medical analysis - simply to the nature of the compensation and complications arising from a "reserve fund." The Court of Common Please upheld the appeal and thereby altered the nature of the compensation, but the connectedness of one disease to another held![19]

## *Conclusion*

What has this brief essay — buttressed with statistics and case studies — demonstrated? The author would like to think that the historian as detective can do some work for the early 20th century that the modern practitioner of occupational medicine does for the 1980s. True, the modern medical detective utilizes the aid of highly trained epidemiologists and toxicologists to link problems in the work environment to worker ailments. At the same time, ironically, the final "proof" even today often resides in drawing reasonable inferences from collections of statistics. This falls short — as any attorney will tell you — of establishing an iron-clad, cause-and-effect relationship. Given this ongoing limitation, it does not seem unreasonable to use early 20th century data on occupation, disease, and mortality the way they have been used in this essay.

The fact remains that the modern industrial revolution that gave us a higher standard of living and a declining mortality rate also managed to create some new work environments that are consistently plagued with hazardous dust particles and toxic fumes. Even when these dusts and fumes do not directly lead to a recognized disease, they can be expected to weaken the lungs and render the worker susceptible to disease. This process is often subtle and thus often overlooked by medical science even today, but its subtlety renders it no less lethal.

## *Notes*

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1. René Dubos, "The Romance of Death," American Lung Association *Bulletin,* Vol. 68, #2, March, 1982, 6.
2. *Reports of the Board of Health,* City of Lancaster, 1900, p. 67, and 1908 p. 21.
3. Ibid.
4. See *The First 75 Years,* American Lung Association of Lancaster County, 1983.
5. Data compiled and calculated from Patient Record Cards, Rossmere Sanatorium, Lancaster, Pa.
6. For a brief discussion concerning a connection between disease and the cotton mill district in the 19th century see Thomas R. Winpenny, *Industrial Progress and Human Welfare: The Rise of the Factory System in 19th Century Lancaster* (Washington, 1982.)
7. See *Occupational Lung Diseases,* a substantial pamphlet published by the American Lung Association, 1983.
8. Mimi Conway, *Rise Gonna Rise* A Portrait of Southern Textile Workers (New York, 1979), 21.
9. See *Reports of the Board of Health* cited earlier.
10. *Select* and *Common Council Minutes,* City of Lancaster, 1898 and 1903.
11. "Lizzie Hollinger v. U.S. Asbestos Co.," *Lancaster Law Review,* 1930-31, 9-13.
12. Ibid.
13. "Susan Stauffer v. Hubley Manufacturing Company," *Lancaster Law Review,* 1942-43, 115-116.
14. Ibid.
15. "Dawson M. Rhoads v. U.S. Asbestos Division of Raybestos Manhatten," *Lancaster Law Review,* 1938-39, 641-43.
16. Ibid.
17. Ibid.
18. Ibid. The conclusion that asbestosis was the "cause of tuberculosis" is a compelling half truth. It was the "cause" to the extend that it weakened the lungs of Dawson Rhoads leaving the unusually vulnerable to the TB germ — tubercle bacillus. On the other hand, many victims of asbestosis never contracted TB.
19. Ibid.